STANDARD TREATMENT GUIDELINES

AND

ESSENTIAL DRUGS LIST

FOR

SOUTH AFRICA

HOSPITAL LEVEL PAEDIATRICS

2006 EDITION
FOREWORD

It is the vision of the National Department of Health to ensure that every citizen has access to good quality and affordable health care, including the access to medicines.

The goal of the National Drug Policy is to ensure an adequate and reliable supply of safe and efficacious medicines of acceptable quality in the most cost-effective manner to all citizens of South Africa. Resources are not unlimited and the appropriate management and use of drugs has often been underestimated and is increasingly being identified as a critical component of an efficient health care system. Thus affordability is a key element in ensuring access.

The National Department of Health through the Cluster: Pharmaceutical Policy and Planning has reviewed the Standard Treatment Guidelines and Essential Drugs List at hospital level for adults and paediatrics. These provide a vital tool to guide prescribers, particularly doctors working in district and regional hospitals.

More attention has been given to address healthy lifestyles, mental health conditions, neonatal conditions, palliative care and to strengthen the implementation of the Department’s Comprehensive HIV and AIDS Prevention, Care, Management and Treatment Plan. More in depth emphasis has been placed on the review of the endocrine, hypertension, infections and tuberculosis chapters. Evidence-based decision-making has been strengthened in the selection of drug entities.

The National EDL Committee has endeavoured to consult widely with colleagues within the Department, Provincial Pharmacy and Therapeutic Committees, universities, experts in different specialities, relevant societies and other stakeholders. I would like to take this opportunity to thank the National Essential Drugs List Committee, the Expert Review groups and all those who have contributed for their dedication and hard work. Congratulations to all role players on this achievement.

I hope this edition of the Standard Treatment Guidelines and Essential Drugs List for Hospital Level will guide you daily in treating all patients optimally.

DR MANTO TSHABALALA-MSIMANG
MINISTER OF HEALTH
The Department of Health is committed to providing quality and affordable healthcare including access to medicines to all citizens in South Africa. This is a challenging task in our health care system.

One of the goals of the National Drug Policy is to develop the full potential of drugs to improve the health status of South Africans within the available resources. The second edition of the Standard Treatment Guidelines (STGs) and Essential Drugs List (EDL) at Hospital Level for adults and paediatrics is a vehicle for the implementation of the National Drug Policy. Legislation has been adapted to address issues of affordability and improved access to medicines.

Advocacy and training are vital elements for the successful utilisation of the Hospital Level STGs and EDL. The concepts of evidence based selection of medicines and cost-effective treatment protocols need to be included in the training of doctors, pharmacists, nurses and other health care professionals. Pharmacovigilance remains an important aspect of ensuring the safety of medicines used. A reporting form in this regard is included in the book. The inclusions of the ICD-10 codes for conditions should facilitate analysis, peer review, billing etc.

The Hospital Level STGs and EDL are aimed for use at District and Regional Hospitals. Formularies remain the responsibility of Provincial Pharmacy and Therapeutics Committees. The Hospital Level STGs and EDL should be used as guidelines to develop these formularies. Updating the STGs and EDL is an ongoing process. Suggestions for improvement will be welcomed and considered.

The intention of the STGs and EDL is to strengthen priority health interventions. The implementation of the Department’s Comprehensive HIV and AIDS Care, Management and Treatment Plan is encapsulated in this edition, particularly with regard to the use of antiretrovirals and treatment of opportunistic infections.

It should not be forgotten that patients must take full responsibility for their own health, including adherence to prescribed treatment and lifestyle changes.

I wish to record a special word of appreciation to the chairpersons of the expert groups, the groups themselves and all other contributors to this edition of the STGs and EDL.

Mr. T.D. Mseleku
Director-General: Health
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It is impossible to name all who have played a part in producing this edition. The treatment guidelines and essential drugs list which appears in this book have been compiled after a lengthy consultative process. They include recommendations and advice from numerous individuals and groups including professional societies, expert committees, medical schools and secondary and tertiary hospitals.

We offer sincere thanks to those who contributed appropriate information and comments and to the members of the National Essential Drugs List Committee.
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CHAPTER 19 – PREMATURITY AND NEONATAL CONDITIONS

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The WHO describes Essential medicines as those that satisfy the priority health care needs of the population. Essential medicines are intended to be available within the context of functioning health systems at all times in adequate quantities, in the appropriate dosage forms, with assured quality and adequate information, and at a price the individual and the community can afford.

Effective health care requires a judicious balance between preventive and curative services. A crucial and often deficient element in curative services is an adequate supply of appropriate medicines. In the health objectives of the National Drug Policy, the government of South Africa clearly outlines its commitment to ensuring availability and accessibility of medicines for all people. These are as follows:

- To ensure the availability and accessibility of essential medicines to all citizens.
- To ensure the safety, efficacy and quality of drugs.
- To ensure good prescribing and dispensing practices.
- To promote the rational use of drugs by prescribers, dispensers and patients through provision of the necessary training, education and information.
- To promote the concept of individual responsibility for health, preventive care and informed decision-making.

Achieving these objectives requires a comprehensive strategy that not only includes improved supply and distribution, but also appropriate and extensive human resource development. The implementation of an Essential Drugs Programme (EDP) forms an integral part of this strategy, with continued rationalisation of the variety of medicines available in the public sector as a first priority. The private sector is encouraged to use these guidelines and drug list wherever appropriate.

The criteria for the selection of essential drugs in South Africa were based on the WHO guidelines for drawing up a national EDL. They include the following:

- any drug included must meet the needs of the majority of the population
- sufficient proven scientific data regarding effectiveness must be available
- any drug included in the EDL should have a substantial safety and risk/benefit ratio
- all products must be of an acceptable quality, and must be tested on a continuous basis
- the aim, as a rule, is to include only products containing single pharmacologically active ingredients
- combination products, as an exception, will be included where patient compliance becomes an important factor, or two pharmacologically active ingredients are synergistically active in a product
- products will be listed according to their generic names only
- where drugs are clinically equally effective, the drugs will be compared using the following:
  - the best cost advantage
  - the best researched
  - the best pharmacokinetic properties
  - the best patient compliance
  - the most reliable local manufacturer
THE ESSENTIAL DRUG CONCEPT

- a request for a new product to be included on the EDL must be supported by scientific data and appropriate references on its advantages and benefits over an existing product.

The implementation of the concept of essential drugs is intended to be flexible and adaptable to many different situations. It remains a national responsibility to determine which medicines are regarded as essential.

It should be noted that the Essential Drugs List (EDL) reflects only the minimum requirements for facilities. In keeping with the objectives of the National Drug Policy, provincial and local Pharmacy and Therapeutics Committees should provide additional drugs from the Hospital level EDL based on the services offered and the competency of the staff at each facility.
HOW TO USE THIS BOOK

It is important that you become familiar with the contents and layout of the book to use the standard treatment guidelines effectively.

Where relevant this book is consistent with the Standard Treatment Guidelines for Primary Health Care, Integrated Management of Childhood Illness Strategy (IMCI) and other National Programme treatment guidelines.

The ICD-10 number, included with the conditions, refers to an international classification method used when describing certain diseases and conditions. A brief description and diagnostic clinical, radiological and laboratory tests are included to assist the medical officer to make a diagnosis. These guidelines also make provision for referral of children with more complex and uncommon conditions to facilities with the resources for further investigation and management.

It is important to remember that the recommended treatments provided in this book are guidelines only and are based on the assumption that prescribers are competent to handle patients’ health conditions presented at their facilities.

The treatment guidelines are presented in chapters according to the organ systems of the body. In order to find the relevant sections in the book easily, use the indices at the back of the book. These have been divided into indices of disease conditions and drugs. Some of the drugs listed are only examples of a therapeutic class. In such cases the Provincial Pharmacy and Therapeutics Committees (PTCs) will decide on their drug of choice within that therapeutic class.

All suspected adverse drug reactions must be reported. In this book, only the common adverse effects have been mentioned. Information on the reporting of adverse drug reactions is provided in the section Guidelines for Adverse Drug Reaction Reporting. The purpose of ADR reporting is to reduce the risks associated with the use of drugs and ultimately improve patient care.

Potentially toxic drugs, drugs with narrow therapeutic indices and those with variable pharmacokinetics should be monitored regularly to optimise dosing, obtain maximum therapeutic effect, limit toxicity and assess compliance. The section on Patient Education in Chronic Conditions aims to assist health workers improve patient compliance and health generally.

As most paediatric doses are given as mg/kg all children must be accurately weighed at each consultation. All doses of drugs in children should be calculated to take into account their size and are based either on weight or body surface area. Modifications of dosage according to organ maturity should also be taken into account. In resource poor settings where a scale is not available, the following formula (though inaccurate in wasting or obesity) may be a useful guide:
**How to Use This Book**

Weight (kg) = (age (years) x 7) + 4.

Body surface area \( (m^2) \) = \( \sqrt{\frac{\text{height (cm)} \times \text{weight (kg)}}{3600}} \)

A number of drugs are not registered for paediatric use. None-the-less it is common practice to use such drugs in children where norms for such use have been established, and where adequate alternatives are not available. This is termed “off label” use. The responsibility for adverse outcomes associated with such practices lies with the prescriber.

Comments that aim to improve these treatment guidelines will be appreciated. The submission form and guidelines for completing the form are included in the book. Motivations will only be accepted from the Provincial PTC. Comments from persons and institutions outside the public service should be sent to:

The Essential Drugs Programme  
Pharmaceutical Programmes and Planning  
Department of Health  
Private Bag X828  
Pretoria  
0001

**Prescription Writing**

Drugs should be prescribed only when they are necessary for treatments following clear diagnosis. Not all patients or conditions need prescriptions for drug. In certain conditions simple advice and non-drug treatment may be more suitable.

In all cases carefully consider the expected benefit of a prescribed medication against potential risks. This is important during pregnancy where the risk to both mother and foetus must be considered.

All prescriptions should:
- be written legibly in ink by the prescriber with the full name and address of the patient, and signed with the date on the prescription form
- specify the age and weight of the patient in the case of children
- have contact details of the prescriber e.g. name and telephone number

In all prescription writing the following should be noted:
- the name of the drug or preparation should be written in full using the generic name and
- no abbreviations should be used due to the risk of misinterpretation.
- Avoid unnecessary use of decimal points and only use where decimal points are unavoidable. A zero should be written in front of the decimal point where there is no other figure, e.g. 2 mg not 2.0 mg or 0.5 mL and not .5 mL
• State the treatment regimen in full:
  ▪ drug name and strength
  ▪ dose or dosage
  ▪ dose frequency
  ▪ duration of treatment

  e.g. amoxicillin 250 mg 8 hourly for 5 days

• In the case of “as required” a minimum dose interval should be specified, e.g. every 4 hours as required
Poor therapeutic outcome of chronic conditions such as asthma, diabetes, epilepsy and hypertension can, in many cases, be ascribed to:

- poor or non-adherence to an otherwise sound therapeutic regimen;
- lack of communication between the various health care providers involved in the patient’s management;
- lack of effective communication between health care provider and patient;
- ineffective and/or insensitive regimens;
- inconsistency of medicine supply.

Patient Compliance

A patient’s compliance to his or her therapeutic regimen may be influenced by:

- medicine selection - prescribing should be the result of a process of concordance whereby the patient’s needs and preferences are matched to the available therapeutic alternatives;
- patient education - this empowers the patient to make an informed decision as to whether he or she should comply or not.

Although both of the above require longer consultation time, this investment is rewarded many times over during the subsequent years of management.

Other influencing factors might be

- adverse side effects of the medicines;
- lifestyle behaviour;
- level of responsibility to manage and control the disease.

Patients behaviour patterns contributing toward poor compliance

Patients may perceive treatment as unnecessary.

In conditions that are asymptomatic, e.g. hypertension, or those that only produce transient symptoms such as epilepsy:

- the patient often questions the validity of complying with therapy where there are no obvious results. As a result he or she decides to abandon therapy particularly where the therapy introduces new symptoms (side effects);
- the patient is compliant in a cyclical fashion - for a short period following transient symptoms (eg. seizure) or increased awareness (eg. following a BP reading at the clinic) but after a period returns to being non-compliant until the next episode of symptoms or clinic visit.

In conditions where symptoms show no improvement and where therapy merely controls the pathophysiological process.

- the patient often feels that his/her therapy has not contributed toward quality of life and in many ways has placed certain demands upon his/her lifestyle.

To be compliant on a sustained basis means that the patient must adjust his/her lifestyle in such a fashion that the regimen becomes habit. Inclusion of a regimen into the patient’s lifestyle is determined by the magnitude with which this adaption intrudes upon his/her established pattern. The greater the demand, the less likely the patient is to comply.
Thus for example a lunchtime dose in a school-going child who remains at school for extramural activity is unlikely to succeed. A shift worker may need to take a sedating medicine in the morning when working night shifts, and at night, when working day shifts.

Some patients’ lifestyles make certain adverse responses acceptable which others may find intolerable. Sedation is unlikely to be acceptable to a student but an older patient with insomnia may welcome this side effect. This is where concordance plays a vital role.

Education points to consider
- Focus on the positive aspects of therapy whilst being encouraging regarding the impact of the negative aspects and offer support to deal with the latter.
- Provide realistic expectations regarding:
  - normal progression of the illness - especially important in those diseases where therapy merely controls the progression.
  - the improvement that therapy and non-drug treatment can add to the quality of life.
  - Establish therapeutic goals and discuss them openly with the patient.
  - Any action to be taken with loss of control or when side effects develop.
  - In conditions that are asymptomatic or where symptoms have been controlled, reassure the patient that this reflects therapeutic success, and not that the condition has resolved.
  - Where a patient raises concern regarding anticipated side effects, attempt to place this in the correct context with respect to incidence, the risks vs. the benefits, and whether or not the side effects will disappear after continued use.

Towards concordance when prescribing
- Establish the patient’s
  - occupation
  - daily routine
  - recreational activities;
- past experiences with other medicines
- expectations of therapeutic outcome
Balance these againsts the therapeutic alternatives identified based on clinical findings. Any clashes with the chosen therapy should be discussed with the patient in such a manner that the patient will conform to a changed lifestyle.

Note:
Education that focuses on these identified problems is more likely to be successful than a generic approach toward the condition/medicine.

Improving Continuity of Therapy
- Clear and concise records.
- Patient involvement in the care plan.
- Every patient on chronic therapy should know:
  - his/her diagnosis
  - the name of every medicine
  - the dose and interval of the regimen
  - his/her BP or other readings
GUIDE TO PATIENT EDUCATION IN CHRONIC CONDITIONS

Note: The prescriber should reinforce this only once management of the condition has been established.
- When the patient seeks medical attention for any other complaints such as a cold or headache he/she must inform that person about any other condition/disease and its management
- If a patient indicates that he/she is unable to comply with a prescribed regimen, consider an alternative - not to treat might be one option, but be aware of the consequences e.g. ethical

Notes on prescribing in chronic conditions.
- Don’t change doses without good reason.
- Never blame anyone or anything for non-adherence before fully investigating the cause
- If the clinical outcome is unsatisfactory - investigate compliance (remember side effects may be a problem here).
- Always think about side effects and screen for them from time to time.
- When prescribing a new medicine for an additional problem ask yourself whether or not this medicine is being used to manage a side effect.
- Compliance with a once daily dose is best. Twice daily regimens show agreeable compliance. However once the interval is decreased to 3 times a day there is a sharp drop in compliance with poor compliance to 4 times a day regimens.
- Keep the total number of tablets to an absolute minimum as too many may lead to medication dosing errors and may influence compliance
1.1 PAEDIATRIC EMERGENCIES

An algorithmic approach to Paediatric Emergencies is provided at the beginning of this chapter. Certain emergencies of the airway, breathing, circulation and neurological system will be dealt with in the chapters on respiratory, cardiac and nervous system respectively. This section deals only with the unresponsive child (cardiorespiratory arrest), anaphylaxis, shock and foreign body inhalation. These guidelines are provided as an aid. All doctors should ensure that they have received appropriate training in providing basic (and preferably advanced) life support to children.

1.1.1 APPROACH TO THE CHILD IN AN EMERGENCY SITUATION

In approaching a child with potential severe illness or injury a structured approach will improve the child’s chances of a best possible outcome in the shortest possible time. The following is a diagrammatic overview derived from the Advanced Paediatric Life Support approach.

A brief summary of the approach and primary survey adapted from the APLS documentation is provided. For comprehensive competence an advanced paediatric life support course should be attended.
Rescuer in order of detection and keep rechecking effective support. Secondary survey delayed until stable.

Prepare for arrival of child if not already on site

SAFE approach: Shout for help, Approach with care, Free from danger, Evaluate ABC

Ensure adequate equipment and staff present
Estimate weight and dosages of resus response
Consider likely scenario and needs

[Weight kg = 2(x age yrs = 4) (birth 3.5kg)]
[ETT (int diam) = (Age yrs + 4) + 4mm]

(only 1 - 10 yrs of age, Neonates usually 3 - 3.5mm)

[Volume Expander = 20 mL/kg]
[Adrenaline dose = 0.1 mL: 1:10 000 solution]
[Defibrillation initial shock = 2 J/kg]

Consider need for immediate cardiac arrest protocol

Signs of circulation/life = Response to stimulation, cough, breathing, spontaneous movement

Primary Survey - ABCD Approach

Stabilise cervical spine if possibility of trauma

Airway & Cervical Spine

If no trauma - child lift/head tilt
If suspected trauma - jaw thrust

Look listen feel for effect airway - air movement

Breathing

Rate, recession, flare, air entry, cyanosis, oximetry

Circulation

Heart rate, pulse volume, capillary filling time, control bleeding

Establish IV Access (for trauma 2 good lines), send bloods

Disability

Level of consciousness score (AVPU), pupils, position glucose

Exposure

Rashes, Temperature, Evidence Poisoning

Secondary Survey

Focused Examination and response

Detailed review and response

Choking Protocol

Airway opening Protocol

Hi Flow Oxygen/reservoir mask

Ventilatory Support

Stridor, Wheeze or Pneumothorax Protocol

Shock, pulseless and cardiac failure protocol

Hi Flow Oxygen/reservoir mask

AVPU Scoring

A - Alert
V - response to voice (GCS ~ 12 - 13 or less)
P - response to pain (GCS ~ 8 or less)
U - unconscious

Normals for Age

Age   Resp   Pulse
<1y   30-40  110-160
1-2yr  25-35  100-150
2-5yr  25-30  95-140
5-12yr 20-25  80-120
>12yr  15-20  60-100
1.1.2 ANAPHYLAXIS

DESCRIPTION
An acute, potentially life-threatening hypersensitivity reaction starting within seconds to minutes after administration of or exposure to a substance to which the individual has been sensitised. Clinical manifestations range from mild urticaria and angioedema to upper airway obstruction, bronchospasm, hypotension, shock and death.
The reaction can be short-lived, protracted or biphasic, i.e. acute with recurrence several hours later. Immediate reactions are usually the most severe and/or life threatening.

DIAGNOSTIC CRITERIA
Clinical
- acute onset of signs and symptoms
- dizziness, paraesthesia, syncope, sweating, flushing, arrhythmias, swelling of eyes, lips and tongue
- angioedema with upper airway obstruction, stridor
- hypotension, shock
- bronchospasm, wheezing, dyspnoea, chest tightness
- gastrointestinal symptoms such as nausea, vomiting, diarrhoea

Facilities to initiate treatment must be available at all health centres.

NON-DRUG TREATMENT
To maintain arterial oxygen saturation ≥ 95% and to abolish cyanosis, administer
- oxygen, 100%, at least 1–2 L/minute by nasal prong
  In severe anaphylaxis nasal oxygen is unlikely to be adequate.
  High concentration oxygen by facemask is essential and the flow must be considerably more than 2 L/minute.
Place hypotensive or shocked patient in flat position.

Secure an open airway. Bag and/or intubate if necessary.
If several intubation attempts have failed, consider laryngeal mask or crico-thyrotomy or tracheotomy.
Observe for 24 hours.

DRUG TREATMENT
- adrenaline 1:1 000, IM, 0.01 mL/kg.
  Can be repeated every 5 minutes if necessary
  Maximum dose: 0.5 mL.
  Do not administer IV unless there is failure to respond to several doses of IM.
  Appropriate monitoring of urine output is required.
In non-response to shock or asystole
See Section 1.1.3

**Intravenous fluids**
Crystalloid solutions, e.g.:
- sodium chloride 0.9%, IV, 20 mL/kg as a bolus
  Repeat if necessary until circulation, tissue perfusion and blood pressure improve.
- promethazine, IV/IM, 0.25–0.5 mg/kg/dose
  Continue with
- promethazine, oral, 0.25–0.5 mg/kg, 6 hourly for 24–48 hours

If associated bronchospasm
- salbutamol, nebulised, 1 mL salbutamol respirator solution in 3 mL sodium chloride 0.9%
  Nebulise at 20-minute intervals.
- hydrocortisone, IV, 4–6 mg/kg, 4–6 hourly for 12–24 hours

**PREVENTATIVE MEASURES AND HOME BASED TREATMENT**
- obtain a history of allergies/anaphylaxis on all patients before administering medication/immunisation
- identify offending agent and avoid further exposure
- wear allergy identification disc/bracelet
- train patients to self-administer adrenaline subcutaneously
- educate patient and parent/caregiver on allergy and anaphylaxis

**REFERRAL**

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<td>Do not refer the patient during the acute phase.</td>
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<td>Transfer can only be done once patient is stable.</td>
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1.1.3 **CARDIORESPIRATORY ARREST**
I46.9

**DESCRIPTION**
Cardiorespiratory arrest in children is usually the end result of a period of circulatory or respiratory insufficiency and is seldom due to a sudden precipitous event. It is therefore important to pre-empt cardiorespiratory arrest in children by recognising and urgently treating respiratory or circulatory failure. Cardiorespiratory arrest is diagnosed clinically in the unresponsive child who displays no respiratory effort and in whom there is no palpable pulse and no signs of circulation, i.e. cough or spontaneous movement.
NON-DRUG TREATMENT
Always call immediately for help from your colleagues on site.

Ensure an open airway.
If there is still no respiration then artificial breathing must be commenced using a self-inflating bag, with a reservoir and an appropriate mask. The bag should be connected to a high flow oxygen source.
The chest should be seen to move in response to artificial breaths. If there is no or inadequate movement with bag-valve-mask ventilation, reassess the airway and place an appropriate sized endotracheal tube. In the event of an unexpected arrest or an arrest where there are no witnesses, consider the possibility of a foreign body obstruction. See Inhalation, Foreign Body: Section 1.1.5
Once effective breathing has been established chest compressions should be provided at a rate of 100/minute for all children excluding neonates. Artificial breaths should be provided at a ratio of one breath to five compressions in children less than 8 years. In older children the ratio is 15 compressions to 2 breaths.
A cardiac monitor should be attached to the child and an intravenous line inserted. See Intraosseous Infusion: Section 1.1.6.1.

DRUG TREATMENT
Asystole or pulseless electrical activity

- adrenaline 1:10 000, IV/intraosseous, 0.1 mL/kg
  0.1 mL = 10 mcg
  Dilute a 1 mL ampoule of adrenaline 1:1 000 in 9 mL of sodium chloride 0.9% or sterile water to give a 1: 10 000 solution.

  OR
  adrenaline 1:1 000, endotracheal, undiluted 0.1 mL/kg down an endotracheal tube. This is a higher dose due to the route of administration.

The dose of adrenaline may be repeated every 3 minutes if asystole persists.
If the arrest was preceded by circulatory shock a bolus of 20 mL/kg of sodium chloride 0.9% may be given.

Note:
There is no evidence to support the routine use of any of the following in asystolic cardiac arrest:
- sodium bicarbonate
- calcium
- high dose IV adrenaline (100 mcg/kg/dose)

Ventricular fibrillation or pulseless ventricular tachycardia
Proceed to immediate defibrillation or cardioversion.
The first two shocks are provided at 2 J/kg.
The third and all subsequent shocks at 4 J/kg.
For pulseless ventricular tachycardia the defibrillator should initially be set to synchronised mode. If it does not discharge then asynchronous shocks should be used.
Shocks are provided in cycles of 3 shocks with re-evaluation of the ECG trace between each shock and re-evaluation of the circulation in the event of a change in the ECG rhythm. Between each cycle of 3 shocks basic life support should continue uninterrupted.

If there is no return to sinus rhythm after the first cycle of 3 shocks, give:

- adrenaline 1:10 000, IV, 0.1 mL/kg.
  - May be repeated every 3 minutes in the face of persistent arrhythmia or asystole.
  - 0.1 mL = 10 mcg
  - Dilute a 1 mL ampoule of adrenaline 1:1 000 in 9 mL of sodium chloride 0.9% or sterile water to give a 1: 10 000 solution.

If ventricular fibrillation or pulseless ventricular tachycardia persists consider the possibility of hypothermia, tricyclic antidepressant toxicity or hyperkalaemia.

Each of these entities requires specific management.

If none of these is likely, give:

- amiodarone, IV/intraosseus, 5 mg/kg

Allow one minute of cardiopulmonary resuscitation between the administration of any drug and a repeat cycle of shocks.

**REFERRAL**

- to an intensive care unit after recovery from an arrest

### 1.1.4 CONVULSIONS, NOT FEBRILE CONVULSIONS

See seizures: section 13.1

### 1.1.5 INHALATION, FOREIGN BODY

T17.9

**DESCRIPTION**

Accidental inhalation of solid organic or inorganic objects that may obstruct the airway at any level.

**DIAGNOSTIC CRITERIA**

- initial symptom is frequently a sudden onset of choking followed by persistent unilateral wheeze, chronic cough, stridor and/or sudden death a few days later
- segmental or lobar pneumonia failing to respond to standard therapy
- signs of shift of the mediastinum
- chest X-ray on full expiration showing hyperinflation and/or collapse or sometimes radio-opaque foreign body
NON-DRUG TREATMENT
ACUTE EPISODE
• if moving air, provide oxygen and refer for bronchoscopy urgently
• if the child is unable to cough or breathe, attempt to dislodge the foreign body by back slaps, chest compressions, or the Heimlich manoeuvre

CAUTION
Blind finger sweeps are dangerous and absolutely contraindicated.

Infants
• check the mouth for any obstruction
• lay the infant on an arm in the head down position and strike the back 5 times with the heel of the hand
• if no response, turn the infant around and give 5 chest thrusts with 2 fingers in the midline just below the nipple line. Repeat sequence if necessary.
• refer if no response

Children
• clear the mouth
• strike the back 5 times while the child sits or lies prone or kneels
• if no response, attempt Heimlich manoeuvre: standing behind the child, pass your arms around the body and form a fist just below the sternum. Thrust upwards 5 times. Repeat as necessary.
• attempt removal under direct visualisation with Magills forceps if skilled clinician available

DRUG TREATMENT
Antibiotic therapy
Required pre and post removal of the foreign body, especially if it has been present for a long period of time.
Total duration of antibiotic therapy is 5–10 days.

• ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly
AND
• gentamicin, IV/IM, 7.5 mg/kg once daily

When child improves follow with
• amoxicillin, oral, 30 mg/kg/dose, 8 hourly

REFERRAL
• all cases for the removal of retained foreign bodies
• pneumonia with respiratory failure requiring ventilatory support
• pneumonia with complications not responding to therapy
1.1.6 SHOCK

DESCRIPTION
An acute syndrome that reflects the inability of the cardiopulmonary and circulatory system to provide adequate perfusion, oxygen and nutrients to meet the physiological and metabolic demands of organs, tissues and cells.

In compensated shock, the blood pressure is relatively well maintained but the patient still requires urgent resuscitation.

Depending on the nature and the intrinsic aetiology, shock can be divided into:

- **Hypovolaemic shock**: loss of intravascular fluid, e.g. dehydration, haemorrhage or fluid shifts
- **Distributive shock**: e.g. sepsis and anaphylaxis
- **Cardiogenic shock**: e.g. cardiac dysfunction
- **Dissociative shock**: e.g. profound anaemia and carbon monoxide poisoning
- **Obstructive shock**: e.g. pneumothorax and cardiac tamponade
- **Septic shock**: Many mechanisms are operative in septic shock.

Complications of shock include multiorgan dysfunction and/or failure.

DIAGNOSTIC CRITERIA
Evidence of compensated shock includes:

- mild agitation/confusion
- skin pallor
- increased heart rate
- cold peripheries
- prolonged capillary filling, i.e. greater than 3 seconds
- diminished urinary output
- signs and symptoms of underlying conditions

In uncompensated shock:

- BP falls and failure of urgent action will result in irreversible shock, i.e. death.

Facilities to initiate treatment of shocked patients must be available at all health centres.

NON-DRUG TREATMENT

- follow the algorithm at the beginning of the chapter
- identify and treat the underlying cause
- consider the need for controlled airway and ventilation in unresponsive, severe shock and if reduced breathing or neurological stability (rapid sequence induction intubation)
- ensure good intravenous or intraosseous access with two large bore lines - See Intraosseous Infusion: Section 1.1.6.1
- take appropriate bloods, e.g. cross match, urea and electrolytes, coagulation studies, full blood count and blood cultures
• monitor:
  ○ and maintain vital signs
  ○ and correct metabolic parameters
  ○ urinary output – aim for at least 1 mL/kg/hour

To maintain arterial oxygen saturation ≥ 95% and to improve oxygenation, administer
• oxygen, hi flow, 15 L/minute via facemask with reservoir bag or 6–10 L/minute via head box or intubation with respiratory support if necessary.
Maintain PaO₂ ≥ 80 mmHg or oxygen saturation ≥ 92%.
Maintain a normal PaCO₂

DRUG TREATMENT
Hypovolaemic shock
Intravenous fluids
Choose the type of replacement fluid to resemble the type of fluid lost from the body.
Give IV fluids to:
• correct the intravascular fluid deficit
• improve circulation
• restore blood pressure
Administer a rapid IV fluid bolus of 20 mL/kg. Repeat bolus fluid until improvement of tissue perfusion, circulation, blood pressure and central venous pressure is achieved. If there is an inadequate response after 40 mL/kg has been given, a third bolus can be started and the patient should be moved to ICU for CVP monitoring and inotropic support.
After stabilisation of the circulation, continue with maintenance fluid volumes according to the age of the patient.

For blood loss
• packed red cells, 10 mL/kg or whole blood, 20 mL/kg, until the haemoglobin is 12 g/dL.
  Maintain haematocrit at 36% (haemoglobin 12 g/dL) or higher.
  While awaiting blood for replacement begin volume resuscitation with crystalloid fluid preparations.

Fluid loss other than blood
Crystalloid, e.g.
• sodium chloride 0.9% or Ringer-Lactate, IV, 20 mL/kg given as a bolus over 10–20 minutes.
Repeat the bolus fluid until improvement is achieved x 3.

Cardiogenic shock
Ideally children receiving treatment for cardiogenic shock should be in high care or ICU.
Inotropic support
When perfusion is poor and blood pressure response unsatisfactory, despite adequate fluid replacement and a central venous pressure of > 10 cm H$_2$O.

- dopamine, IV, 2–6 mcg/kg/minute
  AND/OR
- dobutamine, IV, 2–6 mcg/kg/minute

Chronotropic support
For cardiogenic shock, when myocardial injury/damage is suspected or when the heart rate is:
- child $\leq$ 100/minute
- neonate $\leq$ 120/minute

Adrenaline and/or afterload reduction can be considered if tissue perfusion and blood pressure do not improve satisfactorily on adequate fluid volume replacement, inotropic and/or chronotropic support.

Note:
Do not introduce inotropic and/or chronotropic support unless serum calcium and potassium are in the physiological range and the fluid volume deficit has been corrected.

- adrenaline, IV infusion, 0.01–1 mcg/kg/minute

Septic shock
Children receiving treatment for septic shock should be in an ICU. The resuscitation and treatment of these children must be aggressive and with continuous reassessment to ensure that progression through the therapeutic options is rapid and appropriate to the response.

Antibiotic therapy
Start antibiotics early to cover Gram-positive and Gram-negative organisms. Before initiating antibiotic therapy, take blood, and urine specimens, if appropriate, for culture and sensitivity testing. Reconsider antibiotic and/or antifungal therapy when culture and sensitivity results become available, or when sepsis has been ruled out as a cause of the shock syndrome.

3$^{rd}$ generation cephalosporins, e.g.
- cefotaxime, IV, 25–50 mg/kg/dose, 6–8 hourly
  OR
- ceftriaxone, IV, 50 mg/kg/dose, 12 hourly

AND
Aminoglycoside, e.g.
- gentamicin, IV, 7.5 mg/kg once daily
Give fluid boluses and monitor response
- sodium chloride 0.9% or Ringer-Lactate, IV, 20 mL/kg rapidly within 10 minutes
  Repeat boluses of 10–20 mL/kg as required according to response.
Unremitting shock without signs of congestion of the circulatory system require repeated boluses of volume expander solution.
Children with septicaemia may require up to or more than their blood volume, 80 mL/kg, in a 24 hour period to achieve adequate circulation.

Monitor for persistence of shock:
- decreasing BP
- increasing pulse rate/ decreasing volume
- increasing capillary filling time

Monitor for fluid or circulatory overload:
- increasing respiratory rate
- increasing basal crepitations
- increasing pulse rate
- increasing liver size/tenderness
- increasing JVP

If more than 40 mL/kg boluses given, consider CVP monitoring.

If shock persists after 40 mL/kg of boluses or if signs of cardiac failure
- dopamine, IV infusion, 5 mcg/kg/minute
  AND/OR
- dobutamine, IV infusion, 5 mcg/kg/minute

With poor response increase infusion rate incrementally for either or both infusions to a maximum of 15 mcg/kg/minute.

Failure of the above
- adrenaline, continuous IV infusion, 0.01 –1 mcg/kg/minute

In unresponsive septicaemic shock
Consider low doses steroids
- hydrocortisone, IV, 1 mg/kg/dose, 6 hourly

REFERRAL
- all

CAUTION
Patients must be resuscitated and stabilised before referral

1.1.6.1 Intraosseous Infusion

If an intravenous drip cannot be set up within 5–10 minutes, set up an intraosseous infusion. Use an 18 X 1.5 or 20 X 1.5 lumbar puncture needle or an intra-osseous infusion needle if available.

1. Grasp the thigh and knee above and lateral to the insertion site with the palm of the left hand (if right-handed). Wrap the fingers around the knee to stabilise the proximal tibia. Do not allow any portion of your hand to rest behind the insertion site.
2. Find the site of insertion i.e. feel the tibial tuberosity. The site of insertion is 2 cm below this tuberosity on the broad flat medial surface of the tibia.
3. Careful surgical preparation of the injection site as for lumber punctures.
4. Insert the needle through the skin over the flat surface of the tibia.
5. Holding the needle low down near the skin, advance the needle through the bony cortex of the tibia, directing the needle perpendicular, i.e. 90° to the long axis, using a gentle but firm twisting or drilling motion.
6. Stop advancing the needle when a sudden decrease in resistance to forward motion of the needle is felt.
7. If a spinal needle is used, remove the stylet from the needle.
8. Slowly inject 10 mL sodium chloride 0.9% through the needle. Check for any signs of increased resistance to injection, increased circumference of the soft tissues of the calf, or increased firmness of the tissue.
   If an injection needle has been used, the needle might be blocked by a core of bone, which may need to be flushed through. The flow rate should rapidly increase after flushing through.
9. If the test injection is successful, disconnect the syringe and join an infusion set to the needle. Secure the needle and tubing with tape and support it with a bulky dressing.
10. If the test injection is unsuccessful, i.e. infiltration of the normal saline into the leg tissue is observed, remove the needle and try again on the other leg.

**Signs of successful insertion:**
- Sudden decrease in resistance to insertion as the needle passes through the bony cortex.
- The needle remains upright without support.
- Fluid flows freely through the needle without evidence of subcutaneous infiltration.
1.2 TRAUMA

1.2.1 BURNS

DESCRIPTION
Skin and tissue damage caused by:
- exposure to extremes of temperature
- contact with an electrical current
- exposure to a chemical agent
- radiation

ASSESSMENT OF BURNS

<table>
<thead>
<tr>
<th>Depth of burn</th>
<th>Degree</th>
<th>Surface/colour</th>
<th>Pain sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superficial (Partial loss of skin)</td>
<td>1&lt;sup&gt;st&lt;/sup&gt;</td>
<td>Dry, minor blisters, erythema</td>
<td>Painful</td>
</tr>
<tr>
<td>Partial A (Superficial dermal)</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; A</td>
<td>Blisters</td>
<td>Painful</td>
</tr>
<tr>
<td>Partial B (Deep dermal)</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; B</td>
<td>Moist white slough, red mottled</td>
<td>Painful</td>
</tr>
<tr>
<td>Full thickness (Deep/complete loss of skin)</td>
<td>3&lt;sup&gt;rd&lt;/sup&gt;</td>
<td>Dry, charred whitish</td>
<td>Painless</td>
</tr>
</tbody>
</table>

DIAGNOSTIC CRITERIA
Burns are classified as minor or major burns.

Major burns:
- partial thickness burns of > 10% body surface area
- full thickness burn of > 3% body surface area
- any burn involving the head and face, hands, feet and perineum
- inhalation injuries
- circumferential burns
- electrical burn injuries
- neonatal burns
- burns in patients with serious pre-existing or concomitant injuries

Minor burns:
- partial thickness burns of < 10% body surface area in a child over 1 year of age
Estimation of percentage of burns:
Body surface area % according to age

<table>
<thead>
<tr>
<th>Age in years</th>
<th>Head (A/D)</th>
<th>Thigh (B/E)</th>
<th>Leg (C/F)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>10</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>1</td>
<td>9</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>6</td>
<td>5</td>
<td>3</td>
</tr>
</tbody>
</table>

Examine carefully to determine:
- other injuries
- respiratory signs due to smoke inhalation

NON-DRUG TREATMENT
Emergency treatment
Soak or immerse the affected area in cold water for the first hour after the accident to limit the extent of the burn.
Remove clothing and gently clean the wound with running water.

BSA burns >20% are often associated with paralytic ileus, leading to gastric distension.
Free nasogastric drainage should be in place.
Concomitant nasojejunal feeding may be attempted within 6 hours under expert supervision.

Fluid resuscitation
Intravenous fluid resuscitation is indicated in:
- shocked patients
- children < 1 year with > 5% burns
- children > 1 year with > 10% burns
- the presence of haemoglobinuria
Avoid circumferential taping when securing infusion lines, as oedema under the eschar may decrease the venous return.

**If in shock**
- sodium chloride 0.9% or Ringer-Lactate, IV, 20 mL/kg immediately as a bolus
  Repeat if needed.
  Continue resuscitation fluid over first 24 hours.

**IV Fluids for replacement and maintenance**
Give estimated losses for each 24-hour period due to the burn + maintenance requirement for 24 hour period.

**Replacement fluids for burns**
**First 24 hours**
- Ringer-Lactate, IV, 4 mL/kg x % burned BSA
  Urine output should be 1–2 mL/kg/hour
  Catheterise patients with >20% burns

**Second 24 hours**
- sodium chloride 0.9% or Ringer-Lactate, IV, 2 mL/kg x % burned BSA

**PLUS**
**Maintenance fluids**
Can be given orally or intravenously.
Dextrose-containing maintenance fluids are given according to age:

<table>
<thead>
<tr>
<th>≤1 year</th>
<th>120 mL/kg/24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>All children older than 1 year – the sum of the following:</td>
<td></td>
</tr>
<tr>
<td>• first 10 kg body weight</td>
<td>100 mL/kg/24 hours</td>
</tr>
<tr>
<td>• second 10 kg body weight</td>
<td>50 mL/kg/24 hours</td>
</tr>
<tr>
<td>• additional weight greater than 20 kg body weight</td>
<td>20 ml/kg/24 hours</td>
</tr>
</tbody>
</table>

**Example: 24 kg child with 10% burns**

<table>
<thead>
<tr>
<th>1st 24 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>• replacement for expected losses: 4 mL/kg x 24 kg x 10% = 960 mL</td>
</tr>
<tr>
<td>• maintenance: first 10 kg = 10 kg x 100 mL/kg/24 hours = 1 000 mL second 10 kg = 10 kg x 50 mL/kg/24 hours = 500 mL remaining 4 kg = 4 kg x 20 mL/kg/24 hours = 80 mL Total maintenance = 1 580 mL Total fluids in 1st 24 hours = 960 mL + 1 580 mL = 2 540 mL</td>
</tr>
</tbody>
</table>
2nd 24 hours

- Replacement for expected losses:
  \[2 \text{ mL/kg} \times 24 \text{ kg} \times 10\%\]
  \[480 \text{ mL}\]

- Maintenance:
  - First 10 kg = 10 kg \times 100 \text{ mL/kg/24 hours} = 1000 \text{ mL}
  - Second 10 kg = 10 kg \times 50 \text{ mL/kg/24 hours} = 500 \text{ mL}
  - Remaining 4 kg = 4 kg \times 20 \text{ mL/kg/24 hours} = 80 \text{ mL}

Total maintenance = 1580 mL

Total fluids in 2nd 24 hours = 480 mL + 1580 mL = 2060 mL

Anaemia
  - Packed red cells, 10 mL/kg over 3 hours if haemoglobin < 7 g/dL

Hypoalbuminaemia
  Prevent by starting enteral feeds early. If indicated,
  - Albumin 20%, IV, 2 g/kg/day
    \[2 \text{ g} = 10 \text{ mL}\]

Inhalation injury
  In addition to other treatment, the degree of inhalation injury may warrant:
  - Monitoring of blood gases
  - Warm humidified oxygen and/or intubation
  - Positive pressure ventilation

Suspect carbon monoxide poisoning in all fire victims. Obtain carboxyhaemoglobin level. Treat by administering 100% oxygen.

Prevent heat loss
  Nurse all major burns in a warm room.

Nutritional support
  A dietician should preferably be involved as children with burns require a higher than usual intake of nutrients.
  Start enteral feeds within 6 hours, especially in patients with burns > 20%.

Estimate daily energy and protein needs using the formulae:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kJ)</td>
<td>[250 \text{ kJ/kg body mass} + (150 \text{ kJ x % burned BSA})]</td>
</tr>
<tr>
<td>Protein:</td>
<td>[3 \text{ g/kg body mass} + (1 \text{ g x % burned BSA})]</td>
</tr>
</tbody>
</table>

Maximum % burn area used for calculation should not exceed 50%

Give iron and vitamins routinely until burn wounds are healed and/or skin grafting has successfully been completed.

Note:
  Do not supplement iron during sepsis or infection.

Also provide
  - Psychological support
  - Physiotherapy
  - Occupational therapy
  - Waterbeds and cradles

16
**DRUG TREATMENT**

**Analgesia**
Children with large burns need effective pain relief. Provide analgesia cover at each dressing change. Major burns dressings should be changed under general anaesthesia.

**Change of dressing** medications at least half an hour before:

Midazolam + paracetamol + tilidine  
OR  
Midazolam + paracetamol + ketamine (orally)  
OR  
General anaesthesia

**Note:**  
The intravenous formulations of ketamine and midazolam can be given orally.

- midazolam, oral, 0.5 mg/kg/dose (anxiolysis only)
- paracetamol, oral, 15 mg/kg/dose
- tilidine, 1 mg/kg/dose  
  1 drop = 2.5 mg  
  Number of drops = body weight ÷ 2.5  
  Not recommended for infants less than one year.
- ketamine, oral, 2–5 mg/kg/dose

**Background pain analgesia**  
See Pain Syndromes: Section 20.2

**Gastric erosions**  
See Section 2.2.7.

**Local treatment of burns**  
Gently clean the wounds with running water. Remove loose skin and debride dead tissue. Next day, rinse with running water and dress with topical antiseptic cream and non-adherent dressing.

**Superficial (partial thickness) burns**  
These will heal spontaneously.  
- silver sulphadiazine 1%, topical  
  Cover with paraffin gauze.

**Full thickness burns**  
Topical antiseptic, e.g.  
- silver sulphadiazine 1%, topical, on non-adhesive dressings  
  Cover with paraffin gauze.  
  Change dressings daily.
All full thickness or deep dermal burns should be excised and grafted as soon as the patient is stable. Wounds not healed in two weeks require skin grafting.

**Antibiotics**
Consider if signs of infection are present. The choice of antibiotics is based on the culture and sensitivity results of wound, urine and blood cultures. Not all positive wound cultures indicate systemic infections requiring antibiotic treatment.

Burns > 50% BSA, inhalation injury with respiratory tract damage, proven burn wound sepsis, septicaemia or other infections
- ceftazidime, IV, 15–25 mg/kg/dose, 8 hourly for 5–14 days
- AND
- amikacin, IV, 15–20 mg/kg once daily for 5–14 days provided renal function is satisfactory

**Tetanus prevention**
Patients with no previous immunisation in the last 5 years
- tetanus toxoid, IM, 0.5 mL. Complete course in previously unvaccinated patients.

Where deep necrotic lesions are part of the burn and if the immunological status is not known
- tetanus immunoglobulin, IM, 500 IU

**Prior to transport/referral**
- commence resuscitative measures if necessary.
- administer 100% humidified oxygen by facemask for inhalation injuries
- cover wounds with clean dressings after hot or smouldering clothing has been removed

**REFERRAL**
- major burn injuries
- burns covering more than 10% of body surface
- all burns involving the hands, joints, face, eyes, ears, feet and perineum
- all inhalation injuries
- electrical or chemical injuries
- all children less than 3 months
- infected burns
2.1 DENTAL AND ORAL DISORDERS

2.1.1 HERPES GINGIVOSTOMATITIS

DESCRIPTION
Inflammation of the mouth structures with multiple small ulcers, caused by Herpes simplex virus infection. The normal course of the disease is 7–10 days.

DIAGNOSTIC CRITERIA
Clinical
• general inflammation of the mouth with multiple small ulcers on the buccal mucosa, palate, anterior tonsillar pillars, tongue, inner lips and gingiva
• fever, malaise and dysphagia
• tender, enlarged cervical lymph nodes

NON-DRUG TREATMENT
• maintain adequate nutrition and hydration. Maintain hydration with oral/nasogastric and/or IV fluids if necessary

DRUG TREATMENT
• chlorhexidine 0.2%, 10 mL as a mouthwash or gargle, 12 hourly
  Do not swallow.

• paracetamol, oral, 10–15 mg/kg/dose 6 hourly
  OR
  ibuprofen, oral, 5–10 mg/kg/dose 6 hourly

If immunocompromised or very severe infection, under specialist supervision
• aciclovir, IV, 5–10 mg/kg/dose 8 hourly for 7–14 days
  Change to oral as soon as possible

• aciclovir, oral, 10–20 mg/kg/dose 4–6 hourly

If poor response or suspected super infection
• amoxicillin/clavulanic acid, oral, 35–45 mg/kg/dose of amoxicillin component, 8 hourly

For extensive oral herpes
• lidocaine 2% gel applied every 3 to 4 hours. Apply a thin layer on the affected areas only
REFERRAL
- herpes gingivostomatitis not responding to therapy
- progressive disease, especially if associated with encephalopathy or increasing liver span

2.2 GASTROINTESTINAL DISORDERS

2.2.1 CHOLERA
A00.9
* Notifiable condition.

DESCRIPTION
An acute diarrhoeal disease caused by *Vibrio cholerae*.

DIAGNOSTIC CRITERIA
Clinical
- sudden onset of severe, watery diarrhoea, i.e. ‘rice water’ diarrhoea
- low-grade or no fever
- persistent vomiting not associated with nausea
- rapid fluid and electrolyte losses with dehydration, acidosis and hypovolaemic shock with or without renal failure
- history of contact with a cholera case or the presence of cholera in the community

Investigations
- positive stool culture
- agglutinating or toxin-neutralising antibodies in the serum

NON-DRUG TREATMENT
- isolate patient and institute barrier nursing
- ensure adequate nutrition
- ensure adequate hydration - See Acute Diarrhoea: Section 2.2.4

DRUG TREATMENT
- ciprofloxacin, oral, 20 mg/kg as a single dose

Chemoprophylaxis
For household and close contacts
children:
- ciprofloxacin, oral, 20 mg/kg as a single dose

adults:
- ciprofloxacin, oral, 1 000 mg as a single dose

REFERRAL
- cholera with complications, e.g. persistent shock, renal failure and severe electrolyte disturbances
### 2.2.2 CONSTIPATION / FAECAL LOADING

**K59.0**

#### DESCRIPTION

**Constipation:** the infrequent passage of hard stools.  
**Faecal soiling:** the involuntary leakage of small amounts of soft or watery stools secondary to faecal loading.

Causes include:
- incorrect diet
- lack of exercise
- certain medicines
- metabolic, endocrine, neurogenic and lower bowel abnormalities
- psychogenic disorders
- chronic use of enemas

#### DIAGNOSTIC CRITERIA

- non-tender deformable faecal masses palpable
- confirm on a straight abdominal X-ray

#### NON-DRUG TREATMENT

- determine and treat the underlying cause
- treatment involves 3 steps:
  - initial clearance of stools
  - prevent reaccumulation of hardened retained stool
  - retraining of the gut to achieve regular toilet habits
- management is long-term, and requires the active involvement of the parents

#### DRUG TREATMENT

**Initial therapy**

Faecal clearance if faecal loading
- phosphate-containing enema twice daily for 3 days
  - polyethylene glycol solution with sodium sulphate and electrolytes, oral/via nasogastric tube, 10–25 mL/kg/hour until clear fluid is passed rectally  
  - Do not use sweeteners containing sugar.  
  - Confirm the bowel is empty.

**Maintenance therapy**

**Bowel re-training**

Diet change with additional natural fibre from fruit, vegetables and bran.

Additional fibre:
- ispaghula husk, oral, 1.75–3.5 g, stirred in water with breakfast
  - liquid paraffin, oral, 2 mL/kg/day
  - Do not use sweeteners containing sugar.
  - Confirm the bowel is empty.
In refractory cases
- lactulose, oral, twice daily
  - < 1 year 2.5 mL
  - 1–6 years 5 mL
  - > 6 years 10 mL

If faecal loading, maintenance therapy should be continued for months to years.

REFERRAL
- suspected organic cause e.g. constipation from birth in a breast-fed baby
- inadequate response to therapy

2.2.3 CYSTIC FIBROSIS
E84.9

DESCRIPTION
An autosomal recessive disorder of exocrine glands, mainly affecting the gut and lungs.

DIAGNOSTIC CRITERIA
Clinical
- recurrent infections of the respiratory tract with later bronchiectasis, respiratory failure and cor pulmonale
- bulky, greasy and foul-smelling stools
- occasionally present with constipation
- malabsorption with weight loss and failure to thrive
- meconium ileus
- family history, rare unless in a sibling

Investigations
- sweat test
  - quantitative analysis of sodium and chloride concentrations in sweat collected after stimulation by pilocarpine iontophoresis with chloride > 60 mmol/L
- DNA analysis for delta F508 and a few other mutations

NON-DRUG TREATMENT
- nutritional support
  - well balanced diet
  - oral intake of at least 120% of recommended daily allowance
  - nutritional supplements
  - occasionally nocturnal supplemental feeding by nasogastric or gastrostomy tube
- physiotherapy and postural drainage
- psycho-social support
- genetic counselling
DRUG TREATMENT
Drug treatment is specialised and individualised and should be under the supervision of a subspecialist.

REFERRAL
• all to a recognised cystic fibrosis centre and/or specialist health facility for confirmation of diagnosis and initiation of treatment
• management of exacerbations

2.2.4 DIARRHOEA, ACUTE
A09

DESCRIPTION
Diarrhoea is a serious common childhood illness evidenced by the passing of frequent profuse loose watery stools. Vomiting may or may not be present. Diarrhoeal disease is often caused by viral infection but may be due to bacterial infection, dietary or other causes. Dehydration and metabolic disturbances are common if treatment is not instituted early and may result in severe disease, irreversible organ damage and death in children. Malnutrition is a serious co-morbidity and/or result of diarrhoeal disease and must be managed correctly employing ongoing feeding while treatment is given except during ileus or shock.

DIAGNOSTIC CRITERIA
Clinical
Adequate initial assessment and frequent reassessment (4 hourly if dehydration is present – more often in the presence of shock) is vital in the care of these children.
• shock
  ○ children with shock at first compensate by decreasing their peripheral circulation, seen by increased capillary filling time (> 3 seconds) or cool peripheries, and increasing pulse rate
  ○ late signs of shock include decreased blood pressure and decreased level of consciousness
  ○ assess capillary filling time and pulse volume/rate
• dehydration
  ○ use the table to assess dehydration

First assess **shock**, then **dehydration** then **no visible dehydration**

<table>
<thead>
<tr>
<th>Signs of dehydration</th>
<th>Shock</th>
<th>Severe dehydration</th>
<th>Dehydration</th>
<th>No visible dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>one of the signs below</td>
<td>two of the signs below</td>
<td>two of the signs below but not severe dehydration</td>
<td>none of the signs of dehydration</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>decreased level of consciousness</td>
<td>lethargic or unconscious</td>
<td>restless or irritable</td>
<td>well, alert</td>
</tr>
<tr>
<td>Eyes sunken</td>
<td>eyes sunken</td>
<td>eyes sunken</td>
<td>eyes sunken</td>
<td>eyes not sunken</td>
</tr>
<tr>
<td>Ability to drink</td>
<td>drinks poorly or not able to drink</td>
<td>thirsty, drinks eagerly</td>
<td>drinks normally, not excessive thirst</td>
<td></td>
</tr>
<tr>
<td>Skin pinch (turgor)</td>
<td>severe decrease in skin turgor; skin pinch returning in &gt; 2 seconds</td>
<td>moderate decrease in skin turgor; skin pinch returning in &lt; 2 seconds</td>
<td>skin pinch goes back immediately</td>
<td></td>
</tr>
<tr>
<td>Capillary filling time</td>
<td>capillary filling time &gt; 3 seconds</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>decreased BP rapid thready pulse</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

• assess for signs of metabolic, nutritional and other comorbidities:
  ○ level of consciousness
  ○ decreased bowel sounds
  ○ tone for floppiness
  ○ recalcitrant or bile stained vomiting
  ○ abdominal distension
  ○ urine for leucocytes or nitrites
  ○ respiratory rate and chest indrawing

**Investigations**
- Na⁺, K⁺, urea, creatinine, blood acid base assessment and other investigations as indicated, in all children with severe dehydration, shock or other signs of metabolic, nutritional or other co-morbidities after resuscitation
• stool culture if at a sentinel site for infectious GIT disease, or suspected dysentery, typhoid, cholera
• urine test strip on fresh/clean urine specimen for leucocytes, nitrites and blood

**NON-DRUG TREATMENT**
- adequate initial assessment and frequent reassessment is vital
- reassess the condition of the patient continuously while shock persists
- if dehydration is present 4 hourly reassessment and immediate correction of shock or deterioration
- monitor and maintain:
  - blood pressure
  - blood electrolytes
  - fluid balance
  - acid–base status
  - blood glucose within physiological ranges
- monitor urine output, should be at least 1 mL/kg/hour
- monitor body mass regularly, weigh daily
- educate caregivers about hygiene, oral rehydration solution and danger signs of diarrhoea
- continue oral feeds during period of diarrhoea:
  - if the child is breast fed, continue breast feeds and encourage the child to feed longer at each feed
  - if the child is exclusively breastfed, give ORS in addition to each feed
  - if the child is not exclusively breast fed, give ORS and other appropriate feeds, e.g. breast milk substitutes or food based fluids
  - if the child is severely dehydrated or shocked feeding may be withheld until stable, usually a few hours only

**DRUG TREATMENT**

There is no place for anti-diarrhoeal medications, i.e. kaolin and pectin, atropine and diphenoxylate, loperamide or antiemetics in the management of acute diarrhoea.
## Outline of Practical Fluid Therapy of Dehydrating Watery Diarrhoea

<table>
<thead>
<tr>
<th>Evaluation</th>
<th>Shock</th>
<th>Severe dehydration</th>
<th>Moderate dehydration</th>
<th>Not obviously dehydrated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Needs resuscitation</td>
<td>Needs urgent fluids and resuscitation</td>
<td>Needs oral rehydration</td>
<td>Potential dehydration for home treatment</td>
</tr>
<tr>
<td>Evaluation</td>
<td>Start IV drip and give: Ringer–Lactate, IV, 20 mL/kg in 10–20 minutes</td>
<td>Start IV drip and give: Ringer–Lactate, IV, 30 mL/kg in 1 hour Reassess after 1 hour: pulse, circulation and capillary filling time. Still severely dehydrated or in shock?</td>
<td>Give supervised ORS for 4–6 hours. Start with small amounts; increase to offer 15–20 mL/kg/hour in small frequent sips. If patient wants more, offer more. Do not allow child to drink large volumes because of risk of vomiting. If child vomits, wait 10 minutes and give again in small frequent quantities. Reassess after 4 hours: Hydration better, not vomiting, wanting food?</td>
<td>Give extra fluids after small feeds and after each diarrhoeal stool. Continue breastfeeding or formula feeding and give food as tolerated. Offer ORS after each stool and after feeds: 10 mL/kg. If patient wants more, offer more in frequent small sips to avoid vomiting May need to disguise the taste of ORS with juice etc. Explain how ORT works: replacement of water losses but not treatment of diarrhoea per se. Explain natural history of disease.</td>
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<td>Reassess after 20 minutes: pulse, circulation, capillary filling time: Still in shock?</td>
<td>• YES Repeat bolus of Ringer–Lactate, 20 mL/kg Refer to ICU if not responding. Do blood tests as below.</td>
<td>• YES Move to column 1 • IMPROVED, PASSING URINE: CONTINUATION PHASE Change drip to Darrows half strength with dextrose 5% at 10 mL/kg/hour Reassess in 4 hours: General state better, able to take oral fluids?</td>
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<tr>
<td>INVESTIGATION</td>
<td>Reduce drip rate to 5 mL/kg/hour and start oral rehydration (next column)</td>
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<tr>
<td></td>
<td>• NO Evaluate blood test results, stool and urine output. Increase drip rate to 10–15 mL/kg/hour, if necessary</td>
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<td>Reassess in 4 hours: Hydration better, able to take oral fluids?</td>
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<td></td>
<td>• YES Reduce drip rate to 5 mL/kg/hour and start oral rehydration (next column)</td>
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<td></td>
<td>well on drip rate &lt; 5 mL/kg/hour, consider stopping the drip.</td>
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<td></td>
<td>• NO Evidence of shock? Resuscitate as before</td>
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<td>Hydration worse? Check fluid administration (how much given?). Consider drip or increase oral fluids.</td>
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<td></td>
<td>In hospital: Review hydration twice daily. Weigh daily: weight loss reflects dehydration. Discharge once hydration maintained without drip and stools becoming less watery</td>
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<td></td>
<td>Home management: Diarrhoea must stop within a week. Give extra food for nutritional recovery. To come back if stools become bloodstained, diarrhoea not stopping in a week or if caregiver still concerned.</td>
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</tbody>
</table>
1. First treat shock, if present
   - Ringer-Lactate, IV, 20 mL/kg given as a bolus over 10–20 minutes.
     Repeat the fluid bolus until improvement is achieved up to 3 times.
     After each bolus reassess for shock.
     After the second bolus, i.e. total of 40 mL/kg has been given with inadequate
     response, the third bolus is started and the patient should be moved to ICU for
     CVP monitoring and inotropic support.
     After stabilisation of the circulation, continue with maintenance fluid volumes
     according to the age of the patient – see: 2. Severe dehydration

```
If an IV infusion cannot be set up within 5–10 minutes use an intraosseous infusion.
See section 1.1.6.1
```

2. Severe dehydration
   - Ringer-Lactate, IV, 20 mL/kg given as a bolus over 10–20 minutes or 30 mL/kg in first
     hour
   Continue with
   - Darrows half strength with dextrose 5%, IV at 10 mL/kg/hour
     Give more if stool output is very high.

Allow oral sips once shock is controlled and no ileus.
Review after 4 hours: general condition, capillary filling time, passing urine, number of
watery stools and level of consciousness.

If no improvement, repeat fluid bolus and increase fluid administration to 15 mL/kg/hour
depending on the extent of ongoing diarrhoea.

Review after 4 hours.

If improved and alert and not vomiting, introduce oral rehydration fluid at an increasing rate
of 5–15 mL/kg/hour or more in small frequent sips and reduce IV fluid rate by 5 mL/kg/hour.
Review in 4 – 6 hours.
As patient takes more ORS without vomiting, reduce the IV rate.

Once hydration corrected, offer small feeds as tolerated and supplement freely with ORS after
feeds and extra for every watery stool. Discontinue IV drip once rate less than 5 mL/kg/hour.

3. Moderate dehydration for oral rehydration
   - ORS, oral, 80 mL/kg over 4 hours using frequent small sips
     Give more if the child wants more.
     Show the caregiver how to give ORS with a cup and spoon.
     If child vomits wait 10 minutes and then continue more slowly.
     Encourage caregiver to continue feeding the child, especially breast-feeding.
Chapter 2

Review after 4 hours.

After 4 hours:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Action</th>
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</thead>
<tbody>
<tr>
<td>If there are signs of shock</td>
<td>treat for shock, and change to the IV regimen as in 2 above</td>
</tr>
<tr>
<td>The dehydration has continued without improvement or if it has become worse</td>
<td>change to IV regimen indicated in 2 above</td>
</tr>
<tr>
<td>If still some dehydration signs but improving</td>
<td>continue same protocol</td>
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</tbody>
</table>

4. No visible signs of dehydration
Show the caregiver how to give ORS with a cup and spoon using frequent small sips. Encourage caregiver to give 10 mL/kg after each diarrhoeal stool until diarrhoea stops, i.e.
- child age up to 2 years, 50–100 mL
- child age 2 years or more, 100–200 mL after each loose stool
Instruct the caregiver how to make ORS/SSS at home and to continue treatment. Home made sugar and salt solution may be used if oral rehydration formula is not available.

<table>
<thead>
<tr>
<th>HOMEMADE SUGAR AND SALT SOLUTION (SSS)</th>
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<tr>
<td>½ level teaspoon salt</td>
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<tr>
<td>+</td>
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<tr>
<td>8 level teaspoons sugar</td>
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<td>+</td>
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<tr>
<td>1 litre of boiled then cooled water</td>
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</tbody>
</table>

Encourage the caregiver to continue feeding the child, especially breast-feeding. Instruct the caregiver to give the child extra feeds after the diarrhoea has stopped to make up for the period of inadequate intake.
Child should return immediately if:
- no improvement
- condition deteriorates
- poor drinking or feeding
- slow skin pinch
- blood in stool
- fever develops
- sunken eyes

5. Dehydration in severely malnourished patient
The assessment of dehydration is much more difficult in a malnourished patient. Confirm a history of watery stool losses. Avoid intravenous infusions if possible.
In resuscitation from shock, reduce the bolus volume to 15 mL/kg over 1 hour. Reassess pulse, size of liver and respiratory rate during infusion. Stop infusion if there is deterioration. Consider change to nasogastric drip infusion of Darrows half strength with dextrose 5% at 10 mL/kg/hour.
Metabolic disturbances

Acidosis

Metabolic acidosis does not require correction unless extremely severe, i.e. pH < 7.1, or if the body is unable to correct the deficit, e.g. salicylate poisoning and renal failure. Correction should only be considered with expert supervision. Correction of renal circulation and shock will lead to self-correction in almost all cases.

If correction is necessary: volume of sodium bicarbonate 4.2% required
\[ = 0.3 \times \text{base deficit} \times \text{weight in kg} \]
Low serum potassium

- If potassium is less than 3.5 mmol/L but greater than 2.5 mmol/L
  - Potassium chloride, oral, 25–50 mg/kg/dose 8 hourly
    - If potassium is less than 2.5 mmol/L
  - Darrow’s half strength with dextrose 5%, 200 mL plus potassium chloride 15%, 1 mL, IV
    - (1 mL potassium chloride 15% = 2 mmol)
    - Mix well before administration.
    - Run additional Darrow’s half strength with dextrose 5% containing potassium chloride at the appropriate rehydration rate (see above).

Oral potassium may also be given during this period

- Potassium chloride, oral, 25–50 mg/kg/dose eight hourly

Monitor serum potassium 8 hourly. Once above 3.0 meq/L, stop IV potassium and continue with oral.

High serum sodium

Continue to rehydrate with Darrow’s half strength with dextrose 5%.

Repeat serum Na⁺ every 12 hours to monitor progress.

Failure to decrease Na⁺ usually means the rehydration rate is too slow.

Fall of more than 1 mmol/hour on average means the rehydration rate is probably too rapid.

Low serum sodium

Replace Darrow’s half strength with dextrose 5% with Ringer-Lactate or sodium chloride 0.9% to give a total volume of sodium chloride 0.9% calculated as follows:

\[ \text{Volume of sodium chloride 0.9\%} = (130 - \text{Na}^+) \times \text{body weight in kg} \times 4 \]

- Sodium chloride 0.9%, 200 mL plus potassium chloride 15%, 2 mL plus dextrose 50%, 20 mL, IV
  - Mix well before administration.

After the calculated volume has been given, resume Darrow’s half strength with dextrose 5% at the appropriate rate according to treatment of dehydration process above and recheck the serum electrolytes (Na⁺).

Antibiotic therapy

Note:
During diarrhoea absorption of antibiotics may be impaired due to intestinal hurry. Give antibiotics orally if administered for intraluminal effect.
Other antibiotics are best administered parenterally.
Consider urinary tract infection or septicaemia in malnourished and immunocompromised children and infants less than 3 months old.
For dysentery: treat initially as shigella dysentery
• ciprofloxacin, oral, 15 mg/kg/dose 12 hourly for 3 days. Consider the risk benefit ratio as highlighted in the package insert when using this medication in children.
  OR
  cefotaxime, IV 50 mg/kg/dose 6 hourly for 5 days
  OR
  ceftriaxone, IV, 20–80 mg/kg as a single daily dose for 5 days

If entamoeba histolytica seen or failed response
• metronidazole, oral, 7.5 mg/kg/dose 8 hourly

For cholera
• ciprofloxacin, oral, 20 mg/kg as a single dose

For typhoid
• ceftriaxone, IV, 50–75 mg/kg once daily for 7–10 days

With severe malnutrition
• ampicillin, IV, 25–50 mg/kg/dose 6 hourly for 5 days
  PLUS
  • gentamicin, IV, 7.5 mg/kg as a single daily dose for 5 days

Very young infants less than 28 days old
• ampicillin, IV, 25–50 mg/kg/dose 6 hourly for 5 days
  PLUS
  • gentamicin, IV, 7.5 mg/kg as a single daily dose for 5 days

Mineral and micronutrient deficiencies
• zinc acetate, oral,
  < 10kg  10 mg/day
  > 10kg  20 mg/day

• potassium chloride, oral, 8 hourly
  < 6 months  125 mg
  > 6 months  250 mg
  Unless hyperkalaemic or anuric.

If recurrent diarrhoea
• vitamin A, oral as a single dose
  6–12 months  100 000 IU
  > 12 months  200 000 IU
REFERRAL

- inability to correct shock, metabolic complications or ongoing diarrhoea at current level of care

Always continue appropriate therapy for shock and dehydration therapy before and during referral.

2.2.5 DIARRHOEA, CHRONIC / PERSISTENT

K52.9

DESCRIPTION

Persistent diarrhoea: an episode that begins acutely and lasts at least 7 days.

Chronic diarrhoea: four or more loose stools per day for longer than two weeks.

Prolonged diarrhoea results in significant morbidity and mortality associated with poor nutrition.

Chronic/persistent diarrhoea is most frequently due to temporary loss of disaccharidase activity in the intestinal microvillous brush border, usually lactase loss, or luminal infection/infestation, which may be non-specific bacterial overgrowth. Rare causes include food allergies, cystic fibrosis and coeliac disease.

DIAGNOSTIC CRITERIA

Clinical

- diarrhoea without weight loss or dehydration – consider Toddler’s diarrhoea
- diarrhoea with weight loss and dehydration – consider small bowel mucosal injury, e.g. lactose intolerance or small bowel bacterial overgrowth
- diarrhoea with weight loss but no dehydration – consider a malabsorption syndrome, e.g. celiac disease, allergic enteropathy, cystic fibrosis, etc.
- consider the possibility of HIV infection

Investigations

Where weight gain falters, dehydration recurs, the child is ill or the diarrhoea continues

- full blood count
- serum proteins
- stool-reducing substances > 0.5% reducing sugar is abnormal if on a lactose-containing diet
- urine and stool microscopy
- culture and sensitivity tests (MCS)

NON-DRUG TREATMENT

Treatment strategy includes a stepwise approach with modification of the diet, which are not mutually exclusive and are applied according to local resources.

- monitor hydration, stools, nutritional status, weight gain, growth and other nutritional parameters such as serum proteins
- nutritional support
  - aim to provide at least 110 kcal/kg/day orally within three days to protect nutrition. In the step-wise protocol (see table/box below) this becomes formalised in the formula.
prior to this or where the stepwise approach is not possible:

- **under 4 months:**
  Encourage exclusive breastfeeding if lactose intolerance is not severe. If not exclusive breastfeeding, give ORS in addition to a breast milk substitute that is low in lactose, e.g. yoghurt or amasi or specialised formulae or lactose-free milk formula.

- **children aged 4 months and older:**
  Feeding should be restarted as soon as the child can eat, with small meals 6 times a day. Nasogastric feeding may be required in children who eat poorly. Where commercial special formulae as used in the step wise protocol are not available consider use of:

<table>
<thead>
<tr>
<th>Diet A: Starch-based, low lactose diet</th>
<th>Diet B: Lactose free diet with reduced starch.</th>
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<tbody>
<tr>
<td>• full-fat dried milk 11 g</td>
<td>• finely ground cooked chicken 12 g</td>
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<tr>
<td>OR</td>
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<tr>
<td>whole liquid milk 85 mL</td>
<td>whole egg 64 g</td>
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<td>AND</td>
<td>AND</td>
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<tr>
<td>• cooked rice 15 g</td>
<td>• cooked rice 3 g</td>
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<tr>
<td>• vegetable oil 3.5 g</td>
<td>• vegetable oil 4 g</td>
</tr>
<tr>
<td>• cane sugar 3 g</td>
<td>• glucose 3 g</td>
</tr>
<tr>
<td>• water to make 200 mL</td>
<td>• water to make 200 mL</td>
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<tr>
<td>Mix together in a liquidiser.</td>
<td>Mix together in a liquidiser.</td>
</tr>
</tbody>
</table>

100 g provides:
- energy: 83 kcal
- protein: 11% of calories
- lactose: 3.7g/kg/day

100 g provides:
- energy: 70 kcal
- protein: 11% of calories

Feed at 120 mL/kg/day

Feed at 150 mL/kg/day

If the response is good:
Give additional fruit and well-cooked vegetables to children who are responding well.

After 7 days of treatment with an effective diet, resume an appropriate diet for age, including milk, which provides at least 110 calories/kg/day.

Follow up regularly to ensure recover from diarrhoea, continued weight gain and adherence to feeding advice.
DRUG TREATMENT

CAUTION
The following agents are NOT recommended:
Antidiarrhoeals
Antibiotics for non-typhi Salmonella.

Antibiotic therapy
Antibiotics are only indicated when specific infections are suspected or where they are used in the STEP-WISE DRUG BASED EMPIRIC PROTOCOL FOR MANAGEMENT OF DIARRHOEA.
Antibiotics are not indicated for salmonella, except S. typhi
All persistent diarrhoea with blood in stool should be treated for Shigellosis: Section 2.2.6

For campylobacter
• erythromycin, oral, 10 mg/kg/dose 6 hourly for 7 days

For G. lamblia
• metronidazole, oral, 7.5 mg/kg/dose 8 hourly for 5–7 days

For Yersinia enterocolitica
• trimethoprim/sufamethoxazole, oral, 5 mg/kg/dose of trimethoprim component 6 hourly for 5 days

For Cryptosporidium
• no effective treatment available

STEP-WISE EMPIRIC PROTOCOL FOR MANAGEMENT OF DIARRHOEA
Commence management at the most appropriate step according to previous management – many infants with persistent diarrhoea will already have failed the “day 3–5” stage and will commence management on “day 6–8”.

DAY 0
Rehydration: Recomence breast or full-strength formula feeds within 12–24 hours.
Additional oral rehydration solution (ORS) to maintain hydration.

DAY 1-2
Continue full-strength feeds with additional ORS as required.

DAY 3-5
Change to lactose-free feeds.
Continue additional fluids as required.
If diarrhoea resolves, discharge, but continue with lactose-free feeds for 2 weeks.
DAY 6-8
• gentamicin, oral, 8 mg/kg/dose 4 hourly for 3 days only. Specialist initiated.
PLUS
• cholestyramine, oral, 1 g 6 hourly for 5 days only. Specialist initiated.
  Continue lactose-free feeds and additional fluids as needed.
  If diarrhoea resolves, discharge, but continue lactose-free feeds for 2 weeks.

DAY 9-11
Semi-elemental formula, sucrose- and lactose-free, protein hydrolysate,
medium chain triglyceride.
Continue additional fluids as required.
If diarrhoea resolves, discharge on semi-elemental feeds for at least 2 weeks.

If giardia is not excluded
• metronidazole, oral, 7.5 mg/kg/dose 8 hourly for 5 days

In HIV infected children: *Isospora belli* and *Cyclospora*
• trimethoprim/sulfamethoxazole, oral, 0.625 mL/kg/dose 12 hourly for 10 days

DAY 12-13
• gentamicin, oral, 8 mg/kg/dose 4 hourly for 3 days only. Specialist initiated.
PLUS
• cholestyramine, oral, 1 g 6 hourly for 5 days only. Specialist initiated.

DAY 14+
Commence total parenteral nutrition until diarrhoea has stopped. Thereafter gradually reintroduce semi-elemental feeds.

After success as indicated by weight gain, return of appetite and decrease of diarrhoea, less elemental diets can be judiciously and slowly reintroduced.

**Mineral and micronutrient deficiencies**
• zinc acetate, oral,
  < 10kg  10 mg/day
  > 10kg  20 mg/day
• magnesium, oral, 0.2 mmol/kg as a single daily dose
• folic acid, oral, 5mg as a single daily dose

If recurrent diarrhoea
• vitamin A, oral as a single dose
  6–12 months  100 000 IU
  > 12 months  200 000 IU
2.2.6 DYSENTERY
A03.9

DESCRIPTION
Passage of blood and mucus in the stools. Shigella infection is the most common serious cause in children in South Africa. Amoebic dysentery is less common.

Complications include:
- dehydration
- shock
- acidosis
- renal failure
- convulsions
- toxic megacolon
- rectal prolapse
- haemolytic uraemic syndrome

DIAGNOSTIC CRITERIA
Clinical
- sudden onset
- abdominal cramps, peritonism, urgency, fever and diarrhoea with blood and mucus in the stools
- meningismus and convulsions may occur
- exclude intussusception. Evidence of intussusception includes:
  - pain or abdominal tenderness
  - bile-stained vomitus
  - red currant jelly-like mucus
  - appearance of the intussusceptum through the anus

Investigations
- stool culture to confirm diagnosis of Shigellosis
- stool microscopy reveals many polymorphs and blood
- immediate microscopy of warm stool to diagnose amoebic dysentery

NON-DRUG TREATMENT
- monitor fluid and electrolyte balance
- ensure adequate nutrition and hydration

DRUG TREATMENT
Fluid and electrolyte replacement
See Acute Diarrhoea: Section 2.2.4
Antibiotic therapy
Treat as Shigella during an epidemic of Shigellosis, or if the child is febrile, “toxic”-looking, has seizures or if Shigella is cultured from the stool and the child is still ill.

- ciprofloxacin, oral, 15 mg/kg/dose 12 hourly for 3 days
  Consider the risk benefit ratio as highlighted in the package insert when using this medication in children.

  OR
  If hospitalised or if unable to take oral antimicrobial agents
  ceftriaxone, IV, 20–80 mg/kg as a single daily dose for 5 days

If amoebic dysentery, seen on stool microscopy
- metronidazole, oral, 15 mg/kg/dose 8 hourly for 7 days.
  In severe disease 10 days therapy is recommended.

REFERRAL
- dysentery with complications, e.g. persistent shock, haemolytic uraemic syndrome and toxic megacolon

2.2.7 GASTRO-OESOPHAGEAL REFLUX DISEASE (GORD)
K21

DESCRIPTION
Gastro-oesophageal reflux is repetitive regurgitation/reflux of gastric contents into the oesophagus.
It is termed “Uncomplicated GOR” if the only symptom is frequent small vomits, in which case no further investigation or treatment is needed.
It is termed “Complicated GOR” or “GORD” if associated with the diagnostic criteria below.

DIAGNOSTIC CRITERIA
Clinical
- recurrent vomiting or regurgitation and any of the following:
  - respiratory symptoms
  - recurrent wheeze or cough, chronic obstructive airway disease, recurrent aspiration, pneumonia, stridor, apnoea and apparent life-threatening event
  - failure to thrive
  - abnormal posturing or opisthotonus (Sandifer syndrome)

Investigations
- 24-hour oesophageal pH monitoring – the most accurate method of assessing significant reflux
- endoscopy to confirm oesophagitis
- barium swallow – easy and accessible, but not very sensitive
- isotope studies ‘milk scan’ – oesophageal and gastric scintiscanning
chapter 2

NON-DRUG TREATMENT
- postural treatment – lying on the left side is currently recommended
- dietary measures such as feed thickeners – if not breastfeeding, frequent small volume feeds and early introduction of solids

DRUG TREATMENT
Note:
Evidence in support of the following recommendations is weak:

- sodium alginate/antacid combination, oral, 1–2 g in 120–240 mL feed, mixed immediately before use

- omeprazole, oral. Specialist initiated.
  - neonate 0.5–1 mg/kg, 12–24 hourly
  - 1 month–2 years 2.5mg, 12 hourly
  - 2–6 years 5 mg, 12 hourly
  - 7–12 years 10 mg, 12 hourly

REFERRAL
- for diagnostic investigations if not available locally
- GORD not responding to treatment

2.2.8 INFLAMMATORY BOWEL DISEASES (IBD)

DESCRIPTION
Chronic incurable inflammatory diseases of the intestine that are of unknown aetiology.
IBD is sub-classified into:
- ulcerative colitis
- Crohn’s disease
- indeterminate colitis

DIAGNOSTIC CRITERIA
Clinical
- Ulcerative colitis
  - abdominal pain
  - chronic diarrhoea with blood in stools
  - urgency and tenesmus
  - fever
  - weight loss
  - arthritis/arthritis

- Crohn’s disease
  - postprandial pain
  - weight loss
  - abscess
  - uveitis/conjunctivitis
  - entero-enteric/enterocutaneous fistulae
Investigations
- blood tests show moderate anaemia, leucocytosis, raised ESR and decreased serum proteins
- in ulcerative colitis, colonoscopy with biopsy and in Crohn’s disease barium studies are helpful diagnostic procedures

NON-DRUG TREATMENT
- enteral nutrition to achieve optimal growth
- elemental or parenteral nutrition may be required in some patients under sub specialist supervision

REFERRAL
- all patients with suspected inflammatory bowel disease for assessment and initiation of therapy

2.3 HEPATIC DISORDERS

2.3.1 BLEEDING OESOPHAGEAL VARICES

NON-DRUG TREATMENT
- for secondary prophylaxis after a bleed, endoscopic injection sclerotherapy or variceal banding every 2 weeks until eradicated
  If either or both treatments fail then surgical over-sewing is done.
- for local control of acute bleeds that are not controlled with medicine treatment, Sengstaken tube is used

DRUG TREATMENT
- octreotide, IV bolus, 1–2 mcg then 1–5 mcg/kg/hour by infusion. Specialist initiated.

Post bleed prophylactic management
- omeprazole, oral. Specialist initiated.
  neonate: 0.5–1 mg/kg, 12–24 hourly
  1 month–2 years: 2.5mg, 12 hourly
  2–6 years: 5 mg, 12 hourly
  7–12 years: 10 mg, 12 hourly

AND
- propranolol oral, 2–8 mg/kg/24 hours in 3 divided doses
  Aim to reduce the pulse rate by 25%.
Previously bled but not actively bleeding:
Surgical oversewing if endoscopy and sclerotherapy or banding have failed.

- omeprazole, oral. Specialist initiated.
  
<table>
<thead>
<tr>
<th>Age</th>
<th>Dose</th>
</tr>
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<tbody>
<tr>
<td>neonate</td>
<td>0.5–1 mg/kg, 12–24 hourly</td>
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<tr>
<td>1 month–2 years</td>
<td>2.5mg, 12 hourly</td>
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<tr>
<td>2–6 years</td>
<td>5 mg, 12 hourly</td>
</tr>
<tr>
<td>7–12 years</td>
<td>10 mg, 12 hourly</td>
</tr>
</tbody>
</table>

AND
- propranolol oral, 2–8 mg/kg/24 hours in 3 divided doses
  Aim to reduce the pulse rate by 25%.

Never bled
Expectant management only.
No prophylaxis nor elective endoscopy/sclerotherapy.

REFERRAL
- all to establish diagnosis and initiate treatment
- bleeding varices - only after commencement of resuscitation and octreotide, if available

2.3.2 CIRRHOSIS

DESCRIPTION
The end result of irreversible damage to the liver tissue, causing a widespread, diffuse process of fibrosis with regenerating nodule formation. The fibrosis and abnormal portosystemic vascular connections that result cause ongoing damage. The progression rate is variable, but ultimately results in liver failure.

Causes are divided into biliary cirrhosis due to bile duct obstruction and post necrotic cirrhosis where the lesion is hepatocellular.

Complications include:
- fat malabsorption
- liver failure
- portal hypertension
- ascites secondary to hypoalbuminaemia or portal hypertension

DIAGNOSTIC CRITERIA
Clinical
- clubbing
- jaundice
- hepatomegaly and/or splenomegaly and/or ascites
- signs and symptoms of complications
Investigations
- liver enzymes may be normal
- FBC shows signs of hypersplenism with reduced circulating red cells, white cells and platelets
- prolonged prothrombin time
- hypoalbuminaemia
- ultrasound of the liver and spleen may be abnormal
- liver biopsy confirms cirrhosis

NON-DRUG TREATMENT
- ensure adequate nutrition
  - consult dietician, if available
  - overnight nasogastric feeding may be helpful
  - if not encephalopathic, high protein diet, i.e. 3 g/kg/day and medium chain triglyceride supplementation
  - high carbohydrate diet, supplement with glucose polymers
  - if serum cholesterol high or if xanthelasma, low cholesterol diet

DRUG TREATMENT
- multivitamin, oral, 5 mL as a single daily dose
  If prothrombin time is abnormal
- vitamin K₁, oral, 5 mg daily

2.3.2.1 Ascites due to Hypoalbuminaemia and/or Portal Hypertension

R18

NON-DRUG TREATMENT
- restrict sodium intake, 1–2 mmol/kg/24 hours
- if respiratory efforts are compromised by abdominal distension, careful removal of fluid by abdominal paracentesis may be necessary

DRUG TREATMENT
- albumin 20%, IV, 5 mL/kg over 4 hours
  PLUS
  If severe symptoms
  - furosemide, IV, 1 mg/kg
Once stabilised, continue with
- furosemide, oral, 1–3 mg/kg as a single daily dose
  OR
  hydrochlorothiazide, oral, 1 mg/kg/dose 12–24 hourly
  AND/OR
  - spironolactone, oral, 1–3 mg/kg as a single daily dose
  Continue for as long as needed to control ascites.
  Monitor serum potassium.
REFERRAL
- for determination of the underlying cause of the cirrhosis, portal hypertension and initiation of treatment
- cirrhosis, portal hypertension and/or liver failure not responding to adequate therapy
- hepatic encephalopathy

2.3.3 PORTAL HYPERTENSION
K76.6

DESCRIPTION
Increased portal venous pressure above vena cava pressure. Most commonly secondary to cirrhosis, but causes without cirrhosis may be divided into:
- prehepatic portal vein obstruction
- intrahepatic presinusoidal (e.g., bilharzia)
- intrahepatic postsinusoidal

DIAGNOSTIC CRITERIA
Clinical
- splenomegaly with recurrent ascites, variceal haemorrhage or hypersplenism

Investigations
- FBC shows hypersplenism
- Doppler assisted ultrasound and angiography may be diagnostic
- venacavagram may be diagnostic

NON-DRUG TREATMENT
- determine and manage underlying cause

2.3.4 HEPATITIS, VIRAL, ACUTE
B16.9
* Notifiable condition

DESCRIPTION
Acute inflammation of the liver with varying degrees of hepatocellular necrosis caused by hepatitis A, B and less commonly C, D and E viruses.

DIAGNOSTIC CRITERIA
Clinical
- prodromal phase
  - nausea
  - vomiting
  - fever
- jaundice, tender hepatomegaly and dark urine

Investigations
- raised transaminases and bilirubin
- serological evidence of hepatitis virus infection
NON-DRUG TREATMENT

- isolate patient
- low fat, high carbohydrate diet or any diet that the patient tolerates
- bed rest does not alter the course of the disease

DRUG TREATMENT

Prophylaxis

- hepatitis B vaccine, IM, 0.5 mL
  - < 1 year outer side of the right thigh
  - > 1 year upper arm
  Use opposite side to that for the DPT/DT injection.
  Give at 6, 10 and 14 weeks.

Neonatal transmission:
Babies born to mothers with acute hepatitis B infection at the time of delivery or to mothers who are HBsAg-positive or HBeAg-positive

- hepatitis B immunoglobulin, IM, 0.5 mL within 12 hours of delivery

PLUS

- hepatitis B vaccine, IM, first dose within 12 hours of delivery
Continue hepatitis B immunisation according to the recommended immunisation schedule.

REFERRAL

- acute hepatitis with bleeding tendency and altered consciousness – isolation recommended
- chronic hepatitis with/without cirrhosis

2.3.5 HEPATITIS, TOXIN INDUCED, ACUTE

K71.6

DESCRIPTION
Liver damage attributed to a toxin or drug. The most common herbal toxin in South Africa is atractyloside (Impila), which causes a Reye-like syndrome, with liver failure. Senecio ingestion is also still seen but this causes endothelial damage in hepatic veins, resulting in veno-occlusive disease with secondary cirrhosis and portal hypertension.

There are many medications that are hepatotoxic. In high doses the commonest are:

- anticonvulsants
- cytotoxics
- analgesics
- antiretroviral medication
- immunosuppressants
- anti-inflammatory medication
- antituberculous medication
chapter 2 alimentary tract

DIAGNOSTIC CRITERIA
• depends on the toxin, but the history is usually diagnostic
• Impila poisoning, given orally or rectally, results in anicteric hepatic encephalopathy
  Presents with onset of severe vomiting, followed by anuria then rapid depression of
  level of consciousness, progressing to seizures and/or coma within a day.

NON-DRUG TREATMENT
• stop medication and if medication was otherwise appropriate, review dosage
• education regarding herbal toxins, if appropriate

DRUG TREATMENT
For paracetamol poisoning
See Section 18.1.7

Hepatic encephalopathy
See Section 2.3.8

For seizures
• phenytoin, IV, 5–20 mg/kg infused over 30 minutes

REFERRAL
• all cases of hepatic encephalopathy due to toxin ingestion

2.3.6 HEPATITIS, CHRONIC, AUTOIMMUNE
K75.2

DESCRIPTION
Autoimmune induced hepatitis.

DIAGNOSTIC CRITERIA
Clinical
• jaundice
• hepatosplenomegaly
• cutaneous features of chronic liver disease
• extrahepatic manifestations of the autoimmune process

Investigations
• elevated bilirubin and transaminases
• hypoalbuminaemia and prolonged prothrombin time
• autoimmune marker screen
• protein electrophoresis shows increased gammaglobulin > 25 g/L
• diagnosis confirmed on liver biopsy
DRUG TREATMENT
• corticosteroids. Specialist initiated.
AND/OR
• azathioprine. Specialist initiated.

REFERRAL
• for confirmation of diagnosis and initiation of treatment

2.3.7 HEPATITIS B, CHRONIC
B18.1

DESCRIPTION
Persistently elevated transaminases after hepatitis B infection.

DIAGNOSTIC CRITERIA
• liver biopsy is characteristic
• transaminases are double upper limit of normal

REFERRAL
• for confirmation of diagnosis and initiation of treatment

2.3.8 LIVER FAILURE, ACUTE
K72.0

DESCRIPTION
Acute liver failure is a devastating clinical syndrome which has a high mortality. It results from massive necrosis of liver cells leading to the development of hepatic encephalopathy. The clinical appearance can be deceptive and it is easy to under-estimate how critically ill these patients are. Patients should be referred to secondary or tertiary hospitals early.

The following complications can occur:
• coagulopathy
• cerebral oedema
• encephalopathy
• metabolic acidosis
• hypoglycaemia
• renal failure
• cardiorespiratory failure
• sepsis

DIAGNOSTIC CRITERIA
Clinical
Appears deceptively well in the early stages. Progressive features include:
• malaise
• stupor
• encephalopathy
• bleeding tendency
• jaundice. The absence of jaundice suggests another process, such as Reye’s syndrome.
Investigations
- raised or low liver enzymes, low serum albumin, raised bilirubin, raised blood ammonia, hypoglycaemia
- prolonged prothrombin time
- low fibrinogen

NON-DRUG TREATMENT
- admit to high care or intensive care unit
- monitor:
  - blood pressure
  - urine output
  - heart rate
  - neurological state
  - respiration
  - gastrointestinal bleeding
  - haematocrit
  - blood glucose, 3 hourly if comatose
  - acid–base status
  - liver and renal functions
  - coagulation competence (INR)
  - electrolytes: sodium, potassium, calcium and phosphate
- maintain hydration
- with encephalopathy, aim to reduce ammonia production by the gut and optimise renal excretion
- withdraw protein completely initially followed by restricted intake if level of consciousness improves, i.e. 0.5–1 g/kg/24 hours
- stop medium chain triglyceride supplements but maintain an adequate energy intake
- stop sedatives, diuretics and hepatotoxic drugs, if possible

DRUG TREATMENT
To reduce intestinal protein absorption
- lactulose, oral, 1 g/kg/dose 4–8 hourly via nasogastric tube, then adjust dose to produce frequent soft stools daily
  - OR
  - polyethylene glycol solution with sodium sulphate and electrolytes, oral/via nasogastric tube, 10–25 mL/kg/hour over 6 hours. Follow with lactulose.
- gentamicin, oral, 12.5 mg/kg/dose 6 hourly for 5 days
  - The intravenous formulation can be given orally.

Cerebral Oedema:
See Section 13.4.

For pre-operative use or with active bleeding
- fresh frozen plasma, IV, 20 mL/kg over 2 hours
  - PLUS
  - cryoprecipitate
  - vitamin K, IV/oral, 2.5–10 mg daily
    - Never give IM.
    - Monitor response to vitamin K with INR and PTT

If platelet count < 10 x 10^9/L or if < 50 and with active bleeding
- platelet transfusion
For gastrointestinal bleeding:
• ranitidine, IV/oral 3–4 mg/kg/day 8 hourly

OR
omeprazole, oral. Specialist initiated.
neonate 1–2 mg/kg, 12–24 hourly
1 month–2 years 5 mg, 12 hourly
2–6 years 10 mg, 12 hourly
7–12 years 20 mg, 12 hourly

AND/OR
• sucralfate, oral, 250–500 mg 6 hourly

For hypoglycaemia
• dextrose 10%, IV bolus 2 mL/kg
  Administer maintenance as below.

For electrolyte imbalance, maintenance volumes of
• maintenance solution or Darrows half strength with dextrose 5%, IV, 60–80 mL/kg/day
  Ensure a minimum of 3–6 mmol/kg/day of potassium.
  Avoid diuretics.

For anaemia
• packed red cells, 10 mL/kg over 3 hours if haemoglobin < 7 g/dL

For Shock:
See Section 1.1.6

For sedation, if essential
• midazolam, IV, 0.1 mg/kg

Amelioration of liver injury, especially in idiopathic/toxin cases
• treat as for paracetamol poisoning, See Section 18.1.7

Antibiotic therapy
Where there is a sepsis tendency, prevent and treat aggressively with intravenous broad-spectrum antibiotics. Empiric antibiotic therapy until cultures are known.

• ampicillin, IV, 25 mg/kg/dose, 6 hourly

PLUS
• cefotaxime, IV, 25–50 mg/kg/dose, 6–8 hourly

• nystatin 100 000 units/mL, oral, 0.5 mL after each feed. Keep nystatin in contact with affected area for as long as possible.

REFERRAL
• for determination of the underlying cause and initiation of treatment
• hepatic encephalopathy
2.4 MALNUTRITION
E40–E43

2.4.1 MALNUTRITION, SEVERE
E40–E43

Admit all cases with severe malnutrition

DESCRIPTION
Severe Malnutrition: A multideficiency state of severe undernutrition of protein, energy and various other minerals, micronutrients and vitamins that includes the clinical entities of Kwashiorkor, Marasmus and Marasmic-Kwashiorkor. It is associated with a high but significantly modifiable mortality.

Kwashiorkor: usually below the 3rd percentile of weight for age, peripheral oedema, skin changes, fine pale sparse hair, potential high mortality.
Marasmus: under 60% expected weight for age or less than 3 standard deviations (< 70% expected weight for height), visible severe wasting, loss of muscle bulk and subcutaneous fat due to severe under-nutrition in children.
Marasmic-Kwashiorkor: children with features of both Kwashiorkor and Marasmus.

Danger Signs
Any of these indicate need for intensive management:
- dehydration
- shock
- lethargy
- weeping skin lesions
- hypothermia
- hypoglycaemia
- jaundice
- refusing feeds
- respiratory distress
- bleeding

Time frame for management of the child with severe malnutrition

<table>
<thead>
<tr>
<th></th>
<th>Stabilisation</th>
<th>Rehabilitation</th>
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<tbody>
<tr>
<td></td>
<td>Days 1–2</td>
<td>Days 3–7</td>
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<tr>
<td>Hypoglycaemia</td>
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<tr>
<td>Hypothermia</td>
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<tr>
<td>Dehydration</td>
<td></td>
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<tr>
<td>Electrolytes</td>
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<tr>
<td>Infection</td>
<td></td>
<td></td>
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<tr>
<td>Micronutrients</td>
<td>no iron</td>
<td>with iron</td>
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<tr>
<td>Initiate Feeding</td>
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<tr>
<td>Catch up growth</td>
<td></td>
<td></td>
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<tr>
<td>Sensory stimulation</td>
<td></td>
<td></td>
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<tr>
<td>Prepare for follow up</td>
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</tr>
</tbody>
</table>
NON-DRUG TREATMENT
Dehydration and severe diarrhoea – See Acute Diarrhoea: Section 2.2.4

Stabilisation phase

- feeding
  - immediate: stabilisation phase
    - begin feeding immediately – do not miss feeds
    - use “start up formula” 130 mL/kg/day divide into 3 hourly feeds, i.e. 8 times daily
    - “start up formula”:
      
      | Ingredient                          | Formula A | Formula B |
      |-------------------------------------|-----------|-----------|
      | whole dried milk                    | 25 g      | -         |
      | fresh cows milk                     | -         | 300 mL    |
      | sugar                               | 100 g     | 100 g     |
      | vegetable oil                       | 20 g      | 20 mL     |
      | trace element mix*                  | 20 mL     | 20 mL     |
      | Water to make up to:                | 1 000 mL  | 1 000 mL  |

100 mL contains:
  - energy: 75 kcal
  - protein: 0.9 g
  - sodium: 0.6 mmol

* Trace element mix
  - CuSO4 (0.5% solution) 10 mL 0.1 mg/mL
  - ZnSO4 18 g 36 mg/mL
  - MgSO4 140 g 280 mg/mL
  - Aqua chlorof conc 12.5 mL
  - Water to 500 mL

  - if danger signs, hypothermia or hypoglycaemia present, feed the same daily
  volume but divided into 2 hourly feeds, i.e. 12 times daily
  - if feeds refused/not finished feed via nasogastric tube

  rehabilitation phase
  - when appetite returns, usually within a week, change to “rebuilding formula” to increase the calories/ protein content in the feeds introduce a balanced soft mixed high-energy diet and add oil or margarine or peanut butter to meals. Prepare food without added salt.
  - for first two days replace the initial feeds with equal amounts of “rebuilding formula”, then gradually increase the volume by 10 mL per feed until some formula remains unfinished, usually ± 200 mL/kg/day
- "rebuilding formula":

<table>
<thead>
<tr>
<th></th>
<th>Formula C</th>
<th>Formula D</th>
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</thead>
<tbody>
<tr>
<td>whole dried milk</td>
<td>80 g</td>
<td>-</td>
</tr>
<tr>
<td>fresh cows milk</td>
<td>-</td>
<td>880 mL</td>
</tr>
<tr>
<td>sugar</td>
<td>50 g</td>
<td>75 g</td>
</tr>
<tr>
<td>vegetable oil</td>
<td>60 g</td>
<td>20 mL</td>
</tr>
<tr>
<td>trace element mix</td>
<td>20 mL</td>
<td>20 mL</td>
</tr>
<tr>
<td>Water to make up to</td>
<td>1 000 mL</td>
<td>1 000 mL</td>
</tr>
</tbody>
</table>

100 mL contains:
- energy: 100 kcal
- protein: 2.9 g
- sodium: 1.9 mmol

- detect and treat hypoglycaemia
  - test blood glucose level 3 hourly in severely ill child for 1st 24 hours and until stable
  - if blood glucose <3 mmol/L in asymptomatic child, give:
    - immediate feed of “start up formula”, or
    - dextrose, 10%, IV, bolus, or
    - sugar solution, oral, 5 mL/kg
  - monitor blood glucose and maintain above 3 mmol/L. Continue feeds.
  - if symptomatic or unresponsive hypoglycaemia, give dextrose 10%, IV, 5 mL/kg
    - Continue feeds.

These children have poor cardiac reserves and are easily volume overloaded
- do not maintain IV infusions unless absolutely necessary

- prevent and treat hypothermia
  - prevent hypothermia
    - use mother-child skin-skin contact, i.e. Kangaroo care, to keep child warm
    - keep child, especially the head, covered at all times especially at night. Protect the airway.
    - avoid drafts and change wet napkins regularly
    - avoid exposure e.g. bathing
    - care for child in a warm area, i.e. 25–30°C
    - feed immediately and 3 hourly as this provides energy to generate heat
  - treat hypothermia
    - check underarm temperature 3 hours post feed
    - axillary temperature < 36°C indicates urgent need to warm child
    - use mother-child skin-skin contact, i.e. Kangaroo care, to keep child warm and wrap both with blankets
    - if no mother, clothe and wrap child, including the head with warmed blanket
    - Protect the airway.
    - place heater nearby
• if severely hypothermic and not improving use other heating measures but do not apply direct heat to the skin as they may burn the child, e.g. hot water bottles
• check temperature 2 hourly until > 36.5°C using a low reading thermometer
• consider infection and sepsis (see below)
• exclude HIV and TB (consider empiric treatment)
• ensure immunisation, especially measles
• counsel parents or caregivers regarding regular and appropriate feeding
• before discharge, ensure parent/caregiver is able to access food for the child, referral to a primary health care nutritional support centre, and all financial supports and grants have been accessed

DRUG TREATMENT
Acute management
Treat all admissions as infected as signs of infection are usually absent.
• gentamicin, IV, 6 mg/kg once daily for 7 days
PLUS
• ampicillin, IV, 50 mg/kg/dose 6 hourly for 2 days
Follow with
• amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5 days

For gastrointestinal infection/infestation
• metronidazole, oral, 7.5 mg/kg/dose 8 hourly for 5–7 days

For dysentery
• cefotaxime, IV, 25–50 mg/kg/dose 6–8 hourly
OR
ceftriaxone, IV, 50–75 mg/kg once daily

Mineral and micronutrient deficiencies
Serum potassium does not indicate body potassium status.
Formulae may have the potassium and trace elements included in the feeds.
• potassium chloride solution, 25–50 mg/kg/dose, oral, three times daily until oedema subsides
  < 10 kg  250 mg
  > 10 kg  500 mg
• magnesium sulphate 50%, oral, 0.2 mL/kg as a once daily dose for a week
• vitamin A, oral, as a single dose
  < 6 months   50 000 IU
  6–12 months  100 000 IU
  > 12 months  200 000 IU

• folic acid, oral, 2.5 mg as a single daily dose

• multivitamin, oral, 5 mL as a single daily dose

If child does not improve clinically in 48 hours
• refer

Non-acute management
Iron supplementation is only given once gaining weight and oedema has resolved.
• iron, oral, 2 mg/kg elemental iron per dose 8 hourly with meals

For intestinal infestation
children under 2 years
• albendazole, oral, 200 mg as a single dose immediately

children 2–5 years
• mebendazole, oral, 100 mg twice daily for three days

children over 5 years
• mebendazole, oral, 500 mg as a single dose immediately

2.4.2 RICKETS
E55.0

DESCRIPTION
Failure to mineralise osteoid tissue in a growing child, usually due to deficiency of vitamin D, its active metabolites, calcium, phosphorus or other rare causes. This leads to bony deformity.

Occurs in ex-premature babies during infancy and in children with developmental disability, on anticonvulsants or not exposed to sunlight.

DIAGNOSTIC CRITERIA
Clinical
• bowing of long bones, widening of metaphyses, cranial bossing, and occasionally convulsions or tetany due to hypocalcaemia

Investigations
• elevated alkaline phosphatase
• serum calcium and/or phosphate abnormalities
• X-ray of wrists
NON-DRUG TREATMENT
- prevent vitamin D deficiency
- exposure to sunlight, at least 3 hours a week

Note:
Breast milk does not contain adequate vitamin D to prevent deficiency.
Ensure adequate sunlight exposure of infant or provide vitamin D until weaning.
- lactating mothers should be on a normal vitamin D containing diet

DRUG TREATMENT
Prophylaxis
For premature babies
- vitamin D, oral, 800 IU, once daily

Infants who are exclusively breast-fed or not on adequate volume of commercial milk formula
- vitamin D, oral, 400 IU, once daily

Treatment of active rickets
Treat only after confirmation of active rickets on X-ray.
- vitamin D, oral, 5 000 IU, once daily, in addition to milk in the diet
  Repeat X-ray after 6–8 weeks.
  If no radiological improvement, further investigation required.
  If healing occurs, continue for 3 months and confirm complete healing and adequate diet for the future.

Low birth weight babies
- phosphate, oral, 0.25 mmol every 12 hours
  Titrate against response.
  Aim to maintain the serum phosphate at 1.8–2.5 mmol/L

REFERRAL
- no radiological response to treatment after 6–8 weeks
- incomplete radiological response
- rickets secondary to other disease processes
### WHO/NCHS Normalised Reference Weight-for-Length (49-84 cm) and Weight-for-Height (85-110 cm), by Sex

<table>
<thead>
<tr>
<th>Boys’ weight (kg)</th>
<th>Girls’ weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-3 SD</td>
<td>-2 SD</td>
</tr>
<tr>
<td>70%</td>
<td>80%</td>
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<tr>
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<td>13.8</td>
<td>15.4</td>
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</tbody>
</table>

**Notes:**
1. SD=standard deviation score or Z-score; although the interpretation of a fixed percent-of-median value varies across age and height, and generally, the two scales cannot be compared, the approximate percent-of-the median values for –1 and –2 SD are 90% and 80% of median, respectively (Bulletin of the World Health Organization, 1994, 72:273-283).
2. Length is measured below 85 cm and above. Recumbent length is on average 0.5 cm greater than standing height, although the difference is of no importance to the individual child. A correction may be made by deducting 0.5 cm from all lengths above 84.9 cm if the standing height cannot be measured.
3.1 ANAEMIA, APLASTIC
D61.9

DESCRIPTION
Anaemia caused by bone marrow failure.
Fanconi anaemia has specific associated clinical features and chromosome abnormalities.

DIAGNOSTIC CRITERIA
Clinical
• pallor, petechiae, purpura, bleeding, with frequent or severe infections.

Investigations
• pancytopenia, with anaemia (may be macrocytic), leucopaenia and thrombocytopenia
• hypoplastic bone marrow on trephine biopsy

NON–DRUG TREATMENT
Blood products (washed/filtered packed red cells and/or single donor platelets) as needed.
Limit the use of blood and blood products as the patient may be sensitised for future bone marrow transplant.

DRUG TREATMENT
Any fever 37.5°C twice or 38°C once
Take blood cultures first.
Broad spectrum antibiotics
• ceftriaxone, IV, 50–75 mg/kg once daily

AND
• amikacin, IV, 15–20 mg/kg once daily

Fanconi anaemia
Androgens (specialist initiated)
• metenolone acetate, oral, 2–5 mg/kg daily as a single dose for at least 3 months

SURGICAL TREATMENT
Bone marrow transplant (specialised centres only).

REFERRAL
• all cases of suspected aplastic anaemia
Stabilise patient before transport (blood, platelets, if necessary, after consultation with an expert).
3.2 ANAEMIA, HAEMOLYTIC

DESCRIPTION
Anaemia caused by destruction of red blood cells. Destruction may be due to:
- abnormalities of the cell membrane (e.g. hereditary spherocytosis)
- enzyme abnormalities (e.g. G6PD deficiency)
- abnormal haemoglobin (e.g. sickle cell anaemia, thalassaemia)
- extracellular factors such as auto-immune antibodies or mechanical factors (e.g. Disseminated Intravascular Coagulation (DIC), hypersplenism, haemolytic uraemic syndrome).

DIAGNOSTIC CRITERIA
Clinical
- pallor, jaundice, fatigue
- spleen may be palpable

Investigations
- haemoglobin below normal for age
- evidence of haemolysis:
  - anaemia
  - reticulocytosis
  - decreased haptoglobin
  - unconjugated hyperbilirubinaemia
  - increased lactate dehydrogenase (LDH)
- Coomb’s test (direct antiglobin) is usually positive with autoimmune haemolysis
- renal function is abnormal in haemolytic uraemic syndrome

NON–DRUG TREATMENT
- do not transfuse prior to appropriate investigations, unless life-threatening
- Coomb’s-positive haemolytic anaemia may require expert blood cross-matching
- in G6PD deficiency, avoid medicines known to cause haemolysis e.g. aspirin, sulphonamides and primaquine

DRUG TREATMENT
Autoimmune haemolytic anaemia
Under specialist supervision:
- prednisone, oral, 2 mg/kg/24 hours until a satisfactory response is obtained and then taper to stop over 14 days

AND/OR
In patients not responding to steroids
- gamma globulin, IV, 400 mg/kg/24 hours for 5 days

AND/OR
To suppress the haemolytic process
- azathioprine, oral, 1–5 mg/kg/24 hours as a single daily dose, may be needed for a variable time
Sickle-cell disease
**Prophylaxis**
Pre-splenectomy
- pneumococcal vaccine (polysaccharide), IM, children > 2 years, 0.5 mL (single dose)
If not fully immunised
- Influenza vaccine

Post splenectomy
Give indefinitely
- benzathine benzylpenicillin (depot formulation), IM, 1.2 million units every 28 days
  **OR**
  - phenoxymethylpenicillin, oral,
    - <5 years 125 mg twice daily
    - >5 years 250 mg twice daily

**Chronic haemolytic anaemia**
Give all patients indefinitely
- folic acid, oral, 5 mg daily

**SURGICAL TREATMENT**
Not indicated for patients under 5 years.
- splenectomy for those, e.g. with spherocytosis, who are likely to respond

**REFERRAL**
- all cases with anaemia that is developing rapidly or is associated with evidence of haemolysis as above
- all cases to be managed in consultation with a paediatrician or paediatric haematologist

### 3.3 ANAEMIA, MEGALOBLASTIC

**DESCRIPTION**
Anaemia caused by a deficiency of folate and/or vitamin $B_{12}$.

**DIAGNOSTIC CRITERIA**

**Clinical**
- pallor and fatigue
- chronic diarrhoea

**Investigations**
- megaloblastic anaemia: elevated MCV (mean corpuscular volume) and MCH (mean corpuscular haemoglobin)
- macro-ovalocytes on blood smear, polysegmentation of neutrophils
- decreased serum vitamin $B_{12}$ or red blood cell folate
- investigations to identify reason for folate or $B_{12}$ deficiency, e.g. malabsorption
- pancytopenia in severe cases
- actively exclude leukaemia and aplastic anaemia which may cause macrocytosis
NON–DRUG TREATMENT

• dietary modifications to ensure adequate intake of folate and vitamin B\textsubscript{12}
• packed red blood cells may be needed if haemoglobin is very low and the patient is in cardiac failure. Try to avoid blood transfusion until all investigations have been done.
• educate caregiver on dietary requirements

DRUG TREATMENT

Folic acid deficiency

• folic acid, oral, 5 mg daily until haemoglobin returns to normal value for age.
  Prolonged treatment may be needed for malabsorption states and congenital deficiencies.

Vitamin B\textsubscript{12} deficiency

• vitamin B\textsubscript{12}, IM, 200–1000 mcg monthly until haemoglobin returns to normal value for age, thereafter 3–6 monthly

REFERRAL

• all case of megaloblastic anaemia, except clear nutritional folate deficiency

3.4 ANAEMIA, IRON DEFICIENCY

D50.9

DESCRIPTION

Anaemia due to iron deficiency is the most common cause of a haemoglobin below the age related norm. Common causes of iron deficiency are poor nutritional intake and blood loss due to parasites (whipworm and hookworm).

LOWER LIMITS OF NORMAL HAEMOGLOBIN

<table>
<thead>
<tr>
<th>Age</th>
<th>Haemoglobin (g/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>birth</td>
<td>13.5</td>
</tr>
<tr>
<td>6 weeks</td>
<td>9.5</td>
</tr>
<tr>
<td>3 months</td>
<td>10.0</td>
</tr>
<tr>
<td>6–12 months</td>
<td>10.5</td>
</tr>
<tr>
<td>12–18 months</td>
<td>10.5</td>
</tr>
<tr>
<td>18 months–4 years</td>
<td>11.0</td>
</tr>
<tr>
<td>4–7 years</td>
<td>11.0</td>
</tr>
<tr>
<td>7–12 years</td>
<td>11.5</td>
</tr>
<tr>
<td>12 years and older</td>
<td>12 (F) : 13 (M)</td>
</tr>
</tbody>
</table>
DIAGNOSTIC CRITERIA
Clinical
Symptoms and signs vary with the severity of the deficiency:
- pallor
- fatigue
- irritability
- behavioural and cognitive effects

Investigations
- haemoglobin below normal for age
- hypochromic microcytic anaemia
- low MCV (mean corpuscular volume) and MCH (mean corpuscular haemoglobin), raised red cell distribution width
- decreased serum iron, ferritin and transferrin saturation
- elevated total iron binding capacity
- stool examination to identify intestinal parasites or to confirm occult blood loss
- routine investigations are unnecessary if nutritional iron deficiency anaemia is strongly suspected and sinister features of other conditions are absent, e.g. splenomegaly, bleeding tendency. A response to trial of iron therapy should be documented to confirm the diagnosis.

NON–DRUG TREATMENT
- dietary adjustment
- counselling

DRUG TREATMENT
Treatment
- iron, oral, 2 mg/kg elemental iron per dose 8 hourly with meals

Elemental Iron per Preparation

<table>
<thead>
<tr>
<th>Weight kg</th>
<th>Elemental iron mg</th>
<th>Ferrous gluconate 40 mg/5mL</th>
<th>Ferrous sulphate</th>
</tr>
</thead>
<tbody>
<tr>
<td>3–6 kg</td>
<td>10 mg</td>
<td>85 mg (1.5 mL)</td>
<td>50 mg</td>
</tr>
<tr>
<td>6–10 kg</td>
<td>20 mg</td>
<td>170 mg (2.5 mL)</td>
<td>100 mg</td>
</tr>
<tr>
<td>10–18 kg</td>
<td>40 mg</td>
<td>340 mg (5 mL)</td>
<td>200 mg</td>
</tr>
<tr>
<td>18–25 kg</td>
<td>60 mg</td>
<td>513 mg (7.5 mL)</td>
<td>300 mg</td>
</tr>
<tr>
<td>25–50 kg</td>
<td>80 mg</td>
<td>680 mg (10 mL)</td>
<td>400 mg</td>
</tr>
</tbody>
</table>

Follow up at monthly intervals.
The expected response is an increase in Hb of 2 g/dL or more in 3 weeks.
Continue for 3–4 weeks after Hb is normal to replenish body iron stores.

Treat worms
- albendazole, oral, daily for three days
  - 1–2 years  200mg
  - > 2 years  400mg
CAUTION
Iron is extremely toxic in overdose, particularly in children
All medication should be stored out of reach of children

Prophylaxis
All premature babies, day 15 to 1 year
- elemental iron, oral, 2 mg/kg daily
- multivitamin, drops, oral, 0.3 mL daily, increase per age

Full term babies after 2 months
- elemental iron, oral, 1 mg/kg daily for one year
- multivitamin, drops, oral, 0.6 mL daily

REFERRAL
- where the underlying cause cannot be established
- patients not responding to adequate therapy and easily treatable causes for non-response are excluded, e.g.:
  - non-adherence to therapy
  - ongoing blood loss
  - ongoing infection

3.5 ANAEMIA OF MALNUTRITION, CHRONIC INFECTION OR DISEASE
D53.9

DESCRIPTION
Anaemia caused by malnutrition, chronic infection or disease. This may be due to interference with nutrient supply or suppression of haemopoiesis.

DIAGNOSTIC CRITERIA
Clinical
- pallor, fatigue
- features of malnutrition or chronic infection e.g. TB, HIV, or auto-immune disease may be present

Investigations
- haemoglobin low with normocytic, normochromic red cells
- ESR, PPD, chest X-ray

NON–DRUG TREATMENT
- emphasise a nutritionally balanced diet that is adequate in protein, vitamins and minerals for nutritional rehabilitation
- transfusion of packed red cells only in severely anaemic patients with infection

DRUG TREATMENT
- treat underlying infection e.g. TB
- defer iron treatment until infections are controlled
- provide extra iron (see above) and multivitamins
REFERENCES
- all cases with unresolving anaemia and no cause found

3.6 HAEMOPHILIA A AND B, VON WILLEBRAND’S DISEASE
D66/7

DESCRIPTION
Haemophilia A, haemophilia B and von Willebrand’s disease are chronic bleeding disorders caused, respectively, by a lack of clotting factor VIII, clotting factor IX and von Willebrand factor (carrier protein for factor VIII).

SUB CLASSIFICATION (FACTOR VIII AND IX DEFICIENCY):

<table>
<thead>
<tr>
<th>Class</th>
<th>Clotting factor</th>
<th>% of normal</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>VIII or IX</td>
<td>5–25%</td>
<td>Occasional bleeds</td>
</tr>
<tr>
<td>Moderate</td>
<td>VIII or IX</td>
<td>1–5%</td>
<td>Less frequent bleeds post trauma/dental extraction</td>
</tr>
<tr>
<td>Severe</td>
<td>VIII or IX</td>
<td>&lt;1%</td>
<td>Trauma/spontaneous bleeds</td>
</tr>
</tbody>
</table>

Complications:
- haemarthrosis with later chronic arthropathy
- intracranial haemorrhage
- soft tissue and muscle haematomas

DIAGNOSTIC CRITERIA

Clinical
- major bleeds:
  - CNS
  - gastrointestinal neck and throat
  - severe injury
  - advanced joint and soft tissue
  - forearm compartment
  - hip and ilio-psoas
- minor bleeds:
  - early joint bleed
  - muscle
  - soft tissue
  - epistaxis
  - mouth and gum
  - haematuria
- pain/tingling in the joints suggests bleeding into the joint in a known haemophiliac

Investigations
- prolonged partial thromboplastin time (PTT)
- factor VIII or factor IX concentration < 25% of normal activity
- prolonged bleeding time (Von Willebrand’s)

NON–DRUG TREATMENT
- haemophilia register
- alert bracelet
- dental care (see below for management of tooth extraction)
Acute bleeds into joints
- apply ice packs
- bed rest and rest of affected joint/limb until pain free and no further bleeding
- no weight bearing
- splint (no circumferential casts)

DRUG TREATMENT

CAUTION
- taking blood from internal jugular, posterior fontanelle and femoral veins is absolutely contra-indicated
- avoid IM injections
- avoid lumbar punctures
- exercise great caution when taking blood specimens
- when immunising press on injection site for at least 5 minutes after injection
- avoid aspirin and NSAIDS

For pain
Non-aspirin containing medicines.
- paracetamol, oral, 10 mg/kg 4–6 hourly as required
  OR
  paracetamol, oral, 10 mg/kg 4–6 hourly
  PLUS
  codeine phosphate syrup (25 mg/5 mL), oral, 0.5–1 mg/kg/dose 4 hourly as required

For bleeds
Emergency treatment while awaiting transfer, if indicated
If serious bleeding with known haemophilia, and no Factor VIII available
- fresh frozen plasma, IV, 10–20 mL/kg
  OR
  cryoprecipitate, IV, 20 units/kg

Factor VIII deficiency (with no inhibitor present)
Give 12 hourly until patient is pain free and has movement of joint/limb.
Minor bleeds
- factor VIII, IV, 15–25 units/kg
Major bleeds
- factor VIII, IV, 40 units/kg

Factor IX deficiency (with no inhibitor present)
Give daily until patient is pain free and has movement of the joint/limb.
Minor bleeds
- factor IX, IV, 15–20 units/kg
Major bleeds
- factor IX, IV, 40 units/kg
Haemophilia with inhibitors
Refer for assessment and planning with a haematologist.
• factor VIII inhibitor-bypassing activity (FEIBA) under haematologist supervision only

For dental extraction
Check that inhibitors are absent.
Admit for 3 days.

Haemophilia A
• factor VIII, IV, 40 units/kg, immediately before extraction

Haemophilia B
• factor IX, IV, 40 units/kg
AND
• tranexamic acid 40 mg/kg/day in 3 divided doses for 5 days

For mucous membrane bleeds
• tranexamic acid, IV/oral, 25 mg/kg/dose 6 hourly
  Contraindicated in haematuria, factor IX deficiency and with prothrombin complex concentrate.

Mild von Willebrand’s disease or established responders of mild factor VIII deficiency
• desmopressin, IV, 0.3 mcg/kg in at least 30 mL sodium chloride 0.9% over 30 minutes

REFERRAL
• all cases with suspected haemophilia (prolonged PTT and normal INR), for assessment, genetic counselling and planning of management to a haemophilia treatment centre

3.7 HAEMORRHAGIC DISEASE OF THE NEWBORN
P53
See Section 19.4

3.8 IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)
D69.3
DESCRIPTION
Common bleeding disorder of childhood due to iso-immune destruction of platelets.

Complications include severe haemorrhage and bleeding into vital organs.
CHAPTER 3  BLOOD AND BLOOD-FORMING ORGS

DIAGNOSTIC CRITERIA

Clinical
- sudden onset of bruising and bleeding, either spontaneously or after minor trauma, into the skin and mucous membranes and rarely into the organs in an otherwise well child.
- the lesions may range from pinpoint petechial bleedings to large ecchymoses, and are often increased on pressure points:
  - epistaxis is common
  - exclude child abuse.
- the presence of the following makes the diagnosis of ITP unlikely:
  - splenomegaly
  - masses
  - joint swelling
  - lymphadenopathy
  - rashes other than petechiae or ecchymoses

Investigations
- thrombocytopenia with normal white cell count and red cell series excluding the effects of blood loss
- normal INR (PT) and partial thromboplastin time (PTT)
- abundant megakaryocytes on bone marrow aspiration with normal erythroid and myeloid cellularity
- indications for bone marrow: Prior to starting steroids or any other abnormality on FBC or any atypical cells of differential count.

NON–DRUG TREATMENT
- avoid:
  - platelet transfusions unless life-threatening bleeds
  - contact sport, injury and trauma
  - dental procedures in acute phase
- reassurance that resolution usually occurs

DRUG TREATMENT
Avoid medication that affects platelet function, e.g. NSAIDs and aspirin.

Acute ITP
Platelets > 20 x 10⁹/L and no bleeding
- observe and follow up

Platelets < 20 x 10⁹/L, no bleeding
- prednisone, oral, 2 mg/kg/24 hours as a single daily dose for 2 weeks after bone marrow aspiration, and then taper to stop, regardless of the platelet count

Active bleeding
- prednisone, oral, 2 mg/kg/24 hours as a single daily dose for 2 weeks, after bone marrow aspiration and then taper to stop, regardless of the platelet count
  OR
  methylprednisolone, IV, 20 mg/kg/dose as single daily dose for 5 days

AND
- refer
Other indications for treatment
• non-elective surgical procedures
• associated coagulation defect

Chronic ITP
Intermittent treatment if platelets ≤ 10 x 10⁹/L and significant bleeding episodes
• prednisone, oral, 2 mg/kg/24 hours as a single daily dose for 2 weeks, after bone marrow aspiration and then taper to stop, regardless of the platelet count
  OR
  methylprednisolone, IV, 20 mg/kg/dose as single daily dose for 5 days
AND/OR
• gamma globulin, IV, 400 mg/kg/24 hours for 5 days

Acute life-threatening bleeds (e.g. intracranial bleeding): (acute or chronic ITP)
• platelet transfusions are only indicated prior to emergency splenectomy
• methylprednisolone, IV, 20 mg/kg/dose as a single daily dose for 5 days
AND
• refer

SURGICAL TREATMENT
• splenectomy should be considered in children 5 years or older in the presence of:
  ○ substantial limitation in activities as a result of the ITP
  ○ failure to recover after a period of 6–12 months
  ○ symptoms not controlled by medical management

Pre-splenectomy
• pneumococcal vaccine (polysaccharide), IM, children > 2 years, 0.5 mL (single dose)
AND
• Influenza vaccine

Post splenectomy
Give indefinitely until at least until 18 years
• benzathine benzylpenicillin (depot formulation), IM, 1.2 million units every 28 days
  OR
  phenoxymethylpenicillin, oral,
  < 5 years  125 mg twice daily
  > 5 years  250 mg twice daily

REFERRAL
• suspected ITP with unusual features such as splenomegaly or lymphadenopathy
• ITP complicated by severe haemorrhage, bleeding into vital organs or an intracranial haemorrhage
• ITP that fails to resolve in 6–12 months on adequate treatment (chronic ITP)
• if there is no local capacity to manage the condition
4.1 CARDIAC ARRHYTHMIAS

DESCRIPTION
A heart rate that is abnormally slow or fast for age or irregular.

Normal heart rate/minute for age:
- Newborn: 100–160
- < 1 year: 110–160
- 1–2 years: 100–150
- 2–5 years: 95–140
- 5–12 years: 80–120
- > 12 years: 60–100

DIAGNOSTIC CRITERIA

Clinical
- Presenting features may vary with the age of the patient:
  - Infants:
    - Colour changes (pale, mottled)
    - Irregular pulse
    - Irritability
    - Tachycardia
    - Feeding difficulties
    - Bradycardia
    - Sweating
    - Signs of cardiac failure
    - Tachypnoea/apnoeic spells
  - Children:
    - Dizziness
    - Tachycardia
    - Palpitations
    - Bradycardia
    - Fatigue
    - Syncope
    - Signs of cardiac failure

Investigations
- ECG is essential for diagnosis, preferably a 12 lead ECG
- Monitors are inadequate to diagnose most arrhythmias.

TACHYARRHYTHMIAS

Sinus tachycardia

![ECG Graph]

ECG Criteria
- Rate: > upper limit for age
- P wave: present and normal
- Rhythm: regular
- QRS: normal
Supraventricular Tachycardia

**ECG Criteria**
- **Rate:** usually > 200 beats per minute
- **Rhythm:** regular
- **P wave:** abnormal
- **QRS:** normal

Ventricular Tachycardia

**ECG Criteria**
- **Rate:** generally 100–220 beats per minute
- **Rhythm:** generally regular
- **P wave:** mostly not seen
- **QRS:** abnormal, width of QRS > 120 millisecond

BRADYARRHYTHMIAS
Common causes:
- drug ingestion
- congenital
- post operative
- excessive vagal stimulation

Sinus Bradycardia

**ECG Criteria**
- **Rate:** < lower limit for age
- **Rhythm:** regular
- **P wave:** present, all look the same
- **QRS:** normal, 80–120 millisecond
Heart Block (Complete)

ECG Criteria
Rate: low, usually < 60 beats per minute  
P wave: independent P waves and QRS’s with no relationship between the two (AV dissociation)
Rhythm: regular  
QRS: can be normal or wide, depending on escape rhythm

NON-DRUG TREATMENT
• sinus tachycardia usually requires management of the underlying condition
• ABC of resuscitation
• admit to high care or intensive care unit
• monitor:
  o ECG  
  o blood pressure  
  o heart rate  
  o respiratory rate  
  o oxygen saturation  
  o haemoglobin  
  o acid–base status  
  o blood gases
• maintain adequate nutrition and hydration
• treat pyrexia

DRUG TREATMENT

TACHYARRHYTHMIAS
Emergency treatment
Narrow Complex Tachycardia
Stable patient: Attempt vagal stimulation  
  Place icebag on face, or  
  Infants: immerse face in ice-cold water for a few seconds  
  Older children: try a valsalva manoeuvre, e.g. ask the patient to blow through a straw.  
  Eye-ball pressure and carotid massage is contraindicated in children.
• adenosine, IV, 0.1 mg/kg initially, increasing in increments of 0.05 mg/kg to 0.25 mg/kg.  
  Telephonic consultation with cardiologist/paediatrician.  
  Follow with a rapid flush of at least 5 mL sodium chloride 0.9%.  
  Because adenosine is rapidly metabolised, one needs to inject the adenosine in a good drip, followed with a rapid flush of a fluid bolus. It is sometimes helpful to have both the syringe with adenosine and the fluid bolus connected to the giving set and having as short as possible line between the syringes and the patient.
Unstable patient – heart failure / shocked
   DC synchronised cardioversion in increments of 0.5–1–2 J/kg
   If possible, empty the stomach before cardioversion is attempted. Resuscitation facilities must be available.
   Midazolam for sedation, if necessary.

Broad Complex Tachycardia
Causes include electrolyte disturbances and drug ingestion.
Stable patient (rare):
   Fax ECG to paediatric cardiologist.
   If unsure whether narrow or wide angle tachycardia, attempt adenosine as in narrow complex tachycardia.

Unstable patient – heart failure/shock
   Pulseless treat as ventricular fibrillation
   DC asynchronised cardioversion in increments of 0.5–1–2 J/kg
   Resuscitation facilities must be available.
   Midazolam for sedation, if necessary.

If DC cardioversion fails
   • amiodarone, IV, 5 mg/kg slowly over 20 minutes – NEVER as a rapid infusion

BRADYARRHYTHMIAS
Stable patient: observe

Unstable patient: Treat as impending arrest:
   • adrenaline, IV/IO, 10 mcg/kg
      Repeat if necessary conferring with referral institution.
      Try and correct underlying causes.

REFERRAL
   • all children with tachyarrhythmias after acute treatment, excluding sinus tachycardia due to other causes
   • bradycardia unresponsive to medical treatment, or heart block

4.2 CYANOTIC CONGENITAL HEART DISEASE WITH HYPOXAEMIC ATTACKS/ SPELLS (HYPERCYANOTIC SPELLS)
Q24.9

DESCRIPTION
Acute worsening of central cyanosis in patients with a confirmed or suspected underlying cyanotic congenital heart disease such as Tetralogy of Fallot.
DIAGNOSTIC CRITERIA
Clinical
• rapid worsening of central cyanosis, tachypnoea/dyspnoea, anxiety and alteration in consciousness in the presence of congenital cyanotic heart disease
• restless and crying in the presence of congenital cyanotic heart disease
• decrease in intensity or disappearance of the systolic murmur in Tetralogy of Fallot

NON-DRUG TREATMENT
• calm patient and keep on mother’s lap, if possible
• oxygen, 100%, by facemask or by nasal cannula
• place patient in knee-chest position to raise systemic blood pressure and increase systemic venous return
• monitor \( SaO_2 \), heart rate, respiratory rate and acid-base status
• ensure adequate hydration

DRUG TREATMENT
• sodium chloride 0.9% or Ringer-Lactate, 20 mL/kg bolus over 5 minutes

• morphine, IV, 0.1–0.2 mg/kg for 1 dose
  May cause impairment of airway reflexes and respiratory depression.

If clinically acidotic or pH < 7.2
• sodium bicarbonate 4.2%, IV, 2 mL/kg

If available:
• esmolol, IV
  Loading: 500 mcg/kg
  Maintenance: 50 mcg/kg/min.
  If inadequate response, increase as necessary by 50 mcg/kg every 1–4 minutes to a maximum of 300 mcg/kg/min.
  If no response, intubate and ventilate

After resolution of spell:
If Hb <10 g/dL, child is anaemic
• packed red cells, 10 mL/kg over 3 hours

• propranolol, oral, 0.5–1 mg/kg/dose 6 hourly. Increase to a maximum of 5 mg/kg/day as required

4.2.1 TETRALOGY OF FALLOT
Q21.3

DESCRIPTION
Most common cyanotic heart disease after infancy.
Chapter 4 Cardiovascular System

Diagnostic Criteria
Clinical
- child with central cyanosis
- may be plethoric due to polycythemia – normal haemoglobin represents relative anaemia
- possible history of cyanotic spells
- heart not clinically enlarged
- right ventricular hypertrophy usually not palpable
- single second heart sound
- coarse, ejection systolic murmur over right ventricular outflow tract
- chest X-ray
  - normal/small heart
  - boot shaped/pulmonary bay - concavity where pulmonary artery should be
  - oligaemic lung fields
- ECG
  - right axis deviation and right ventricular hypertrophy

Non-Drug Treatment
- good dental hygiene

Drug Treatment
- elemental iron, oral, 1 mg/kg/dose three times daily
- folic acid, oral, 2.5–5 mg/day
- propranolol, oral, 0.5–1 mg/kg/dose 6 hourly. Increase to a maximum of 5 mg/kg/day as required

Endocarditis prophylaxis: See Section 4.3

Referral
- all children with cyanotic heart defects

4.3 Endocarditis, Infective
I33.0

Description
Infection of the endothelial surface of the heart.
Suspect infective endocarditis in all children with persistent fever and underlying heart disease.

Diagnostic Criteria
Clinical
- an underlying heart defect and a persistent low grade fever without an obvious underlying cause
- associated other findings include: fatigue, joint pain, new murmurs, clubbing, splenomegaly and haematuria
- must be differentiated from acute carditis due to rheumatic fever
- the Duke criteria have been suggested as a guide to diagnosis, but have definite limitations as they were developed for use in adult patients
# Table 1: Major and Minor Clinical Criteria Used in the Modified Duke Criteria for Diagnosis of Infective Endocarditis (IE)

<table>
<thead>
<tr>
<th>Major Criteria</th>
<th>Minor Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>• positive blood culture</td>
<td>• predisposing heart condition or IV drug use</td>
</tr>
<tr>
<td>○ typical micro-organisms from two separate blood cultures:</td>
<td>• fever ≥ 38°C</td>
</tr>
<tr>
<td>S. viridans, including nutritional variant strains, S. bovis, HACEK group,</td>
<td>• vascular phenomena</td>
</tr>
<tr>
<td>S. aureus, or</td>
<td>○ major arterial emboli</td>
</tr>
<tr>
<td>▪ Enterococci, in the absence of a primary focus, or</td>
<td>○ septic pulmonary infarcts</td>
</tr>
<tr>
<td>▪ persistently positive blood culture with a micro-organism consistent</td>
<td>○ mycotic aneurysm</td>
</tr>
<tr>
<td>with IE from blood cultures drawn &gt; 12 hours apart, or</td>
<td>○ intracranial haemorrhage</td>
</tr>
<tr>
<td>▪ all 3 or a majority of 4 or more separate blood cultures, with the</td>
<td>○ conjunctival haemorrhages</td>
</tr>
<tr>
<td>first and last drawn at least one hour apart, or</td>
<td>○ Janeway lesions</td>
</tr>
<tr>
<td>▪ positive serology for Q fever</td>
<td></td>
</tr>
<tr>
<td>• evidence of endocardial involvement</td>
<td>• immunologic phenomena</td>
</tr>
<tr>
<td>○ positive echocardiogram for IE: oscillating intracardiac mass, on valve</td>
<td>○ Osler’s nodes</td>
</tr>
<tr>
<td>or supporting structures, or in the path of regurgitant jets, or on</td>
<td>○ Roth spots</td>
</tr>
<tr>
<td>implanted materials, in the absence of an alternative anatomic explanation,</td>
<td>○ glomerulonephritis</td>
</tr>
<tr>
<td>▪ abscess, or</td>
<td>○ rheumatoid factor</td>
</tr>
<tr>
<td>○ new partial dehiscence of prosthetic valve, or</td>
<td>• microbiologic evidence</td>
</tr>
<tr>
<td>○ new valvular regurgitation</td>
<td>○ positive blood culture but not meeting major criterion or</td>
</tr>
<tr>
<td></td>
<td>○ serologic evidence of active infection with organism consistent with IE</td>
</tr>
</tbody>
</table>
Table 2: Modified Duke Criteria for Diagnosis of Infective Endocarditis (IE)

<table>
<thead>
<tr>
<th>DEFINITE IE</th>
<th>POSSIBLE IE</th>
<th>REJECTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathological criteria</td>
<td>• at least one major and one minor criterion, or</td>
<td>• alternative diagnosis for manifestation of endocarditis, or</td>
</tr>
<tr>
<td>• micro-organisms</td>
<td>• 3 minor</td>
<td>• resolution of manifestations, with antibiotic therapy ≤ 4 days, or</td>
</tr>
<tr>
<td>o by culture or histology in a vegetation, or</td>
<td></td>
<td>• no pathologic evidence of IE at surgery or autopsy, after antibiotic therapy for ≤ 4 days</td>
</tr>
<tr>
<td>o in a vegetation that has embolised, or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o in a intracardiac abscess, or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lesions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• vegetation or intracardiac abscess present - confirmed by histology showing active IE</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical criteria - see Table 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 2 major criteria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 1 major and 3 minor or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• 5 minor</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Limitations of the Duke Criteria in Children
The clinical criteria rely heavily on relatively rare clinical features. In contrast, splenomegaly, seen in about 70% of children with infective endocarditis, clubbing and haematuria have not been included. Investigations like CRP or ESR, which may be of value, have not been included.

Investigations
• blood cultures
  o Sterile blood culture technique is essential. Take three blood cultures (venous) from different sites within 2 hours if very ill, otherwise over 24 hours. There is little benefit of doing more than five blood cultures. Child does not necessarily have a temperature as patients are mostly constantly bacteraemic.
• urine test strips - haematuria
• CRP/ESR may be helpful

NON-DRUG TREATMENT
• bed rest/limit physical activity
• ensure adequate nutrition
• maintain haemoglobin > 10 g/dL
• measures to reduce fever
DRUG TREATMENT
Heart failure: See Section 4.7

For pyrexia
- paracetamol, oral, 20 mg/kg at once, then 10–15 mg/kg/dose, 6 hourly as required

Antibiotic therapy
Antibiotics are always given IV, according to culture and sensitivity.
If culture is available treat according to sensitivities.

Empiric treatment
If culture is not yet available or is negative
- benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 6 hourly for 4 weeks
  PLUS
  - cloxacillin, IV, 12.5–25 mg/kg/dose 6 hourly for 4 weeks
  PLUS
  - gentamicin, IV, 1 mg/kg/dose 8 hourly for 4 weeks
  Daily gentamicin has not been proven to be equivalent to 8 hourly dosages in infective endocarditis.

If culture available

*S. viridans*
- benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 6 hourly for 4 weeks
  PLUS
  - gentamicin, IV, 1 mg/kg/dose 8 hourly for 2 weeks

*Enterococci*
- benzylpenicillin (Penicillin G), IV, 75 000 units/kg/dose, 6 hourly for 4–6 weeks
  PLUS
  - gentamicin, IV, 1 mg/kg/dose 8 hourly for 4–6 weeks

Cloxacillin sensitive staphylococcus
- cloxacillin, IV, 50 mg/kg/dose 6 hourly for 4–6 weeks

If the organism is gentamicin sensitive
ADD
- gentamicin, IV, 1 mg/kg/dose 8 hourly for 3–5 days

*Multi Resistant Staph Aureus* (MRSA)
- vancomycin, IV, 10 mg/kg/dose infused over 1 hour, 6 hourly for 6 weeks

HACEK organisms
- ceftriaxone, IV, 100 mg/kg once daily for 4 weeks
  OR
  - ampicillin, IV, 50 mg/kg/dose, 6 hourly for 4 weeks
  PLUS
  - gentamicin, IV, 1 mg/kg/dose 8 hourly for 4 weeks
Enteric bacilli, e.g. *Klebsiella*
- piperacillin, IV, 50 mg/kg/dose 6 hourly
  OR
  ceftazidime, IV, 50 mg/kg/dose 6 hourly

**PLUS**
- gentamicin, IV, 1 mg/kg/dose 8 hourly for 6 weeks

**Penicillin allergy**
- vancomycin, IV, 20 mg/kg/dose infused over 1 hour, 12 hourly

**PLUS**
- gentamicin, IV, 1 mg/kg/dose 8 hourly for 6 weeks

**Prophylaxis**
For children with an underlying cardiac lesion undergoing procedures that may induce bacteraemia.

**Dental, oral or upper respiratory tract procedures**
- amoxicillin, oral, 50 mg/kg (maximum 2 g) 1 hour before the procedure

Patients unable to take oral medication
- ampicillin, IV, 50 mg/kg (maximum 2 g) ½ hour before the procedure

**Penicillin allergy**
- clindamycin, oral, 20 mg/kg (maximum 300 mg) 1 hour before the procedure
  OR
  clindamycin IV, 20 mg/kg (maximum 300 mg), ½ hour before the procedure, then half the dose after 6 hours

**Genito-urinary or gastrointestinal procedures**
- ampicillin, IV, 50 mg/kg (maximum 2 g) ½ hour before the procedure
  OR
  amoxicillin, oral, 50 mg/kg 1.5 gm 1 hour before procedure

**PLUS**
- gentamicin, IV 1.5 mg/kg (maximum 120 mg) ½ hour before the procedure

**Penicillin allergy**
- vancomycin, slow IV, 20 mg/kg (maximum 1 g) 1 hour before the procedure
  Maximum rate of administration 500 mg over 30 minutes.

**PLUS**
- gentamicin, IV/IM, 1.5 mg/kg (maximum 120 mg) ½ hour before the procedure

**REFERRAL**
- all patients with suspected and confirmed infective endocarditis within a few days
4.4 RHEUMATIC FEVER, ACUTE

I01.9

* Notifiable condition.

DESCRIPTION
Rheumatic fever is a common cause of acquired heart disease with significant morbidity and mortality rates, both in the acute phase of the disease and as result of chronic valvular sequelae.

DIAGNOSTIC CRITERIA

- revised Jones criteria: Evidence of recent streptococcal infection:
  - elevated ASO-titre or other streptococcal antibody titres
  - positive throat culture for group A beta haemolytic streptococcus

  PLUS
  - two major manifestations plus supporting evidence of a recent streptococcal infection

  OR
  - one major and two minor manifestations plus supporting evidence of a recent streptococcal infection, justifies the presumptive diagnosis of acute rheumatic fever

<table>
<thead>
<tr>
<th>Major manifestations</th>
<th>Minor manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>• polyarthritis</td>
<td>• polyarthralgia</td>
</tr>
<tr>
<td>• carditis</td>
<td>• fever</td>
</tr>
<tr>
<td>• erythema marginatum</td>
<td>• acute phase reactants: increased erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP)</td>
</tr>
<tr>
<td>• subcutaneous nodules</td>
<td>• ECG: prolonged PR-interval, ≥ 0.18 seconds in the absence of carditis</td>
</tr>
<tr>
<td>• Sydenham’s chorea</td>
<td></td>
</tr>
</tbody>
</table>

- rheumatic fever can be diagnosed without supporting evidence of a recent streptococcal infection if Sydenham’s chorea is the only manifestation of rheumatic fever
- must differentiate acute rheumatic carditis with fever and heart involvement from infective endocarditis

NON-DRUG TREATMENT

- hospitalise with bed rest until sleeping pulse is normal and signs of rheumatic activity have resolved
- restrict physical activity for at least 2 weeks after the evidence of rheumatic activity has resolved
DRUG TREATMENT

Antibiotic therapy
To eradicate any streptococci
- benzathine benzylpenicillin (depot formulation), IM, as a single dose
  - < 30 kg \( \text{600 000 IU} \)
  - > 30 kg \( \text{1.2 MU} \)
  - OR
    - phenoxymethylpenicillin, oral, 250–500 mg 6 hourly for 10 days

Penicillin allergy
- erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 10 days

Anti-inflammatory therapy
Do not start until a definite diagnosis is made. The use of steroids remains controversial.

Severe arthritis
- aspirin soluble, oral, 75 mg/kg/24 hours in 4 divided doses for 2–6 weeks
  - OR
    - ibuprofen, oral, 5–10 mg/kg/dose, 6 hourly

Heart failure: See Section 4.7

Chorea
See Sydenham’s Chorea: Section 13.8

Prevention of repeated attacks
Any patient with documented rheumatic fever must receive prophylaxis up to 35 years age. Intramuscular penicillin is superior to other forms of prophylaxis.

- benzathine benzylpenicillin (depot formulation), IM, every 21–28 days (3–4 weeks)
  - < 30 kg \( \text{600 000 IU} \)
  - > 30 kg \( \text{1.2 MU} \)
  - OR
    - phenoxymethylpenicillin, oral, 250 mg twice daily

Penicillin allergy
- erythromycin, oral, 250 mg twice daily

REFERRAL
Rheumatic fever:
- with residual valvular damage electively for planning of care
- with symptomatic valvular damage
- unresponsive to treatment
4.5 MYOCARDITIS/DILATED CARDIOMYOPATHY
I40/I42.0

DESCRIPTION
Myocarditis is an acute inflammation of the cardiac muscle. Dilated cardiomyopathy refers to a group of conditions of diverse aetiology in which both ventricles are dilated with reduced contractility. It is difficult and sometimes impossible to distinguish myocarditis from dilated cardiomyopathy and these terms are sometimes used interchangeably.

DIAGNOSTIC CRITERIA
Clinical
• clinical signs of heart failure
• may present with cardiogenic shock

Investigations
• chest X-ray:
  ○ pulmonary congestion
  ○ cardiomegaly
  ○ there may be pleural effusion
• ECG:
  ○ mostly non-specific
  ○ arrhythmias or extra-systole may occur

NON-DRUG TREATMENT
• recognise and treat the underlying condition, e.g. infection, hypertension, cardiac tamponade, fluid overload.
• oxygen via face mask, nasal cannula or head box to prevent hypoxia
• fluid restriction (75% of daily requirements) – not at expense of adequate caloric intake
• ensure adequate nutrition, tube-feeding may be necessary

REFERRAL
• all

4.6 PERICARDITIS/PERICARDIAL EFFUSION
I31.9

DESCRIPTION
Inflammation of the pericardium.

DIAGNOSTIC CRITERIA
Clinical
• most patients present with a prolonged history and signs of pericardial tamponade:
  ○ low cardiac output
  ○ distended neck veins
  ○ muffled or diminished heart sounds
• patients with HIV may be asymptomatic and incidentally diagnosed when having a chest X-ray
• is often associated with TB
• acute septic pericarditis may occur in patients with septicaemia
Investigations

- **ECG:**
  - small complexes tachycardia
  - diffuse T wave changes
- **chest X-ray:**
  - in pericardial effusion – “water bottle” heart, or triangular heart with smoothed out borders.
  - must differentiate from a dilated heart due to cardiomyopathy
- **echocardiogram**
- **tuberculin skin test**
- **diagnostic pericardiocentesis**
  - in all patients with suspected bacterial or neoplastic pericarditis, and in all others in whom the diagnosis is not readily obtained
  - include cell count and differential, culture, gram stain
  - an elevated adenosine deaminase (ADA) may be helpful in diagnosing TB

**NON-DRUG TREATMENT**

- **pericardiocentesis**
  - preferably under ultrasound guidance
  - should preferably be performed by an experienced person
  - indicated in children with symptomatic pericardial effusion
  - may not be indicated in a child that has been unwell for a long time, and is not haemodynamically compromised
  - in an emergency, drainage by using a large bore intravenous cannula
  - technique:
    - ensure that full resuscitation equipment is available as well as an IV line and cardiac monitor
    - if the patient is restless, it may be necessary to sedate the patient. In an emergency situation, this is unnecessary.
    - position the patient in a 30° sitting-up position
    - prepare and drape a large area centered at the subxiphoid, if time permits. The preferred site for pericardiocentesis is just to the left of the xiphoid process, 1 cm inferior to the bottom rib.
    - infiltrate this area with 1% lidocaine
    - maintaining negative pressure on the syringe, insert the needle at a 45° angle to the skin, advancing in the direction of the patient’s left shoulder
    - observe closely for ventricular ectopics, a sign of myocardial contact, while advancing the needle. If this is noted, the needle should be withdrawn 1–2 cm
    - once air or fluid begins to fill the syringe, advance the intravenous cannula, withdraw the needle, attach the syringe to the hub of the cannula and slowly aspirate the pericardial fluid
    - potential complications include: haemopericardium (from laceration of the heart wall or coronary artery), cardiac arrhythmias, pneumothorax, and pneumopericardium
CHAPTER 4  
CARDIOVASCULAR SYSTEM

DRUG TREATMENT
If hypotensive, rapidly administer intravenous fluids.
Treat all pericardial disease as TB, give antituberculosis drugs for 6 months.

As soon as cultures are proved negative for organisms that can cause purulent pericarditis
• prednisone, oral, 2 mg/kg/day for 4 weeks

Antibiotic therapy
Empiric therapy should be provided until culture and sensitivity results available.
Antibiotic therapy should be continued for 3–4 weeks.

In case of purulent pericarditis
• cloxacillin, IV, 50 mg/kg/dose 6 hourly
PLUS
• ceftriaxone, IV, 100 mg/kg as a single daily dose

Heart Failure: See Section 4.7

REFERRAL
• all

4.7 HEART FAILURE
I50.9

DESCRIPTION
Clinical syndrome reflecting the inability of the myocardium to meet the oxygen and nutritional/metabolic requirements of the body.
Causes include:
• volume overload
  L-R shunt lesions
  mitral/aortic regurgitation
• pump failure
  myocarditis cardiomyopathy
• high output failure
  septicaemia
  severe anaemia

DIAGNOSTIC CRITERIA
Clinical
• acute cardiac failure may present with shock – refer to section on shock
• history of recent onset of:
  ○ poor feeding
  ○ tachypnoea
  ○ sweating
  ○ poor or excessive weight gain
  ○ breathlessness
  ○ cough
• physical findings:
  ○ tachycardia ○ cardiomegaly
  ○ hypotension ○ cool peripheries
  ○ weak pulses ○ reduced urinary output
  ○ gallop rhythm with/without a cardiac murmur
  ○ pulmonary venous congestion and fluid retention:
    ▪ tachypnoea
    ▪ dyspnoea
    ▪ orthopnoea
    ▪ recession
    ▪ wheezing
    ▪ coarse crepitations
    ▪ cyanosis
  ○ systemic venous congestion:
    ▪ hepatomegaly
    ▪ periorbital oedema - not seen in infants
    ▪ abnormal weight gain
  ○ signs and symptoms of underlying condition/disease

Investigations
• chest X-ray: cardiomegaly is almost always present
• electrocardiogram may show evidence of hypertrophy/enlargement of one or more heart chambers and/or dysrhythmias

NON-DRUG TREATMENT
• recognise and treat the underlying condition, e.g. infection, hypertension, cardiac tamponade, fluid overload
• oxygen via face mask, nasal cannula or head box to prevent hypoxia
• fluid restriction (75% of daily requirements) – not at expense of adequate caloric intake
• ensure adequate nutrition, tube-feeding may be necessary

DRUG TREATMENT
Combination drug therapy is usually indicated, i.e. start with diuretic, then add digoxin then add ACE inhibitor.

Diuretic therapy
Side effects:
  hypokalaemia
  hypochloraemic alkalosis – may increase digitalis toxicity

Monitor blood potassium levels.
Potassium supplements are necessary if furosemide is used without an aldosterone antagonist, i.e. spironolactone.

• furosemide, IV/oral, 1–3 mg/kg/24 hours in 2–3 divided doses
AND/OR
In refractory failure
• spironolactone, oral, 2–4 mg/kg/24 hours in 2 divided doses
Continue diuretic therapy as long as needed to control heart failure.
**Digoxin**  
Digoxin is contraindicated in bradycardia, heart block, cardiac tamponade or hypertrophic cardiomyopathy. Use with caution in myocarditis.  
Monitor digoxin blood levels and ECG.  
**Intravenous digoxin is dangerous and inappropriate.**  
Because of the potential confusion with digitalising dose of digoxin it is best to start with a maintenance dose.  
- digoxin, oral, 0.005 mg/kg/dose twice daily. (0.005 mg = 0.1 mL)  
  In older children a once daily dose can be given, i.e. 0.01 mg/kg/day.

**ACE inhibitor**  
For afterload reduction.  
Consider in persistent heart failure where other measures have failed, only after consultation with a paediatrician or paediatric cardiologist.  
Monitor blood potassium levels and consider stopping potassium supplements while patient is on an ACE inhibitor.  
- captopril, oral, 0.5 mg/kg/24 hours in 3 divided doses (8 hourly) for 24–48 hours - initial dose  
  Increase by 0.5 mg/kg/24 hours every 24–48 hours until maintenance dose of 3–5 mg/kg/24 hours is reached.  
  Continue as long as needed to control the cardiac failure.  
OR  
enalapril, 0.2–1 mg/kg/day as single or 2 divided doses

### 4.7.1 ACUTE SEVERE HEART FAILURE (ACUTE PULMONARY OEDEMA/ PULMONARY VENOUS CONGESTION)

**NON-DRUG TREATMENT**  
- treat the underlying disorder/condition. Where the primary cause of acute pulmonary oedema is renal failure treat as under renal failure.  
- restrict fluids – beware of IV fluids  
- upright or semi-upright sitting position  
- intubate and ventilate  
- administer 100% oxygen via face mask or nasal cannula

**DRUG TREATMENT**  
- furosemide, IV, 1–3 mg/kg immediately

For patients not responding to furosemide  
- morphine, IV, 0.1 mg/kg
**Inotropic support**

Inotropic support may help to stabilise patients with severe myocardial dysfunction, hypotension or low cardiac output. May be lifesaving in severe myocarditis or cardiogenic shock.

- **dobutamine**, IV infusion, 2–15 mcg/kg/minute
  Continue until myocardial function and blood pressure improve.

Once patient stable and maintaining blood pressure,

- **captopril**, oral, 0.5 mg/kg/24 hours in 3 divided doses (8 hourly) for 24–48 hours – initial dose
  Increase by 0.5 mg/kg/24 hours every 24–48 hours until maintenance dose of 3–5 mg/kg/24 hours is reached.
  Continue as long as needed to control the cardiac failure.

**REFERRAL**

- for determination of the underlying cause, where this is not known and initiation of treatment after stabilisation
- deterioration despite adequate treatment

### 4.8 DYSLIPIDAEMIA

**E78.9**

**DESCRIPTION**

Dyslipidaemia is a broad term used to describe disorders of fat metabolism. Hypercholesterolaemia associated with increased levels of apolipoprotein B100-containing lipoproteins (low density lipoprotein LDL, intermediate density lipoprotein IDL) and hypertriglyceridaemia (chylomicrons, very low density lipoproteins VLDL) have the most serious clinical implications. Hypercholesterolaemia promotes atherosclerosis and hypertriglyceridaemia is a major component of the metabolic syndrome.

Clinical features depend on the type of hyperlipidaemia.

- **Increased chylomicrons** are associated with eruptive xanthomas and hepatosplenomegaly.
- **Hypertriglyceridaemia** is associated with pancreatitis.
- **Increased levels of VLDL** are associated with familial hypercholesterolaemia (FH).
- **Heterozygous FH phenotype** lacks physical signs.
- **Homozygous phenotype** displays physical signs e.g. cutaneous xanthoma.
- **Increased LDL** is associated with glucose intolerance and hyperuricaemia.

Increased levels of HDL cholesterol protects against coronary heart disease.

**DIAGNOSTIC CRITERIA**

**Clinical**

- most hyperlipidaemia seen in clinical practice in children is secondary to chronic kidney/liver disease, diabetes mellitus, hypothyroidism and drug treatment e.g. calcineurin inhibitors (transplant patient) and protease inhibitors (ARV treatment)
- screening for hyperlipidaemia is indicated for children at risk of developing premature atherosclerosis, including:
CHAPTER 4 CARdiovascular SYSTEM

○ positive family history in parent/grandparent of any of the following conditions presenting <55 years of age:
  • familial hypercholesterolaemia
  • cardiovascular disease
  • metabolic syndrome
○ overweight or obese children

• a high-risk familial hypercholesterolaemia is perceived to be the concomitant occurrence of 3 or more risk factors:
  ○ LDL > 8 mmol/L (TChol > 10 mmol/L) or
  ○ HDL < 0.9 mmol/L and
  ○ positive family history of premature coronary heart disease (myocardial infarct in non-smoking parent < 35 years of age)
  ○ male gender
  ○ Lp(a) > 0.3 g/L
  ○ thick carotid intima (IMT)

Investigations
• to exclude secondary hyperlipidaemia: urine test strips, liver function tests, fasting blood glucose and thyroid function test
• in most cases non-fasting total cholesterol is determined in children at risk if level is higher than upper limit, lipid profile is done after 12 hours of fasting.
  ○ upper limit of S-cholesterol and triglycerides:
    total cholesterol
    4–6 years   4.5 mmol/L
    6–14 years  5.4 mmol/L
  ○ triglycerides (after 12 hours of fasting)
    • influenced by lifestyle – needs attention if > 2.5 mmol/L
    • pancreatitis risk if > 10 mmol/L

NON-DRUG TREATMENT
• schedule for integrated cardiovascular health promotion in children
  ○ obesity
    • See Section 7.15
  ○ blood pressure
    • with family history of hypertension < 55 years: routine BP measurement from 3 years once a year
    • if BP ≥ 95th percentile for sex, age, and height follow up and investigate if persistently elevated
  ○ diet
    • hypertriglyceridaemias need dietary intervention to restrict triglyceride
    • refer to dietician
    • learning a healthy eating behaviour is an important preventative measure
    • moderate salt intake
  ○ physical activity
    • advise prudent lifestyle choices including lifestyle and family activities
    • encourage active child-parent play.
    • limit child’s sedentary behaviour such as time watching TV and playing video computer games to maximum 2 hours per day or 14 hours per week
▪ children should not be allowed to eat while watching TV, i.e. “no grazing”
▪ daily moderate to vigorous activity for all school going children
▪ organised sport 3–4 times per week for 20–30 minute periods
  ○ smoking
    ▪ if household smoking, counsel to quit

**DRUG TREATMENT**
Drug therapy should only be considered after failure of non-drug treatment to lower the cholesterol.
Treatment with suitable statin for child with dyslipidaemia should be guided by decisions made in tertiary centre under supervision of a specialist.

Secondary hypercholesterolaemia
ACE inhibitor for persisting nephrotic range proteinuria

Chronic kidney disease (increased risk of cardiovascular disease)
▪ folic acid, oral, 5 mg/day for empiric treatment of increased plasma homocysteine
  PLUS
  ▪ pyridoxine, oral, 6.25 mg/day

**REFERRAL**
▪ children with familial hypercholesterolaemia or primary underlying metabolic disorder
▪ for initiation of therapy

### 4.9 HYPERTENSION IN CHILDREN

**DESCRIPTION**
Hypertension is defined as systolic and/or diastolic blood pressure ≥ the 95th percentile for gender, age and height percentile on at least three consecutive occasions. A sustained blood pressure of > 115/80 is abnormal in children between 6 weeks and 6 years of age.

In children it is easier to monitor the systolic blood pressure because of better correlation and less technical pitfalls than diastolic blood pressure.

In the majority of children hypertension is due to an identifiable cause. The likelihood of identifying a secondary cause is directly related to the level of BP and inversely related to the age of the child. Severe hypertension suggests renal disease.

**Hypertensive emergency/crisis** exists when CNS signs of hypertension appear such as encephalopathy, convulsions, retinal haemorrhages or blindness. Great care is required to reduce the blood pressure in a controlled manner to avoid potentially serious consequences of impaired auto-regulation of cerebral blood flow.

**Hypertensive urgency** is defined as a significant elevation of blood pressure without accompanying end organ damage. Patients are generally symptomatic with complaints of headache, blurred vision and nausea, despite the lack of end organ involvement.
The blood pressure level at which these changes may occur is not predictable and will vary between patients. It rather depends on the rate of developing the increase in blood pressure.

A valid assessment of the blood pressure is of extreme importance. The blood pressure is measured by standard auscultation technique in children older than 1 year.
In children less than 1 year old, a flush technique is usually used, although Doppler measurement would be preferable.
One should use the widest cuff that can be applied to the upper arm. The cuff bladder must encircle at least 80% of the upper arm and should cover at least 75% of the distance between the acromion and the olecranon. It is better to use a cuff that is slightly too large than one that is too small. Large cuffs, if covered with linen-like material, can be folded to the appropriate size in smaller infants as long as the bladder encompasses the arm.

**DIAGNOSTIC CRITERIA**

**Clinical**
- symptoms and signs of any of the following systems:
  - central nervous
  - cardiovascular
  - respiratory
  - urogenital system
- the most common associated features are:
  - oedema, haematuria, proteinuria
  - skin sores (impetigo)
  - convulsions, coma and visual symptoms
  - acute heart failure and pulmonary oedema
  - acute respiratory distress, cyanosis and apnoea
  - some children may be asymptomatic
- blood pressure in children correlates with body size and increases with age

<table>
<thead>
<tr>
<th>Age of child</th>
<th>95th Percentile of Systolic and Diastolic Blood Pressure</th>
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<tbody>
<tr>
<td></td>
<td>First 12 hours</td>
</tr>
<tr>
<td>newborn prem</td>
<td>65/45 mmHg</td>
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<tr>
<td>newborn fullterm</td>
<td>80/50 mmHg</td>
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<tr>
<td></td>
<td>Systolic mmHg</td>
</tr>
<tr>
<td>6 weeks–6 years</td>
<td>115</td>
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<td>8 years</td>
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<tr>
<td>12 years</td>
<td>135</td>
</tr>
<tr>
<td>14 years</td>
<td>140</td>
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</tbody>
</table>
95th Percentile Of Systolic And Diastolic BP Relation To Height Of Child (Ref 1)

<table>
<thead>
<tr>
<th>Height cm</th>
<th>Systolic mmHg</th>
<th>Diastolic mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
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<td>70</td>
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<tr>
<td>170</td>
<td>140 (133)</td>
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</tr>
<tr>
<td>180</td>
<td>145 (135)</td>
<td>83</td>
</tr>
</tbody>
</table>

Ref 1. Adapted from Andre et. al. Data from 17067 French children and adolescents. Boys and girls have been merged into one table, because in most instances data was similar except for systolic BP when height is ≥ 160 cm (girls 95th percentile given in brackets).

NON-DRUG TREATMENT

- there is a strong association with overweight and high blood pressure
  - The majority of these patients have mild hypertension and usually only need lifestyle modification.
- acute hypertension:
  - bed rest – Fowler’s position
  - control fluid intake and output (restriction)
  - restrict dietary sodium
  - manage end organ effects
- chronic hypertension
  - advise a change in lifestyle
  - institute and monitor a weight reduction programme for obese individuals
  - regular aerobic exercise is recommended in essential hypertension
  - dietary advice
    ▪ limit salt and saturated fat intake
    ▪ increase dietary fibre intake

4.9.1 HYPERTENSION, ACUTE SEVERE

For acute on chronic hypertension blood pressure needs to be lowered cautiously.
Medications for sustained control should be initiated as soon as possible so that the effect will be maintained when the emergency measures are discontinued.

Rate of BP reduction depends upon starting BP and age of the child.
In the absence of central nervous system signs, acute hypertension can be rapidly controlled over 24 hours. If in doubt about duration of hypertension, reduce BP slower over 48 hours.
Aim to reduce the systolic BP with not more than ⅓ of the interval between the patient’s systolic level from the presenting systolic blood pressure and the above the 95th percentile for that age or height in the first 8 hours, then further gradual decline over the next 24–48 hours. Do not decrease BP to < 95th percentile in first 24 hours.

**NON-DRUG TREATMENT**
- admit patient to paediatric intensive care unit, if possible
- monitor BP every 10 minutes until stable – thereafter every 30 minutes for 24 hours
- patient needs two peripheral intravenous drips

**DRUG TREATMENT**
Do not combine drugs of the same class.
- furosemide, IV, 1–2 mg/kg as a bolus slowly over 5 minutes
  - If oliguric, maximum dose: 5 mg/kg/dose.
  - Repeat appropriately for fluid overload.

**AND**
- labetolol, IV, 0.5–3 mg/kg/hour
  - 100 mg labetolol in 80 mL sodium chloride 0.45% = 1 mg/mL
  - Infuse with infusion pump.
  - Give bolus of 0.5 mg/kg and then titrate the dose slowly upwards until the desired blood pressure is achieved.
  - Repeat based on BP response.

**AND/OR**
- amlodipine, oral, 0.2 mg/kg/dose
  - May be repeated 6 hours later.
  - Thereafter every 12 hours.

**AND**
- prazosin, oral, 0.05–0.15 mg/kg/dose once daily

**4.9.2 HYPERTENSION, CHRONIC**

**DESCRIPTION**
**Primary/Essential hypertension**
Occurs most commonly in adolescents.
The patient is asymptomatic and well.
It is diagnosed by excluding underlying causes of hypertension.
Mild hypertension is confirmed by sustained hypertension on 3 follow-up occasions.

**Chronic secondary hypertension**
All children with incurable forms of persistent secondary hypertension require drug treatment over and above non-drug treatment.

**DIAGNOSTIC CRITERIA**
**Investigations**
- urine tests strips for protein, blood, leucocytes and nitrites
  - If latter two are positive, do urine MCS.
  - Positive urine findings indicate secondary hypertension and should be managed accordingly.
• blood urea, calcium, creatinine and electrolytes
• chest X-ray, ECG and abdominal sonar.
If all tests are negative, start lifestyle intervention.

NON-DRUG TREATMENT
• introduce physical activity, diet management and weight reduction, if obese
• advise against smoking in teenager
• follow up to monitor blood pressure and educate patient on hypertension
  o if blood pressure decreases, continue with non-drug management and follow up
  o if BP is increasing progressively, reinvestigate to exclude secondary causes or refer
  o if BP is stable but persistently > 95th percentile and secondary causes have been excluded, start drug treatment after failed non-drug management for 6 months
• consider earlier initiation of drug treatment if positive family history for cardiovascular disease, essential hypertension or diabetes mellitus

DRUG TREATMENT
Aim to achieve control BP over 48–72 hours in symptomatic patients.

For ambulatory patients start at the lowest dose of the preferred drug and increase the dose until control is achieved.

Once the highest recommended dose is reached or if the patient experiences side effects from the drug, a second drug from a different class should be added.
For patients with persistent hypertension despite the use of first line drugs, a second/third drug should be added. There is no specific order in which drugs should be added.

Specific classes of antihypertensive drugs should be used according to the underlying pathogenesis or illness.
For patients with predominantly fluid overload: use diuretics with/without ß-blocker.
All patients with hypertension and persistent proteinuria should be treated with an ACE inhibitor.
  For patients with predominantly renin induced hypertension, start with ACEI with/without diuretic. Always exclude bilateral renal artery stenosis before treating with an ACE inhibitor.
  Renal function must be monitored when an ACE inhibitor is prescribed because it may cause a decline in GFR resulting in deterioration of renal function and hyperkalaemia.

Patients with hypertension due to a neuro-secretory tumour (pheochromocytoma or neuroblastoma), should receive an a-blocker either as single drug or in combination with ß-adrenergic blocker.
**ACE inhibitor**
Side-effects include:
- hyperkalaemia and decreased GFR
  - check renal function and Se-K periodically
  - not used in bilateral renal artery stenosis
- captopril, oral, 0.1 mg/kg/dose 8 hourly – initial dose
  - Maximum 2 mg/kg/dose
  - OR
  - enalapril, oral, 0.04 mg/kg/dose 12 hourly
  - Maximum 0.3 mg/kg/dose up to 40 mg/day.

**β-blocker**
Contraindicated in severe heart failure and asthma.
- propranolol, oral, 0.25–1 mg/kg/dose 8–12 hourly
  - Maximum 2.5 mg/kg/dose.
  - OR
  - atenolol, oral, 0.5–1 mg/kg/dose once daily.
  - Maximum 2 mg/kg/day.

**Calcium channel blocker**
Can be used for control of acute hypertension
- amlodipine, oral, 0.1–0.2 mg/kg/dose once daily
Diuretic
- hydrochlorothiazide, oral, 0.5–1 mg/kg/dose once daily
  May cause hypokalaemia.
  OR
  furosemide, oral, 0.5–1.5 mg/kg/dose 12–24 hourly
  Maximum 6 mg/kg/day.
  May cause hypokalaemia.
  OR
  spironolactone, oral, 1–3 mg/kg/day 12–24 hourly
  May cause hyperkalaemia.

Vasodilator
Causes tachycardia and fluid retention.
- hydralazine, oral, 1–6 mg/kg/daily dose 8-12 hourly
  Maximum 200 mg/day.

α-blocker
Also indicated in patients with phaeochromocytoma-associated hypertension.
- prazosin, oral, 0.1–0.3 mg/kg/day 8–12 hourly
  Maximum 0.4 mg/kg/day.
  OR
  doxazosin, oral, 0.02–0.1 mg/kg/dose once daily

URGENT REFERRAL
- severe hypertension in for specific diagnosis and treatment

REFERRAL
- all children with acute and chronic hypertension for specific diagnosis, planning of treatment and long-term follow-up
- persistent cough on treatment with ACE inhibitor
Skin lesions are best characterised by their morphologic appearance which allows consideration of a suitable differential diagnosis.

5.1 BULLAE

5.1.1 EPIDERMOLYSIS BULLOSA

Q81.9

DESCRIPTION
Congenital, hereditary blistering skin lesions with onset in the newborn. Lesions do not have erythematous base. Loss of nails may occur.

NON-DRUG TREATMENT
- may require care in high or intensive care unit
- do not rupture bullae
- prevent infection by appropriate wound care
- attend to fluid and nutrition balance

REFERRAL
- all cases

5.1.2 STAPHYLOCOCCUS SCALDED SKIN SYNDROME

L00

DESCRIPTION
Blistering skin infection that appears as scalded skin.

NON-DRUG TREATMENT
- appropriate wound care

DRUG TREATMENT
- cloxacillin, IV, 50 mg/kg/dose 6 hourly for 5 days
  - neonates:
    - week 1–2  12 hourly
    - week 2–4  8 hourly
  - OR
  - flucloxacillin, oral, 12.5–25 mg/kg/dose 6 hourly for 7 days

REFERRAL
- recalcitrant cases
5.1.3 CHRONIC BULLOUS DISEASE OF CHILDHOOD
L12.2

DESCRIPTION
Tense blisters that lead to ulceration involving the groin, face and trunk.

DIAGNOSTIC CRITERIA
• skin biopsy with immunofluorescence

NON-DRUG TREATMENT
• appropriate wound care

REFERRAL
• all cases

5.2 ERYTHEMA AND DESQUAMATION

It is a continuum ranging from Erythema Multiforme (EM) to Stevens Johnson Syndrome and then to the potentially lethal Toxic Epidermal Necrolysis (TEN).

5.2.1 ERYTHEMA MULTIFORME/STEVENS-JOHNSON SYNDROME
L51

DESCRIPTION
Acute, vesico-bullous disorder with numerous manifestations on the skin, mucous membranes and, occasionally, internal organs caused mainly by:
• medicines, e.g. sulphonamides, phenytoin, phenobarbitone
• exposure to toxic substances
• infections, e.g. herpes simplex and mycoplasma.

Complications include:
• conjunctivitis
• corneal scarring
• infections
• oesophageal strictures
• uveitis
• fluid loss
• anaemia

DIAGNOSTIC CRITERIA
Iris or target lesions consisting of a dark centre, an inner pale ring and an erythematous outer border. In erythema multiforme lesions are pathognomonic.

Erythematous macules evolve into papules, vesicles, bullae, urticarial plaques or patches of confluent erythema. The centre of the lesion may be vesicular, purpuric or necrotic.

Erythema multiforme minor
Prodromal symptoms are generally absent. Symmetric crops of skin lesions of diverse morphology, primarily on the extensor surfaces of the arms and legs and often including soles and palms with relative sparing of the mucous membranes and the trunk.
Erythema multiforme major (Stevens-Johnson syndrome)
A serious, systemic condition involving the skin and at least two mucous membranes. Eruption may be preceded by non-specific prodromal symptoms like:
- malaise
- fever
- chills, or
- upper respiratory infection.
Cutaneous lesions tend to rupture, leaving the skin denuded, with fluid loss, anaemia and high risk of infection. Involvement of oral mucosa is common.

NON-DRUG TREATMENT
- may require care in high or intensive care unit
- examine daily for systemic involvement, infection and ocular lesions. If infection is suspected, send blood and skin lesion specimens for culture and sensitivity before initiating antibiotic therapy.
- do not puncture bullae or vesicles
- frequent mouth washes for oral lesions
- eye care
- maintain fluid balance, beware of shock
- nasogastric feeds if unable to eat, IV alimentation if enteral feeds are not possible
- cool compresses and wet dressings

DRUG TREATMENT
- paediatric maintenance with dextrose solution, IV

Antibiotic therapy
Reconsider choice of antibiotic when the results of cultures become available or the child does not improve.
Use IV antibiotics if the oral route cannot be used.
- erythromycin, IV/oral, 6.25–12.5 mg/kg/dose, 6 hourly for 10 days
AND
- promethazine, oral, 0.125 mg/kg, 6 hourly
  OR
  promethazine, oral, 0.5 mg/kg, as a single dose at night
  OR
  hydroxyzine, oral, 0.5 mg/kg/dose as a single dose at night

For pain
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as needed
  OR
  tilidine, oral, 1 mg/kg/dose
    1 drop = 2.5 mg
    Number of drops = body weight ÷ 2.5
    Not recommended for infants less than one year.
    Maximum: 10 drops/dose.
  OR
  Change of dressing protocol: See Section 20. 2.1
Note:
The use of systemic corticosteroids is not recommended.

- chlorhexidine 0.2%, 15 mL as a mouthwash
  Use as needed.
  Do not swallow.

REFERRAL
- erythema multiforme not responding to adequate therapy or with ocular involvement

5.3 MACULES AND PAPULES

5.3.1 DRUG REACTIONS
L27.0

Commonly associated with:
- sulphur containing agents
- penicillin
- carbamazepine
- NSAIDs
- TB drugs
- non-nucleoside reverse transcriptase inhibitors

A variety of rashes occur, ranging from (worst) erythema multiformae with mucosal involvement, target lesions, blistering and fever, through itchy or painful urticarial eruptions, measles-like maculopapular rashes, to erythema and flat, symmetrical macular lesions (fixed drug reactions) These are commonly flat or slightly raised lesions of < 0.5 cm in size.

Lesions recur upon re-exposure to the offending agent.

NON-DRUG TREATMENT
- stop causative agents

DRUG TREATMENT

Antihistamines:
- promethazine, oral, 0.125 mg/kg, 6 hourly
  OR
  hydroxyzine, oral, 2 mg/kg/dose, 6–8 hourly

Corticosteroids:
- prednisone, oral, 1–2 mg/kg/day, for 5–7 days

REFERRAL
- systemic involvement with organ dysfunction

5.3.2 ACNE
L70

DESCRIPTION
An inflammatory condition of hair follicles leading to comedone formation that can lead to scarring and post inflammation hyper pigmentation.
DIAGNOSTIC CRITERIA
• black or white heads – comedones

NON-DRUG TREATMENT
• avoid greasy and oily topical products

DRUG TREATMENT
• benzoyl peroxide 5 %, topical, applied to affected areas as needed
AND
• doxycycline, oral, 2.5 mg/kg/dose, 12 hourly

If ineffective
Topical retinoids, e.g. tretinoin gel/cream, must be introduced gradually at night to limit skin irritation.
Apply sunscreen to avoid sun irritation.
Oral contraceptives are useful in young females with premenstrual flare.

REFERRAL
• recalcitrant and/or fulminant acne
• psychologically disturbed or depressed patient

5.3.3 CELLULITIS
L03.9

DESCRIPTION
Infection of the skin and subcutaneous tissue usually caused by streptococci, H influenzae or staphylococci.

Erysipelas
The affected area is:
• well demarcated with firm borders
• very tender and warm
• bright red and swollen

Erysipelas must be distinguished from necrotising fasciitis where there is infection and inflammation usually by a gas-forming organism that spreads rapidly along the fascial tissue.
Complications may lead to septicaemia.

DIAGNOSTIC CRITERIA
• acutely ill, child with fever and malaise
• involved area is swollen, indurated, erythematous and painful/tender with regional lymphadenopathy

NON-DRUG TREATMENT
• ensure adequate nutrition and hydration
• elevate the affected limb to reduce swelling
DRUG TREATMENT
Choice of intravenous or oral antibiotics depends on the severity of the condition.

Severe disease
For streptococci or haemophilus
• benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 6 hourly for 5 days
AND
For staphylococci
• cloxacillin, IV, 50 mg/kg/dose 6 hourly for 5 days

For peri-orbital cellulitis
• ceftriaxone, IV, 50 mg/kg, 12 hourly
AND
For staphylococci
• cloxacillin, IV, 50 mg/kg/dose 6 hourly for 5 days

For pain
• ibuprofen, oral, 5–10 mg/kg/dose, 6 hourly for 72 hours
  < 30 kg, maximum: 500 mg/day
OR
• tilidine, oral, 1 mg/kg/dose, 6–8 hourly for 48–72 hours
  Maximum: 10 drops/dose
  1 drop = 2.5 mg
  Number of drops = body weight ÷ 2.5
  Not recommended for infants less than one year.

Non-severe disease
For streptococci or haemophilus
• amoxicillin, oral, 30 mg/kg/dose, 8 hourly for 5 days

For other organisms
• flucloxacillin, oral, for 6 hourly 7 days
  < 2 years  62.5 mg
  2–10 years  125 mg
  >10 years    250 mg

For pain
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

Penicillin allergy
• erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 5 days

URGENT REFERRAL
• necrotising fasciitis

REFERRAL
• poor response to therapy
• recurrent cellulitis
Exclude eczema, immunocompromised state, diabetes and underlying osteomyelitis.
5.3.4 ECZEMA

DESCRIPTION
An inflammatory itchy skin condition characterised by:
• vesicles, weeping and crusting in the acute stage
• scaling and lichenification during the chronic stage.
May be allergic or non-allergic.

DIAGNOSTIC CRITERIA
• family history of allergies
• reaction after exposure to allergens
• typical distribution: face, flexure of knees and elbows, creases of neck

NON-DRUG TREATMENT
• avoidance measures: use neutral soaps and rinse clothes properly after wash
• cut nails short and don't scratch
• wrap with dressings soaked in sodium chloride 0.9%
• avoid sunlight and use sunscreen

DRUG TREATMENT
• relieve skin dryness with emulsifying ointments, e.g. aqueous cream
• hydrocortisone 1%, topical, applied to face
• betamethasone 0.1%, topical, diluted in buffered cream in 1:10, applied to body
  OR
  betamethasone 0.1%, topical, applied once daily to body for 3, 5 or 7 days until eczema
  has cleared
  Moisturise with emulsifying ointments during therapy and for remaining weeks.

Secondary bacterial infection
• erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 14 days
  • povidone iodine cream, apply twice daily

For pruritis
• promethazine, oral, 0.125 mg/kg, 6 hourly
  OR
  promethazine, oral, 0.5 mg/kg, as a single dose at night

Note:
Short term use of topical steroids is recommended.
Oral corticosteroids do not have a role in the management of this condition.

REFERRAL
• recalcitrant cases
5.3.5 FUNGAL INFECTIONS

5.3.5.1 Candidiasis

DESCRIPTION
Skin infection involving face, neck and perineum. Commonly occurs in immunocompromised individuals. Involvement of mouth and perineal regions suggest systemic disease.

DIAGNOSTIC CRITERIA
Clinical
- scaly lesions with an erythematous base and satellite pustular/vesicular lesions with clear edges
- mucosal involvement

Investigations
- wet preparation with potassium hydroxide or biopsy and culture

NON-DRUG TREATMENT
- control underlying immunosuppressive state e.g. diabetes, HIV
- personal hygiene of mothers prior to breast-feeding

DRUG TREATMENT
- imidazole cream 2%, e.g. clotrimazole, topical, applied three times daily for 14 days
  OR
  nystatin 100 000 IU/g, topical, applied three times daily for 14 days

If no response
- fluconazole, oral, 3–6 mg/kg/day

Note:
Deep and systemic fungal infections should be referred to tertiary centres providing dermatological services, e.g.:
- mycetomas
- sporotrichosis
- blastomycosis
- cryptococcus
- histoplasmosis

REFERRAL
- recalcitrant infection

5.3.6 PSORIASIS

DESCRIPTION
An inflammatory condition of the skin and joints.
DIAGNOSTIC CRITERIA
- scaly, red itchy papules and plaques over scalp, perineum, nails and skin folds and extensor surface
- occasional pustules are seen

NON-DRUG TREATMENT
- avoid precipitants e.g. drugs

DRUG TREATMENT
Local plaques
To remove scales
- salicylic acid 2% and coal tar in white soft paraffin three times a day
  OR
dithranol 0.1–1.0% in soft paraffin (dermatologist only).

Severe pustular psoriasis
- hydrocortisone 1%, topical, applied 1–2 times daily
  OR
prednisone, oral, 1–2 mg/kg as a single daily dose for 7 days

Severe psoriasis
Refer to dermatologist for use of:
- calcipotriol
- acitretin
- UVB
- hydroxycarbamide
- PUVA
- methotrexate or azathioprine

REFERRAL
- recalcitrant cases

5.3.7 URTICARIA
L50.9

DESCRIPTION
An itchy inflammatory skin and mucosal condition recognised by wheal and flare reaction that may be acute or chronic. Often due to irritants, insect bites or allergens. Secondary infective features include excoriation, vesicles and pigmentary changes. Chronic papular eruptive urticaria is often seen in HIV infected individuals.

DIAGNOSTIC CRITERIA
- history of allergen exposure
- wheal and flare reaction (“hives”)
- positive skin test if due to allergy

NON-DRUG TREATMENT
- limit exposure to precipitants, e.g. drugs, allergens and toxins
- limit exposure to insects by using topical insect repellent which contains more than 10% diethyltoluamide (DET)
- wrap with dressings soaked in sodium chloride 0.9%
DRUG TREATMENT
- promethazine, oral, 0.125 mg/kg, 6 hourly
  OR
  promethazine, oral, 0.5 mg/kg, as a single dose at night
  OR
  hydroxyzine, oral, 0.5mg/kg as a single dose at night

- hydrocortisone 1%, topical, applied twice daily as required.
  Useful when applied immediately after insect bite.

Persistent disease
- tar-steroid combination (Modified Adamson’s ointment), applied at night

Severe chronic urticaria
- prednisone, oral, 1–2 mg/kg as a single daily dose

REFERRAL
- recalcitrant and chronic cases

5.4. PURPURA
D69.9

5.4.1 MENINGOCOCCAEMIA
A39.2

DESCRIPTION
Palpable bleeding into skin caused by *N. meningitides* and is associated with rapid spread. This is a medical emergency and can be fatal.

See also Sepsis: Section 8.25

NON-DRUG TREATMENT
- monitor blood pressure and capillary filling time
- ensure adequate hydration

DRUG TREATMENT
- benzylpenicillin (Penicillin G), IV, 100 000 units/kg/dose, immediately, then 4 hourly
  AND
  hydrocortisone, IV, 4–6mg/kg/dose, immediately, then 4–6 hourly

REFERRAL
- associated septic shock
5.5. VESICLES AND PUSTULES

5.5.1 INFECTIONS
R23.8/L08.9

5.5.1.1 Herpes group: Varicella, herpes zoster and simplex

DESCRIPTION
Itchy, umbilicated vesicles that occur in:
• crops on the trunk (varicella), or
• painful vesicles in a linear distribution (herpes zoster), or
• a group of vesicles that coalesce to form an ulcer with an erythematous base on the lips or mouth (simplex).
Often secondarily infected with bacteria.

DIAGNOSTIC CRITERIA
• Tzanck smear – multinucleated giant cells are seen

NON-DRUG TREATMENT
• condition is infectious – avoid spread
• avoid rubbing when eye involved

DRUG TREATMENT
Varicella
• calamine lotion, applied on the skin

For immunosuppressed and newborns
• aciclovir, IV, 10–20 mg/kg/dose 8 hourly administered over 1 hour for 7–14 days

If there is evidence of good clinical response, change to:
• aciclovir, oral, 10–20 mg/kg/dose 4–8 hourly

Prophylaxis for close contacts
• varicella-zoster immunoglobulin, IM, 0.5 mL, single dose immediately

Herpes zoster
• aciclovir, IV, 5–10 mg/kg/dose 8 hourly for 7–14 days
  OR
• aciclovir, oral, 10–20 mg/kg/dose 4–8 hourly for 7 days

For pain
• carbamazepine, oral, 5 mg/kg/dose, 8 hourly
Herpes simplex
- aciclovir, oral, 10–20 mg/kg/dose 4–6 hourly for 7 days
- aciclovir 5%, topical, applied 4 hourly
- chlorhexidine 0.2 %, 15 mL as a mouthwash
  Use as needed.
  Do not swallow.

Secondary bacterial infection
- erythromycin, oral, 6.25–12.5 mg/kg/dose 6 hourly for 5 days
CHAPTER 6
GENITO-URINARY SYSTEM

6.1 NEPHROLOGICAL/UROLOGICAL DISORDERS

6.1.1 POST STREPTOCOCCAL GLOMERULONEPHRITIS
N05.9

DESCRIPTION
Acute post-streptococcal glomerulonephritis is an immune mediated inflammatory condition caused by the deposition of immune complexes in the glomerular basement membrane and/or mesangium of the glomeruli.

DIAGNOSTIC CRITERIA
Clinical features
- predominantly occurs in children 3–12 years old
- manifests 1–3 weeks after preceding pharyngitis or impetigo
- characteristic features include:
  - facial or generalised oedema
  - painless macroscopic haematuria (smoky or tea coloured urine)
  - oliguria, and
  - hypertension

Special investigations to confirm APSGN

<table>
<thead>
<tr>
<th>Urine analysis</th>
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<tbody>
<tr>
<td><strong>Macroscopic appearance</strong></td>
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<tr>
<td><strong>Urine test strips</strong></td>
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<tr>
<td><strong>Microscopic examination</strong></td>
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<table>
<thead>
<tr>
<th>Blood investigations</th>
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</thead>
<tbody>
<tr>
<td><strong>Streptococcus serology</strong></td>
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<tr>
<td>ASO or Anti-DNAseB titre</td>
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<td><strong>Complement study</strong></td>
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<td>(C_4)</td>
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<tr>
<td><strong>S-biochemistry</strong></td>
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<tr>
<td>Serum Electrolytes</td>
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<tr>
<td>S-Urea &amp; creatinine</td>
</tr>
<tr>
<td>Full blood count</td>
</tr>
</tbody>
</table>
NON-DRUG TREATMENT

- bed rest is necessary with:
  - severe hypertension
  - left heart failure
  - pulmonary oedema, or
  - central nervous system symptoms.

- monitor fluid balance:
  - no fluids while pulmonary oedema is present
  - restrict fluid intake to 300–400 mL/m²/24 hours (25 mL/kg/24 hours) while oliguric, i.e. urine flow < 1 mL/kg/hour or fluid overloaded
  - fluid should only be given orally or via nasogastric tube
  - only if anuric and enteral feeds are impossible, give IV fluids, i.e. 5–10% dextrose water, with a volumetric controller

- weigh daily and record intake and output strictly. In small children fluid balance is best monitored with regular weighing.

- dietary measures. Restrict:
  - potassium intake until result of serum electrolytes are available. Bread and jam is relatively safe.
  - sodium while oedema and/or hypertension is present
  - protein to 0.8 g/kg/day if urea exceeds 20 mmol/L

DRUG TREATMENT

Eradication of streptococci

- phenoxymethylpenicillin, oral, 12.5 mg/kg/dose 6 hourly for 10 days
  OR
  - benzathine benzylpenicillin (depot formulation), IM, 600 000–1.2 million units, two doses given 5 days apart

Penicillin allergy

- erythromycin, oral, 10 mg/kg/dose, 6 hourly for 10 days

Hypertension

Hypertension usually develops acutely and is mostly related to fluid overload.

Hypertensive crisis: Patient with signs of hypertensive encephalopathy, i.e.:
- convulsions
- retinal haemorrhages
- blindness

Hypertensive urgency: Symptomatic patient with significant elevation of blood pressure with complaints of headache, blurred vision and nausea but lacks the above clinical manifestations.

Initiate treatment for Acute Hypertension: See Section 4.9
CAUTION

Great care is required to reduce the blood pressure in a controlled manner to avoid potentially serious consequences of impaired auto-regulation of cerebral blood flow. Do not lower blood pressure precipitously – rather titrate small doses against response.

Medicines for sustained control should be initiated as soon as possible so that the effect will be maintained when the emergency measures are discontinued. Rate of BP reduction depends upon starting BP and age of the child.

For management of acute hypertensive emergency – Post streptococcal glomerulonephritis

- furosemide, IV, 1–2 mg/kg
  - If oliguric
  - furosemide, IV, 5 mg/kg/dose
    - IV bolus must be administered slowly over 5 minutes due to risk of ototoxicity.

  **AND**
  - amlodipine, oral, 0.2 mg/kg/dose. May be repeated 6 hours later, thereafter every 12 hours
  - **OR**
    - atenolol, oral, 1 mg/kg/dose. If no improvement, repeat after 6 hours. Once improved, give once daily at the appropriate dosage.

If no hypertensive crisis but persistent significant hypertension

- atenolol, oral, 12 mg/kg/24 hours as single dose preferably at night
  - **OR**
    - propranolol, oral, 0.5–4 mg/kg/dose, 12 hourly
      - Maximum dose: 8 mg/kg/24 hours
  - **OR**
    - hydralazine, oral, 0.25–1.25 mg/kg/dose, 6 hourly

**Volume overloaded** - hypertensive, orthopnoea and raised JVP

- restrict sodium chloride intake
- restrict fluid intake equal to 50% of urine output plus insensible loss, i.e. 400 mL/m²/day
- if pulmonary oedema - do not give fluids

For anuric patient with acute volume overload and unresponsive to furosemide – refer urgently.

- furosemide, slow IV, 1–2 mg/kg/dose. Repeat after 30 minutes, if needed and 4–6 hourly if required.
  - Maximum dose: 5 mg/kg/dose.
  - Do not give an IV infusion after administering furosemide.
  - Refer.
  - Place patient in Fowler’s position and give oxygen via nasal prongs.
- morphine, IV, 0.1 mg/kg. Repeat after 4 hours if required.
**REFERRAL**

**Urgent**

- anuric patient with acute volume overload and unresponsive to furosemide
- uncontrolled hypertension
- progressive or severe renal failure
- cardiac failure or pulmonary oedema not responding to treatment

**For specialist advice**

- macroscopic haematuria persisting for more than 4 weeks or persistent proteinuria
- family history of renal disease
- streptococcal aetiology unproven (ASOT and anti-dnase B negative, normal C$_3$ levels, decreased C$_4$ levels)
- decreased complement levels not normalised within 6 weeks

---

**6.1.2 URINARY TRACT INFECTION**

**N39.0**

**DESCRIPTION**

Bacterial infection of the urinary tract.

**Simple urinary tract infection** – infection is limited to the lower urinary tract and there are no associated urological anomalies.

**Complicated urinary tract infection** – infection of the urinary tract involving the renal parenchyma or which is associated with underlying urological anomalies.

**DIAGNOSTIC CRITERIA**

**Clinical**

Signs and symptoms are related to the age of the child and are often non-specific. Uncomplicated urinary tract infections may cause very few signs and symptoms. Complicated infections may present with a wide range of signs and symptoms.

**Neonates may present with:**

- fever
- hypothermia
- poor feeding
- sepsis
- vomiting
- prolonged jaundice
- failure to thrive
- renal failure

**Infants and children may present with:**

- failure to thrive
- persisting fever
- abdominal pain
- frequency
- dysuria
- enuresis or urgency

In any child with fever of unknown origin, the urine must be examined.
Special investigations
If a bag specimen reveals the following, a urine specimen must be collected aseptically for culture and sensitivity:

- positive leukocytes or nitrites on dipsticks
- motile bacilli and increased leukocytes or leukocyte casts on urine microscopy

Urine specimen is collected aseptically

- by suprapubic aspiration or transurethral bladder catheterisation in acutely ill children less than 2 years of age or in smaller children who are unable to co-operate
- by mid-stream clean catch method in older children

Criteria for the diagnosis of UTI

- any culture from a suprapubic urine sample
- a culture of $> 10^4$ col/mL urine of a single organism from a catheter specimen
- a pure culture of $> 10^5$ col/mL in a mid-stream clean catch sample or consistent culture of a pure growth even with counts as low as $10^4$ col/mL.

NON-DRUG TREATMENT

- exclude complications of urinary tract infection
- ensure adequate nutrition and hydration. Maintain hydration with oral and/or IV fluids if necessary.
- for recurring infections:
  - avoid irritant soaps and bubble baths
  - prevent constipation
  - treat pinworm
  - perineal hygiene
  - regular complete emptying of the bladder and/or double voiding, i.e. making an additional attempt at voiding after the initial flow of urine has ceased.

DRUG TREATMENT

Antibiotic therapy

All acutely ill babies must be treated parenterally for the first few days until clinically well and able to tolerate feeds. Children > 3 months old, who are unwell but not acutely ill and who are not vomiting may be treated with oral antibiotics.

Duration of oral antibiotic therapy is for a minimum of 7–10 days.

The choice of antibiotics used depends on the expected culture and sensitivity of the organism.

Review antibiotic choice once culture and sensitivity results become available.

- cefuroxime, IV, 25 mg/kg/dose 8 hourly for 7 days
  OR
  amoxicillin/clavulanic acid, IV, 25 mg/kg/dose 8 hourly

If there is evidence of good clinical response, change to:

- amoxicillin/clavulanic acid, oral, 30 mg/kg/dose of amoxicillin component 8 hourly
  OR
  cefuroxime, oral, 15 mg/kg/dose 12 hourly for 7 days
Prophylactic antibiotic therapy for UTI

Indications:
• infants and young children from 2 months–2 years until the imaging studies are completed
• recurrent infections
• structural and/or functional abnormality of the urinary tract

For continent children
• cephalexin, 10 mg/kg/dose as a single dose at night

For children not yet continent
• cephalexin 5 mg/kg/dose, 12 hourly
  OR
  nitrofurantoin, oral, 12 mg/kg at night
  Contra-indicated in children with renal impairment.
  Do not use for no longer than 4 weeks continuously.

For pain
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required
  Avoid NSAIDs.

URGENT REFERRAL
• if obstruction is suspected refer for consideration of an MCUG

REFERRAL
• all children with proven urinary tract infections for renal and bladder ultrasound assessment
• boys with recurrent urinary tract infections to exclude obstructive causes (posterior urethral valves)
• poor response to adequate therapy
• complications such as renal failure

6.1.3 NEPHROTIC SYNDROME

DESCRIPTION
Nephrotic syndrome is a clinical syndrome associated with massive proteinuria due to increased permeability of the glomerular basement membrane.
In children it is mostly idiopathic, e.g.:
• minimal change nephrotic syndrome (MCNS)
• focal segmental glomerular sclerosis (FSGS).

Features of nephrotic syndrome are:
• massive proteinuria of > 40 mg/m²/hour or a protein to creatinine ratio on a random urine sample of > 0.2 g/mmol
• hypo-albuminaemia < 25 g/L
• oedema
• hyperlipidaemia (hypercholesterolaemia)
• haematuria or hypertension may be present, but is not diagnostic criteria
DIAGNOSTIC CRITERIA
Special investigations
- urine test strips: 3–4+++ proteinuria with or without trace to 1+ haematuria
- urine microscopy: hyaline and lipid casts. May have occasional red and white blood cells.
- urine protein: creatinine ratio: > 0.2 g/mmol
- serum albumin: < 25 g/L
- S-urea and creatinine and electrolytes usually normal
- S-complement usually normal
- S-cholesterol: increased
- exclude infections e.g. Streptococcal antibody, Hepatitis B antigen carrier, syphilis, HIV and CMV
- exclude connective tissue disorder, e.g. SLE

A presumptive diagnosis of MCNS can be made in children:
- in whom secondary causes have been excluded
- between 2–6 years of age
- with:
  - normal blood pressure
  - normal renal function
  - or only a trace of haematuria, but no red cell casts
  - normal complement levels
  - no evidence of chronic infection or connective tissue disease

NON-DRUG TREATMENT
- assess hydration status
  Normovolaemic – normal moist mucosa and normal blood pressure with well perfused limbs
  - restrict salt intake
  - no fluid restriction
  - weigh daily (1 kg = 1 L of fluid)
  Hypovolaemic – often preceded by diarrhoea, vomiting, dry mucosa, hypotensive, cyanosed, cold extremities
  - check urine Na, K, creatinine and osmolarity
  - give fluid bolus: sodium chloride 0.9%, IV, 20 mL/kg, immediately over 10 minutes
  - replace fluid loss as for dehydrated child e.g. oral rehydration for gut losses, etc.
  - monitor urine output strictly and weigh regularly

Continued weight gain or anuria is an indication for referral.

- dietary measures
  - restrict sodium. No salt should be added to food and salt preserved foods are restricted.
  - normal energy intake
  - adequate protein diet with normal serum creatinine
  - in patients with raised creatinine and non remitting nephrotic syndrome protein intake needs to be restricted to 0.8 g/kg/day plus equivalent of protein lost in urine per day
DRUG TREATMENT

Symptomatic treatment of oedema
Loop diuretics should not be prescribed routinely.

For patients with severe oedema with a low albumin
- albumin, human 20% (salt free), IV, 1 g/kg administered over 2–4 hours

AND
Follow with or simultaneously
- furosemide, IV, 2 mg/kg, slow IV infusion over 5 hours
  i.e. 0.4 mg/kg/hour

Mild to moderate oedema
- spironolactone, oral, 1.5–2.5 mg/kg/dose, 12 hourly

WITH/WITHOUT
- hydrochlorothiazide, oral, 1 mg/kg, once daily. Do not exceed 25 mg daily

Non-remitting nephrotic syndrome
For thrombotic complications
- aspirin, soluble, oral, 1–2 mg/kg, once daily

Supplementation of multivitamins and minerals in non-remitting nephrotic syndrome
- multivitamin, oral, 5 mL daily
  inclusive of pyridoxine, other water soluble vitamins of B group and vitamin C 30 mg
  and vitamin D 400 IU
- folic acid, oral, 5 mg daily
- calcium, oral, 10–15 mg/kg/dose, twice daily
  Maximum dose: 1 000 mg daily.

All children with non-remitting nephrotic syndrome should receive renoprotective treatment as for patients with chronic renal failure

ACE inhibitor
To decrease proteinuria, irrespective of presence or absence of systemic hypertension. Monitor renal function and potassium especially in children with impaired renal function or volume depletion.

Adverse effects of ACE inhibitor:
- hyperkalaemia (higher risk when potassium sparing diuretic is used simultaneously)
- acute renal failure in volume depleted patients.

Begin with low dosage of ACE inhibitor and titrate against response and blood pressure.

- enalapril 0.1 mg/kg once daily
  Dose may be increased to 0.5 mg/kg/day, as a single dose or two divided doses.
  OR
  captopril, oral, 0.5–2.5 mg/kg/dose, twice daily
  OR
  perindopril 0.05-0.15 mg/kg once daily
**Immunisation**
All routine vaccinations should be given.
Once in remission
- pneumococcal vaccine (23 strain), IM, 0.5 mL in children > 2 years
- varicella zoster vaccine, SC, 0.5 mL. Repeat once after 4 weeks.

**Note:**
Live virus vaccine should not be given while the patient is receiving steroids or other immunosuppressive treatment.

**Antibiotics**
During periods of severe oedema
- phenoxy methylpenicillin, oral, 125–250 mg, 12 hourly

**Corticosteroids**
Corticosteroid treatment should only be initiated in consultation with a paediatric nephrologist or paediatrician.
Steroids are indicated in children with histologically confirmed MCNS or in those in whom this diagnosis is highly probable.
The response to corticosteroid treatment is an indication of the underlying histology and may give some information regarding the long-term prognosis. A rapid response to steroid treatment is usually indicative of MCNS.

Urine should be tested every morning and should remain protein free before decreasing the dose. If proteinuria recurs, go back one step in the suggested dose.
If the first course is tapered too rapidly, the child tends to develop more frequent relapses. Relapses occur in up to 85% of all children with MCNS.

If there is no response to steroid treatment after 6 weeks, the patient is steroid resistant and should be referred.

Start with high dose
- prednisone, oral, 2 mg/kg/dose as a single dose in the morning
  Maximum dose: 80 mg daily.
  Once the urine test strips is negative for proteinuria on 3 consecutive days, give the same dose every alternative day and then taper dose slowly over the next 4 months.

<table>
<thead>
<tr>
<th>Dose (mg/kg) alternative days</th>
<th>Period of treatment (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>4–6</td>
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<tr>
<td>1.5</td>
<td>4</td>
</tr>
<tr>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>0.5</td>
<td>4</td>
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</table>

Additional steroids or steroid supplementation is necessary during periods of acute stress, e.g. surgery or septic shock.
Under subspecialist supervision or advice, cyclophosphamide and pulse steroid therapy may be considered.
All other immunosuppressive medications should only be used once a histological diagnosis has been made.

**REFERRAL**
- where a presumptive diagnosis of MCNS cannot be made
  The patient should be referred for renal biopsy to make a definite diagnosis and to plan treatment.

### 6.1.4 RENAL FAILURE, ACUTE

**DESCRIPTION**
Acute renal failure is a syndrome characterised by a rapid decline in glomerular filtration rate and retention of nitrogenous waste products. It is important to differentiate prerenal, renal and postrenal failure.

**DIAGNOSTIC CRITERIA**

**Clinical features**
- in neonates exclude congenital abnormality of the urinary tract
- oliguria is the most common manifestation, i.e. :
  - neonates  output < 1 mL/kg/hour
  - older children  output ≤ 0.3 mL/kg/hour
- prerenal – shock and dehydration
- postrenal – exclude obstructive uropathy
- renal – oedema and volume overload
- hypertension
- signs of an underlying infection/septicaemia, e.g. fever, skin rash, etc.

**DIAGNOSTIC CRITERIA**
- urine culture – to exclude acute complicated pyelonephritis
- urine:
  - macroscopic appearance: brownish with acute tubular necrosis
  - microscopic appearance: red blood cell casts, leukocyte, hyaline and granular casts
- urine test strips:
  - haematuria
  - proteinuria
  - glycosuria
  - leucocytes
  - nitrites
- urine biochemistry:

<table>
<thead>
<tr>
<th></th>
<th>Pre-renal failure</th>
<th>Intrinsic renal failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>U-Osmol (mOsmol/L)</td>
<td>↑ &gt; 320</td>
<td>equal to serum Osmol</td>
</tr>
<tr>
<td>FeNa % *</td>
<td>&lt; 1 %</td>
<td>≥ 3 %</td>
</tr>
</tbody>
</table>

* FeNa %  = fractional excretion of Na (%)
  = \([U-Na/U-Creatinine \times S-Creatinine/S-Na]\) \times 100

**Note:**
S-creatinine is measured in micromol/L and urine creatinine in millimol/L
To convert millimol/L to micromol/L ÷ by 1 000
Special investigations
- ultrasound of kidneys and bladder
- S-urea, urate, creatinine, electrolytes and osmolarity, glucose, calcium, phosphate and ALP usually reveals:
  - hyperkalaemia
  - hypocalcaemia
  - hyponatraemia
  - hyperphosphataemia
  - metabolic acidosis
- full blood count, differential and platelet count
- clotting profile
- cultures and DIC workup as indicated
- check ECG on the vital signs monitor to exclude life threatening hyperkalaemia
- chest X-ray to evaluate cardiomegaly, pleural effusions and pulmonary oedema

NON-DRUG TREATMENT
- treat the underlying cause
- monitor fluid intake and output, blood pressure
- weigh daily
- nutritional support
  - high-energy diet with supplementary nasogastric feeds, if required
  - infants should preferably be given breast feeds or a milk formula
  - daily requirements
    - protein     0.8–1 g/kg maximum
    - carbohydrate 2–3 g/kg
    - fat         2 g/kg
  - restrict NaCl, K and phosphate intake
  - restrict protein intake when S-urea > 25 mmol/L

DRUG TREATMENT
- Avoid nephrotoxic or renally excreted medications, e.g. NSAIDs, aminoglycosides, vancomycin, cough and cold mixtures, radiocontrast drugs, etc.

Fluid management
For a well-hydrated patient without abnormal fluid losses, give maintenance fluid only.
For an anuric patient use an electrolyte free solution only to replace insensible losses, i.e. dextrose 5% or 10 %.
Insensible water loss:
- neonate and young baby       30–40 mL/kg/day
- older children               25 mL/kg/day (400 mL/m2/day)
Replace fluid losses with an appropriate solution, e.g. of diarrhoea or naso-gastric drainage.

Severe polyuria, i.e. urine output > 4 mL/kg/hour, due to tubular dysfunction and impaired urinary concentration occurs during the recovery (diuretic) phase of acute tubular necrosis.
Replace fluid and electrolyte losses, e.g. K, Cl and Na. Darrows half strength with dextrose 5% is usually the appropriate solution to use in this case.
Treat shock
See Section 1.1.6

Hyperkalaemia
Monitor ECG for signs of hyperkalaemia.
Discontinue all sources of intake of potassium.
Treat when serum potassium > 6.5 mmol/L
Monitor response to treatment and adjust accordingly.

- salbutamol, solution, 2.5–5 mg/dose, nebulise over 20 minutes
  0.5–1 mL salbutamol in 1 mL sodium chloride 0.9%
- sodium bicarbonate 4.2 %, IV, 4 mL/kg
  Do not mix calcium and sodium bicarbonate containing solutions.
- sodium polystyrene sulfonate, oral/rectal, 1 g/kg in dextrose water
- calcium gluconate 10 %, IV, 0.5–1 mL/kg/dose slowly over 3–5 minutes
- dextrose water 50%, IV, 2 mL/kg over 20 minutes ± insulin, 0.1 units/kg
  Check for hypoglycaemia hourly if insulin is used.

If hyperkalaemia persists despite above treatment
- refer for dialysis

Other complications
Metabolic acidosis - if S-pH ≤ 7.1
- sodium bicarbonate 4.2 %, IV, 4 mL/kg over 2–4 hours
  Do not mix calcium and sodium bicarbonate containing solutions.

Hypertension
See Section 4.9

Infection
Avoid nephrotoxic antibiotics.

Uraemic convulsions – See Section 13.4.
Refer for urgent dialysis
Exclude specific causes of convulsions, e.g. hypoglycaemia, hyper/ hyponatraemia, hypocalcaemia or hypertension and treat accordingly.

Anaemia – for acute blood loss/active haemolysis and Hb < 7 g/dL
- packed red cells, 10 mL/kg over 6 hours

Pulmonary oedema, acute heart failure, volume overload and hypertension
No IV fluid.
  Pulmonary oedema is an indication for dialysis.
  Digitalis is dangerous as it cannot be excreted.
  Intubate and initiate positive pressure ventilation as necessary.
- furosemide, IV, 2–5 mg/kg over 5 minutes
- morphine, IV, 0.1 mg/kg
- oxygen, 100%, 2–3 L/minute by nasal cannula
then
- refer
REFERRAL
Urgent for dialysis when:
- fluid overload causing pulmonary oedema
- anuria > 24 hours
- central nervous system signs, e.g. convulsions or coma
- uraemic diaphesis
- uraemic pericarditis
- hyperkalaemia or hyponatraemia not responding to conservative treatment
- persistent metabolic acidosis pH < 7.1 or serum bicarbonate < 10 mmol/L
- uncontrollable hypertension
- severe hyperphosphataemia and hypocalcaemia

6.1.5 RENAL FAILURE, CHRONIC
N18.9

DESCRIPTION
Chronic renal failure is that stage of renal function in which the kidney is unable to maintain the integrity of the internal environment. Chronic renal failure has been arbitrarily defined according to repeated measurements of creatinine clearance over time:
- persistent S-creatinine > 88 micromol/L in all infants (whole first year of life)
The following clearances can be calculated:

<table>
<thead>
<tr>
<th>Levels of chronic renal failure</th>
<th>Creatinine clearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>chronic renal impairment</td>
<td>&gt; 60 and &lt; 80 mL/minute</td>
</tr>
<tr>
<td>chronic renal failure</td>
<td>&gt; 20 and ≤ 60 mL/minute</td>
</tr>
<tr>
<td>end stage renal failure</td>
<td>≤ 20 mL/minute</td>
</tr>
</tbody>
</table>

DIAGNOSTIC CRITERIA
Clinical
Renal function may deteriorate without clinical symptoms.
- poor weight gain and stunting is often present over a long period
- children are likely to present with renal failure during episodes of acute intercurrent illness
- signs and symptoms may be due to:
  - disordered fluid and electrolyte excretion, or
  - disordered regulatory functions
- obligatory salt wasting may cause severe dehydration and metabolic acidosis:
  - obstructive uropathy           tubulo-interstitial nephropathy
  - chronic pyelonephritis         hypoplastic/dysplastic kidneys
- respiratory distress may be caused by compensatory tachypnoea due to acidosis
- poor appetite, chronic constipation, polydipsia and polyuria
- chronic anaemia
- renal osteodystrophy, i.e. bone pain and skeletal deformities
- volume overload: oedema, hypertension, heart failure, pulmonary oedema
• Uraemic symptoms and signs:
  ○ Nausea
  ○ Vomiting
  ○ Itching
  ○ Uraemic pigmentation, i.e. brownish skin pigmentation
• Bleeding tendency (mucosa)
• Convulsions due to hyponatraemia, hypernatraemia, hypocalcaemia, uraemia or hypertension.

**Special Investigations**

• Urine volume
  ○ Normal, or
  ○ Increased: > 6 mL/kg/day, or
  ○ Oliguric: <1.0 mL/kg/day
• Urine test strips
  ○ May be normal or reveal proteinuria, haematuria, glycosuria
  ○ Nitrites and leucocytes may indicate UTI – do urine MCS
• Urine microscopy
  ○ May be normal or reveal casts
  ○ Pus cells, leukocyte casts and bacteria may indicate UTI - do urine MCS
• S-urea
  ○ Increased, depending on hydration, nutritional state and protein intake
• S-creatinine
  ○ Increased – depends on age and muscle mass
• Theoretical creatinine clearance = [Constant x height (cm)] ÷ S-creatinine (micromol/L)

<table>
<thead>
<tr>
<th>Age group</th>
<th>Constant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-pubertal children</td>
<td>40</td>
</tr>
<tr>
<td>Adolescent girl</td>
<td>45</td>
</tr>
<tr>
<td>Adolescent boy</td>
<td>55</td>
</tr>
</tbody>
</table>

• S-electrolytes
  ○ Hyperkalaemia
  ○ Increased chloride
  ○ Decreased bicarbonate
• Urine Osmol
  ○ ISO-osmolar, i.e. 300–350 mOsm/L
• Calcium, phosphate and ALP
  ○ Decreased calcium
  ○ Increased phosphate
  ○ Increased ALP
• Parathyroid hormone
  ○ Increased
• Sonar
  ○ To exclude obstruction
  ○ Small shrunken kidneys are indicative of chronic renal failure
• There is no place for renal biopsy in patients with end stage renal failure
NON-DRUG TREATMENT

- determine and treat the underlying cause
- monitor fluid intake and output, blood pressure
- weigh daily
- if in respiratory distress
  - place in Fowler’s position, and give
  - oxygen, 100%, 2–3 L/minute by nasal prongs
- dietary management
  - potassium
    - Monitor serum potassium levels closely.
    - Limit potassium intake if serum potassium >5.5 mmol/L.
    - Restrict fruit juices, dried fruit, all citrus fruits, bananas, guavas and tomatoes.
    - All vegetables should either be soaked for 24 hours before cooking or water should be decanted twice during cooking.
  - phosphate
    - Restrict intake when blood levels reach or exceed the upper limit of normal for age, usually > 1.6 –1.8 mmol/L.
    - Limit dairy products, protein intake, grains and cereals, soft drinks, etc.
  - protein
    - Restrict once blood urea exceeds 20 mmol/L.
    - If urea exceeds 25 mmol /L protein, restrict protein intake to 0.8 g/kg/24 hours to alleviate acidosis, nausea and vomiting.
  - restrict salt intake
    - No salt added to food during preparation and consumption or salty foods. Generally, salt is restricted for hypertensive, oedematous patients, but not for patients with salt losing nephropathies who are polyuric.
  - high-energy diet with supplementary nasogastric feeds or nocturnal fluids is necessary for children with poor appetite and polyuria/nocturia

DRUG TREATMENT

Avoid nephrotoxic or renally excreted medications, e.g. NSAIDs, aminoglycosides, vancomycin, cough and cold mixtures, radiocontrast drugs, etc.

Fluid management

Volume required depends on the underlying cause of the renal failure. For ambulatory patients fluid management is guided by type of renal failure and presence or absence of oedema and hypertension.

Do not give parenteral fluids to hospitalised patients who are volume overloaded and oliguric/anuric.

Replace urine output and losses, volume for volume, with an appropriate solution, usually a potassium free solution, e.g. sodium chloride 0.45%.

Insensible water loss:
- neonate and young baby 30–40 mL/kg/day
- older children 25 mL/kg/day (400 mL/m²/day)
If dehydrated and hypotensive, give:
- sodium chloride 0.9%, IV, immediately as a bolus and reassess.
A repeat fluid bolus may be necessary, but strict monitoring of urine output and fluid losses is required.

For an anuric patient use an electrolyte free solution only to replace insensible losses, i.e. 5 or 10 % dextrose water.

- multivitamin, oral, 5 mL, daily
  Containing vitamins B₁, B₆, B₁₂ and C.
  **AND**
- folic acid, oral, 5 mg daily

**Hyperphosphataemia/osteodystrophy**
In combination with restricted dietary intake of phosphate:
- calcium carbonate, oral, 1–4 tablets chewed 3 times daily with meals
  1 tablet is equivalent to 0.168 g elemental calcium

In patients with serum calcium < 2.2 mmol/L, give activated Vitamin D supplementation early. If serum phosphate is > 2.5 mmol/L, treat the hyperphosphataemia first to decrease below this level before beginning the alfalcacidol.  – See above.
- alfalcacidol oral, 0.25 mcg, initially twice weekly
  Increase dose as necessary to maintain serum calcium in upper normal range.
  Doses as high as 0.5 mcg twice daily may be required.

**Chronic metabolic acidosis**
If serum bicarbonate < 18 mmol/L
- sodium bicarbonate, oral, 1 mmol/kg/dose 2–3 doses per day after meals
  Adjust according to response.
  **Note:**
  The intravenous formulation can be given orally.
  **OR**
  Shohl’s solution, oral, 1-2 mmol/kg/dose, 2–3 times daily after meals
  Adjusted according to response.

<table>
<thead>
<tr>
<th>Ingredient</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>citric acid</td>
<td>140 g</td>
</tr>
<tr>
<td>sodium citrate</td>
<td>98 g</td>
</tr>
<tr>
<td>water to</td>
<td>1 L</td>
</tr>
<tr>
<td><strong>1 mL = 1 mmol of alkali</strong></td>
<td></td>
</tr>
</tbody>
</table>

**Hyperkalaemia**
Discontinue all drugs that may cause hyperkalaemia, e.g. potassium sparing diuretics, spironolactone, ACE inhibitors.
Exclude volume depletion as an underlying cause for hyperkalaemia.
If serum potassium remains > 5.5 mmol/L
- sodium polystyrene sulfonate, oral/rectal, 1 g/kg/dose in dextrose water, once or twice daily
Treat accompanying metabolic acidosis.
Anaemia
Ensure adequate intake of haematinics.
Ensure adequate iron stores - check levels of serum ferritin, transferrin, transferrin saturation and total iron binding capacity.
Check levels of serum B₁₂ and red cell folate before starting erythropoetin treatment.
For persistent anaemia – refer to tertiary centre for nephrologist assessment.

Hypertension: See Section 4.9

Renoprotective treatment
All children with persistent proteinuria, i.e. creatine clearance more than 60 mL/min should receive the following under nephrologist supervision:

ACE inhibitor, e.g.:
- enalapril, oral, 0.1 mg/kg/dose, once daily
  Dose may be increased to 0.5 mg/kg/day, as a single dose or two divided doses.
  OR
captopril, oral, 0.5–2.5 mg/kg/dose twice daily
  OR
perindopril, oral, 0.05-0.15 mg/kg/dose, once daily

ACE inhibitors may cause hyperkalaemia, worsening metabolic acidosis and declining renal function.
Monitor serum urea and electrolytes, i.e. serum potassium and bicarbonate, and renal function within 7 days.
If serum creatinine has doubled, hydration status should be checked, diuretics should be stopped and dose of ACE inhibitors halved.
If renal function does not improve, or hyperkalaemia > 5.5 persists, stop ACE inhibitor treatment.

Immunisation
All children should receive routine immunisation according to EPI schedule.
Check immunity against Hepatitis B.
In the absence of any immunity, vaccinate as for any non-immune individual.
  • hepatitis B vaccine, IM, 1 mL, 3 doses at monthly intervals
    1 mL = 3 mcg
If the antibody level is considered non-protective or insufficient, give 2 booster doses one month apart.

REFERRAL
  • all children with chronic kidney disease, including those with:
    o persistent proteinuria or haematuria
    o inherited kidney diseases
    o renal tubulopathies
    o congenital malformation of kidneys
    o chronic bilharziasis, etc.
  • patients with dyslipidaemia or hypercholesterolaemia
6.1.6 ENURESIS

DESCRIPTION
Enuresis is bedwetting after the age of 5 years. Primary monosymptomatic enuresis refers to incontinence during sleep only. It is of great importance to differentiate between monosymptomatic enuresis and enuresis with associated bladder dysfunction during daytime, because the treatment of these two conditions is totally different.

DIAGNOSTIC CRITERIA

Clinical
- clinical evaluation of all enuretic children should begin with a structured interview
- exclude symptoms of underlying systemic disease e.g.:
  - diabetes mellitus
  - diabetes insipidus
  - urinary tract infections
  - neurological disturbances
  - structural abnormalities

Special investigations
- urine examination should be done in all patients
- exclude organic causes
- ultrasound investigation may be necessary to identify structural abnormalities of the kidneys, pelvis and ureters

NON-DRUG TREATMENT
Enuresis is a benign condition with a spontaneous annual resolution rate. Intervention must carry no risk or have minimal side effects. The cure rate of “treatment” should be significantly greater than the spontaneous cure rate before it can be considered effective.
- motivate, counsel and reassure child and parents
- advise against punishment and scolding
- spread fluid intake throughout the day
- restrict excessive fluid intake before retiring to bed
- diapers should never be used as this will lower the self esteem
- bell systems are effective but should only be used in older children
- consider behaviour modification and bladder training exercises in children with diurnal enuresis

DRUG TREATMENT
For short term treatment only for a patient who was abused and who has enuresis – in consultation with a specialist
- desmopressin, oral, 200–400 mcg at night for 3 months
  Adverse effects include fluid retention, hyponatraemia and cerebral oedema.

In children over 5 years with voiding dysfunction and accompanying diurnal enuresis
- oxybutinin, oral, 2.5–5 mg, 8–12 hourly
REFERRAL
- suspected underlying systemic illness or chronic kidney disease
- persistent enuresis in a child over 8 years

6.2. GENITAL CONDITIONS

6.2.1 CONTRACEPTION
Z30

Adolescents
In general, adolescents are eligible to use any method of contraception and must have access to a variety of contraceptive choices. Age alone does not constitute a medical reason for denying contraception. Dual method use, i.e. use of hormonal contraceptives (oral or injectable) as well as barrier contraception is advisable as precaution against pregnancy and sexually transmitted infections. Adolescents are not good candidates for intra-uterine contraceptive devices.

Safety issues
While some concerns have been expressed regarding the use of certain contraceptive methods in adolescents, e.g. the use of progestogen-only injectables by those < 18 years, these concerns must be balanced against the advantages of avoiding pregnancy. It is clear that many of the same issues regarding appropriate contraceptive use applicable to older clients apply to adolescents.

Some non-contraceptive advantages of oral contraceptives include less menstrual blood loss, regulated cycles and decreased incidence of ovarian and breast cysts. Return to fertility is rapid once the medication is discontinued. There is no evidence of increased risk of infertility, malignancy of the cervix, uterus, ovaries or breasts or of increased risk for STIs when oral contraceptives are used.

Emergency contraception
Should be provided to all females with signs of breast development who have a negative pregnancy test.
- norgestrel 0.5 mg and ethinyl oestradiol 0.05 mg, oral, 2 tablets immediately and 2 tablets 12 hours later

6.2.2 ABNORMAL UTERINE BLEEDING
N93.8

REFERRAL
- all adolescent patients with oligomenorrhea or amenorrhea or dysfunctional uterine bleeding associated with unexplained symptoms and signs, including those with:
  - accompanying hypertension
  - features of Cushing’s syndrome
  - striae
  - galactorrhoea
  - male pattern alopecia
• family history of infertility and hirsutism
• if polycystic ovary syndrome cannot be excluded. Clinical features include:
  ○ oligo-ovulation or anovulation, usually manifests as oligomenorrhoea or amenorrhoea
  ○ clinical manifestations of androgen excess (hyperandrogenism) including hirsutism, acne and male pattern of hair loss
  ○ acanthosis nigricans due to hyperinsulinism
  ○ elevated levels of circulating androgens (hyperandrogenaemia)
  ○ polycystic ovaries as defined by ultrasonography
  ○ substantial proportion of women are obese
• exclude pregnancy

6.2.3 VAGINAL DISCHARGE IN PREPUBESCENT CHILDREN
N89.8

DESCRIPTION
Vaginal discharge may be thin grey and foul smelling as caused by Gardnerella vaginalis or due to anaerobic bacteria such as Bacteroides and Peptostreptococcus. Other pathological organisms include Chlamydia and Trichomonas.

In prepubescent girls it may be due to poor hygiene or irritants, such as bubble baths, deodorants and detergents used to wash underwear. Foreign bodies, pinworms and sexual abuse should always be excluded in the prepubescent girl. Gonorrhoea in the prepubescent girl is almost invariably due to sexual abuse.

DIAGNOSTIC CRITERIA
Clinical
• presence of overt discharge
• absence of foreign body/allergy
• specific diagnosis dependent on microbiological investigation

Special Investigations
• vaginal aspirate or pus swab should be sent for microscopy and cultures
  ○ presence of Gonococci, Trichomonas or Chlamydia indicates the likelihood of sexual abuse
• serological testing for syphilis (STS) and HIV (with consent)

NON-DRUG TREATMENT
• if the likelihood of sexual abuse exists, ask child about history of previous abuse, if possible when caregiver is not present
  ○ if there is a history of sexual abuse, manage as Sexual Abuse: See Section 6.2.5
  ○ if there is no history do not force disclosure
• exclude pinworm infestations
• advise parents and child regarding hygiene, toilet habits and avoidance of irritants

DRUG TREATMENT
Treat STIs appropriately.
For Monilial infection
- nystatin cream 100,000 IU/g, topical, apply 8 hourly for 7 days

Indigenous bacterial vaginosis
- metronidazole, oral, 7.5 mg/kg/dose, 8 hourly for 7 days
  AND
- amoxicillin, oral, 30 mg/kg/dose, 8 hourly for 7 days

For resistant discharge
- conjugated oestrogen cream 0.625 mg/g, vaginally, at night for a maximum of two weeks. Warn parents about bloody discharge due to withdrawal afterwards.

6.2.4 SEXUALLY TRANSMITTED INFECTIONS

Sexual abuse should be excluded in young children who have acquired gonorrhoea.

Gonorrhoea
- ceftriaxone, IM, immediately as a single dose
  - < 25 kg 125 mg
  - > 25 kg 250 mg
  OR
  - If not available and > 2 years old and not allergic to penicillin
    amoxicillin, oral, 50 mg/kg immediately
    Maximum dose: 3 g

Syphilis
Only if early disease
- benzathine benzylpenicillin (depot formulation), IM, 50,000 unit/kg, single dose
  Maximum dose: 2.4 million units

If present for more than one year
- benzathine benzylpenicillin (depot formulation), IM, 1.2 million units, weekly for 3 doses

Penicillin allergy
- erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly
  If infection is present for less than 1 year treat for 15 days.
  If longer than 1 year treat for 30 days.

Do titres after 6 months and 1 year to confirm decrease.
Treat again if:
- clinical signs and symptoms persist
- sustained or increase of titre

Trichomonas vaginalis and gardnerella vaginalis
- metronidazole, oral, 7.5 mg/kg/dose, 8 hourly for 7 days
  OR
  - Older children
    metronidazole, oral, 2 g, immediately
Chlamydia trachomatis
- erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 10–14 days
ANDEX > 8 years old
- doxycycline, oral, 100mg twice daily for 7 days

6.2.5 SEXUAL ABUSE AND PREVENTION OF INFECTION/CONCEPTION
T74.2

DESCRIPTION
The following indicate that sexual abuse has or may have occurred:
- sexually transmitted or vaginal infections
- painful urination, frequency of micturition or frequent urinary infections
- pregnancy in children under the age of 16
- pain, itch, bruises or bleeding from the external genitalia or anal area
- sexualised behaviour or other unexplained behavioural problems
- unexplained difficulty in walking or standing
- recurrent unexplained abdominal pain
- unexplained behavioural changes, e.g. depression, anxiety disorders, aggression, fear, parasuicide, enuresis, encopresis and pseudoseizures

MANAGEMENT OBJECTIVES
- psychological support of the victim and family
- prevent or minimise the unwanted complications of the assault
  - physical trauma
  - psychosocial trauma
  - sexually transmitted infections
  - pregnancy
- support the due legal process
  - medical documentation of evidence
  - collection of appropriate specimens
- conduct baseline investigations
  - HIV test
  - RPR
  - hepatitis screening
  - vaginal swabs for acid phosphatase and microbiology after consent

NON-DRUG TREATMENT
- obtain informed consent from the patient and written consent from parent/guardian in case of minors before HIV testing and PEP. Children over the age of 14 years may sign their own consent. Every effort should be made to encourage testing.
- the patient's HIV-status should be determined before initiating PEP. Prophylaxis given to a previously infected HIV person will have no clinical benefit and may lead to the development of viral resistance.
- it is the patient’s choice to have immediate HIV testing. However, no PEP will be given in the case of refusal of HIV testing.
- a patient presenting after 72 hours will not be given PEP but should be counselled about the possible risk of transmission. HIV testing should still be offered at the time of presentation and 3 months later.
perform a pregnancy test before initiating PEP
HIV Elisa positive tested sexually abused children under the age of 15 months must be referred to have an HIV DNA PCR (polymerase chain reaction) performed. If HIV uninfected or if the child has no access to PCR, they should receive prophylaxis.
explain the side effects of the ARV drugs, e.g. tiredness, nausea and flu-like symptoms
emphasise the importance of compliance with ARV treatment
counsel all sexually assaulted patients and caregivers in the case of children
psychosocial support
medical risks, e.g. transmission of sexually transmitted infections including HIV, hepatitis-B and pregnancy
psycho-emotional-social effects of the sexual assault according to their level of understanding and maturity
identify need for support and refer if needed
discuss issues relating to stress management at subsequent visits
post traumatic stress may eventually cause exhaustion and illness. Inform the patient of the signs and symptoms of post traumatic stress, including:
  - general irritability
  - trembling
  - pain in neck and/or lower back
  - change in appetite
  - change in sleep pattern
medico-legal assessment of injuries
complete appropriate registers

Note:
Refer very young or severely traumatised children to a specialised unit or facility.
Children with external signs of genital trauma may need an examination under anaesthesia and should be referred. Trauma to the genital area increases transmission. The character of the exposure should be classified as:
  - low risk – non receptive or non traumatic intercourse
  - high risk – penetration and traumatic intercourse

Blood tests
the patient should sign a consent form for both testing and PEP
voluntary rapid HIV testing should be made available and should be done on all opting for PEP
further blood tests should include full blood count (FBC)
a full blood count should be repeated at 2 and 4 weeks
blood should be taken at 6 weeks, 3 months and 6 months for HIV testing
DRUG TREATMENT

Note:
• if the patient presents within 72 hours of being raped, PEP should be offered
• consent for HIV testing must be obtained from all patients before initiating PEP
• initiate PEP as soon as possible provided the patient is not HIV-infected prior to the incident
• for low risk exposure, initiate dual therapy
• for high risk exposure and children with physically traumatic assaults, refer for management of these physical injuries and to consider the use of triple therapy. During referral dual therapy should be initiated immediately.
• in children under the age of 15 months antiretroviral therapy should be used while arranging transfer and awaiting confirmation of HIV results
• initiating therapy within 24 hours is most likely to be effective at preventing transmission of HIV
• for those refusing an HIV test, no PEP will be provided
• do a pregnancy test in all women and female adolescents. In the case of children who are clearly pre-pubertal this is omitted.

If not pregnant:
STI prophylaxis
children under 8 years
• ceftriaxone, IM, immediately as a single dose
  < 25 kg  125 mg
  > 25 kg  250 mg

• Hepatitis-B vaccination: See Section 2.3.4

PEP treatment
As the body surface area is very difficult to calculate, the following guidelines are provided:
• zidovudine, oral, 12 hourly. Maximum 300 mg/dose.
  6 months–3 years  9 mg/kg/dose
  4–12 years    7.5 mg/kg/dose

• lamivudine, oral, 4 mg/kg/dose 12 hourly. Maximum 150 mg/dose.
AND
If significant exposure has occurred
• lopinavir/ritonavir 80/20, oral, 230 mg/m²/dose of lopinavir component 12 hourly
  Administer with food.
  A high-fat meal increases absorption, especially of the solution.

Dosages may be varied by up to 1 mg/kg/dose more or less to allow a convenient volume of medication.

Follow up visits should be at 6 weeks, 3 months and 6 months after the rape. HIV testing should be performed at each of these visits.
REFERRAL
• all patients with severe physical or psychological injuries
• pregnant rape patients
• infants with significant evidence of sexual assault need referral after beginning dual therapy as soon as possible

Note:
Refer as soon as possible within 24 hours if there are inadequate resources with regard to:
• counselling
• laboratory for testing
• medico-legal examination
• drug treatment
7.1 ADRENAL HYPERPLASIA, CONGENITAL
E25.0

DESCRIPTION
Autosomal recessive enzymatic defects of the cortisol biosynthetic pathways in the adrenal gland. The presentation depends on the severity and type of the enzyme defect.

DIAGNOSTIC CRITERIA
Clinical
- neonates with ambiguous genitalia
- adrenal insufficiency – See Section 7.2
- accelerated growth velocity or precocious pseudopuberty

Investigations
See Acute adrenal Insufficiency: Section 7.2
- elevated 17-hydroxyprogesterone in the serum
- elevated serum renin

NON-DRUG TREATMENT
- surgical correction of genital abnormalities after endocrine treatment
- psychological support for child and family

DRUG TREATMENT
Glucocorticoid and mineralocorticoid replacement. To be initiated in consultation with subspecialist.

- hydrocortisone, oral, 0.5 mg/kg/day in three divided doses. Specialist initiated.
  The morning dose should be given as early as possible.

- fludrocortisone acetate, oral, 5 mcg/kg/day as single daily dose
  Range: 50–200 mcg daily.

For salt losing patients
- sodium chloride, oral, 0.5–1 g for every 10 kg body weight per day

Glucocorticoids are administered for life. Once growth is complete, prednisone may be given once or twice daily or betamethasone given as a single daily dose.
The dose is individualised by monitoring growth, bone age and hormonal levels.

REFERRAL
- all cases for confirmation of the diagnosis, counselling and initiation and monitoring of treatment
7.2 ADRENAL INSUFFICIENCY, ACUTE
E27.4

DESCRIPTION
Acute failure of adrenal function, suspected when a patient presents with hypotension, hypoglycaemia, hyponatraemia, hyperkalaemia, and metabolic acidosis.

Patients at present or recently on chronic steroid therapy are at risk for adrenal insufficiency if they fail to augment the steroid dose during times of stress (fever, trauma, and surgery).

DIAGNOSTIC CRITERIA

Clinical
- acute circulatory collapse. The features include:
  - tachycardia
  - hypotension
  - pallor
  - poor peripheral perfusion
  - cool clammy skin
  - disturbed consciousness
  - coma
  - hyperkalaemia
  - signs of dehydration
  - hyponatraemia
  - metabolic acidosis

- a history of weakness, anorexia, vomiting, weight loss, salt craving, hyperpigmentation (primary adrenal insufficiency), auto-immune endocrinopathies and steroid-dependence
- ambiguous genitalia

Investigations
Take blood for estimation of
- serum electrolytes and blood glucose
- In all suspected cases, take a sample of clotted blood for estimation of plasma cortisol prior to treating the patient. Send this sample with the patient to the central hospital if laboratory facilities are not locally available.

DRUG TREATMENT

Stabilisation
- dextrose 5% in sodium chloride 0.9%, IV, 20 mL/kg bolus as needed
  OR
  Ringer-Lactate with dextrose 5%, IV, 20 mL/kg bolus as needed
  OR
  dextrose 10%, IV, 3 mL/kg glucose as needed

- hydrocortisone, IV, 2–3 mg/kg immediately, then 2–3 mg/kg/day every six hours

Manage hyperkalaemia – See Section 7.8

Prevention
In patients on chronic steroid therapy, it is important to increase corticosteroid dose in all stressful situations, e.g. pre-surgery, burns, trauma and dental procedures.

Adrenal insufficiency is a life threatening emergency
REFERRAL
- all cases immediately after stabilisation

7.3 DIABETES INSIPIDUS
E23.2

DESCRIPTION
Diabetes insipidus should be suspected in any child with polydypsia and polyuria. Infants may present with failure to thrive. Central diabetes insipidus is due to deficiency of antidiuretic hormone. Nephrogenic diabetes insipidus occurs if the kidney is unable to respond to antidiuretic hormone.

DIAGNOSTIC CRITERIA
- pathological polyuria defined as excretion of > 1.5 L/m² of urine
  In infants the corresponding value is > 2.5 L/m²
- serum osmolality > 300 mOsm/kg, with urine osmolality < 300 mOsm/kg is suggestive of diabetes insipidus
- a positive water deprivation test - only conducted under specialist supervision

DRUG TREATMENT
Central diabetes insipidus
- desmopressin intranasal solution, intranasal, 5–30 mcg/day 12–24 hourly
  OR
desmopressin, oral, 50–300 mcg/day twice daily
  Increase the dose to the lowest amount which gives an antidiuretic effect.
  The patient must have a phase of urinary dilution or breakthrough urination before the next dose to ensure that water intoxication does not result

Nephrogenic diabetes insipidus
Treat the underlying cause.

REFERRAL
- all cases for evaluation

7.4 DIABETES MELLITUS

DESCRIPTION
A syndrome of abnormal carbohydrate metabolism, associated with a relative or absolute impairment of insulin secretion with varying degrees of peripheral resistance to the action of insulin.

7.4.1 DIABETES MELLITUS, INSULIN DEPENDENT (TYPE 1)
E10

DESCRIPTION
Most diabetic children have type 1 diabetes, and:
- have auto-immune destruction of the pancreatic beta cells as the underlying cause
- have an absolute requirement for insulin therapy
- will develop diabetic ketoacidosis (DKA) if not given insulin
CHAPTER 7

ENDOCRINE SYSTEM

DIAGNOSTIC CRITERIA
- polydipsia
- polyphagia
- heavy glycosuria
- random blood glucose of ≥ 11.1 mmol/L
- polyuria – this can present as 2º enuresis in young children
- fasting blood glucose of ≥ 7.0 mmol/L – fasting is not usually needed for the diagnosis
- an oral glucose tolerance test is not needed

NON-DRUG TREATMENT
- general measures
  - educate child and caregiver about all aspects of the disease
  - medical alert bracelet should be worn at all times
  - follow-up by medical practitioner or at clinic/hospital at least every 3 months
- diet: healthy lifelong eating habits
  A newly diagnosed patient and family must be referred to a dietician.
  Principles of the prudent diet:
  - children should be encouraged to reduce the intake of fats and salt and to increase dietary fibre content.
  - All diabetics should be given a meal plan, e.g. “constant carbohydrate meal plan” or “carbohydrates counting meal plan”. There is no one ‘diabetic’ diet. The diet should be individualised with consideration given to usual eating habits and other lifestyle changes.
  - Six main nutrition factors contribute to better sugar control, i.e. lower HbA1c levels. These are:
    1. following a meal plan. Keep day to day intake consistent.
    2. avoiding extra snacks that are not part of the meal plan
    3. avoiding over-treatment of low blood sugars (hypoglycemia)
    4. prompt correction of high blood sugars
    5. adjusting insulin levels for meals in patients using the “carb counting meal plan”
    6. consistency of night snacks
**CONSTANT CARBOHYDRATE (CARB) MEAL PLAN**

Consistency is the key. The amount of insulin, usually two or three doses per day, is kept relatively constant from day-to-day. Carbohydrates should be manipulated to match the relatively constant insulin dose. The amount of carbs (types can vary) is kept about the same for each meal and each snack from one day to the next.

As part of the educational process of the family, labels must be read to know the grams (g) of carbs being eaten. The dietician may give a range of carbs for each meal.

**Examples of carbohydrate content of some foods**
The following foods have 15 g of carbohydrate per serving:

<table>
<thead>
<tr>
<th>FOOD</th>
<th>SERVING SIZE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beans (cooked, canned)</td>
<td>½ cup</td>
</tr>
<tr>
<td>Bread (white, brown)</td>
<td>1 slice</td>
</tr>
<tr>
<td>Maize (cooked)</td>
<td>½ cup</td>
</tr>
<tr>
<td>Pasta (cooked)</td>
<td>½ cup</td>
</tr>
<tr>
<td>Potato (mashed)</td>
<td>½ cup</td>
</tr>
<tr>
<td>Rice (cooked)</td>
<td>½ cup</td>
</tr>
<tr>
<td>Apple (small)</td>
<td>1</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>½ cup</td>
</tr>
<tr>
<td>Grapes (small)</td>
<td>17</td>
</tr>
<tr>
<td>Orange (small)</td>
<td>1</td>
</tr>
<tr>
<td>Banana (small)</td>
<td>1</td>
</tr>
<tr>
<td>Milk</td>
<td>1 cup</td>
</tr>
<tr>
<td>Yoghurt (light)</td>
<td>1 cup</td>
</tr>
<tr>
<td>Pizza</td>
<td>1 slice</td>
</tr>
<tr>
<td>Potato chips</td>
<td>8–12</td>
</tr>
</tbody>
</table>

- the advice should be tailored to the patients' lifestyle, economic circumstances and usual diet and, where possible, should avoid drastic changes
- no particular food should be forbidden as this may lead to disturbed attitudes to food, e.g. carbohydrates are not forbidden but can be taken before exercise, incorporated into a main meal or used as a source of energy during illness when children have a poor appetite
- diet should provide adequate nutrition for growth and development
Dietary composition
It is recommended that:
- approximately 35% of dietary energy should be derived from fat, mono and polyunsaturated
- 15% from protein
- 50% from carbohydrates. Carbohydrates should always provide at least 40% of the total calories.

Timing of meals and snacks
Children receiving twice daily injections of combined short and intermediate acting insulin regimens need three main meals and three snacks (midmorning, mid afternoon and prior to bed time).

Eat meals and snacks at the same time each day. The timing of insulin injections may need to be adjusted according to the patients’ own circumstances.

Preschool aged children may have unpredictable eating habits and may require frequent small meals.

• exercise
  - regular exercise helps increase insulin sensitivity, maintains proper weight, blood pressure, blood glucose and blood fat levels
  - exercise must be regular, i.e. daily. The same amount of exercise should ideally be done at the same time of the day.
  - some form of carbohydrate is necessary before and after intense exercise to reduce the risk of hypoglycaemia. Blood glucose monitoring may be necessary before and after intense exercise.

• blood glucose testing, record keeping and review of records
  - glucometers should be available with compatible strips and bloodletting devices.
  - children should be encouraged to perform their own finger-prick blood glucose testing
  - finger prick should be performed at the side of the fingertips
  - the child should be encouraged to monitor his/her blood glucose prior to each main meal and at bedtime. A daily record of all testing performed should be recorded in a logbook. This should be reviewed frequently to ensure optimal adjustments in management are made.
  - more frequent blood glucose testing is indicated if the child is unwell, partaking in unusual amounts of physical activity or feels hypoglycaemic

• glycaemic targets
  Glycaemic targets for young children should not be as strict as for adults. Balance the ability of the family to avoid recurrent hypoglycaemia. A paediatrician should assist in setting practical goals.
  Ideally 80% of the pre-meal blood glucose values should fall within the target range during home monitoring, but targets may need to be altered based on the age of the child and the ability of the family.
  - babies 6–10 mmol/L
  - toddlers 5–8 mmol/L
  - older children 4–7 mmol/L
The aim is to maintain HbA1C as close as possible to the normal range.

**Desired Ranges of HbA1c for Diabetic Children**

<table>
<thead>
<tr>
<th>AGE</th>
<th>HbA1c</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5 years</td>
<td>7.5–9.3%</td>
</tr>
<tr>
<td>5–11 years</td>
<td>&lt;8.5%</td>
</tr>
<tr>
<td>12–21 years</td>
<td>&lt;7.8%</td>
</tr>
</tbody>
</table>

- **Urinary Ketone Testing**
  The presence of hyperglycaemia and substantial ketones (+++) indicates that DKA is present.
  Urine should be tested for ketones in the following circumstances:
  - if vomiting occurs
  - any time the blood glucose is above 15 mmol/L, especially if the child is unwell and particularly if the blood glucose has been high for more than 24 hours
  - if unusual drowsiness is present
  - in the presence of high temperature, vomiting or diarrhoea, even when the glucose is < 15 mmol/L
  - if abdominal pain occurs
  - if the breathing is deep and rapid or smells of acetone

**Drug Treatment**

**Insulin Therapy**

- **Principles of Insulin Therapy**
  - to provide sufficient insulin throughout the 24 hour period to cover basal requirements
  - to deliver higher boluses of insulin in an attempt to match the glycaemic effect of meals

- **The Most Suitable Areas for Insulin Injection Are:**
  - the upper, outer area of the arms
  - the front and side of the thigh
  - the upper, outer surface of the buttocks, and
  - the abdomen, except the area close to the navel

- **Establish a Pattern for Injecting**, i.e. horizontally or vertically. Vary the site of injection according to this pattern. When the area has been fully covered move to another area.

- **Patients Doing Strenuous Exercise Should Not Inject Into Their Legs**
Insulin injection technique:

Insulin injection by syringe is usually given into deep subcutaneous tissue through a two-finger pinch of skin at an angle of 45–90 degrees. The subcutaneous fat layer should be thicker than the needle length. There is significant risk of accidental intramuscular injections and hence more rapid absorption especially in lean individuals. This can be minimised by using a two-finger pinch technique, an injection angle of 45 degrees and 8 mm needles. Five or 6 mm needles may be appropriate in lean children or those using pens. Disinfection of the skin is not necessary prior to insulin injections, however injections should be given through clean, healthy skin. Needles should not be used for more than 3–6 injections. Prefilled insulin syringes are recommended for children. Pen devices delivering less than 1 unit should be available for infants and young children. All insulin suspensions must be thoroughly mixed before injection by rolling or inverting the vial ten times so that the cloudy suspension mixes thoroughly and uniformly.

### Duration of Action of Standard Insulins and Insulin Analogues

<table>
<thead>
<tr>
<th>INSULIN</th>
<th>ONSET OF ACTION</th>
<th>PEAK ACTION</th>
<th>EFFECTIVE DURATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>STANDARD INSULIN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Regular/short acting</td>
<td>30–60 minutes</td>
<td>2–3 hours</td>
<td>8–10 hours</td>
</tr>
<tr>
<td>Intermediate acting</td>
<td>2–4 hours</td>
<td>4–12 hours</td>
<td>12–20 hours</td>
</tr>
<tr>
<td>ANALOGUE INSULIN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rapid</td>
<td>5–15 minutes</td>
<td>30–90 minutes</td>
<td>4–6 hours</td>
</tr>
</tbody>
</table>

### Choice of insulin regimen

- no insulin injection regimen satisfactorily mimics normal physiology
  
  The choice of insulin regimen should be individualised and will depend on age, duration of diabetes, lifestyle (dietary patterns, exercise schedules, school, work commitments, etc), targets of metabolic control, and particularly, individual patient/family preferences.
The choice of an insulin regimen is determined by the patient’s circumstances. Depending on the patient’s scope to undertake insulin therapy, a number of alternatives will allow insulin therapy to be tailored to their lifestyle. Discussion with parents should provide the basis for such important decisions.

**Whichever insulin regimen is chosen should be supported by comprehensive education appropriate for the age, maturity and individual needs of the child and family.**

### Insulin Regimens

The following regimen choices are listed in order of simplicity and flexibility:

- **Regimen 1: Two Injections daily**
  - a mixture (premixed combination) of short and intermediate acting insulins (before breakfast and the main evening meal)
  - The total daily dose is divided so that \(\frac{2}{3}\) is given in the morning and \(\frac{1}{3}\) in the evening

<table>
<thead>
<tr>
<th>Regimen 1: Premixed 70/30</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breakfast</strong></td>
</tr>
<tr>
<td>intermediate acting (\frac{2}{3}) of dose \ (+ \ short acting insulin (\frac{1}{3}) of dose)</td>
</tr>
<tr>
<td>(\frac{2}{3}) of total daily dose</td>
</tr>
<tr>
<td><strong>Supper</strong></td>
</tr>
<tr>
<td>intermediate acting (\frac{2}{3}) of dose \ (+ \ short acting insulin (\frac{1}{3}) of dose)</td>
</tr>
<tr>
<td>(\frac{1}{3}) of total daily dose</td>
</tr>
</tbody>
</table>

- OR

- **Regimen 2: Three injections daily**
  - a mixture of short and intermediate acting insulin before breakfast; short acting insulin alone before an afternoon snack or main evening meal; intermediate acting insulin before bed; or variations of this.

<table>
<thead>
<tr>
<th>Regimen 2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Breakfast</strong></td>
</tr>
<tr>
<td>short acting insulin (\frac{1}{3}) of dose \ (+ \ intermediate acting (\frac{2}{3}) of dose)</td>
</tr>
<tr>
<td>(\frac{2}{3}) of total daily dose</td>
</tr>
<tr>
<td><strong>Supper</strong></td>
</tr>
<tr>
<td>short acting insulin (\frac{1}{3}) of dose</td>
</tr>
<tr>
<td>(\frac{1}{3}) of total daily dose</td>
</tr>
<tr>
<td><strong>At night (± 21h00)</strong></td>
</tr>
<tr>
<td>intermediate acting (\frac{2}{3}) of dose</td>
</tr>
<tr>
<td>(\frac{1}{3}) of total daily dose</td>
</tr>
</tbody>
</table>

- OR

- **Regimen 3: Basal-bolus regimen**
  - rapid acting insulin analogue with main meals or short acting insulin 15–30 minutes before a meal, e.g. breakfast, lunch and main evening meal; intermediate acting insulin before bed
Normally, 30 – 40% of the total daily dose of insulin is given at bedtime as intermediate acting insulin. The remaining insulin is given prior to breakfast, lunch and evening meal in the form of short acting insulin.

**Regimen 3: Basal-bolus regimen**

Rapid acting insulin is indicated in the child (especially under 3 years) with erratic eating habits despite adequate education

<table>
<thead>
<tr>
<th>Time</th>
<th>Type of Insulin</th>
<th>Percentage of Total Daily Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breakfast</td>
<td>rapid (or short acting)</td>
<td>20% of total daily dose</td>
</tr>
<tr>
<td>Lunch</td>
<td>rapid (or short acting)</td>
<td>20% of total daily dose</td>
</tr>
<tr>
<td>Supper</td>
<td>rapid (or short acting)</td>
<td>20% of total daily dose</td>
</tr>
<tr>
<td>At night (± 21h00)</td>
<td>intermediate acting</td>
<td>40% of total daily dose</td>
</tr>
</tbody>
</table>

○ Questions to be considered when choosing a regimen

**What scope does the patient have for insulin therapy?**

- Will the patient be able to undertake, financially and culturally, an advanced insulin regimen if necessary?
- Is a responsible person available to give insulin injections at all times of the day or only at certain times?
- How goal orientated is the patient/caregiver in terms of diabetes control?

**What is the patient’s eating pattern?**

- What is the typical pattern of meals?
- What type of food do they typically eat at each meal, and how much?
- Is their eating pattern relatively constant, or does it vary?
- Can they or do they want to change their eating habits?

None of these regimens can be optimised without frequent assessment by blood glucose monitoring.

Achieving a balance between food intake, insulin levels and energy expenditure is an essential pre-requisite for achieving glycaemic control.
Adjustment of insulin dosage for regimens 1 and 2

The insulin dose should not be changed after a single abnormal blood glucose reading. Only once a pattern has been established should the dose be adjusted. Which dose is to be adjusted depends on the time of abnormal glucose readings, as indicated in the table below:

### Regimens 1 and 2

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Timing of the unsatisfactory blood glucose level</th>
<th>Before breakfast</th>
<th>Before Lunch</th>
<th>Before supper</th>
<th>At ± 21h00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose too high</td>
<td>Which Insulin dose to be increased</td>
<td>Supper or 21h00 dose: intermediate acting insulin</td>
<td>Breakfast dose: short acting insulin</td>
<td>Breakfast dose: intermediate acting insulin</td>
<td>Supper dose: short acting insulin</td>
</tr>
<tr>
<td>Glucose too low</td>
<td>Which Insulin dose to be reduced</td>
<td>21h00 dose: intermediate acting insulin</td>
<td>Breakfast dose: rapid (or short acting) insulin</td>
<td>Lunch dose: rapid (or short acting) insulin</td>
<td>Supper dose: rapid (or short acting) insulin</td>
</tr>
</tbody>
</table>

### Regimen 3

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Timing of the unsatisfactory blood glucose level</th>
<th>Before breakfast</th>
<th>Before lunch</th>
<th>Before supper</th>
<th>At ± 21h00</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose too high</td>
<td>Which Insulin dose to be increased</td>
<td>21h00 dose: intermediate acting insulin</td>
<td>Breakfast dose: rapid (or short acting) insulin</td>
<td>Lunch dose: rapid (or short acting) insulin</td>
<td>Supper dose: rapid (or short acting) insulin</td>
</tr>
<tr>
<td>Glucose too low</td>
<td>Which Insulin dose to be reduced</td>
<td>21h00 dose: intermediate acting insulin</td>
<td>Breakfast dose: rapid (or short acting) insulin</td>
<td>Lunch dose: rapid (or short acting) insulin</td>
<td>Supper dose: rapid (or short acting) insulin</td>
</tr>
</tbody>
</table>

**Total daily Insulin dose:**

This is individualised and varies according to age, puberty development, stress and individual variability. Usual range is 0.5 – 1 units/kg/day, but may be higher or less.

**REFERRAL**

- management of all children with diabetes should be supervised by a paediatrician and under ideal circumstances should involve a multidisciplinary team, i.e. paediatrician, dietician, nurse educator, psychologist, etc. at a district or regional hospital
- complications
- periodic screening of eyes by an ophthalmologist:
  - prepubertal onset of diabetes: 5 years after onset or at age 11 years, or at puberty (whichever is earlier), and annually thereafter
  - pubertal onset of diabetes: 2 years after onset and annually thereafter
7.4.1.1 Guidelines for Management of Diabetics on Sick Days

DESCRIPTION
Illness associated with fever tends to raise blood glucose because of higher levels of stress hormones, gluconeogenesis and insulin resistance.
Illness associated with vomiting and/or diarrhoea may lower blood glucose, with the possibility of hypoglycaemia.
Illness may result in ketone production.

DIAGNOSTIC CRITERIA
- unstable blood glucose measurements as a result of illness or stress
- increased insulin requirements are induced by a catabolic state and stress
- ketonuria may also indicate the need for extra insulin
  - ketonuria in the presence of hyperglycaemia is indicative of severe insulin deficiency and calls for urgent therapy to prevent progression into ketoacidosis
  - ketonuria in the presence of low blood glucose levels is indicative of a starvation state or is the result of a counter-regulatory response to hypoglycaemia

NON-DRUG TREATMENT
- monitor glucose more frequently
- test urine for ketones
- ensure adequate intake of calories on sick days to prevent ketogenesis. If not enough calories are consumed, ketones will appear in the urine without the development of hyperglycaemia. In this circumstance, it is appropriate to encourage the patient to eat whatever he/she feels like.
- treat underlying intercurrent illness
- special circumstances:
  - Gastroenteritis
    - if hypoglycaemia occurs especially with gastroenteritis, and there is mild ketonuria, ensure that the child takes regular frequent amounts of carbohydrate using oral rehydration solution or intravenous fluids
  - Loss of appetite
    - replace meals with easily digestible food and sugar-containing fluids
  - Vomiting
    - if the patient has difficulty eating or keeping food down and the blood glucose is below 10 mmol/L, the patient should be encouraged to take sugar-containing liquids. Small volumes should be given. Some glucose will be absorbed. If there is no vomiting, increase the amount of liquid.

DRUG TREATMENT
Insulin therapy
Insulin must always be given each day. – Do not skip an insulin injection because of sickness and/or vomiting. If vomiting occurs, IV fluids may be needed to avoid hypoglycaemia. Generally the body will require more energy during illness. More insulin allows more glucose to enter into the cells, providing more energy to fight infection.
General guidelines when giving extra insulin:
• if the blood glucose is rising or if there are ketones in the urine increase, the patient must seek urgent medical attention.

Moderate urine ketones
• the extra dose of insulin is usually 10–20% of the total daily dose
  This extra insulin is given as short (or rapid) acting insulin every three hours.
  If the blood glucose drops below 8.3 mmol/L, it may be necessary to sip regular juice or other sugared drinks. This is done to raise the blood glucose before giving the next insulin injection.

Large amount of urine ketones
• give 20% of the total daily insulin dose
  Repeat as above if necessary.

Extra fluids
In addition to taking extra insulin, extra fluids, e.g. water and fruit juices are important in the prevention of acidosis. These fluids replace the fluids lost in the urine and prevent dehydration.

REFERRAL
• In a child with intercurrent illness urgent specialist medical or nursing advice must be obtained when:
  ○ patient is unable to carry out the advice regarding sick days
  ○ the diagnosis is unclear
  ○ vomiting is persistent, particularly in young children
  ○ blood glucose continues to rise despite increased insulin
  ○ hypoglycaemia is severe – see grading of hypoglycaemia
  ○ ketonuria is heavy or persistent
    • the child is becoming exhausted, confused, hyperventilating, dehydrated or has severe abdominal pain

7.4.2 DIABETES MELLITUS, INSULIN DEPENDENT: ACUTE COMPLICATIONS

7.4.2.1 Cerebral Oedema in Diabetic Coma

DESCRIPTION
A condition of brain swelling during the course of treatment of hyperglycaemic coma. Cerebral oedema usually occurs 4–12 hours after the initiation of treatment and often follows an initial period of clinical and biochemical improvement.

Cerebral oedema causes significant neurological morbidity and has a mortality of approximately 80%.

The cause of cerebral oedema during treatment remains unclear. However, too rapid reduction in intravascular osmolality may aggravate the process. Therefore rehydration should occur more slowly in children with DKA than in other causes of dehydration.
CHAPTER 7

ENDOCRINE SYSTEM

DIAGNOSTIC CRITERIA

Clinical

• signs and symptoms of cerebral oedema include:
  ○ headache
  ○ irritability
  ○ small pupils
  ○ slowing pulse
  ○ respiratory impairment

• the risk of cerebral oedema is increased if the $\text{PCO}_2$ is persistently low, i.e. < 20 mmHg or urea levels are increased

NON-DRUG TREATMENT

• admit to ICU, if possible
• restrict intravenous fluids to $\frac{2}{3}$ maintenance and replace deficit over 72 hours rather than 48 hours pending ICU admission
• exclude hypoglycaemia
• refrain from using bicarbonate
• exclude thrombosis, haemorrhage or infection

Do not delay treatment while waiting for a CT scan to confirm cerebral oedema.

DRUG TREATMENT

• mannitol 20%, IV, 2.5 mL/kg, immediately over 15 minutes
  This needs to be given within 10 minutes.

7.4.2.2 Hyperglycaemic Ketoacidosis
E10.1

DESCRIPTION

Diabetic ketoacidosis occurs with relative or absolute insulin deficiency, either caused by non-compliance with insulin regimens or by excessive secretion of counterregulatory hormones during stress, e.g. infection, trauma and surgery.

DIAGNOSTIC CRITERIA

• heavy glycosuria
• hyperglycaemia, i.e. blood glucose usually > 15 mmol/L and ketonuria pH < 7.3
• bicarbonate <15 mmol/L AND who are 5% or more dehydrated
• ± vomiting
• ± drowsiness

Note:
In rare cases blood glucose is not elevated.
Children < 5% dehydrated and not clinically unwell usually tolerate oral rehydration and subcutaneous insulin.
See Guidelines For Sick Day Management: Section 7.4.1.1
NON-DRUG TREATMENT
- admit all children and adolescents to an ICU or ward experienced in the management of DKA in children and adolescents, if possible
- ensure that the airway is patent
- if the child is comatose, insert an artificial airway and a urinary catheter
- if comatose or recurrent vomiting insert oro/nasogastric tube and apply free drainage
- oxygen via facemask or airway

DRUG TREATMENT
The objectives of fluid and sodium replacement therapy in diabetic ketoacidosis are:
- restoration of circulating volume
- replacement of sodium and extracellular fluid and intracellular fluid deficits of water
- the restoration of glomerular filtration rate with enhanced clearance of glucose and ketones from the blood
- to reduce the risk of cerebral oedema

Fluids
For resuscitation in shock
- sodium chloride 0.9%, IV, 10–20 mL/kg over 10–30 minutes. Repeat if peripheral pulses remain poor.

Fluid requirements after resuscitation

<table>
<thead>
<tr>
<th>CALCULATION OF FLUID REQUIREMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid requirement = deficit + maintenance</td>
</tr>
<tr>
<td>Calculate deficit = estimated % dehydration x body weight (kg and equivalent in mL)</td>
</tr>
<tr>
<td>Calculate maintenance (mL):</td>
</tr>
<tr>
<td>≤1 year</td>
</tr>
<tr>
<td>All children older than 1 year – the sum of the following:</td>
</tr>
<tr>
<td>• first 10 kg body weight</td>
</tr>
<tr>
<td>• second 10 kg body weight</td>
</tr>
<tr>
<td>• additional weight greater than 20 kg body weight</td>
</tr>
</tbody>
</table>

Add deficit to 48-hour maintenance and replace this volume evenly over 48 hours with sodium chloride initially.

Assess hydration status every 4–6 hourly

When blood glucose falls to 12–15 mmol/L the infusion should be changed to a dextrose-containing maintenance fluid, e.g. dextrose 5% in sodium chloride 0.45% or 0.9%.

Bicarbonate
Bicarbonate use is associated with increased risk of cerebral oedema.

CAUTION
Bicarbonate should never be given without prior discussion with a specialist
Potassium
Potassium replacement should be commenced immediately unless anuria is present. Early addition of potassium in the fluid regimen (20–40 mmol/L) is essential even if the serum concentration is normal as insulin will drive glucose and potassium into the cells.

<table>
<thead>
<tr>
<th>Serum potassium (mmol/L)</th>
<th>Potassium supplement mmol/L of fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3</td>
<td>40</td>
</tr>
<tr>
<td>&gt; 3–4</td>
<td>30</td>
</tr>
<tr>
<td>&gt; 4–5</td>
<td>20</td>
</tr>
<tr>
<td>&gt; 5–6</td>
<td>10</td>
</tr>
<tr>
<td>&gt; 6</td>
<td>none</td>
</tr>
</tbody>
</table>

DKA protocol: Two-bag system – Alternative fluid and electrolyte treatment
Under supervision of a specialist.
The two-bag system consists of 2 bags of identical electrolyte content but different dextrose concentrations, 0% and 10%, administered simultaneously into a single IV line. Variations in dextrose delivery are achieved through differential proportions of the 2 bags contributing to the total rate, which is determined by the patient’s degree of dehydration.

- sodium chloride 0.9%, IV, 10–20 mL/kg
  May be repeated if necessary.
Then switch to “two bag” system

<table>
<thead>
<tr>
<th>BAG 1 (dextrose 0%)</th>
<th>BAG 2 (dextrose 10%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>sodium chloride 0.45%, 1 L</td>
<td>dextrose 10%, 1 000 mL</td>
</tr>
<tr>
<td>potassium chloride, 20 mL</td>
<td>sodium chloride 5%, 90 mL</td>
</tr>
<tr>
<td></td>
<td>potassium chloride, 20 mL</td>
</tr>
</tbody>
</table>

Run these two riders for easy titration of dextrose from dextrose 10% to dextrose 0%

<table>
<thead>
<tr>
<th>Fluid</th>
<th>Blood glucose &gt;15</th>
<th>Blood glucose 10–15</th>
<th>Blood glucose &lt;10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bag 1</td>
<td>100%</td>
<td>50%</td>
<td>0%</td>
</tr>
<tr>
<td>Bag 2</td>
<td>0%</td>
<td>50%</td>
<td>100%</td>
</tr>
</tbody>
</table>

Insulin
- insulin short-acting, 0.1 unit/kg/hour as a continuous IV infusion
  Add insulin, 50 units (0.5 mL) to sodium chloride 0.9%, 50 mL in a syringe pump to get a solution of 1 unit/mL.
  Attach this using a Y-connector to the IV fluids already being administered.
  Do not add insulin directly to the fluid bags.
  The solution should then be administered at a rate of 0.1 mL/kg/hour (0.1 unit/kg/hour).
If the rate of blood glucose fall exceeds 5 mmol/L/hour, or the blood glucose falls to 14 mmol/L,
  ○ add a dextrose-containing fluid
    Do not stop the insulin infusion while dextrose is being infused.
    If the blood glucose falls below 4 mmol/L, give a bolus of 2 mL/kg of dextrose 10% and increase the concentration of dextrose in the infusion.
Continue with IV insulin until:
  ▪ base deficit is < 5 or bicarbonate is 15 mmol/L
  ▪ there is no ketonuria (or ketonemia if you can measure it)
  ▪ blood glucose is 10 mmol/L

When syringe pumps are not available a separate low-dose infusion may be given, e.g.
• insulin short-acting, 50 units in sodium chloride 0.9%, 500 mL
  i.e. 1 unit insulin per 10 mL sodium chloride
  Change bag every 24 hours to avoid inactivation of insulin.

**Alternative to insulin infusion**
Where there are no facilities for insulin infusion, e.g. no syringe pumps, staff constraints, etc.
• insulin short-acting, IM, 0.1 unit/kg, hourly if not in shock
  If patient in shock – give IV 0.1 unit/kg, hourly
  Increased, hourly contact with the patient may be of advantage.

**Changing from intravenous to subcutaneous insulin**
Intravenous fluids should be continued until the child is drinking well and able to tolerate snacks.
When oral fluids are tolerated intravenous fluids should be reduced.
Subcutaneous insulin can be started once the child is well hydrated and able to tolerate a normal diet. The most convenient time to change to subcutaneous insulin is just before a mealtime. During the changeover, the insulin infusion should be continued after the meal for a total of 90 minutes after the subcutaneous insulin injection to prevent rebound hyperglycaemia.
In newly diagnosed diabetics regimen 1 is chosen at a low range dose (total daily dose of insulin being 0.7 units/kg and 1 unit/kg in prepubertal and pubertal children respectively, divided in the usual way.
In established diabetics, give usual insulin.
Supplemental subcutaneous short acting insulin is given before meals if the blood glucose exceeds 11 mmol/L:

<table>
<thead>
<tr>
<th>Blood glucose</th>
<th>Short-acting insulin (units/kg/dose)</th>
</tr>
</thead>
<tbody>
<tr>
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<td>0.09</td>
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<td>16</td>
<td>0.12</td>
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</tbody>
</table>
REFERRAL
- no improvement
- deterioration of condition, i.e.:
  - pH < 7.1
  - hyperventilation
  - shock
  - depressed level of consciousness
  - persistent vomiting
  - age < 5 years
- rising blood glucose

7.4.2.3 Hypoglycaemia in Diabetics
E16.0

DESCRIPTION
In hypoglycaemia the level of blood glucose is so low that neurological dysfunction occurs.

Neuroglycopaenia (impaired thinking, change of mood, irritability, dizziness, headache, tiredness, confusion, and later convulsions and coma) may occur before autonomic activation (causing hypoglycaemia unawareness).

Causes of hypoglycaemia include:
- a missed or delayed snack or meal
- exercise without appropriate dietary preparation
- alcohol
- overdose of insulin
- impaired food absorption e.g. gastroenteritis
- Addison’s disease - recurrent hypoglycaemia may necessitate investigation for this condition

Nocturnal hypoglycaemia
Nightmares and headaches may be suggestive of nocturnal hypoglycaemia. Blood glucose concentrations fall to their lowest levels between 03h00 and 04h00.

DIAGNOSTIC CRITERIA
- blood glucose < 3.5–4 mmol/L with symptoms in a known diabetic patient
  Good glycaemic control is likely to be associated with occasional hypoglycaemic episodes.
- grading of severity:
  Mild (Grade 1)
  - child or adolescent is aware of, responds to and self-treats the hypoglycaemia
  - children under six years can rarely be classified as grade 1 because they are unable to help themselves
  Moderate (Grade 2)
  - child or adolescent cannot respond to hypoglycaemia and requires help from someone else, but oral treatment is successful
  Severe (Grade 3)
  - child or adolescent is semi-conscious or unconscious or in coma with/without convulsions and may require parenteral therapy with glucagon or intravenous glucose
CHAPTER 7

ENDOCRINE SYSTEM

NON-DRUG TREATMENT

- determine underlying cause
- patient education on diabetes and its complications
- if patient is fully alert and conscious, give sugar-containing soft drink and/or snack (carbohydrate)
- monitor blood glucose every 15 minutes until blood glucose is 6–8 mmol/L

DRUG TREATMENT

Mild or moderate hypoglycaemia,

immediate oral rapidly absorbed simple carbohydrate, e.g.

- glucose, oral, 5–15 g
  - Wait 10–15 minutes.
  - If no response, repeat above.
  - As symptoms improve, the next meal or oral complex carbohydrate should be ingested, e.g. fruit, bread, cereal, milk, etc.

Severe hypoglycaemia

Outside hospital

- glucagon, IM/SC, 0.1–0.2 mg/10 kg
  - < 12 years  0.5 mg
  - > 12 years  1.0 mg

In hospital

If there is an unsatisfactory response or inability to take oral carbohydrate and signs of disorientation, stupor, convulsions, coma

- dextrose 10%, IV, 2–5 mL/kg
  - OR
  - dextrose 50%, IV, 0.5 mL/kg.
    - Dilute dextrose 50% solution before use to 10% strength
    - 0.5–1 mL of dextrose 50% = 250–500 mg
    - OR
    - 2.5 mL of dextrose 10% = 250 mg

If IV dextrose cannot be given

- glucagon, IM/SC, 0.1–0.2 mg/10 kg
  - < 12 years  0.5 mg
  - > 12 years  1.0 mg
  - Monitor blood glucose every 15 minutes until stable, then repeat 1–2 hourly.
  - Keep blood glucose between 6 and 8 mmol/L.
7.4.2.4 Nephropathy
N08.3

**DIAGNOSTIC CRITERIA**
- albumin/creatinine ratio in the first voided morning urine sample
  - The upper limit of the normal reference range in early morning urine is 1.5 mg albumin/mmol of creatinine.

<table>
<thead>
<tr>
<th>albumin/creatinine ratio</th>
<th>diagnostic action</th>
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</thead>
<tbody>
<tr>
<td>1.5–3.5 mg/mmol</td>
<td>check albumin/creatinine ratio annually</td>
</tr>
<tr>
<td>&gt; 3.5 mg/mmol</td>
<td>timed collection of urine to confirm AND check albumin/creatinine ratio six monthly</td>
</tr>
<tr>
<td>repeated values &gt; 3.5 mg/mmol</td>
<td>requires therapy</td>
</tr>
</tbody>
</table>

**DRUG TREATMENT**
If albumin/creatinine ratio is greater than 3.5 mg/mmol
ACE inhibitor, e.g.:
- enalapril, oral, 0.5 mg/kg/dose as a single dose or two divided doses

**Note:**
Exclude non-diabetic nephropathy.

**REFERRAL**
- all patients with significant proteinuria

7.4.3 DIABETES MELLITUS IN ADOLESCENTS
E10

**DESCRIPTION**
Adolescence is that period between puberty and when the patient leaves school to join the workforce. The adolescent and the transition should be managed with special planning, i.e.:
- the admission policy of the hospital
- the wishes of the adolescent
- emotional and physical maturity
- presence of any coexisting medical, surgical or psychiatric disorder that may be more appropriately managed in the paediatric service

**NON-DRUG TREATMENT**
- promotion of:
  - normal growth and pubertal development
  - psychological development
  - maintenance of glycaemic control and adherence
  - normal lifestyle
  - avoidance of risk taking behaviours (smoking, substance abuse)
  - sex education
DRUG TREATMENT
Failure of current insulin regimens are attributed to the endocrine changes of puberty which results in poor glycaemic control.
Insulin resistance occurs during puberty, being maximal in late puberty. The insulin dose should be increased in line with requirements and may reach 1.5–2.0 units/kg/day. After puberty, the insulin requirements fall to prepubertal levels. Failure to reduce insulin requirements in the late adolescent stages may result in aggressive weight gain.

7.4.4 DIABETES MELLITUS, (TYPE 2)

DESCRIPTION
Type 2 diabetes in adolescents is becoming increasingly prevalent with the increase in the incidence of obesity. It is characterised by varying degrees of insulin resistance.

DIAGNOSTIC CRITERIA
Clinical
- symptoms of diabetes plus random plasma glucose above 11 mmol/L or a fasting glucose greater than 7 mmol/L
- type 2 diabetes may have minimal symptoms or signs for months or even years before the diagnosis

Investigations
- 2–hour post prandial glucose of ≥ 11 mmol/L on an oral glucose tolerance test. An oral glucose tolerance test requires the equivalent of 1.75 g/kg to a maximum of 75 grams of glucose dissolved in water.
- screening. Only children at substantial risk for the presence or development of type 2 diabetes should be tested.
  - when routine urine test strips shows glycosuria
  - if an individual is overweight, i.e.
    - BMI > 85th percentile for age and sex, or
    - weight for height > 85th percentile, or
    - > 120% of ideal weight for height
  AND
  - has the following risk factors:
    - family history of type 2 diabetes in first or second degree relatives
    - signs of insulin resistance or conditions associated with insulin resistance, e.g. acanthosis nigricans, hypertension, dyslipidaemia or polycystic ovary syndrome

NON-DRUG TREATMENT
- lifestyle modification
  Patients who are not ill at diagnosis can be managed initially with advice on nutrition and exercise, but most will eventually require drug therapy.
- education on routine blood glucose monitoring. A daily record of all testing performed should be recorded in a logbook. Record prebreakfast fasting and 2–hour postprandial dinner levels which is sufficient in most cases.
**DRUG TREATMENT**
Refer for initiation of therapy.

Biguanides, e.g.:
- metformin, oral, 500 mg twice daily (adolescent dose)
  Contraindications include:
  - renal failure
  - hepatic disease
  - hypoxaemia e.g. in severe respiratory disease
  - severe infection
  - alcohol abuse

If monotherapy fails, alternatives include sulfonylurea or addition of insulin.

If albumin/creatinine ratio is greater than 3.5 mg/mmol
ACE inhibitors, e.g.:
- enalapril, oral, 0.5 mg/kg/dose as a single dose or two divided doses

**Hyperlipidaemia**
See Section 4.8.

**7.5 HYPOGLYCAEMIA IN OLDER CHILDREN**

**DESCRIPTION**
Infants and small children have relatively limited glycogen stores with larger brain/body ratios than adults and are therefore at greater risk of hypoglycaemia during starvation.
The causes of hypoglycaemia (outside the neonatal period) include:

- hypopituitarism
- growth hormone deficiency
- glucagon deficiency
- inborn errors of metabolism
- malnutrition
- liver dysfunction
- accelerated starvation (ketotic hypoglycaemia)
- drugs, e.g. alcohol, aspirin, β-blocker, oral hypoglycaemic agents, sulphonylureas, quinine

**DIAGNOSTIC CRITERIA**

**Clinical**
- acute autonomic symptoms: sweating, pallor, tachycardia, abdominal pain and headache
- neuroglycopenic symptoms: confusion, altered level of consciousness, convulsions
- seriously ill patients often asymptomatic
Investigations
- plasma glucose concentrations less than 2.6 mmol/L
- any blood glucose value less than 4 mmol/L with symptoms

Although hypoglycaemia is a clinical emergency requiring prompt therapy, wherever possible, a blood sample for investigation should be drawn prior to the administration of glucose
- collect 5 mL of blood in a plain tube at the earliest opportunity and send for separation and storage of plasma at −20°C.
Such samples may provide clear biochemical evidence of the cause of the hypoglycaemic episode thus avoiding having to subject the child to further investigations.

DRUG TREATMENT
After collection of initial blood samples
- dextrose 10%, IV, 2–4 mL/kg over 4–6 minutes followed by an infusion at an initial rate of 6 mL/kg/hour, i.e. 10 mg/kg/minute
  Dilute 50% dextrose solution before use.
  250 mg/kg = 0.5 mL/kg of 50% dextrose

If the patient remains unconscious despite normalisation of the blood glucose concentrations, in case of undiagnosed adrenal insufficiency
- hydrocortisone, IV, 2–3 mg/kg, immediately

Stabilisation
- dextrose 5% in sodium chloride 0.9%, IV, 20 mL/kg bolus as needed
  OR
  Ringer-Lactate with dextrose 5%, IV, 20 mL/kg bolus as needed
  OR
  dextrose 10%, IV, 2–3 mL/kg glucose as needed

- hydrocortisone, IV, 2–3 mg/kg immediately, then 2–3 mg/kg/day every six hours

Ongoing treatment
Intravenous fluid therapy as needed.

Manage hyperkalaemia
See Section 7.8

REFERRAL
- all patients with confirmed hypoglycaemia not explained by intercurrent illness, drugs or other disease
- persisting or recurrent hypoglycaemia
7.6 GROWTH DISORDERS
R62

DESCRIPTION

Constitutional delay in growth
Bone age is significantly delayed compared to chronological age.

Common features of constitutional delay in growth and familial short stature include:
- short for chronological age
- normal rate of linear growth
- no decline in height percentile

Familial short stature
Bone age equivalent to chronological age.

DIAGNOSTIC CRITERIA
- measurement and plotting of a child’s height and weight on growth charts
  Routine monitoring of height and weight for growth assists in the diagnosis of problems
  which would otherwise be missed or would come to light at a stage where the outcome
  of treatment may be less favourable.

NON-DRUG TREATMENT
- identify non-endocrine causes of stunted growth before referral, e.g.:
  - intra-uterine growth retardation
  - chronic disease
  - psychosocial deprivation
  - skeletal dysplasia and other dysmorphic syndromes

REFERRAL
- child is more than three standard deviations below the mean
- patient’s height significantly below target height
- subnormal height velocity
- history of chronic disease
- dysmorphic syndrome
- endocrine causes of stunted growth
- for consideration of drug therapy

7.7 HYPOCALCAEMIA IN OLDER CHILDREN
E83.5

DESCRIPTION
The main causes of hypocalcaemia in older children are:
- vitamin D deficiency
- calcium deficiency
- reduced parathyroid hormone production
- impaired renal function
DIAGNOSTIC CRITERIA

Clinical
- signs and symptoms include:
  - paraesthesia
  - cramps
  - tetany
  - weakness
  - lethargy
  - positive Chvostek’s sign
  - positive Trousseau’s sign
  - convulsions
  - laryngospasm
  - prolonged QT interval on the ECG

DRUG TREATMENT

Acute hypocalcaemia
- calcium gluconate 10%, IV, 1–2 mL/kg over 5–10 minutes 6–8 hourly
  - Maximum dose: 10 mL.
  - Electrocardiographic monitoring is advised.

Chronic therapy
Long-term therapy depends on the cause.
Manage hypophosphataemia or hyperphosphatemia, depending on the cause of hypocalcaemia, before long-term calcium is initiated.
- elemental calcium, oral, 30 mg/kg/day (calcium carbonate tablets; 1 tablet is equivalent to 0.168 g elemental calcium)
  - If vitamin D deficient
  - vitamin D, oral, 5000 IU/day

For hypoparathyroidism and pseudohypoparathyroidism
- calcitriol, oral, 0.01–0.04 mcg/kg/day
  - OR
  - alfacalcidol, oral, 0.05 mcg/kg/day
    - < 20 kg 0.05 mcg/kg/day
    - > 20 kg 1 mcg/day

REFERRAL
- chronic hypocalcaemia

7.8 HYPERKALAEMIA
E87.5

DESCRIPTION
Serum potassium > 5.5 mmol/L
- pseudohyperkalaemia – haemolysed blood samples, lysis of leukocytes
- decreased renal excretion – renal failure
- drugs – potassium sparing diuretics, digitalis, β-blockers, ACE inhibitors and trimethoprim/sulphamethoxazole
- increased potassium load
- transmembrane shifts
- acidosis
**DIAGNOSTIC CRITERIA**
- generalised signs of hyperkalaemia:
  - weakness
  - paraesthesia
  - cardiac arrhythmias
- history of drugs, renal or adrenal disease

**NON-DRUG TREATMENT**
- reverse causative factors
- eliminate all sources of potassium

**DRUG TREATMENT**

*To reduce potassium acutely*
- salbutamol, solution, 2.5–5 mg (maximum 5 mg/dose), nebulise over 20 minutes
  - 5 mg salbutamol in 2–4 mL sodium chloride 0.9%
- calcium gluconate 10%, IV, 0.5 mL/kg over 3–5 minutes

*If adrenal insufficiency is suspected*
- hydrocortisone, IV, 2–3 mg/kg, immediately

*Correct acidosis*
- sodium bicarbonate 4.2%, IV, 4 mL/kg over 5–10 minutes

*Glucose/Insulin infusion*
- dextrose 50%, IV, 2 mL/kg over 20 minutes ± insulin, 0.1 unit/kg
  Check for hypoglycaemia hourly if insulin is used.

*To increase potassium elimination/excretion*
- sodium polystyrene sulfonate, oral/rectal, 1 g/kg
- furosemide, IV, 1 mg/kg
- dialysis

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**7.9 HYPOKALAEMIA**

**DESCRIPTION**
Causes include:
- prolonged decreased intake and protein energy malnutrition
- increased renal excretion - renal tubular acidosis, amphotericin B and diuretics
- increased extrarenal losses
- transmembrane shifts – β₂ stimulants, alkalosis
- mineralocorticoid excess
DIAGNOSTIC CRITERIA
Clinical
- cardiac arrhythmias, especially with digitalis
- neuromuscular dysfunction, e.g. muscle weakness
- haemolysis
- renal – impairment of urine concentrating or diluting ability

Investigations
- serum potassium < 3.0 mmol/L

DRUG TREATMENT
See Acute Diarrhoea: Section 2.2.4

Severe respiratory paralysis and or cardiac arrhythmias
- potassium chloride, IV, < 1 mEq/kg/hour
  Electrocardiac and potassium monitoring.
  Potassium concentration should not exceed more than 40 mmol/L/infusion.
  Never give potassium as an IV bolus.

Less critical situations
- potassium chloride, oral, 2–6 mEq/kg/day to correct potassium deficit over 2–3 days

If hypomagnesaemia present
- magnesium, oral, 24–48 mg/kg/day

7.10 HYPOPITUITARISM
E23.0

DESCRIPTION
Multiple or isolated deficiencies of adrenocorticoid hormone (ACTH), luteinising hormone, thyroid stimulating hormone, and growth hormone manifesting as hypoglycaemia, abnormal body proportions and failure to grow and develop.
The deficiency may be due to:
- congenital abnormalities with/without midline structural abnormalities of the brain
- central nervous system tumours
- histiocytosis
- complications of radiation therapy

DIAGNOSTIC CRITERIA
Clinical
- neonates with hypopituitarism may present with:
  - persistent hypoglycaemia
  - cholestatic jaundice (related to low cortisol)
  - micropenis
- growth failure with immature body proportions
Investigations
• endocrine evaluation with pituitary function tests under specialist supervision
• diagnosis may be confirmed in older children with stimulation tests

DRUG TREATMENT
To correct hypoglycaemia
• hydrocortisone, IV, 1–2 mg/kg

REFERRAL
• all patients after stabilisation of hypoglycaemia

7.11 HYPOTHYROIDISM, NEONATAL

DESCRIPTION
Congenital deficiency of thyroid hormone due to aplasia/hypoplasia of the thyroid gland, defects in thyroid hormone biosynthesis or intrauterine exposure to antithyroid drugs. Congenital hypothyroidism is one of the common treatable causes of preventable mental retardation in children. Congenital hypothyroidism must be treated as early as possible to avoid intellectual impairment.

DIAGNOSTIC CRITERIA
Clinical
• prolonged unconjugated hyperbilirubinaemia
• feeding difficulties
• lethargy
• somnolence
• apnoeic episodes
• poor cry
• constipation
• wide open fontanels
• enlarged tongue
• short and thick neck
• dry skin
• hypotonia
• delayed physical and mental development
• oedema of the extremities and genitals
• bradycardia
• anaemia
• nasal obstruction
• abdominal distension
• umbilical hernia
• subnormal temperature
• periorbital oedema
• delayed dentition
• broad hands
• hair coarse and scanty
• hoarse voice and goitre

Investigations
• cord blood or serum sample/dried blood spot after day 4: Low $T_4$ and/or elevated TSH
• Any of the above clinical features: Blood sample to identify low $T_4$ and/or high TSH

NON-DRUG TREATMENT
• growth and neurodevelopmental assessment
• regular follow up is necessary
CHAPTER 7

DRUG TREATMENT
Neonates and infants
• levothyroxine, oral, 10–15 mcg/kg as a single daily dose
  Dosage must be adjusted to blood levels of $T_4$ and TSH and decreases with increase in age.
  Treatment is continued indefinitely.

REFERRAL
• all patients for confirmation of diagnosis and initiation of therapy

7.12 HYPOTHYROIDISM IN OLDER CHILDREN AND ADOLESCENTS
E03.9

DESCRIPTION
Acquired hypothyroidism in childhood and adolescents may be due to:
• chronic lymphocytic thyroiditis
• goitrogen induced
• iodine deficiency
• post surgery
• radioactive iodine
• infiltrations, or
• medicines, e.g. antiretrovirals

DIAGNOSTIC CRITERIA
• elevated TSH and low thyroxine levels

DRUG TREATMENT
• levothyroxine, oral, 100 mcg/m$^2$ once daily

REFERRAL
• all cases for investigation and initiation of therapy

7.13 HYPERTHYROIDISM, GRAVES DISEASE
E05.8

DESCRIPTION
Hyperthyroidism is a pathological syndrome in which tissue is exposed to excessive amounts of circulating thyroid hormones. The most common cause is Grave’s disease, although thyroiditis may also present with thyrotoxicosis.
DIAGNOSTIC CRITERIA
Clinical
• fatigue
• nervousness or anxiety
• weight loss
• palpitations
• heat insensitivity

Investigations
• high thyroxine ($T_4$) and suppressed TSH

DRUG TREATMENT
• carbimazole, oral, 0.5 mg/kg once daily
AND
• atenolol, oral, 1–2 mg/kg as a single daily dose

REFERRAL
• all patients for confirmation of diagnosis, initiation and follow up of therapy

7.14 LIPODYSTROPHY/ENDOCRINOPATHIES IN HIV INFECTED CHILDREN
E88.1

DESCRIPTION
Long term survivors with HIV infection may develop unique complications of lipid metabolism usually attributable to HAART, particularly protease inhibitors. Some of the risk factors include virologic response to therapy and pubertal development during protease inhibitor therapy. Lipodystrophy can lead to non-adherence with ARV treatment if patient is embarrassed by his/her physical appearance.

DIAGNOSTIC CRITERIA
• the three main components of lipodystrophy include:
  ○ body fat redistribution
  ○ insulin resistance
  ○ abnormal lipid metabolism

Clinical
• physical features include:
  ○ fat wasting (lipoatrophy) of the face, extremities or buttocks
  ○ fat accumulation (lipohypertrophy) in the abdomen, or over the dorsocervical spine (buffalo hump)
  ○ excessive breast enlargement during puberty.
• insulin resistance may be suspected if there is:
  ○ fasting hyperglycaemia
  ○ frank diabetes or acanthosis nigricans
  ○ biochemical features include an elevated fasting C–peptide or an abnormal glucose/insulin ratio
• abnormal lipid profile – See Dyslipidaemia: Section 4.8
  ○ hypercholesterolaemia, i.e total cholesterol level > 5 mmol/L
  and
  ○ hypertriglyceridaemia, i.e. fasting triglyceride level > 1.7 mmol/L
  The likely consequences are premature atherosclerosis.
NON-DRUG TREATMENT
- dietary modification and exercise

DRUG TREATMENT
- modification of anti-retroviral therapy, e.g. substitute another drug
- lipid lowering agents if hyperlipidaemia is confirmed

REFERRAL
- all patients for confirmation of diagnosis and initiation of therapy

7.15 OBESITY

DESCRIPTION
Most children with obesity do not have an underlying pathological cause and have so-called "simple obesity", i.e. both weight and height are increased. In children with pathological obesity, the height is not usually increased. Causes of pathological obesity include syndromes, hypothalamic damage, endocrine abnormalities, immobility, impaired skeletal growth or drugs.

There has been a dramatic increase in the prevalence of childhood overweight and its resultant comorbidities.

DIAGNOSTIC CRITERIA
Clinical
- measurement of weight alone is inadequate given the influence of height on weight
- severity may be assessed using body mass index (BMI)
  - body mass index = \( \frac{\text{weight (kg)}}{\text{height}^2 (m^2)} \)

The BMI varies with age. Sex-specific BMI charts must be used for accurate identification of obesity. In general, if the BMI exceeds 19 at age 5 years, 20 at age 10 years, and 25 at age 18 years, a diagnosis of obesity is likely.

Investigations
- fasting glucose and lipid profile
- alanine aminotransferase

NON-DRUG TREATMENT
- weight control by:
  - education about the nature of obesity and its longer term consequences
  - healthy eating, e.g. regular meal times, avoidance of excessive "snacking", fried foods, added fats and sugars and high energy drinks while encouraging foods with high fibre content, with modest calorie restriction
  - increasing physical activity
  - reduce sedentary time, e.g. TV watching, computer games, videogames or time on the telephone
  - psychological support
For many obese children, weight loss down to an “ideal body weight for height” is probably unrealistic. Nevertheless, prevention of further weight gain may produce significant longer-term benefits. If the patient is over 7 years, or if complications are present, aim for a 0.5 kg/month weight loss.

**DRUG TREATMENT**
There is little experience with medicines that have been tested in adults. Their use is not currently recommended in children.

Manage hyperlipidaemia – See Dyslipidaemia: Section 4.8

**REFERRAL**
- all cases of pathological obesity
- severe/progressive obesity < 2 years
- serious co-morbidity requiring weight loss

### 7.16 DISORDERS OF PUBERTY

**DESCRIPTION**
Abnormally early or abnormally late development of signs of puberty including the development of breasts, external genitalia and sexual hair. Often associated abnormality of growth velocity.

**DIAGNOSTIC CRITERIA**
- puberty begins after 9 years and usually not later than 14 years in males
- corresponding ages in girls are 8 years and 13.5 years
- problems occurring outside these ages need investigation

**Investigations**
- puberty staging
- radiological bone age
- endocrine investigation

**NON-DRUG TREATMENT**
- psychological support
- treat the cause, e.g. tumours

**REFERRAL**
- all
8.1 HELMINTHIASIS, INTESTINAL
B82.0

DESCRIPTION
Infestation of the intestine with adult worms. The following species are commonly encountered:

- *Ascaris lumbricoides* (round worm)
- *Enterobius vermicularis* (pin worm)
- *Trichuris trichiura* (whipworm)
- *Ankylostoma duodenale* and *Necator americanus* (hookworm)
- *Taenia saginatum* and *solium* (beef and pork tapeworms)

DIAGNOSTIC CRITERIA
- most infestations are asymptomatic and become apparent with the passage of a worm rectally or orally
- signs and symptoms include:
  - vague abdominal pains
  - diarrhoea
  - rectal prolapse
  - protein losing enteropathy
  - surgical complications of mechanical effects in the bowel, pancreatic duct or biliary tree
  - perianal itch
  - vaginitis
  - iron deficiency anaemia
  - migration of worm larvae may cause cutaneous, pulmonary or cerebral symptoms. See *Neurocysticercosis: Section 13.6.*
- definitive diagnosis is based on recognition of the worm or identification of worm eggs or proglottids in stool

NON-DRUG TREATMENT
- prevent infestation by hand washing
- careful preparation of foods by adequate washing and cooking and by wearing shoes (hookworm)
- improved sanitation will protect the environment from contamination

DRUG TREATMENT
All helminths excluding *Taenia*, *Trichuris* and *Enterobius*

- children under 2 years
  - albendazole, oral, 200 mg immediately as a single dose

- children 2–5 years
  - mebendazole, oral, 100 mg twice daily for three days

- children over 5 years
  - mebendazole, oral, 500 mg immediately as a single dose
**Enterobius**
- mebendazole, oral, 100 mg immediately as a single dose
  
  Repeat after 2 weeks.

**Taenia and Trichuris**
- albendazole, oral for three days
  
  1–2 years   200 mg
  
  > 2 years   400 mg

**REFERRAL**
- abdominal complications requiring specialist assessment

### 8.1.1 CUTANEOUS LARVA MIGRANS/ANCYLOSTOMA BRAZILIENSE (DOG HOOKWORM)
B76.9/B76.0

**DESCRIPTION**
Infestation of the skin by dog hookworm larvae. Maturation of the larvae cannot occur. The infection is self-limiting.

**DIAGNOSTIC CRITERIA**
- presents as an itchy “serpiginous” skin lesion

**NON-DRUG TREATMENT**
- regular deworming of dogs
- wearing shoes to protect against infection

**DRUG TREATMENT**
- albendazole, oral for three days
  
  1–2 years   200 mg
  
  > 2 years   400 mg

### 8.2 HYDATID
B67

**DESCRIPTION**
The development of hydatid (*Echinococcus granulosis*) cysts follows ingestion of worm ova that are usually passed in the stools of dogs in sheep farming areas. Cysts may occur in any organ, but are most commonly found in the liver and lungs.

**DIAGNOSTIC CRITERIA**
- typical radiological features
  
  Diagnostic aspiration of an organ cyst should never be attempted.
NON-DRUG TREATMENT
• prevent infestation by:
  ○ hand washing
  ○ adequate food preparation
• surgical removal of cysts may be indicated

DRUG TREATMENT
• albendazole, oral, 5–7.5 mg/kg/dose 12 hourly for three 28 day cycles with a 14-day interval between cycles

REFERRAL
• all cases for specialist assessment

8.3 SCHISTOSOMIASIS (BILHARZIA)
B65.0/B65.1

DESCRIPTION
Disease manifestations caused by infestation by species of the genus Schistosoma. Infestations with *S. haematobium* and *S. mansoni* are endemic in certain areas of South Africa. Nematodes reside in venous plexus draining bladder wall (haematobium) or intestine (mansoni).

Complications include:
- haematuria
- dysuria
- cystitis
- calcifications in the bladder wall
- obstructive uropathy
- bladder stones
- intestinal perforation
- fistules
- spinal cord granulomas with pressure effects

DIAGNOSTIC CRITERIA
Clinical
- transient pruritic papular rash (swimmers itch) after exposure to cercariae in the water
- a few weeks after exposure:
  ○ fever
  ○ chills
  ○ headache
  ○ urticaria
  ○ cough
- haematuria and dysuria
- abdominal pain and diarrhoea often with food
Investigations
- positive serological tests for schistosomiasis
- viable eggs in urine, stools or rectal biopsy specimens

NON-DRUG TREATMENT
- educate patient/caregiver on preventative measures
- symptomatic and supportive treatment
- avoid exposure to water contaminated by schistosoma
- surgical intervention to correct or prevent complications

DRUG TREATMENT
- praziquantel, oral, 40 mg/kg/24 hours as a single dose or in 2 divided doses on the same day

REFERRAL
- schistosomiasis with suspected complications following adequate therapy

8.4 CANDIDIASIS, SYSTEMIC AND OTHER

DESCRIPTION
Superficial and/or disseminated (systemic) fungal infection caused by *C. albicans*, *C. tropicalis*, other candida species or the closely related *Torulopsis*.

Risk factors include:
- prolonged, broad-spectrum antibiotic therapy
- compromised immune system, including patients infected with HIV or on cancer chemotherapy, and the premature baby
- steroid therapy
- diabetes mellitus
- IV hyperalimentation – may directly contaminate solution or as an associated risk factor
- instrumentation, and central or peripheral vascular catheters

DIAGNOSTIC CRITERIA

Clinical
- oral candidiasis (thrush):
  - white plaque adheres to inner cheeks, lips, palate and tongue
  - stomatitis with red mucosa and ulcers may also be present
  - in immunocompromised patients, the lesions may extend into the oesophagus
- oesophageal candidiasis:
  - presents as difficulty swallowing, drooling or retrosternal pain (irritability)
- intertriginous moniliasis:
  - involves the genitocrural fold, gluteal fold, axillae, neck folds and peri-umbilical area
  - is characterised by a sharply circumscribed, erythematous, moist surface with scattered superficial satellite lesions
• skin lesions in the newborn:
  ○ a red, maculopapular or pustular rash is seen in infants born to women with candida amnionitis
• cutaneous dissemination:
  ○ may be represented by scattered, red papules or nodules
  ○ superficial infections of any moist area, such as axillae or neck folds, are common and may present as an erythematous, intertriginous rash with satellite lesions
• vulvovaginitis and candida balanitis:
  ○ a thick cheesy vaginal discharge with intense pruritus, white plaques on glans of penis
  ○ common in diabetics and patients on broad-spectrum antibiotics
  ○ in recurrent vulvovaginitis, exclude diabetes, foreign body or sexual abuse
• paronychia and onychomycosis:
  ○ usually seen in immunocompromised children
• systemic or disseminated candidiasis
  ○ mimics bacterial sepsis but fails to respond to antibiotics
  ○ thrombocytopenia is common
  ○ ophthalmitis with “cotton wool” retinal exudates may also occur
  ○ is usually nosocomial

Investigations
• for oesophageal candidiasis
  ○ scope or barium swallow. It is reasonable to initiate treatment on clinical grounds
• systemic candidiasis
  ○ urine and blood cultures are essential
• budding yeasts and pseudohyphae are seen on microscopy of biopsy specimens, fluid or scrapings of lesions

NON-DRUG TREATMENT
• eradicate or minimise risk factors
• sterilise pacifiers (dummies), teats and bottles, if possible
• encourage cup feeding
• remove all invasive devices, drain abscesses and debride infected tissue

DRUG TREATMENT
Oral candidiasis
• nystatin suspension 100 000 IU/mL, oral, 1 mL 4 hourly
  Keep in contact with affected areas for as long as possible.
  Suspect immunodeficiency if poor response to treatment.
If no response
Imidazole oral gel, e.g.:
• miconazole gel, oral, apply three times daily
Oesophageal candidiasis
- fluconazole, IV/oral, 6 mg/kg immediately, then 3 mg/kg/day for 3 weeks

Skin infection or diaper rash
- nystatin, topical, 100 000 IU/g, applied three times daily for 14 days
  OR  
  Imidazole topical, e.g.
  clotrimazole or miconazole, topical, applied three times daily for at least 7–14 days

Vulvovaginitis
- fluconazole, oral, 6 mg/kg as a single dose
  OR
  Imidazole topical/vaginal, e.g.
  clotrimazole/miconazole, applied locally at night for 7–14 days
  Do not use applicator in pre-pubertal girls, as this may cause injury to the hymen.

Systemic candidiasis
- amphotericin B, IV infusion, 0.5–1 mg/kg/dose once daily over 4 hours for at least 4 weeks depending on disease response.
  Higher dose if CNS involvement.
  Total dose: 30–35 mg/kg over 4–8 weeks.
  Give initial test dose – see package insert.
  Adjust dose if in renal failure.
  Protected from light during infusion.
  Check serum potassium levels every 5 days.

REFERRAL
- systemic candidiasis
- candidiasis not responding to adequate therapy

8.5 CYTOMEGALOVIRUS (CMV) INFECTION
B25.9

DESCRIPTION
Usually asymptomatic infections but may cause mononucleosis syndrome in children and adolescents.
Congenital infections vary from asymptomatic through isolated neural deafness, to severe disease including microcephaly.
Severe disease can occur in immunocompromised children especially HIV-infected children, e.g. pneumonia, encephalitis, retinitis and gastrointestinal infections.

DIAGNOSTIC CRITERIA
Diagnosis can be difficult as presence of antibodies to CMV does not imply active infection or causality.
- PP65 antigen is a sensitive and specific indicator of systemic infection
• intranuclear inclusion bodies may be seen in biopsy material

REFERRAL
• all cases of severe organ-related disease or disseminated disease

8.6 DIPHTHERIA
A36.9
* Notifiable condition

DESCRIPTION
Diphtheria is an acute, communicable infection of the upper respiratory tract, caused by Corynebacterium diphtheriae. Diagnosis is unlikely if the patient shows documented evidence of complete immunisation. Cutaneous diphtheria can also occur. Incubation period is between 2 and 7 days.

Complications include:
• in the first 2 weeks of the disease:
  o cervical lymphadenopathy with peri-adenitis and swelling of the neck (bull neck)
  o upper airway obstruction by membranes
  o myocarditis
• usually after 3 weeks:
  o neuritis resulting in paresis/paralysis of the soft palate and bulbar, eye, respiratory and limb muscles

DIAGNOSTIC CRITERIA
Clinical
• presents with upper airway obstruction and white to grey adherent pseudomembrane, myocarditis or peripheral neuritis

Investigations
• irregular staining Gram positive pleomorphic bacillus on throat swab
• culture of membrane or throat swab

NON-DRUG TREATMENT
• isolate patient in high or intensive care unit until 3 successive nose and throat cultures at 24-hour intervals are negative
  Usually non-communicable within 4 days of antibiotics.
• nutritional support
• if respiratory failure develops, provide ventilatory support
  Tracheostomy if life-threatening upper airway obstruction.
• bed rest for 14 days

DRUG TREATMENT
Note:
Treatment should not be withheld pending culture results.
**Antibiotic therapy**
- benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 6 hourly for 10 days

**Penicillin allergy**
- erythromycin, IV/oral, 10–15 mg/kg/dose, 6 hourly for 10 days

Close contacts (household and regular visitors):
Regardless of immunisation status, isolate patient and swab throat for culture. Keep under surveillance for 7 days.

All patients
- erythromycin, oral, 12.5 mg/kg/dose 6 hourly for 7 days
  Maximum dose: 1 000 mg/day

OR
If contacts cannot be kept under surveillance
benzathine benzylpenicillin (depot formulation), IM, single dose
- < 30 kg 600 000 units
- > 30 kg 1.2 million units

If 1st culture was positive, follow up throat culture after 2 weeks and retreat
- erythromycin, oral, 12.5 mg/kg/dose 6 hourly for 10 days
  Maximum dose: 1 000 mg/day

**REFERRAL**
- all

**8.7 MALARIA**

B53.8
* Notifiable disease

**DESCRIPTION**
Malaria is transmitted by the bite of an infected female Anopheles mosquito. The incubation period varies with the species of the parasite, *P. falciparum* being shortest, usually 7–21 days, and *P. malariae* the longest. The incubation period may be prolonged by use of malaria prophylaxis or certain antibiotics.

Infection is caused by four species of protozoa of the genus Plasmodium, i.e. *P. falciparum, P. vivax, P. malariae* and *P. ovale.* *Plasmodium falciparum* is the most common and causes the most severe disease.

The confirmation of the diagnosis and treatment of malaria is an emergency. Complications develop rapidly. Malaria can be missed outside transmission areas.

**DIAGNOSTIC CRITERIA**
**Clinical**
- a child living in, or with recent travel history to a malaria transmission area
- fever, which may be intermittent
• flu-like symptoms including sweating or rigors, i.e. cold shaking feeling
• body pains and headache
• occasionally diarrhoea, loss of appetite, nausea and vomiting, tachypnoea and cough
• a young child may present with fever, poor feeding, lethargy, vomiting, diarrhoea or cough

Investigations
• clinical features are non specific and overlap with many other infections
• testing is urgent. Obtain the result immediately.
  ○ rapid diagnostic test, e.g. a plasma reagent dipstick or immunochromatographic test for malaria antigen or for lactic dehydrogenase
    In areas where malaria transmission occurs – rapid tests should always be available for malaria screening but cannot be used for monitoring response to treatment as they may remain positive for over 4 weeks.
  ○ malaria parasites in blood smear – thick and thin smears
    One negative malaria test does not exclude the diagnosis.
    Repeat smears if initially negative.
    If severe malaria suspected, commence therapy and repeat smears after 6–12 hours.
    Repeat smears after 24–48 hours and if no improvement in degree of parasitaemia, consider alternative therapy.

If severe malaria is suspected and diagnosis cannot be confirmed immediately, treat while awaiting laboratory results.

8.7.1 P. FALCIPARUM MALARIA, NON-SEVERE, UNCOMPPLICATED

A child with uncomplicated malaria is alert, can tolerate oral medication, can sit, stand or walk unaided as appropriate for age and has no clinical or laboratory evidence of severe malaria.

Ideally treatment should be started in hospital. Initial doses should be directly observed. Observe for 1 hour to ensure dose is not vomited.

DRUG TREATMENT
Treat according to the National Malaria Guidelines.

Option 1:
• quinine, oral, 10 mg/kg/dose 8 hourly for 7–10 days
  2–3 days after initiating treatment with quinine
  children < 8 years
• clindamycin, oral, 10 mg/kg/dose 12 hourly for 7 days
  children > 8 years
• doxycycline, oral, 4 mg/kg immediately then 2 mg/kg/daily with a meal or a full glass of fluid for 7 days or until smears are negative
  Can cause gastrointestinal intolerance and oral aphthous ulceration.
OR
Option 2: Only for clearly uncomplicated, low risk malaria cases

- artemether/lumefantrine, oral, with fat-containing food/milk to ensure adequate absorption
  
  Give first dose immediately, the second dose after 8 hours and subsequent doses twice daily for 2 days.
  
  1 tablet contains 20 mg artemether plus 120 mg lumefantrine

<table>
<thead>
<tr>
<th>Weight</th>
<th>Dose</th>
<th>Total tablets per course</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–15 kg</td>
<td>1 tablet</td>
<td>6</td>
</tr>
<tr>
<td>15–25 kg</td>
<td>2 tablets</td>
<td>12</td>
</tr>
<tr>
<td>25–35 kg</td>
<td>3 tablets</td>
<td>18</td>
</tr>
<tr>
<td>over 35 kg</td>
<td>4 tablets</td>
<td>24</td>
</tr>
</tbody>
</table>

8.7.2 *P. FALCIPARUM* MALARIA, SEVERE, COMPLICATED (OR IF REPEATED VOMITING)

**DIAGNOSTIC CRITERIA**

**Clinical**

- manifests with 2 or more convulsions, which may be subtle, and/or any change in mental state, ranging from irritability to coma
- respiratory distress and metabolic acidosis
- anaemia – can be severe and lead to cardiac failure and a depressed mental state
- shock – cold moist skin, low blood pressure and collapse
- hypoglycaemia – can present with convulsions and a depressed mental state
- jaundice, bleeding, acute renal failure and ARDS are less common in children than adults

**Investigations**

- hyperparasitaemia – > 5% of RBCs infected indicates severe malaria but a lower parasite density does not exclude severe malaria
- low haemoglobin (< 6 g/dL)
- low blood glucose (< 2.2 mmol/L)
  
  Test glucose immediately with a fingerprick test.
- acidosis – serum lactate > 5 mmol/L or bicarbonate < 15 mmol/L
- severe thrombocytopenia – < 50 x 10⁹/L
- in severe cases, repeat smear after 72 hours and after the completion of the course of treatment

**NON-DRUG TREATMENT**

- admit to high care or intensive care unit
- review the child at least twice daily, including holidays
- avoid overhydration
- monitor blood glucose and correct hypoglycaemia with dextrose 10%
- packed red cells, 10 mL/kg over 3 hours if haemoglobin < 7 g/dL
- control convulsions
- ventilatory support, if necessary
- nutritional support
**DRUG TREATMENT**

**URGENT**
- quinine, IV infusion, diluted in 5–10 mL/kg dextrose 5% or sodium chloride 0.9%
  - Administer 20 mg/kg over 4 hours, then 10 mg/kg over 4–6 hours at 8 hourly intervals until able to take oral therapy.

2–3 days after initiating treatment with quinine and able to swallow
- quinine, oral, 10 mg/kg/dose 8 hourly to complete 7–10 day course
  - Ensure that tablets are swallowed.
  - Monitor blood glucose regularly as quinine exacerbates hypoglycaemia.
  - Quinine is cardiotoxic – monitor heart rate/ECG, if available.

**PLUS**

children < 8 years
- clindamycin, oral, 10 mg/kg/dose 12 hourly for 7 days

children > 8 years
- doxycycline, oral, 4 mg/kg immediately then 2 mg/kg/daily with a meal or a full glass of fluid oral for 7 days or until smears are negative.
  - Can cause gastrointestinal intolerance and oral aphthous ulceration.

For concurrent bacterial sepsis
- ceftriaxone, IV, 100 mg/kg as a single daily dose once daily for 10 days
  - Maximum dose: 4 000 mg/24 hours

For fever
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

**Note:**
Fluid loss is often underestimated in a febrile, vomiting, sweating child.

**8.7.3 P. OVALE, P. VIVAX AND P. MALARIAE**

- chloroquine, oral, 10 mg base/kg as a single dose, then 5 mg base/kg given 6, 24 and 48 hours after the first dose

**8.7.4 MALARIA PROPHYLAXIS – SELF PROVIDED CARE**

From October to May, prophylaxis should be used together with preventative measures against mosquito bites in the high-risk malaria areas in Southern Africa. State facilities do not provide prophylactic therapy. It is recommended that persons intending to travel to high-risk areas take the relevant prophylactic therapy.

*Consult the National Malaria Guidelines.*

**CAUTION**
Children under 5 years should avoid visiting malaria-transmission areas, as they are more prone to the serious complications of malaria.
NON-DRUG TREATMENT
- prevent insect bites between dusk and dawn
  - insecticide treated nets, screens, coils or pads
  - insect repellent to exposed skin
  - long sleeved shirts and long trousers
  - socks and shoes
- preferably visit endemic areas only during cold, dry season

DRUG TREATMENT
Chemoprophylaxis
Widespread chloroquine resistance has made prophylaxis with chloroquine plus proguanil substantially less effective.

If > 5 kg
- mefloquine, oral
  - Initiate treatment 8 days before entering a malaria area, continue throughout stay and for a further 4 weeks after leaving the area.

If > 8 years
- doxycycline, oral
  - Initiate treatment 24 hours before entering a malaria area, continue throughout stay and for a further 4 weeks after leaving the area.

<table>
<thead>
<tr>
<th>Weight</th>
<th>Mefloquine weekly</th>
<th>Doxycycline daily</th>
</tr>
</thead>
<tbody>
<tr>
<td>5–20 kg</td>
<td>62.5 mg (¼ tablet)</td>
<td>Contraindicated</td>
</tr>
<tr>
<td>21–30 kg</td>
<td>125 mg (½ tablet)</td>
<td>Contraindicated</td>
</tr>
<tr>
<td>31–45 kg</td>
<td>187.5 mg (¾ tablet)</td>
<td>2 mg/kg</td>
</tr>
<tr>
<td>&gt; 45 kg</td>
<td>250 mg (1 tablet)</td>
<td>100 mg</td>
</tr>
</tbody>
</table>

URGENT REFERRAL
- severe or complicated malaria

REFERRAL
- high risk children under 2 years, splenectomised patients
- malaria not responding clinically to adequate treatment within 48-72 hours (possible resistance)
- children with \textit{P. vivax} or \textit{P. ovale} malaria for primaquine after initial chloroquine treatment
8.8 MEASLES
B05.8
* Notifiable condition

DESCRIPTION
The following case definition is an epidemiological and not a diagnostic tool:
• fever and maculopapular rash with any one of the following:
  ○ cough
  ○ coryza/runny nose
  ○ conjunctivitis
Suspect measles in any child fulfilling the case definition.

An acute, highly contagious, viral, childhood exanthem.
Incubation period: 8–14 days from exposure to 1st symptoms and 14 days between appearance of rash in source and contact.

Complications include:
• pneumonia
• laryngotracheobronchitis (croup)
• encephalitis
• stomatitis
• feeding difficulties
• severe diarrhoea
• otitis media
• corneal ulceration
Subacute sclerosing panencephalitis is a rare long-term complication.

DIAGNOSTIC CRITERIA
Clinical
• prodromal (catarrhal) phase:
  ○ duration 3–5 days
  ○ fever
  ○ runny nose (coryza)
  ○ cough
  ○ conjunctivitis
  ○ Koplik’s Spots, followed 3–5 days later with maculopapular rash
• the rash begins to fade after 3 days in the order of its appearance leaving temporary darker staining
• if fever is still present after the third day of the rash, a complication should be suspected

Investigations
• serum measles IgM antibodies for confirmation of diagnosis

NON-DRUG TREATMENT
• notify provincial EPI manager prior to confirmation
• only admit high risk patients:
  ○ children less than 6 months old
  ○ immune compromised/suppressed children
  ○ children with severe malnutrition
  ○ children with complications
• minimal exposure to strong light, if patient is photophobic
• isolate the patient in a separate room, if possible away from other children
  All entering the room to wear mask, gloves and gown.
  Patient is infectious for 4 days after onset of rash, longer if HIV-infected.
• screen outpatient waiting areas for children with measles
• if pneumonia with hypoxia, give humidified oxygen by means of nasal cannula

**DRUG TREATMENT**
**All patients**
• vitamin A, oral, as a single daily dose for 2 days
  < 1 year 100 000 units
  > 1 year 200 000 units

For fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required until fever subsides

**Complications**
**Pneumonia**
**Antibiotics, empirical**
To cover *S. aureus* and Gram-negative infection.
Total duration of therapy: 5–7 days

• ampicillin, IV, 25–50 mg/kg/dose, 6 hourly
  **PLUS**
  • gentamicin, IV, 7.5 mg/kg once daily
  **PLUS**
  • cloxacillin, IV, 50 mg/kg/dose 6 hourly

when child improves follow with oral therapy to complete 5–7 days treatment
• amoxicillin, oral, 30 mg/kg/dose 8 hourly
  **PLUS**
  • flucloxacillin, oral, 12.5–25 mg/kg/dose, 6 hourly

**Penicillin allergic**
• erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly
  **PLUS**
  • gentamicin, IV, 7.5 mg/kg once daily

In very severe progressive or unresponsive pneumonia consider use of aciclovir for possible herpes infection.

**Croup**
See Laryngotracheobronchitis (Croup): Section 15.5.2
Consider herpes and the need to use aciclovir.

**Diarrhoea**
See Acute Diarrhoea: Section 2.2.4
Encephalitis
See Section 8.12

Convulsions
See Section 13.4

Conjunctivitis and corneal dryness
- chloramphenicol ophthalmic ointment 1%, inserted 6 hourly for 5 days
If corneal clouding/ulceration present obtain urgent ophthalmologic consultation.

Management of Contacts
Immunise children older than 6 months if unvaccinated and less than 72 hours since exposure.
Between 3 and 6 days after exposure and for contacts less than 6 months old
- gamma globulin, IM, 0.25 mL/kg
If immunodeficient
- gamma globulin, IM, 0.5 mL/kg
Immunise all children > 6 months of age if outbreak occurs.

REFERRAL
- children in need of intensive care unit
- children with depressed level of consciousness
- children with corneal ulceration/opacity

8.9 MENINGITIS, ACUTE BACTERIAL
G00
* Notifiable condition. (N. meningitidis and H. influenzae)

This guideline applies to children > 60 days.

DESCRIPTION
Bacterial meningitis most commonly results from haematogenous dissemination of microorganisms from a distant site, e.g. the nasopharynx.
In children, S. pneumoniae and N. meningitides are the usual pathogens.

Note:
Tuberculosis or cryptococcal meningitis should be considered when the clinical and laboratory features are not typical of pyogenic meningitis.

Complications include:
- raised intracranial pressure due to cerebral oedema, subdural effusion/empyema or hydrocephalus
- other acute complications include:
  - cerebral infarctions
  - shock
  - seizures
  - metastatic infection, e.g. arthritis, pneumonia, pericarditis
  - disseminated intravascular thrombosis
  - inappropriate antidiuretic hormone (ADH) secretion
Long-term neurological sequelae include deafness, blindness, mental retardation and motor paralysis, e.g. hemiparesis.

**DIAGNOSTIC CRITERIA**

**Clinical**
- fever
- headache
- vomiting
- convulsions
- feeding problems
- irritability
- lethargy
- signs of meningeal irritation. In young infants signs of meningism are often absent.
- signs of increased intracranial pressure, e.g. bulging fontanel
- papilloedema is not a useful sign in young children with meningitis. It is difficult to elicit and may be absent even with acutely raised ICP.

**Investigations**
- lumbar puncture
  - defer but initiate treatment immediately if:
    - clinical signs of severely raised intracranial pressure, i.e. impending cerebral herniation:
      - deep coma, i.e. GCS < 13, or sudden deterioration of level of consciousness
      - decerebrate or decorticate posturing
      - neurogenic hyperventilation
      - unequal dilated or poorly reactive pupils
      - absent doll eye reflex
    - hemodynamic/respiratory unstable patients
    - clinical meningococcaemia (septicaemia) with petechiae/purpura
      - confirm with skin scrape and Gram stain and blood culture.

**NON-DRUG TREATMENT**
- admit to high or intensive care unit, if appropriate
- monitor, where indicated:
  - neurological status
  - heart rate
  - blood pressure
  - acid–base status
  - blood glucose
  - fluid balance, i.e. hydration
  - respiration
  - body temperature
  - haematocrit
  - electrolytes
  - blood gases
  - serum and urine osmolality
- ensure adequate nutrition by enteral feeding where possible. Use a nasogastric tube if necessary. If enteral feeding is not possible, give intravenous fluids: paediatric or neonatal maintenance solution with dextrose.

**DRUG TREATMENT**

**Antibiotic therapy**

Duration of treatment:

- *N. meningitidis* 5 days
- *S. pneumoniae* 12 days
- *H. influenzae* 7 days

In complicated cases, a longer duration of therapy may be required. Reassess antimicrobial therapy when blood and CSF culture and sensitivity results become available, or when improvement is not evident within 72–96 hours.
- cefotaxime, IV, 25–50 mg/kg/dose, 6–8 hourly
  OR
  ceftriaxone, IV, 50 mg/kg/dose 12 hourly

Seek immediate advice on what treatment to start when ventriculo-peritoneal shunt infection or spread from sinuses, mastoid or direct penetrating source of infection is present. For shunts, 3rd generation cephalosporin, e.g.:
- cefotaxime, IV, 60–70 mg/kg/dose, 8 hourly
  OR
  ceftriaxone, IV, 50 mg/kg/dose 12 hourly
PLUS
- vancomycin, IV, 15 mg/kg/dose, 6 hourly, infused over 1 hour

Steroid therapy
- dexamethasone, IV, 0.15 mg/kg 6 hourly for 3 days starting with or before initial antibiotic dose

Fever and headache
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

Convulsions
See Section 13.4

Raised intracranial pressure or cerebral oedema
Elevate head of bed ± 20 degrees.
Maintain PaCO₂ at 4–5 kPa; intubate and ventilate if necessary.
Avoid fluid overload.
- mannitol, IV, 250 mg/kg administered over 30–60 minutes
- dexamethasone, IV, 0.5 mg/kg twice daily

Chemoprophylaxis for close paediatric contacts
A close contact is defined as someone living in the same household or dormitory, if institution, or children in the same crèche, or any other “kissing” contact.

*N. meningitidis*
- ceftriaxone, IM, single dose
  < 12 years  125 mg
  > 12 years  250 mg
  OR
  ciprofloxacin, oral, 10 mg/kg as a single dose
  6–12 years  250 mg
  > 12 years  500 mg

Note:
If < 6 years of age and able to swallow a tablet a single 250 mg tablet may be considered.
H. influenzae prophylaxis for household and day care contacts under 5 years
- rifampicin, oral, 20 mg/kg/dose, once daily for 4 days
  Maximum dose: 600 mg.
  Neonatal dose: 10 mg/kg/dose, once daily for 4 days.

REFERRAL
- where lumbar puncture is deferred. Start treatment immediately and before referral.
- meningitis with complications
- all cases of suspected shunt infection. Start treatment immediately and before referral.

8.10 MENINGITIS, CRYPTOCOCCAL
G02.1

DESCRIPTION
An uncommon childhood meningitis that may occur in older HIV-infected children with severe CD4 T-cell depletion. Pulmonary and skin involvement can occur.

DIAGNOSTIC CRITERIA
Clinical
- acute or chronic headache in an older HIV-infected child. Meningism need not be present.
- often presents with cranial nerve palsy
- can occur as result of Immune Reconstitution Inflammatory Syndrome (IRIS) after initiation of antiretroviral therapy

Investigations
- all cerebrospinal fluid (CSF) specimens from HIV-infected children with suspected meningitis
  - India ink stain, and/or
  - cryptococcal antigen test – more sensitive than India ink stain
- fungal culture blood and urine
- chest X-ray
- ophthalmological assessment

NON-DRUG TREATMENT
- admit to high or intensive care unit, if appropriate
- serial spinal tap to relieve CSF pressure if raised
- monitor, where indicated:
  - neurological status
  - heart rate
  - blood pressure
  - haematocrit
  - minerals
  - acid–base status
  - fluid balance, i.e. hydration
  - respiration
  - body temperature
  - electrolytes
  - blood glucose
  - blood gases
  - serum and urine osmolality
- ensure adequate nutrition by enteral feeding where possible. Use a nasogastric tube if necessary. If enteral feeding is not possible, give intravenous fluids: paediatric or neonatal maintenance solution with dextrose.
DRUG TREATMENT

- amphotericin B, IV infusion, 0.5–1 mg/kg/dose once daily over 4 hours for 14 days, or longer depending on disease response.
  - Higher dose if CNS involvement.
  - Total dose: 30–35 mg/kg over 4–8 weeks.
  - Give initial test dose – see package insert.
  - Adjust dose if in renal failure.
  - Protect from light during infusion.
  - Check serum potassium levels every 5 days

Follow with

- fluconazole, oral, 12–15 mg/kg/once daily for a further 6–8 weeks
  - Maximum dose: 400 mg

THEN

Secondary prophylaxis

Continue indefinitely.

- fluconazole, oral, 6–10 mg/kg/once daily
  - For adolescents receiving antiretroviral therapy, maintenance fluconazole may be stopped if immune reconstitution occurs, i.e. CD4 count increases to between 100–200 cells/mm$^3$.
  - There is no data available to confirm that stopping maintenance therapy in children is safe.

For continued raised intracranial pressure

- acetazolamide, oral, 50 mg/kg/24 hours in 3 divided doses
  - Maximum dose: 1 g/day.
  - Monitor for metabolic acidosis and serum potassium derangements.

PLUS

- furosemide, oral, 1 mg/kg/24 hours in 3 divided doses for the first month of treatment
  - Taper slowly over 2 weeks if the intracranial pressure has normalised, as indicated by clinical response.

REFERRAL

- all cases

8.11 MENINGITIS, TUBERCULOUS (TBM)

G01

* Notifiable condition.

DESCRIPTION

Tuberculous meningitis is an infection of the meninges caused by *M. tuberculosis*. Early diagnosis and treatment improves the prognosis. Differentiation from acute bacterial meningitis may be difficult. If in any doubt treat for both conditions.
Complications may be acute or long term

- **acute:**
  - raised intracranial pressure
  - cerebral oedema
  - hemi/quadriplegia
  - hyponatraemia due to inappropriate antidiuretic hormone (ADH) secretion or cerebral salt wasting

Cerebral salt wasting and SIADH both present with hyponatraemia; the former responding to fluid replacement, i.e. sodium chloride 0.9% and the latter to fluid restriction.

Cerebral salt wasting has a normal serum uric acid and high urine output. SIADH has lower serum uric acid and low urine output.

**Note:**
Restrict fluid once diuretics are initiated.

- **long term neurological sequelae include:** mental handicap, blindness and deafness

**DIAGNOSTIC CRITERIA**

**Clinical**

- history of contact with tuberculosis
- onset may be gradual with vague complaints of headache, irritability, weight loss and drowsiness
- later symptoms are convulsions and neurological fall out
- older children may present with behavioural changes
- examination may reveal signs of meningeal irritation and raised intracranial pressure, convulsions, cranial nerve palsies, localising signs (such as hemiparesis), altered level of consciousness or coma and choroidal tubercles.
- degree of involvement is classified into 3 stages. Prognosis relates to the stage of the disease.
  - **Stage 1:** non-specific signs, signs of meningeal irritation, conscious, rational, no focal neurological signs, no hydrocephalus
  - **Stage 2:** confusion and/or focal neurological signs
  - **Stage 3:** stupor, delirium, coma and/or neurological signs, i.e. hemiplegia

**Investigations**

- **CSF findings:**
  - may vary depending on the stage
  - protein usually raised
  - chloride and glucose are moderately low
  - lymphocytes usually predominate
  - Gram stain is negative and acid-fast bacilli are seldom found
    - Bacilli may be cultured from the CSF but may take up to 4–6 weeks.
  - Always send for culture, do not perform stain as low diagnostic yield from low concentration of organisms wastes CSF sample.
  - a Mantoux test and chest X-ray must be done – are often negative
  - if depressed level of consciousness or focal neurological signs are present, a CT scan is useful
  - electrolytes – check for hyponatraemia
NON-DRUG TREATMENT
• monitor neurological status on a regular basis
• attend to nutritional status. Initially nasogastric feeding is usually needed
• rehabilitative measures
  ○ most patients need physiotherapy and occupational therapy
• non-communicating hydrocephalus, diagnosed by air encephalogram, should be
treated surgically
• communicating hydrocephalus with severely raised pressure may be managed with
medicines and/or serial lumbar puncture with specialist consultation

DRUG TREATMENT
Differentiation from acute bacterial meningitis may be difficult.
If in doubt treat for both conditions.

Antituberculous Treatment
Requires therapy with a combination of 4 drugs as a special regimen.
Do not use single drugs for therapy.
Single drugs may form part of the regimen to provide the total daily required dose for
each drug by supplementing the combination to give the necessary therapeutic dose per
kilogram.

The following regimen should be used seven days a week for 3 months, not five days a
week:
• rifampicin, oral, 20 mg/kg as a single daily dose
PLUS
• isoniazid, oral, 20 mg/kg as a single daily dose
PLUS
• pyrazinamide, oral, 40 mg/kg as a single daily dose
  Maximum daily dose: 2 000 mg.
PLUS
• ethionamide, oral, 20 mg/kg as a single daily dose
  Maximum daily dose: 1 000 mg.

THEN
After 3 months therapy, use five days a week for a further 6 months
• rifampicin, oral, 20 mg/kg as a single daily dose
PLUS
• isoniazid, oral, 20 mg/kg as a single daily dose
PLUS
• ethionamide, oral, 20 mg/kg as a single daily dose
  Maximum daily dose: 1 000 mg.

Steroid therapy
• prednisone, oral, 4 mg/kg as a single daily dose for 4 weeks.
  Maximum daily dose: 60 mg.
  Taper to stop over 2 weeks.
Hydrocephalus
Avoid low sodium IV fluids in these patients, i.e. < 60 mmol/L.
To differentiate communicating from non-communicating hydrocephalus an air encephalogram is usually required. Communicating hydrocephalus is more common in this condition. In children with a sudden deterioration of level of consciousness and other comatose children with TBM, inform the neurosurgeon before doing the air-encephalogram so that shunt surgery can immediately be done if the hydrocephalus is non-communicating.

Communicating hydrocephalus
- acetazolamide, oral, 50 mg/kg/24 hours in 3 divided doses
  - Maximum daily dose: 1 000 mg.
  - Monitor for metabolic acidosis and serum potassium derangements.

PLUS
- furosemide, oral, 1 mg/kg/24 hours in 3 divided doses for the first month of treatment.
  - Taper slowly over 2 weeks if the intracranial pressure has normalised, as indicated by clinical response or resolution of hydrocephalus on follow-up scan.
  - Do not restrict fluids once on diuretics.

Sudden deterioration of level of consciousness.
- mannitol, IV, 250 mg/kg administered over 30–60 minutes

REFERRAL
- TBM not responding to adequate therapy
- TBM with complications
- noncommunicating hydrocephalus

8.12 MENINGO-ENCEPHALITIS/ENCEPHALITIS, ACUTE VIRAL
A86

DESCRIPTION
A number of viruses cause infection of the brain and meninges. Herpes simplex is the most important because it is treatable. A high mortality and morbidity is associated with untreated disease.

Complications include:
- increased intracranial pressure
- cerebral oedema
- blindness
- inappropriate antidiuretic hormone (ADH) secretion
- permanent neurological deficits
- seizures
- deafness

DIAGNOSTIC CRITERIA
Clinical
- severe headache, fever, nausea, vomiting, lethargy and abnormal behaviour
- alteration in level of consciousness, i.e. drowsiness, confusion, stupor and coma
- generalised and/or focal convulsions
- focal neurological deficits
- abnormal movements, i.e. basal ganglia involvement
• cranial nerve palsies (brainstem involvement), loss of sphincter control, paresis of limbs and segmental sensory loss (spinal cord involvement)
• some patients may have signs of meningeal irritation
• herpes encephalitis may have an acute and fulminant course. It can result from primary or recurrent infection.

Investigations
• laboratory tests are mostly unhelpful
• CSF may reveal:
  ○ slightly raised protein
  ○ normal glucose level, and
  ○ mild pleocytosis, mostly lymphocytes
  ○ a specific virus is sometimes isolated. PCR is helpful, if available
  ○ red cells are seen with Herpes encephalitis
• in some instances, the CSF may be completely normal
• a CT scan of the brain may reveal brain oedema
  CT findings may only be apparent after 3–5 days.
  The Herpes virus preferentially involves the temporal lobe and orbital surfaces of the frontal lobes.
• an EEG may demonstrate changes suggestive of herpes encephalitis

NON-DRUG TREATMENT
• admit to high or intensive care unit, if appropriate
• monitor, where indicated:
  ○ neurological status
  ○ heart rate
  ○ blood pressure
  ○ haematocrit
  ○ acid–base status
  ○ fluid balance, i.e. hydration
  ○ respiration
  ○ body temperature
  ○ electrolytes
  ○ blood glucose
  ○ blood gases
  ○ serum and urine osmolality
• ensure adequate nutrition by enteral feeding where possible. Use a nasogastric tube if necessary. If enteral feeding is not possible, give intravenous fluids: paediatric or neonatal maintenance solution with dextrose.

DRUG TREATMENT
If herpes encephalitis is suspected
• aciclovir, IV, 250 mg/m²/dose 8 hourly administered over 1 hour for 14–21 days
  > 1 month – 1 year: 12.5 mg/kg/dose
  2–6 years: 10 mg/kg/dose
  7–12 years: 7.5 mg/kg/dose

If varicella zoster virus
• aciclovir, IV, 500 mg/m²/dose 8 hourly administered over 1 hour for 10 days
  > 1 month – 1 year: 25 mg/kg/dose
  2–6 years: 20 mg/kg/dose
  7–12 years: 15 mg/kg/dose

Acute Convulsions
See Section 13.4
For fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly until fever subsides

**Raised intracranial pressure or cerebral oedema**
Elevate head of bed ± 20 degrees.
Maintain PaCO$_2$ at 4–5 kPa; intubate and ventilate if necessary.
Avoid fluid overload.
• mannitol, IV, 250 mg/kg administered over 30–60 minutes.
  Do not repeat without consultation with a paediatrician

**REFERRAL**
• deterioration of clinical condition despite adequate treatment
• meningo-encephalitis with complications or loss of consciousness

### 8.13 MUMPS
**A31.0**

**DESCRIPTION**
Mumps is an acute, communicable, viral disease of childhood that commonly affects the salivary glands, chiefly the parotid gland, and frequently the central nervous system. The incubation period is 2–3 weeks.

**Complications** include:
- meningo-encephalitis
- pancreatitis
- orchitis
- facial nerve paresis
- nephritis
- oophoritis
- thyroiditis
- nerve deafness
- myocarditis

**DIAGNOSTIC CRITERIA**

**Clinical**
• a prodrome of 1–2 days may precede the salivary gland involvement and is characterised by fever, malaise, headache and pain in or behind the ear on chewing or swallowing
• painful enlargement of the parotid gland/s with the ear usually displaced upward and outward with the mandibular angle obliterated. The submaxillary and sublingual glands may also be involved.
• pain may be referred to the ear
• papilla of Stensen’s duct opposite the upper second molar may be oedematous and red
• central nervous system involvement may occur alone or may precede, accompany or follow inflammation of the salivary glands

**Investigations**
• leucopaenia with relative lymphocytosis

**NON-DRUG TREATMENT**
• isolate patient until salivary gland enlargement subsides
• maintain adequate nutrition and hydration
  Patient may return to school after swelling has subsided.
DRUG TREATMENT
Treat complications as appropriate.

For pain and fever
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

REFERRAL
- mumps with complications not responding to adequate therapy

8.14 MYCOBACTERIUM AVIUM COMPLEX (MAC) INFECTION
A31.0

DESCRIPTION
Atypical mycobacterium, causing disease in extremely immunocompromised patients.
MAC infection in HIV usually presents with disseminated disease, often enlarged intra-abdominal lymph nodes and pancytopenia.
Pulmonary, GIT or skin disease is less common.

DIAGNOSTIC CRITERIA
- MAC may be isolated from blood, bone marrow, lymph node, other sterile fluids and tissues
- confirm diagnosis with a biopsy for histology and culture

DRUG TREATMENT
To be managed under specialist care.
Therapy consists of a combination of at least two drugs.

New generation macrolide e.g.:
- azithromycin
  OR
  clarithromycin
PLUS
- ethambutol

For extensive disease in severely immunodeficient child
ADD
Quinolone, e.g.:
- ciprofloxacin
PLUS
- amikacin

REFERRAL
- all cases
8.15 PERTUSSIS
A37.9
* Notifiable condition

DESCRIPTION
A communicable respiratory infection usually recognised by a paroxysmal cough followed by an inspiratory whoop and associated vomiting. Subconjunctival haemorrhages may be present. The cough can persist for 3 months or longer with the infectious period between 2 weeks and 3 months. The disease is more severe in young infants where it may present with apnoea rather than the inspiratory whoop. Incubation period: 7–10 days. Range: 6–21 days.

DIAGNOSTIC CRITERIA
• diagnosis is clinical
• a definitive diagnosis often not possible with respect to viral pertussis-like syndrome
• FBC
  ○ usually very high WCC with > 50% lymphocytosis
• use naso-pharyngeal aspirates if possible for special cultures for Bordetella pertussis

NON-DRUG TREATMENT
• isolation during first 2 days whilst on antibiotic therapy
• clear airways by gentle suction taking care not to induce cough
• appropriate respiratory support for apnoea or respiratory distress/failure
• if hypoxic, give oxygen, 1–2 L/minute via nasal prongs
• encourage oral feeding. If unsuccessful provide nasogastric feeds with small volumes.
• immunise infant against pertussis even if diagnosis of pertussis was made

DRUG TREATMENT
• erythromycin, oral, 10–15 mg/kg/dose, 6 hourly for 14 days

For fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

All contacts of presumed pertussis, including adults.

Children:
• erythromycin, oral, 10–15 mg/kg/dose, 6 hourly for 14 days

Adults:
• erythromycin, oral, 250 mg, 6 hourly for 14 days

Vaccinate contacts where appropriate.

REFERRAL
• children with seizures or encephalopathy for further evaluation
• infants requiring intensive care, where none is available on site
8.16 PNEUMOCYSTIS JIROVECI PNEUMONIA (PCP)
B20.6

DESCRIPTION
PCP is an opportunistic respiratory infection most common in infants from 2–6 months. It presents with an acute onset of respiratory distress with minimal/absent chest signs in a child who is HIV exposed. Hypoxaemia and cyanosis are common features as the disease progresses.

DIAGNOSTIC CRITERIA
Clinical
• clinical suspicion in HIV exposed infants

Investigations
• oxygen saturation: usually less than 90% on pulse oximetry in room air
• chest X-ray
  ○ findings can vary
  ○ diffuse bilateral alveolar or interstitial infiltrate
• indirect immunofluorescence of nasal wash or tracheal aspirate/induced sputum may demonstrate Pneumocystis

NON-DRUG TREATMENT
• give oxygen, 1–2 L/minute via nasal prongs
• monitor saturation respiratory rate and other vital parameters
• supportive care, nasogastric feeds and intravenous fluids

DRUG TREATMENT
• trimethoprim/sulfamethoxazole, IV, 5 mg/kg/dose of trimethoprim component 6 hourly for 5 days

When child improves follow with
• trimethoprim/sulfamethoxazole, oral, 5 mg/kg/dose of trimethoprim component 6 hourly for 3 weeks

Note:
PCP prophylaxis should continue after discharge.

If no response
ADD
• clindamycin, IV, 10mg/kg/dose, administered over 30 minutes, 8 hourly
  OR
dapsone, oral, 5 mg/kg 8 hourly

If hypoxic and PCP confirmed or highly suspected
• prednisone, oral, 1–2 mg/kg daily for 2 weeks
  Beware: danger of worsening co-morbid lung CMV infection.

For pain and fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required
REFERRAL
- drug intolerance
- infants and children requiring intensive care, where none is available on site

8.17 POLIOMYELITIS (ACUTE FLACCID PARALYSIS)
A80.3
* Notifiable condition

See also Guillain-Barré Syndrome: Section 13.6.1

DESCRIPTION
Poliomyelitis is caused by polio virus, types 1, 2 and 3. It mainly affects children under 5 years of age, but a person at any age who does not have immunity may be infected. Risk of paralysis increases with age.
Poliomyelitis is uncommon. Most cases of acute flaccid paralysis (AFP) are caused by Guillain-Barré Syndrome, but all cases of AFP should be notified.
Humans are the only reservoir. The faecal-oral route is the major route of transmission, although droplet spread can occur.
Incubation period is between 7–14 days.

DIAGNOSTIC CRITERIA
Clinical
- polio virus infection is asymptomatic in 90–95% of cases
- ± 4–8% will develop abortive polio with some or all of the following symptoms:
  - fever
  - headache
  - stiff neck
  - muscle pain
  - nausea
  - vomiting and diarrhoea.
- ± 1% may present as viral meningitis
- the remaining 1–2% will develop paralysis of sudden rapid onset reaching full development in hours, maximum 3 days
- paralysis is often asymmetrical and always flaccid. Reflexes are absent.
- sensation usually not affected
- lower limbs are affected more than upper limbs and proximal more than distal muscles

Investigations
- send two stool specimens taken 24–48 hours apart to the National Institute of Virology via the local laboratory
- send one specimen after 60 days

NON-DRUG TREATMENT
- isolate patient to prevent faecal-oral spread
- rehabilitative measures
  - most patients need physiotherapy and occupational therapy

DRUG TREATMENT
Prevention
Immunise all children, including HIV-infected children, according to the EPI programme.
REFERRAL
• children requiring intensive care, if none is available on site

8.18 RABIES
A82.9
* Notifiable condition
Inform state veterinarian or local veterinary official.

DESCRIPTION
A viral infection of the central nervous system following transmission of the rabies virus from the saliva of affected animals through bites or contamination of mucosa or skin lesions.
Incubation period 2–8 weeks.
Range: 5 days–1 year.

DIAGNOSTIC CRITERIA
Clinical
• signs and symptoms may begin with:
  ○ fever
  ○ headache
  ○ nausea
  ○ diarrhoea
  ○ irritability
• early signs include paraesthesia or itching at site of bite in ⅓ of cases
• the acute neurologic phase interspersed with lucid periods manifests with:
  ○ agitation
  ○ mania
  ○ hyperactivity
  ○ hallucinations
• seizures may be precipitated by auditory or tactile stimuli
• hypersalivation, hydrophobia or aerophobia may occur
• death is usually due to cardio-respiratory failure

Investigations
• virus specific fluorescent antigen in brain tissue confirms diagnosis in animals
• preserve brain tissue of the dead animal

NON-DRUG TREATMENT
• symptomatic and supportive treatment
• prompt cleansing of the bite wound
• do not suture puncture wounds
• seek advice

<table>
<thead>
<tr>
<th>Telephone Hotline</th>
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<tr>
<td>National Institute of Communicable Diseases</td>
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<tr>
<td>After hours</td>
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</table>
DRUG TREATMENT
Treatment depends on the risk category.

<table>
<thead>
<tr>
<th>RISK CATEGORY</th>
<th>TYPE OF EXPOSURE</th>
<th>ACTION</th>
</tr>
</thead>
</table>
| 1.            | • touching or feeding animal  
                • licking intact skin | • none if reliable history |
| 2.            | • nibbling uncovered skin  
                • superficial scratch without bleeding  
                • licking broken skin | • wound treatment  
                • give rabies vaccine  
                • do not give anti-rabies immunoglobulin  
                Stop vaccination if laboratory tests of animal are negative for rabies or animal, i.e. dog or cat remains well after 10 days observation. |
| 3.            | • bites or scratches penetrating skin and drawing blood.  
                • licking of mucous membranes | • wound treatment  
                • give rabies vaccine  
                • give anti-rabies immunoglobulin (RIG)  
                • give tetanus toxoid vaccine and antibiotic  
                Stop vaccination if laboratory tests of animal are negative for rabies or animal, i.e. dog or cat remains well after 10 days observation. |

Post Exposure Prophylaxis

CAUTION
Start Post Exposure Prophylaxis immediately.  
Do not wait for confirmatory laboratory tests in the animal.

Post exposure prophylaxis may be life saving and should always be given if there is the slightest suspicion that the animal may have been rabid. The decision to give post exposure prophylaxis is based on the risk of rabies transmission, the species and behaviour of the animal and the nature of the bite. No laboratory test on the human victim is possible or available to confirm or exclude possible transmission.

Wound Treatment
Local wound care:
• flush wound thoroughly and clean with soap and water or sodium chloride 0.9% or chlorhexidine 0.05%  
• povidone iodine 10%, topical
For penetrating wounds

- tetanus toxoid vaccine (TT), IM, 0.5 mL

If ≥ 5 years after primary immunisation or immunisation status incomplete

- phenoxybenzylpenicillin, oral, 12.5 mg/kg/dose 6 hourly for 5 days

PLUS

- cloxacillin, oral, 25 mg/kg/dose 6 hourly for 5 days

Rabies Vaccine

Must be given for category 2 and 3 bites.

Vaccine is administered on days 0, 3, 7, 14, 28. Vaccine is ideally given as soon as possible after exposure, but should still be given if patient presents some time after the exposure. If vaccine administration is delayed > 48 hours, a double dose should be given initially.

Rabies vaccine is given IM but never in the buttock. Give to deltoid muscle in adults and antero-lateral aspect of thigh in infants.

Adverse events are uncommon and include:

- local reactions
  - pain
  - erythema
  - swelling or itching at the injection site

- systemic reactions
  - fever
  - arthralgia
  - arthritis
  - angioedema
  - nausea
  - vomiting
  - malaise

Rabies Immunoglobulin (RIG)

Must be given for category 3 bites only. Always give the vaccine first.

Immunoglobulin must be given as soon as possible after exposure, but may be administered up to 7 days after the first vaccine is given.

Do not give RIG if the patient has previously received pre- or post-exposure prophylaxis.

- rabies immunoglobulin, 20 units/kg
  Infiltrate around wound with up to 50% of dose.
  Administer remaining immunoglobulin into deltoid muscle opposite to vaccine administration site.
  If multiple wounds, dilute in sodium chloride 0.9% to 2–3 times so that all wounds are infiltrated.
  **DO NOT** exceed maximum dose as antibody production to the vaccine is inhibited.
  If unavailable, **DO NOT** delay active immunisation.

REFERRAL

- where prophylactic treatment is not immediately available
- all cases of human clinical rabies for appropriate palliative care
8.19 TETANUS
A35
* Notifiable condition

DESCRIPTION
Tetanus is an acute spastic paralytic illness caused by tetanospasmin, the neurotoxin produced by *C. tetani*. The toxin prevents neurotransmitter release from spinal inhibitory neurons.

Complications include:
- asphyxia
- bronchopneumonia
- dehydration
- respiratory failure
- hyperpyrexia
- laryngospasm
- inability to suck, chew and swallow

DIAGNOSTIC CRITERIA
The diagnosis is made on clinical grounds

Clinical
- unimmunised/incompletely immunised child
- history of wound/trauma or unhygienic care of umbilical cord/stump
- trismus
- stiffness of the neck, back and abdominal muscles
- pharyngospasm, laryngospasm, dysphagia, inability to suck, chew and swallow which severely compromises feeding and eating activities
- spontaneous muscle contractions/spasms or muscle contractions/ spasms triggered by minimal stimuli such as touch, sound, light or movement
- no involvement of sensorium, i.e. consciousness is not disturbed
- autonomic nervous system instability with hypertension, tachycardia and dysrhythmias

NON-DRUG TREATMENT
- admit to high or intensive care unit, if available
- oxygen to prevent hypoxia and ventilatory support if needed
- monitor:
  - temperature
  - blood pressure
  - respiration
  - blood glucose
  - heart rate
  - electrolytes
  - blood gases
  - acid–base status
  - SaO₂
- protect the patient from all unnecessary sensory and other stimuli
- ensure adequate hydration and nutrition
- wound care and debridement/umbilical cord care
- educate parents/caregivers regarding prevention of tetanus by vaccination

DRUG TREATMENT
- tetanus immunoglobulin, IM, 500–2 000 IU as a single dose
- benzylpenicillin (Penicillin G), IV, 12 500 –25 000 units/kg/dose, 6 hourly
- diazepam, IV, 0.1–0.2 mg/kg/dose 4–6 hourly, titrated according to response
  Do not exceed dose of 10 mg/dose.
After recovery from tetanus, patients should be actively immunised as the disease does not confer immunity.

**Prevention of tetanus**

**Minor Wounds:**
Children with clean minor wounds do not require tetanus immunoglobulin or antibiotics. Tetanus vaccine should be given, except in fully immunised patients who have received a booster within the past 5 years.

**For more severe wounds:**
If child with penetrating wound not completely immunised
- tetanus immunoglobulin, IM
  - < 5 years  75 IU
  - 5–10 years  125 IU
  - > 10 years  250 IU
- tetanus toxoid vaccine (TT), IM, 0.5 mL
  Not required in immunised patients who have received a booster within the past 5 years.
- phenoxyacetylepenicillin, oral, 12.5 mg/kg/dose 6 hourly for 7 days

Penicillin allergy
- erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 7 days

**REFERRAL**
- all severe cases

### 8.20 TICK-BITE FEVER

**DESCRIPTION**
A febrile illness with exanthem caused by *R. conorii* with the tick as vector. Recently other tick-borne Rickettsial diseases have been identified.

The rash appears on days 3–5 of the illness. It spreads from the extremities to the trunk, neck, face, palms, and soles within 36 hours. The lesions progress from macular to maculopapular and may persist for 2–3 weeks. Atypical cutaneous findings may occur in a few patients.

**Complications** include:
- vasculitis
- thrombosis
- myocarditis
- thrombocytopaenia
- encephalitis
- renal failure
- pneumonitis

**DIAGNOSTIC CRITERIA**
The diagnosis is made on clinical grounds.

**Clinical**
- fever, headache, malaise, myalgia and arthralgia
- maculopapular rash which may involve the palms and soles
- eschar at the site of the tick bite is associated with regional lymphadenopathy and splenomegaly
Investigations
• diagnosis can be confirmed retrospectively by immunofluorescent antibody techniques

NON-DRUG TREATMENT
• remove tick as soon as possible after detection on the body

DRUG TREATMENT
Antibiotic therapy
Treatment must be started before confirmation of diagnosis by serology.
Although not recommended for children < 8 years of age, doxycycline is still regarded as the drug of choice for children with tick-bite fever and least associated with dental staining.

• doxycycline, oral
  < 50 kg  4 mg/kg/24 hours in 2 divided doses on the first day, then 2 mg/kg/24 hours in 2 divided doses for 7–10 days
  > 50 kg  100 mg twice daily for 7–10 days

For children < 8 years and encephalitis
• chloramphenicol, IV, 50 mg/kg/24 hours in divided doses 6 hourly for 7–10 days

Headache and fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

REFERRAL
• patients not responding to adequate therapy
• patients with complications

8.21 TOXOPLASMOSIS
B58.9

DESCRIPTION
Rarely occurs in children.
Usually presents as encephalitis, with focal neurological abnormalities occurring in association with headache.
Ocular and pulmonary disease is also seen.

DIAGNOSTIC CRITERIA
Investigations
• diagnosis may be made on blood and CSF serology
• CSF PCR for toxoplasmosis may also be helpful
• CT scan usually reveals multiple bilateral, focal hypodense ring-enhancing lesions

REFERRAL
• all cases
8.22 TYPHOID
T01.0
* Notifiable condition

DESCRIPTION
A systemic disease caused by S. typhi.

DIAGNOSTIC CRITERIA
Clinical
• fever
• headache
• diarrhoea or constipation
• abdominal pain or tenderness
• cough
• meningeal tenderness
• epistaxis
• cough
• hepatomegaly and/or splenomegaly
• delirium

Investigations
• leucopaenia, anaemia and thrombocytopenia
• positive cultures from blood (1st week), stool (after 1st week), urine and bone marrow
• serology may be helpful

NON-DRUG TREATMENT
• isolate patient until 3 consecutive stools are culture negative
• correct and maintain fluid and electrolyte status
• packed red cells, 10 mL/kg over 3 hours if haemoglobin < 7 g/dL

DRUG TREATMENT
Note:
Relapse and carrier state may occur despite adequate therapy.

Third generation cephalosporin, e.g.:
• ceftriaxone, IV, 50–75 mg/kg once daily for 7–10 days

If cephalosporin allergy consider quinolones.

SURGICAL TREATMENT
Surgical intervention for bowel perforation, osteomyelitis, etc.

REFERRAL
• inadequate response to treatment
• patients with complications

8.23 VARICELLA (CHICKEN POX)
B01.8

DESCRIPTION
An acute, highly contagious, viral disease caused by herpes varicella-zoster. It spreads by
infective droplets or fluid from vesicles. One attack confers permanent immunity. Varicella
is contagious from about 5 days before the onset of the rash until the crusts begin to
disappear.
Reactivation of the virus may appear later as herpes zoster or shingles. Incubation period is 2–3 weeks.

**Complications** are more common in immunocompromised patients and include:
- secondary skin infection
- necrotising fasciitis
- haemorrhagic varicella lesions with evidence of disseminated intravascular coagulation

Two important bacteria causing complications are *S. aureus* and *S. pyogenes*.

**DIAGNOSTIC CRITERIA**

**Clinical**
- mild headache, fever and malaise
- characteristic rash
  - the lesions progress from macules to vesicles in 24–48 hours
  - successive crops appear every few days
  - the vesicles, each on an erythematous base, are superficial, tense ‘teardrops’ filled with clear fluid which dry to form fine crusts
  - the rash is more profuse on the trunk and sparse at the periphery of extremities
  - at the height of eruption, all stages (macules, vesicles and crusts) are present at the same time
  - the rash lasts 8–10 days and heals without scarring, unless secondarily infected
- mucous membranes may be involved
- pruritus may be severe
- patients are most contagious from 1–2 days before onset of the rash until crusting of lesions

**NON-DRUG TREATMENT**
- isolate the patient
- isolate the neonate until the mother is regarded as non-contagious
- maintain adequate hydration

**DRUG TREATMENT**

**Antiviral therapy**
For immunocompetent patients with varicella complications and for all immunocompromised patients. Initiate as early as possible, preferably within 24 hours of the appearance of the rash.

For less severe cases
- aciclovir, oral, 40 mg/kg 8 hourly daily for 5 days
  - Maximum dose: 800 mg/dose
  - or
  - aciclovir, IV, 500 mg/m2/dose 8 hourly administered over 1 hour for 7–10 days
    - > 1 month–1 year  25 mg/kg/dose
    - 2–6 years        20 mg/kg/dose
    - 7–12 years       15 mg/kg/dose

For fever
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required
For pruritus
   Mild:
   • calamine lotion, topical, applied 3 times daily
   Severe:
   • promethazine, oral, 0.25–0.5 mg/kg/dose 6 hourly for 24–48 hours

Secondary skin infection
   • amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5 days
   PLUS
   • flucloxacillin, oral, 12.5–25 mg/kg/dose, 6 hourly for 5 days

Prophylaxis
Post exposure prophylaxis must be given to:
Neonates whose mothers develop varicella from 5 days before delivery to 2 days after delivery:
   • varicella-zoster immunoglobulin, IM, 1 mL (100 units) given within 96 hours of exposure

If varicella-zoster immunoglobulin is not available
   • aciclovir, oral, 20 mg/kg/dose 8 hourly for 10 days
   Note:
   In neonates, prophylaxis may not prevent disease.

Immunocompromised children exposed to varicella
   • aciclovir, oral, 20 mg/kg/dose 8 hourly for 10 days

REFERRAL
   • neonates with varicella
   • patients with complications

8.24 ZOSTER
B02

DESCRIPTION
A vesicular eruption in a dermatomal pattern, which does not cross the midline, due to reactivation of herpes varicella–zoster virus. Occurs commonly in immunocompromised children and occasionally in immunocompetent children.

DIAGNOSTIC CRITERIA
Usually made on clinical grounds.
Investigations
   • confirm diagnosis by viral culture or Tzanck preparation

NON-DRUG TREATMENT
   • isolate patient
**DRUG TREATMENT**

Within 24 hours of the appearance of the rash for less severe cases.
- aciclovir, oral, 40 mg/kg/dose 8 hourly for 5 days  
  Maximum dose: 800 mg/dose.

If oral treatment cannot be taken and for severe cases
- aciclovir, IV, 500 mg/m2/ dose 8 hourly administered over 1 hour for 7–10 days  
  > 1 month–1 year     25 mg/kg/dose  
  2–6 years           20 mg/kg/dose  
  7–12 years         15 mg/kg/dose

In older children where pain may become a problem
- carbamazepine, oral, 5 mg/kg/dose every 8 hours

**REFERRAL**
- disseminated zoster

**8.25 SEPSIS (OUTSIDE THE NEONATAL PERIOD)**

**DESCRIPTION**

Systemic Inflammatory Response Syndrome in the presence of or as a result of suspected or proven infection.

Severe sepsis is an uncontrolled inflammatory response as a result of suspected or proven infection.

Clinical features include:
- raised cardiac output
- decreased systemic resistance
- warm extremities
- a wide pulse pressure

The hyperdynamic state is recognised by hyperpyrexia, hyperventilation, tachycardia and mental confusion.

A widespread scarlatiniform rash with secondary desquamation, conjunctivitis, strawberry tongue, vomiting and watery diarrhoea may be present in cases of toxic shock.

Children 2–3 years of age may present with a history of poor feeding, mottled appearance of the skin, acidosis, and inconsolable crying.

**DIAGNOSTIC CRITERIA**

**Clinical**
- a systemic inflammatory response with at least two of the following four criteria, one of which must be abnormal temperature or leukocyte count:
  - core temperature of > 38.5°C or < 36°C
  - tachycardia
  - tachypnoea
  - elevated leukocyte count
PLUS
• one of the following:
  ○ cardiovascular dysfunction
  ○ acute respiratory distress syndrome
  ○ ≥ 2 other organ dysfunctions

Investigations
• blood culture and identify focus of infection e.g. osteomyelitis, abscess
• investigate for malaria especially in endemic areas or if there is a history of travel
• where meningitis due to meningococcus is suspected, i.e. with petechial rash, lumbar puncture is contra-indicated if the patient is shocked

NON-DRUG TREATMENT
• admit to high care area
• early recognition and treatment of septic shock

DRUG TREATMENT
Empiric antibiotic therapy
Choice of antibiotic depends on the severity of the condition and predisposing factors. Reconsider choice of antibiotic when the results of cultures become available or the child does not improve.
• cefotaxime, IV, 80 mg/kg/dose 8 hourly for 7–10 days
PLUS
• amikacin, IV, 15 mg/kg as single daily dose for 7–10 days
OR
• gentamicin, IV, 7.5 mg/kg/dose as single daily dose for 7–10 days

Suspected meningococcal septicaemia
• benzylpenicillin (Penicillin G), IV, 100 000 units/kg/dose immediately then 4 hourly

Suspected staphylococcal infection (e.g. osteomyelitis)
• cloxacillin, IV, 50 mg/kg/dose 6 hourly
PLUS
• cefotaxime, IV, 50 mg/kg/dose 6 hourly
• hydrocortisone, IV, 2 mg/kg/dose 6 hourly

REFERRAL
• septicaemia with complications
• patients requiring intensive care
Knowledge about HIV/AIDS is constantly being updated. Comprehensive guidelines are available for the use of ARV’s in children and the care of children with HIV infection – these are encompassed in the *Khomanani Document of National Antiretroviral Treatment Guidelines* and should be used for detail on the management of counseling and care of such children.

**DESCRIPTION**

Human Immunodeficiency Virus (HIV) is a retrovirus which infects human immune cells, especially T lymphocytes known as CD4 cells by inserting its RNA into the genetic code of the cell. It is replicated and released in large numbers into the body to infect other lymphocytes. This process results in the destruction of the lymphocytes it infects and the body loses its ability to fight off infections. Acquired Immune Deficiency Syndrome (AIDS) is advanced HIV disease where the body’s ability to fight infections is lost and is defined by the presence of selected defining opportunistic infections or measurable levels of loss of immune competence.

**DIAGNOSTIC CRITERIA**

Suspect HIV infection when risk factors and/or clinical features of symptomatic HIV infection are present.

- risk factors are:
  - exposure to infection from infected mothers
  - sexual abuse
  - adolescents having unprotected sexual encounters with multiple partners

- clinical features of symptomatic HIV infection:
  - persistent/recurrent ear discharge
  - recurrence of unusual pneumonia, e.g. *Pneumocystis jiroveci* (carinii) pneumonia (PCP)
  - low weight for age or unsatisfactory weight gain
  - persistent or recurrent diarrhoea for the past three months
  - enlarged lymph glands in two or more of the following sites: neck, axilla or groin
  - oral thrush
  - parotid gland swelling
  - liver enlargement
  - spleen enlargement
  - recurrent infections
  - severe progressive pneumonia
  - clubbing
  - progressive developmental delay
the combination of multiple problems may also suggest HIV infection

**confirmation of diagnosis**
- 2 separate tests on separate specimens are required to confirm diagnosis
  - two positive HIV Elisa tests > 18 months of age
  - < 18 months, HIV PCR testing should be performed in HIV Elisa positive children
    - Between 6 and 12 weeks of age the HIV PCR may rarely give false negatives results.
    - After 3 months the specificity and sensitivity of the HIV PCR is close to 100%.
  - negative tests do not exclude infection until 6 weeks to 3 months after cessation of breast feeding, birth or exposure to other risk of HIV infection

**Note:**
When testing for HIV be mindful of the implications concerning child, mother and the rest of the family unit. Counseling should be carried out with proper care.

**REFERRAL**
Health care professionals competent in managing HIV infections and its complications should manage all children with HIV infection. The level of referral will depend on the competence at each level.
- all children for antiretroviral assessment and treatment
- all complications of ARV's or failure of clinical improvement

**NON-DRUG TREATMENT**
- counseling
  - an extremely vital part of the successful care of children with HIV infection and their families
  - specific matters requiring attention are:
    - the implications of the disease in the family
    - implications of treatment and understanding of the condition and its care
  - on completion of counseling the family should be able to make informed decisions taking all this information into account
- nutritional advice and support – See Section 2.4.1

**DRUG TREATMENT**

**PROPHYLAXIS**
**Immunisation, deworming and vitamin A programmes**
Continue immunisation as in the normal child.
Do not give BCG after 6 weeks of age and specifically not to children with symptomatic HIV.
**Pneumocystis jiroveci (carinii) pneumonia (PCP) prophylaxis**

Indications:
- any infant born to an HIV-infected woman: start treatment at 4–6 weeks of age
- any infant who is identified as being HIV infected during the first year of life by a PCR test or by a clinical diagnosis of HIV infection with positive serology
- children with:
  - symptomatic HIV disease, or
  - an AIDS-defining illness (WHO category II and III), or
  - CD4 count < 15%
- trimethoprim/sulfamethoxazole, oral, 6-8 mg/kg/dose of trimethoprim component once daily. (8 mg = 1 mL)

When to stop prophylaxis
- HIV infected
  - indefinitely where ARV therapy is not yet available
  - if child is on ARV therapy only when evidence of immune restoration has occurred, i.e. the child is over 18 months and the CD4% > 15 at two measurements 6 months apart
- HIV exposed
  - once HIV infection has confidently been excluded
  - child < 18 months and not breast fed – negative virological HIV testing
  - child < 18 months – negative virological HIV testing 6 weeks after stopping of breastfeeding
  - child > 18 months – negative HIV antibody testing 3 months after stopping breastfeeding
- mother no longer breastfeeding and HIV infection is definitely ruled out

**Tuberculosis**

Actively exclude tuberculosis in all patients especially those in contact with an adult with pulmonary TB, before starting ARV therapy. Where TB has been excluded and the patient is in contact with a person who has TB, prophylaxis should be given.

If ARV therapy is necessary start immediately with
- isoniazid, oral, 1–7.5 mg/kg/dose 5 days a week for 6 months

If not on ARV therapy
- rifampicin/isoniazid 60/30, oral, 10–15 mg/kg/dose of rifampicin component, once daily for 5 days a week for 3 months
  Where rifampicin is used with ARV’s the use of lopinavir/ritonavir must be amended as indicated under TB treatment.

**Nutritional support**

Specific nutritional conditions should be treated appropriately – See Section 2.4
- multivitamin syrup, oral, 5 mL/dose once daily for 5 days per week
  - Syrup to contain vitamins A, B, C and D.
- ferrous gluconate, oral, 0.5 mL/kg/dose once daily for 5 days per week
  - If iron deficiency, See Section 3.4.
- folic acid, 2.5 mg/dose once daily for 5 days per week
TREATMENT
Antiretroviral Therapy (ART)

Do not rush into starting patients on ART.
Spend time on treatment plan.

The preparation of the child and family to start ART is critical to the success of the treatment. Failure to achieve adherence and understanding will lead to resistance and adversely affect both the child and the national outcome of the ARV programme. ARV’s are only used in sites accredited for their use.

Eligibility for Antiretroviral Therapy
- patients must satisfy clinical and social criteria before being accepted for treatment
- clinical criteria:
  - recurrent hospitalisations, > 2 admissions per year, for HIV complications, or
  - a prolonged hospitalisation for HIV, > 4 weeks, or
  - patient satisfies the WHO Stage III or IV disease, or
  - for relatively asymptomatic patients, one can consider:
    - if < 18 months CD 4 percentage < 20% of the total lymphocyte count
    - if > 18 months < 15% of the total lymphocyte count
- social criteria:
  - these criteria are extremely important for the success of the programme and need to be adhered to
  - the principle is that adherence to treatment must be at least probable
  - mandatory:
    - at least one identifiable caregiver who is able to supervise child and/or administer medication. All efforts should be made to ensure that the social circumstances of vulnerable children e.g. orphans be addressed so that they too can receive treatment.
    - disclosure to another adult living in the same house is encouraged so that there is someone else who can assist with the child’s treatment

Adherence
- adherence greater than 95% should be attained to ensure a good virological response and prevent the emergence of viral resistance
- good adherence can be achieved with regular education and support
- may be monitored using effective counseling and other measures
- all efforts to encourage this level of adherence should be made
# INTERIM WHO CLINICAL STAGING OF HIV/AIDS FOR INFANTS AND CHILDREN

For persons aged under 15 years with confirmed laboratory evidence of HIV infection:

- HIV antibody if aged 18 months and above
- virological or p24 antigen testing if under 18 months

### Clinical Stage 1
- asymptomatic
- persistent generalised lymphadenopathy (PGL)

### Clinical Stage 2
- hepatosplenomegaly
- papular pruritic eruptions
- seborrhoeic dermatitis
- extensive human papilloma virus infection
- extensive molluscum contagiosum
- fungal nail infections
- recurrent oral ulcerations
- lineal gingival erythema (LGE)
- angular cheilitis
- parotid enlargement
- herpes zoster
- recurrent or chronic RTI’s, i.e.
  - otitis media
  - otorrhoea
  - sinusitis

### Clinical Stage 3
Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations

- moderate unexplained malnutrition (between the 3rd percentile and 60% of expected weight) not adequately responding to standard therapy
- unexplained persistent diarrhoea (14 days or more)
- unexplained persistent fever (intermittent or constant, for longer than one month)
- oral candidiasis (outside neonatal period)
- oral hairy leukoplakia
- acute necrotising ulcerative gingivitis/periodontitis
- pulmonary TB
- severe recurrent presumed bacterial pneumonia
- conditions where confirmatory diagnostic testing is necessary
- chronic HIV-associated lung disease including bronchiectasis
- lymphoid interstitial pneumonitis (LIP)
- unexplained anaemia (< 8 g/dL), and or neutropaenia (< 500/mm³) and or thrombocytopaenia (< 50,000/mm³) for more than one month
Clinical Stage 4
Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations
- unexplained severe wasting or severe malnutrition not adequately responding to standard therapy
- pneumocystis pneumonia
- recurrent severe presumed bacterial infections, e.g.:
  - empyema
  - pyomyositis
  - bone or joint infection
  - meningitis
  but excluding pneumonia
- chronic herpes simplex infection; (orolabial or cutaneous of more than one month’s duration)
- extrapulmonary TB
- Kaposi’s sarcoma
- oesophageal candidiasis
- CNS toxoplasmosis (outside the neonatal period)
- HIV encephalopathy
Conditions where confirmatory diagnostic testing is necessary
- CMV infection (CMV retinitis or infections of organs other than liver, spleen or lymph nodes; onset at age one month of more)
- extrapulmonary cryptococcosis including meningitis
- any disseminated endemic mycosis, e.g.:
  - extrapulmonary histoplasmosis
  - coccidiomycosis
  - penicilliosis
- cryptosporidiosis
- isosporiasis
- disseminated non-tuberculous mycobacteria infection
- candida of trachea, bronchi or lungs
- visceral herpes simplex infection
- acquired HIV associated rectal fistula
- cerebral or B cell non-Hodgkin lymphoma
- progressive multifocal leukoencephalopathy (PML)
- HIV-associated cardiomyopathy or HIV-associated nephropathy

Treatment of mothers, caregivers and other family members
- always ask about the caregiver’s health, and the health of other members of the family
- ensure that mothers and other family members are able to access medical care timeously, including ART if needed
Requirements before ART is used
- within the child's family/environment parents, caregivers and children should understand:
  - that antiretroviral treatment is life-long
  - the prognosis of the condition
  - side effects of the medicines, their mode of action, and the risk and implications of developing resistance if incorrectly used.

ART Regimens
Regimens are chosen according to age, weight and prior antiretroviral exposure for 1st line care.
2nd line regimens are chosen according to the same criteria and depend on specific adverse events on the 1st line, or specified failure on the 1st line regimens.
Do not change regimens or move to 2nd line therapy without clear guidance from an experienced child ARV practitioner as unnecessary loss of effective regimens for a child is a life shortening outcome and must be avoided.

First line regimens:
**Option 1.1:**
Age 6 months to 3 years or < 10 kg
- stavudine, oral, 1 mg/kg/dose 12 hourly
  **PLUS**
  - lamivudine, oral, 4 mg/kg/dose 12 hourly
  **PLUS**
  - lopinavir/ritonavir 80/20, oral, 230 mg/m$^2$/dose of lopinavir component 12 hourly
    - Administer with food.
    - A high-fat meal increases absorption, especially of the solution.
    - If co-administered with didanosine, didanosine should be given 1 hour before or 2 hours after lopinavir/ritonavir.

**Option 1.2:**
Age > 3 years and > 10 kg
- stavudine, oral, 1 mg/kg/dose 12 hourly
  **PLUS**
  - lamivudine, oral, 4 mg/kg/dose 12 hourly
  **PLUS**
  - efavirenz, oral, 350 mg/m$^2$/dose as a single daily dose
    - OR
    - efavirenz, oral, as a single daily dose
      - | Weight (kg) | Dosage (mg) |
        |------------|------------|
        | 10–15 kg   | 200 mg     |
        | 15–20 kg   | 250 mg     |
        | 20–25 kg   | 300 mg     |
        | 25–32.5 kg | 350 mg     |
        | 32.5–40 kg | 400 mg     |
        | > 40 kg    | 600 mg     |
Second line regimens:

Option 2.1:
If previously on stavudine, lamivudine and lopinavir/ritonavir
- zidovudine, oral, 240 mg/m²/dose 12 hourly
**PLUS**
- didanosine, oral, 12 hourly
  - < 8 months 100 mg/m²/dose
  - > 8 months 120 mg/m²/dose
  Can be given as a single daily dose in older children.
  Do not give simultaneously with other ARV medication. Administer 2 hours before/after other ARV medication.

**PLUS**
If age < 3 years or < 10 kg
- nevirapine, oral, 120 mg/m²/dose as a single daily dose for 2 weeks, then 12 hourly if no rash or severe side effects
OR
If age > 3 years or > 10 kg
- efavirenz, oral, 350 mg/m²/dose as a single daily dose
OR
  efavirenz, oral, as a single daily dose
<table>
<thead>
<tr>
<th>Weight Range</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>10–15 kg</td>
<td>200 mg</td>
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<tr>
<td>15–20 kg</td>
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<td>&gt; 40 kg</td>
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Option 2.2:
If previously on stavudine, lamivudine and efavirenz
- zidovudine, oral, 240 mg/m²/dose 12 hourly
**PLUS**
- didanosine, oral, 12 hourly
  - < 8 months 100 mg/m²/dose
  - > 8 months 120 mg/m²/dose
  Can be given as a single daily dose in older children.
  Do not give simultaneously with other ARV medication. Administer 2 hours before/after other ARV medication.

**PLUS**
- lopinavir/ritonavir 80/20, oral, 230 mg/m²/dose of lopinavir component 12 hourly
  Administer with food.
  A high-fat meal increases absorption, especially of the solution.
  If co-administered with didanosine, didanosine should be given 1 hour before or 2 hours after lopinavir/ritonavir.
General comments

- Where no refrigerator is available the following can be used:
  - stavudine capsules can be used instead of suspension. The capsules can be opened and the contents suspended in 10 mL of water and the required amount administered to the child. Shake well to resuspend for the second dose 12 hours later. Discard the rest.
  - didanosine tablets can be dissolved in at least 30 mL of water. It is important to use 2 tablets didanosine e.g. if child needs 100 mg prescribe 2 x 50mg tablets.
  - lopinavir/ritonavir capsules and suspension needs to be kept cool at < 25º C. Use insulated container/cooler box where temperature is > 25º C.
- switch to tablets or capsules from syrups or solutions as soon as possible
- didanosine must be taken alone, on an empty stomach, at least an hour before or 2 hours after a meal
- children may occasionally need to change a drug from the first line regimen to one from the second line regimen because of intolerance or a serious adverse reaction. The decision to swap must be made by a doctor with antiretroviral experience by telephonic consultation.
- dosage of antiretroviral therapy should be adjusted according to weight during follow up visits
- treatment failure on ARV is defined by clinical criteria, CD4 counts and viral loads. If the CD4 counts decreases or viral load increases or the child has growth faltering, neurodevelopment regression or recurrent opportunistic infections treatment failure should be suspected and adherence checked. If the abnormalities persist then the child should be referred for evaluation of drug resistance.
- if second line therapy fails seek advice without stopping therapy

Tuberculosis and ARV Treatment

TB and HIV are often comorbid conditions. Exclude tuberculosis before starting ART. First treat TB before initiating ARV’s unless special circumstances warrant the risks involved in concomitant treatment.

When initiating ART, beware of interactions of TB Treatment and ARV’s, and the risk of Immune Reconstitution Inflammatory Syndrome (IRIS).

Management of TB and HIV

Child presents with tuberculosis or tuberculosis is likely and cannot be excluded prior to commencing antiretroviral therapy

Complete TB therapy if possible before commencing ART or delay ART for at least 2 months. If the child needs to take concomitant TB and ARV treatment and the regimen includes efavirenz, stavudine and lamivudine

- no adjustment of dosages are required
  - Be alert for Immune Reconstitution Inflammatory Syndrome.
If the child needs to take concomitant TB and ARV treatment and the regimen includes lopinavir/ritonavir, stavudine and lamivudine:

Use ritonavir instead of lopinavir/ritonavir

- ritonavir, oral, 250 mg/m²/dose 12 hourly.
  Increase dose by 50 mg/m²/dose every 2–3 days up to 400 mg/m²/day.
  If < 2 years old increase up to 450 mg/m²/day.
  Take with food and 2 hours apart from didanosine.

**OR**

provide additional ritonavir to the dosage level of lopinavir in the fixed combination of lopinavir/ritonavir while on TB therapy

No adjustment of dosages for stavudine or lamivudine is required. Be alert for IRIS.

**Child develops tuberculosis while on antiretroviral therapy**

If the child is on lopinavir/ritonavir, or other protease inhibitor:
switch to ritonavir at full dose. Start directly at full dose of ritonavir if already on lopinavir/ritonavir.

If the child is unable to tolerate the large number of drugs:
ART may have to be interrupted until TB therapy has been completed – consult an expert for advice.

### Specific Information on ARVs

<table>
<thead>
<tr>
<th>Storage</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nucleoside Reverse Transcriptase Inhibitors (NRTIs)</strong></td>
<td></td>
</tr>
<tr>
<td>zidovudine</td>
<td>room temperature</td>
</tr>
<tr>
<td>didanosine</td>
<td>refrigerate suspension</td>
</tr>
<tr>
<td>stavudine</td>
<td>refrigerate suspension</td>
</tr>
<tr>
<td>lamivudine</td>
<td>room temperature</td>
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<td></td>
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<tr>
<td><strong>Non-nucleoside Reverse Transcriptase Inhibitors (NNRTIs)</strong></td>
<td></td>
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<tr>
<td>nevirapine</td>
<td>room temperature</td>
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<td></td>
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</table>
### Specific Information on ARVs

<table>
<thead>
<tr>
<th>Protease Inhibitors (PIs)</th>
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<tbody>
<tr>
<td>ritonavir</td>
<td></td>
<td>• bitter taste</td>
</tr>
<tr>
<td>lopinavir/ritonavir</td>
<td>oral solution and capsules should be refrigerated. can be kept at room temperature up to 25°C if used within 2 months</td>
<td>• nausea • vomiting • diarrhoea</td>
</tr>
</tbody>
</table>

### Caution

All children with severe skin reaction to nevirapine should never be re-challenged with nevirapine.
## Important Side Effects of ARV’s

<table>
<thead>
<tr>
<th>Side Effect</th>
<th>Monitoring/Action</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Continue ART with careful monitoring.</strong></td>
<td>Consider stopping treatment <strong>URGENTLY.</strong> Consult expert.</td>
</tr>
<tr>
<td><strong>Consider single drug replacement with expert advice.</strong></td>
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</tr>
</tbody>
</table>

- **Lactic acidosis**
  - Lactate 2–5 mmol/L with no signs or symptoms
  - Lactate > 5 mmol/L, or with signs or symptoms or acidosis

- **Anaemia**
  - Hb = 7.0–9.9 g/dL
  - Hb < 7g/dL or cardiac failure

- **Neutropaenia**
  - 0.4–1.2 X 10⁹/L
  - ≤ 0.399 X 10⁹/L

- **Increase liver enzymes and hepatitis**
  - ≤ 9.9 X upper normal limit
  - ≥ 10.0 X upper normal limit

- **Increased serum lipids**
  - 1.54–8.46 mmol/L
  - ≥ 8.47 mmol/L

- **Increased cholesterol**
  - 4.43–12.92 mmol/L
  - ≥ 12.93 mmol/L

- **Severe skin reactions**
  - Diffuse maculopapular rash, or dry desquamation
  - Vesiculation, or ulcers, or exfoliative dermatitis, or Stevens-Johnson syndrome, or erythema multiforme, or moist desquamation, or with elevated ALT or AST

- **Peripheral neuropathy**
- **Myopathy**
- **Abdominal pain**
- **Nausea and vomiting**
- **Pancreatitis**
- **Headache**
- **Fatigue**
- **Sedative effect**
- **Sleep disturbance**
- **Confusion**
- **Abnormal thinking**
- **Probably teratogenic**

- **Clinical evaluation:**
  - Discuss all cases with a paediatrician with antiretroviral experience, before interrupting therapy.
## 9.1.1 MONITORING OF EFFICACY AND SAFETY

Monitoring of the effectiveness, safety of the regimen and their adverse effects is essential. Laboratory monitoring depends on the regimen chosen and includes baseline and preparation assessments and tests.

<table>
<thead>
<tr>
<th>Regimen</th>
<th>Test</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>stavudine/lamivudine/lopinavir+ritonavir</td>
<td>• CD4</td>
<td>• staging, 6-monthly</td>
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<tr>
<td></td>
<td>• VL</td>
<td>• baseline, 6-monthly</td>
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<tr>
<td></td>
<td>• fasting cholesterol</td>
<td>• baseline, 6-monthly</td>
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<tr>
<td></td>
<td>• fasting glucose</td>
<td>• baseline, 6-monthly</td>
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<tr>
<td></td>
<td>• fasting triglycerides</td>
<td>• baseline, 6-monthly</td>
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<tr>
<td>didanosine/zidovudine/nevirapine</td>
<td>• CD4</td>
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<td></td>
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<td>• baseline, 6-monthly</td>
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<td></td>
<td>• FBC</td>
<td>• baseline, then monthly for 3 months, then 6 monthly (with CD4 and viral load) thereafter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• baseline, week 2, 4 and 8, thereafter 6 monthly</td>
</tr>
<tr>
<td></td>
<td>• ALT</td>
<td></td>
</tr>
<tr>
<td>didanosine/zidovudine/efavirenz</td>
<td>• CD4</td>
<td>• 6-monthly</td>
</tr>
<tr>
<td></td>
<td>• FBC</td>
<td>• baseline, then monthly for 3 months, then 6 monthly (with CD4 and viral load) thereafter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• 6-monthly</td>
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<tr>
<td></td>
<td>• ALT</td>
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</tbody>
</table>
9.2 SPECIFIC ADVERSE EVENTS AND COMPLICATIONS

9.2.1 LACTIC ACIDOSIS
E87.2

DESCRIPTION
All nucleoside analogues have been associated with lactic acidosis which rare but life threatening. Initial symptoms are variable and may occur as early as 1 month after starting therapy. Most frequently associated with didanosine and stavudine combinations.

DIAGNOSTIC CRITERIA

Clinical
- clinical prodromal syndrome:
  - generalised fatigue
  - weakness
  - gastrointestinal symptoms:
    - nausea
    - vomiting
    - diarrhoea
    - abdominal pain
    - hepatomegaly
    - anorexia
    - and/or sudden unexplained weight loss
  - respiratory symptoms: tachypnoea and dyspnoea
  - neurologic symptoms, including motor weakness

Special investigations
- laboratory abnormalities:
  - hyperlactataemia
    - moderate abnormal: 2–5 mmol/L
    - severe abnormal: 5–10 mmol/L
    - very severe abnormal: > 10 mmol/L
  - anion gap may be increased
  - measurable acidosis is a severe finding
- lactic acidosis is defined by:
  - lactate > 5 mmol/L
  - bicarbonate < 20 mmol/L
  - severe acidosis, i.e. pH < 7.3
  - increased anion gap
  - associated symptomology

TREATMENT
- obtain expert advice urgently
- discuss management with a treatment expert
- in patients with symptoms and increased lactate levels treatment should be stopped pending this advice
- symptoms associated with lactic acidosis may continue or worsen following discontinuation of antiretroviral therapy
- treatment is primarily supportive, consisting of maintenance fluid, bicarbonate administration to half correct acidosis and respiratory support

REFERRAL
- for adjustment of regimen
9.2.2 LIPODYSTROPHY/ENDOCRINOPATHIES IN HIV INFECTED CHILDREN

See Section 7.14

9.2.3 IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

DESCRIPTION
Paradoxical clinical deterioration after starting HAART due an improvement in the immune system response to organisms that have colonised the body, e.g.

- *M. tuberculosis* (MTB)  
- *M. avium complex*  
- *M. leprae*  
- *P. jiroveci*  
- CMV  
- JC virus

Other organisms include:

- *C. neoformans*
- Aspergillus
- *C. albicans*
- Human Herpes viruses
- Human Papilloma virus
- Hepatitis B and C viruses (HBV, HCV)

DIAGNOSTIC CRITERIA

- presentation:
  - usually during the first 6 weeks after starting HAART
  - clinical presentation depends on the causative organism and the organ-system colonised, e.g. TB presents with: fever, lymphadenopathy, worsening of the original tuberculous lesion, and/or deteriorating chest radiographic manifestations such as miliary pattern or pleural effusion

TREATMENT

Obtain expert advice urgently.

- antimicrobial therapy for specific infections

In severe reactions

- hydrocortisone, IV, 3–6 mg/kg/dose 3–6 hourly on first day
Follow with

- prednisone, oral, 1–2 mg/kg/dose 24 hourly

Temporary discontinuation of antiretroviral agents may be considered.

9.2.4 WASTING SYNDROME

This syndrome appears to be a combination of the direct effects of advanced HIV infection and the occurrence of opportunistic infections.

TREATMENT

- nutritional advice – see chapter on nutrition
- ART may reverse some of the features of HIV wasting syndrome
- exclude chronic infection, e.g. tuberculosis and *M. avium complex*, malabsorption and malignancy
9.3 POST EXPOSURE PROPHYLAXIS FOLLOWING ALLEGED PENETRATIVE SEXUAL ABUSE

See Section 6.2.5: Sexual abuse and prevention of infection/conception
DESCRIPTION
Surgical prophylaxis is the peri-operative and/or intra-operative administration of antibiotics to patients to reduce the risk of post-operative infection. Specific epidemiological considerations may alter the choice of agents.

PRINCIPLES OF SURGICAL PROPHYLAXIS
• the need for prophylactic antibiotic therapy is based on the risk of wound contamination
• antibiotic prophylaxis is not required for clean operations/procedures in immunocompetent patients, who have minimal risk of contamination. In all other situations, prophylaxis should be considered
• the medication chosen should be active against the pathogens most likely to be associated with wound infections
• prophylaxis must be given within 30 minutes of induction – usually at induction of anaesthesia

The prophylactic dose is a single dose equal to the standard therapeutic dose.
A second dose is ONLY given if surgery is prolonged, i.e. > 4 hours for cefazolin OR > 8 hours for metronidazole

ANTIBIOTIC TREATMENT
• cefazolin, IV, 25 mg/kg, 30 minutes before the procedure
  Maximum dose: 1 000 mg
  AND
  For lower limb amputation, colorectal, appendix, biliary and pelvic surgery
  • metronidazole, IV, 7.5 mg/kg
  AND
  If high risk of gram negative contamination, e.g. perforated intestines, urogenital, hepatobiliary
  • gentamicin, IV, 5 mg/kg

  For eye surgery
  • chloramphenicol ophthalmic drops

Penicillin allergy
• clindamycin, IV, 10 mg/kg
  AND
  If abdominal viscus involved
  • gentamicin, IV, 5 mg/kg
11.1 ARTHRITIS, SEPTIC (PYOGENIC)

DESCRIPTION
Septic or pyogenic arthritis is often part of a generalised septicaemia which may involve more than one joint and is caused by pyogenic micro-organisms. The organisms involved vary:

<table>
<thead>
<tr>
<th>Group</th>
<th>Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>S. aureus, Group B Streptococci, E. coli, fungi</td>
</tr>
<tr>
<td>Infants/children</td>
<td>S. aureus, H. influenzae, Group A Streptococci S. pneumonia</td>
</tr>
<tr>
<td>Adolescents - sexually active</td>
<td>N. gonorrhoea</td>
</tr>
<tr>
<td>Chronic septic arthritis</td>
<td>Brucella, tuberculosis, atypical mycobacteria, fungi and other uncommon organisms</td>
</tr>
</tbody>
</table>

DIAGNOSTIC CRITERIA
The diagnosis is largely clinical and confirmed by finding pus in the joint space.

Clinical
- fever, local pain, loss of function and general toxicity
- subtle, non-specific signs of sepsis early in the course of the disease, especially in neonates
- local tenderness, warmth, swelling at a joint with restriction of passive and active movement
- malaise, irritability, feeding problems and pseudo-paralysis
- if lower extremities are involved, development of a limp or refusal to bear weight

Investigations
- aspiration of pus from joint space under ultrasound guidance if possible
- blood and aspirated pus for culture and sensitivity
- raised white-cell count and sedimentation rate

NON-DRUG TREATMENT
- septic arthritis of the hip (emergency) requires prompt open surgical drainage at the time of presentation
- most infections of other sites may be managed by repeated aspiration or open drainage (not antibiotic instillation) if frank pus is obtained on initial diagnostic aspiration
- immobilise affected limb in position of function
- identify other effects of septicaemia / haematogenous spread and treat appropriately
- supportive and symptomatic care

CAUTION
Do not carry out needle aspiration in haemophiliacs.
DRUG TREATMENT

Antibiotic therapy
Minimum duration of therapy is 4–6 weeks.

IV antibiotics
As soon as diagnosis is made, collect blood and pus specimens for microscopy, Gram stain, culture and sensitivity.
Start IV antibiotics immediately.
Review antibiotic choice when culture and sensitivity results become available or if response to antibiotic treatment is unsatisfactory.
IV antibiotics should continue until there is evidence of good clinical response and laboratory markers of infection improve (usually about 2 weeks). Oral antibiotics may then be considered.

Neonates
- cloxacillin, IV, 50 mg/kg/dose,
  - 1st week of life 12 hourly
  - 2nd–4th week 8 hourly
  - > 4 weeks 6 hourly
PLUS
- cefotaxime, IV, 50 mg/kg/dose,
  - preterm 12 hourly
  - 1st week life 8 hourly
  - > 2 weeks 6 hourly

Infants and children
- cloxacillin IV 50 mg/kg/dose, 6 hourly
PLUS
- cefotaxime IV 25–50 mg/kg/dose, 6 hourly

Special Circumstances
If very small neonates or neonates with central lines and if MRSA is likely on the basis of unit experience - replace cloxacillin with vancomycin.
- vancomycin IV, 10 mg/kg/dose 6 hourly, infused over 1 hour

Penicillin allergy – replace cloxacillin with clindamycin
- clindamycin, IV, 10 mg/kg/dose, 8 hourly

Specific culture – treat appropriately to sensitivities

Oral antibiotics
Weekly sedimentation rate should be done. A deterioration of sedimentation rate indicates non-absorption of the oral form.
Continue with oral antibiotics until no signs of infection and white cell count/ sedimentation rate are back to normal.
Antibiotics according to sensitivities. If resistant none are known consider:
• flucloxacillin, oral, 12.5–25 mg/kg/dose, 8 hourly
PLUS
• amoxicillin, oral, 30 mg/kg/dose, 8 hourly

Symptomatic antipyretic and anti-inflammatory therapy
• ibuprofen, oral, 5–10 mg/kg/dose, 6 hourly
  Note:
  Safety of ibuprofen has not been established in children under 2 years and is not recommended for children under 5 years.

Analgesia
See Chapter 20.

REFERRAL
• multi-organ involvement
• failure to achieve progressive improvement on treatment

11.2 ARTHRITIS, JUVENILE IDIOPATHIC
See Section 12.2: Juvenile rheumatoid arthritis (JRA)/ Juvenile idiopathic arthritis (JIA)

11.3 OSTEITIS/OSTEOMYELITIS, ACUTE
M86.1

DESCRIPTION
Osteomyelitis is an infection of the bone, often part of a generalised septicaemia which may involve more than one bone. The organisms involved vary:

<table>
<thead>
<tr>
<th>Description</th>
<th>Organisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>S. aureus, Group B Streptococci, Gram negative (E. coli)</td>
</tr>
<tr>
<td>Infants/children</td>
<td>S. aureus, H. influenzae, Group A Streptococci</td>
</tr>
<tr>
<td>Traumatic direct infection</td>
<td>P. aeruginosa (penetrating foot wounds)</td>
</tr>
<tr>
<td>Co-existing medical conditions</td>
<td>M. tuberculosis, fungi</td>
</tr>
<tr>
<td>Sickle cell disease</td>
<td>Salmonella</td>
</tr>
</tbody>
</table>

DIAGNOSTIC CRITERIA
Clinical
• local pain and tenderness, loss of function, general toxicity and fever
• if lower extremities are involved, development of a limp or refusal to bear weight occurs
• in neonates, early signs may be subtle or non-specific, e.g. irritability, feeding problems, pseudoparalysis
• investigate for multi-organ disease, e.g. endocarditis, pericarditis and pneumonia
Investigations
Diagnostic
• aspiration of pus for microscopy, Gram stain, culture and sensitivity
• blood culture and full blood count
• raised white-cell count and sedimentation rate
The following may be helpful:
• X-ray after 2 weeks
• radionuclide examination (Tc99*)
• MRI

NON-DRUG TREATMENT
• Surgical drainage if:
  ○ frank pus is aspirated from bone
  ○ evidence of clear progression to soft tissues
  ○ when a marked improvement has not occurred within 24-36 hours on adequate IV antibiotic treatment
  ○ associated with septic arthritis
• immobilise affected limb in position of function
• supportive and symptomatic care

DRUG TREATMENT
Antibiotic therapy
Minimum duration of therapy is 4–6 weeks.

IV antibiotics
Start IV antibiotics immediately as soon as diagnosis is made and blood and pus specimens have been collected for microscopy, Gram stain, culture and sensitivity.
Review antibiotic choice when culture and sensitivity results become available or if response to antibiotic treatment is unsatisfactory.
Where a single agent has been found to be sensitive, continue treatment on that single agent.
IV antibiotics should continue until there is evidence of good clinical response and laboratory markers of infection improve (usually about 2 weeks). Oral antibiotics may then be considered.
Ongoing fever could suggest an undrained focus of pus.

Neonates
• cloxacillin, IV, 50 mg/kg/dose,
  1st week of life 12 hourly
  2nd–4th week 8 hourly
  > 4 weeks 6 hourly
PLUS
• cefotaxime, IV, 50 mg/kg/dose,
  Preterm 12 hourly
  1st week life 8 hourly
  > 2 weeks 6 hourly
Infants and children
- cloxacillin, IV, 50 mg/kg/dose, 6 hourly
  **PLUS**
- cefotaxime, IV, 50 mg/kg/dose, 6 hourly

Special Circumstances
*Methicillin resistant staphylococci infection* - replace cloxacillin with vancomycin
- vancomycin, IV, 10 mg/kg/dose, 6 hourly, infused over 1 hour

*Penicillin allergy* - replace cloxacillin with clindamycin
- clindamycin, IV, 10 mg/kg/dose, 8 hourly

*Penetrating foot bone injuries* – replace cefotaxime with ceftazidime plus aminoglycoside
- ceftazidime, IV, 15–25 mg/kg/dose, 8 hourly
  **PLUS**
- gentamicin, IV, 7.5 mg/kg once daily

**Oral antibiotics**
Weekly sedimentation rate should be done. A deterioration of sedimentation rate indicates non-absorption of the oral form.
Continue with oral antibiotics until no signs of infection and white cell count/ sedimentation rate are back to normal.

Antibiotics according to sensitivities. If resistant consider:
- flucloxacillin, oral, 12.5–25 mg/kg/dose, 6 hourly
  **PLUS**
- amoxicillin, oral, 30 mg/kg/dose, 8 hourly

**Symptomatic antipyretic and anti-inflammatory therapy**
- ibuprofen, oral, 5–10 mg/kg/dose, 6 hourly
  **Note:**
  Safety of ibuprofen has not been established in children under 2 years and is not recommended for children under 5 years.

**Analgesia**
Refer to pain chapter

**REFERRAL**
- multi-organ involvement
- failure to achieve progressive improvement on treatment
12.1 HENOCH SCHÖNLEIN PURPURA (HSP)
D69.0

DESCRIPTION
HSP is an acute leucoclastic vasculitis of small blood vessels usually involving skin, gastrointestinal tract, joints and the kidney. Aetiology is unknown.

Complications include:
• acute severe abdominal pain
• nephritis with renal impairment or nephrotic syndrome
• CNS involvement

DIAGNOSTIC CRITERIA
Clinical
• syndrome consisting of :
  ○ purpuric skin rash with a very typical distribution on lower extremities and buttocks Trunk and upper extremities may be involved. It begins as a wheal or erythematous macule/papule, which develops into red-purple palpable purpura.
  ○ arthritis in 60–70% of cases: mostly of large joints, i.e. knees and ankles
  ○ abdominal pain with colic: may develop haematemesis or intussusception or infarction
  ○ renal involvement in 25–50% with haematuria or proteinuria
  ○ angio-oedema of scalp, eyelids, lips and ears
  ○ rarely CNS involvement: seizures, paresis or coma

Investigations
• no diagnostic test
• FBC is usually normal but it is necessary to rule out other conditions with thrombocytopenic purpura
• coagulation studies are usually normal
• urine tests strips to evaluate renal involvement
• serum urea, creatinine, electrolytes and albumin with renal involvement

NON-DRUG TREATMENT
• short period of immobilisation during acute arthritis
• soft diet for acute gastrointestinal involvement

DRUG TREATMENT
For arthritis, oedema, fever, malaise
• ibuprofen, oral, 10 mg/kg 3 times daily

For complicated HSP
• prednisone, oral, 1–2 mg/kg/24 hours in 3 divided doses for 10 days
For patients with rapidly progressive glomerulonephritis
Immunosuppressive treatment, e.g. cyclophosphamide, should be given to control progression of disease and to halt deterioration of renal function. Specialist initiated. All children with persistent proteinuria and/or renal impairment. See Chronic Renal Failure: Section 6.1.5.

REFERRAL
- HSP with complications
- patients with:
  - rapidly declining renal function, or
  - persistent nephrotic range proteinuria, or
  - persistent macroscopic haematuria for kidney biopsy to plan immunosuppressive treatment

12.2 JUVENILE RHEUMATOID ARTHRITIS (JRA)/JUVENILE IDIOPATHIC ARTHRITIS (JIA)

DESCRIPTION
Juvenile rheumatoid arthritis is a chronic non-suppurative inflammatory condition of synovium. Different clinical subgroups are recognised according to the pattern of onset:

- **Systemic onset**
  - extra articular features are most striking
  - characteristic spiking fever and erythematous macular rash
  - serositis, i.e. pericarditis and pleuritis
  - hepatosplenomegaly and lymphadenopathy
  - 50% of patients will have destructive polyarthritis with poor response to treatment

- **Pauciarticular**
  - typical patient is a pre-school girl
  - involves the large joints, i.e. wrists, knees, ankles or elbows
  - often asymmetrical distribution
  - ≤ 4 joints are involved
  - prognosis is good, depending on management
  - 15–30% develop chronic iridocyclitis, which is asymptomatic, but eventually may lead to severe visual impairment/blindness
  
  There is an increased risk of iridocyclitis/uveitis in patients with positive antinuclear antibodies.
  
  All children with pauciarticular disease must be examined at each visit and may need slit lamp examinations 3–4 times yearly for at least the first 5 years of disease.
  - a subgroup will develop polyarthritis, i.e. > 4 joints affected, which is then classified as extended oligo-articular JIA
• **Polyarthritis (Rheumatoid factor negative)**
  ○ affects ≥ 5 joints in first 6 months of disease
  ○ typical patient is a pre-school girl
  ○ symmetric arthritis of multiple joints typically including small joints of the hands
  ○ temporomandibular joints and cervical spine may become involved later on
  ○ onset may be insidious with gradual development of joint stiffness, swelling and loss of motion, or fulminant, with sudden appearance of symptomatic arthritis

• **Polyarthritis (Rheumatoid factor positive)**
  ○ affects ≥ 5 joints in first 6 months
  ○ involves large and small joints
  ○ follows pattern of adult RA with nodules and bony erosions
  ○ aggressive form of disease with chronic course persisting into adulthood

• **Enthesitis related arthritis** (HLA B27 positive or family history thereof)
  ○ mostly pre-teen and teenage boys
  ○ onset of arthritis in boy > 8 years
  ○ asymmetrical arthritis of lower limb joints and enthesitis
  ○ enthesitis, presenting with heel pain, plantar fasciitis, Achilles tendonitis, pain at bases of 1st and 5th metatarsals and tibial tuberosity
  ○ sacroiliac joint tenderness and inflammatory spinal pain
  ○ anterior uveitis associated with pain, redness and photophobia
  ○ family history of arthritis, bad backs or ankylosing spondylitis
  ○ associated with inflammatory bowel disease

**DIAGNOSTIC CRITERIA**

**Clinical**

- exclude other forms of arthritis
- age of onset < 16 years
- arthritis in one or more joints
- duration > 6 weeks
- any of the patterns of onset

**Differential diagnosis**

- JRA is a clinical diagnosis and depends on the persistence of arthritis or typical systemic manifestations for ≥ three consecutive months and excluding other diseases:
  ○ pyogenic and tuberculous joint infection and osteomyelitis
  ○ arthritis associated with other acute infectious illnesses
  ○ acute leukaemia and other malignancies
  ○ acute rheumatic fever
  ○ auto immune disorders, SLE or mixed connective tissue disease
  ○ Reiter syndrome, i.e. arthritis, urethritis and conjunctivitis
  ○ ulcerative colitis or arthritis associated with enteritis
Investigations
- there is no diagnostic test
- full blood count: differential and platelet count
- bone marrow aspiration must be done before starting disease modifying drugs, including steroid therapy and methotrexate
- C-reactive protein and erythrocyte sedimentation rate
- serum urea, creatinine and electrolytes
- muscle enzymes, albumin, calcium, phosphate and alkaline phophatase
- auto-antibodies, complement C₃ and C₄, rheumatoid factor, IgG and IgA levels
- plain X–ray
- specialist may advise arthroscopy, synovial biopsies, ultrasound and CT scan in appropriate circumstances

NON-DRUG TREATMENT
- occupational and physiotherapy are essential to provide:
  - exercises to increase range of movements of joints and to maintain muscle strength
  - daily exercise programmes, hot water baths, swimming pool exercises
  - splints, e.g. nocturnal splints, for pain relief and prevention of contractures
  - shoe inserts/raises
  - advice on aids for activities of daily living
  - orthodontic treatment if joints of jaw are involved

DRUG TREATMENT
NSAID, e.g.:
- ibuprofen, oral, 10 mg/kg/dose 3–4 times daily
  Efficacy is determined within weeks to months unless there is aggressive progression or severe adverse effects, i.e. gastric irritation, peptic ulcer, hepatic toxicity, renal impairment or platelet dysfunction.

If arthritis is not adequately controlled
ADD
- methotrexate, oral, 0.3 mg/kg/week as a single dose on an empty stomach. Specialist initiated.
  Increase at monthly intervals up to 1 mg/kg/week until there is satisfactory response.
  Maximum dose: 25 mg/week.
  Adverse effects include: nausea, mood changes, raised liver enzymes, bone marrow toxicity and protein/haematuria.
  Monitor FBC, LFT, U&E and urine test strips monthly.
PLUS
- folic acid, oral, 5 mg daily for the duration of the treatment

If arthritis still not controlled and to control acute flare
ADD
- prednisone, oral, 2 mg/kg as a single daily dose for 1–2 weeks. Specialist initiated.
  Continue with 0.3–0.5 mg/kg/day as single dose.
  Try to wean off over next 3 months.

Disease modifying drugs
All patients requiring disease-modifying drugs must be referred to specialist rheumatologists.
REFERRAL
- all for confirmation of diagnosis
- patients with pauciarticular disease for slit lamp examination, if not locally available
- patients with iridocyclitis
- all with complicated JRA or uncontrolled disease
- adverse reaction to NSAID
- for orthopaedic or orthodontic treatment

12.3 KAWASAKI SYNDROME
M30.3

DESCRIPTION
Kawasaki syndrome is an acute self-limiting vasculitis of unknown aetiology occurring predominantly in children. It involves the small and medium arteries.

DIAGNOSTIC CRITERIA
Clinical
- the diagnosis is confirmed by the presence of fever for ≥ 5 days and the lack of another known disease process to explain the illness and the presence of 4 of the 5 criteria listed below:
  1. bilateral non supplicative conjunctival infection
  2. changes of the mucous membranes of the upper respiratory tract: reddening of the pharynx and lips, fissured lips, reddening of the oral mucosa and strawberry tongue
  3. polymorphous rash, primarily on the trunk
  4. acute non-purulent swelling of a cervical lymph node >1.5 cm
  5. changes of the extremities, including reddening of the palms and soles, oedema of the hands and/or feet and desquamation from the finger tips
- there is no diagnostic test
- important differential diagnosis which must be excluded:
  o aseptic/bacterial meningitis
  o viral or drug eruption
  o bacterial adenitis
  o diseases mediated by staphylococcal or streptococcal toxins
  o rickettsial diseases
  o urinary tract infection

Investigations
- C-reactive protein
- full blood count: increased white blood cell count 12 000–40 000 with left shift and thrombocytosis
- urine MCS: transient pyuria
- elevated erythrocyte sedimentation rate
- echocardiography detects coronary artery aneurysms: 100% sensitivity, 97% specificity

NON-DRUG TREATMENT
- routine supportive care
- tepid sponging for fever
- copious oral fluid to maintain hydration
DRUG TREATMENT
Within first 10 days from onset of fever
- immunglobulin, IV, 2 g/kg as a single dose infused over 12 hours
  Monitor fluid balance to prevent volume overload.

PLUS
- aspirin soluble, oral, 20–25mg mg/kg/dose 6 hourly in acute stage
  Once patient is afebrile for 3–7 days: decrease to single daily dose of 3 mg/kg/day.
  Continue for 4–6 weeks.

REFERRAL
- all patients for confirmation of diagnosis
- for echocardiography to confirm presence of coronary artery aneurysms

12.4 SYSTEMIC LUPUS ERYTHEMATOSUS
M32

DESCRIPTION
Systemic lupus erythematosus (SLE) is an auto-immune disease with auto-antibodies directed against a number of self components causing widespread vasculitis characterised by fibrinoid necrosis of the vessel wall.
It manifests clinically in multisystem organ damage. In children it predominantly involves kidneys, central nervous system, skin and joints.
Control of acute lupus depends on severity of illness, with more aggressive treatment for CNS, renal and haematologic involvement.

DIAGNOSTIC CRITERIA
Clinical
Diagnosis may be elusive due to its variations in presentation and is confirmed with at least 4 of 11 criteria:
1. malar rash
2. discoid rash
3. photosensitivity
4. oral ulcers
5. non-erosive arthritis
6. pleuro-pericarditis
7. renal disease, i.e. proteinuria and/or cellular casts
8. neurologic disorder, i.e. seizures or psychosis in the absence of precipitating circumstances
9. haematologic disorder: haemolytic anaemia, leucopaenia, lymphopaenia, thrombocytopaenia
10. immunologic disorder
   a) anti-DNA antibody
   b) anti-smooth muscle antibody
   c) positive antiphospholipid antibodies
   d) false positive antitreponema test
11. positive anti-nuclear antibody test
Investigations

Lack of urinary sediment changes do not exclude active ongoing glomerulonephritis, especially interstitial nephritis.

- urine tests strips: haematuria and proteinuria
- urine microscopy: cell casts
- full blood count: differential and platelet count
- complement, antinuclear antibodies
- serum urea, creatinine, electrolytes, albumin and cholesterol
- clotting profile, anti-phospholipid antibody and lupus anti-coagulant
- electrocardiography and chest X-ray
- refer for kidney biopsy

NON-DRUG TREATMENT
- avoid exposure to sunlight: limit outdoor activity
- physiotherapy to relieve arthralgia
- maintain adequate nutrition
- psychological support
- cosmetic management

DRUG TREATMENT
For mild disease without nephritis, NSAID e.g.:
- ibuprofen, oral, 10 mg/kg/dose 6 hourly

To control acute active SLE, after confirmation of diagnosis
- cyclophosphamide, IV bolus, 500–750 mg/m$^2$/dose. Specialist initiated.
  Repeat monthly for 6 months
  OR
cyclophosphamide, oral, 2.5 mg/kg/day for 12 weeks. Specialist initiated.
PLUS
- prednisone, oral, 2 mg/kg/day as single dose in the morning
  Once disease is under control, taper dose slowly.
  Attempt to decrease dose to 0.25 mg/kg/day
  Steroids should not be stopped completely within 3 years of diagnosis.

REFERRAL
- all patients for confirmation of diagnosis and initiation of treatment
- reno-protective treatment, all patients with lupus nephritis, including children with positive urine test strips for proteinuria, haematuria, hypertension, elevated S-urea and S-creatinine
12.5 TAKAYASU ARTERITIS

DESCRIPTION
Takayasu arteritis is a chronic inflammatory disease involving the aorta, arterial branches from the aorta and the pulmonary vasculature. Lesions are typically segmental – obliterative and aneurysmal.

DIAGNOSTIC CRITERIA
Clinical
- unexplained significant hypertension with no obvious kidney disease
- any bruits/discrepancy in pulses
- any signs of unexplained inflammatory activity
- strongly positive PPD
- increased plasma renin
- discrepancy of kidney sizes

Investigations
- C-reactive protein
- erythrocyte sedimentation rate
- plasma renin
- serum urea, creatinine and electrolytes
- PPD to exclude tuberculosis
- electrocardiography and chest X-ray
- radio-isotope study of kidneys to demonstrate split renal function

NON-DRUG TREATMENT
- there is a strong association with overweight and high blood pressure
  The majority of these patients have mild hypertension and usually only need lifestyle modification.
- acute hypertension:
  - bed rest – Fowler’s position
  - control fluid intake and output (restriction)
  - restrict dietary sodium
  - manage end organ effects
- chronic hypertension
  - advise a change in lifestyle
  - institute and monitor a weight reduction programme for obese individuals
  - regular aerobic exercise is recommended in essential hypertension
  - dietary advice
    - limit salt and saturated fat intake
    - increase dietary fibre intake
DRUG TREATMENT

Treat hypertension

**CAUTION**

Never use ACE inhibitor if bilateral renal artery stenosis is present. Avoid ACE inhibitor if possible due to risk of acute renal failure.

- prednisone, oral, 2 mg/kg/day for 2 weeks, then
  - 1 mg/kg/day for 2 weeks, then
  - 1 mg/kg on alternative days for 4 weeks, then
  - 0.5 mg/kg on alternative days for 4 weeks.
- Total duration of steroid therapy: 12 weeks

after confirmation of diagnosis
- cyclophosphamide, IV bolus, 500–750 mg/m²/dose immediately. Specialist initiated.
  - Repeat monthly for 1–2 months.
  **OR**
  - cyclophosphamide, oral, 2.5 mg/kg/day for 4 weeks. Specialist initiated.

If there is activity of disease after cyclophosphamide treatment has been given
- methotrexate, oral, 10 mg/m²/week. Specialist initiated.
  **PLUS**
  - folic acid, oral, 5 mg daily for the duration of the treatment
  **PLUS**
  - aspirin soluble, oral, 1–2 mg/kg/day

**REFERRAL**
- all patients
13.1 SEIZURES

DESCRIPTION
A seizure is a change in movement, attention or level of awareness that is sustained or repetitive and occurs as a result of abnormal neuronal discharges within the brain. When seizures are recurrent or typical of a specific syndrome, then the term epilepsy is used and specific management applies. See Epilepsy: Section 13.2

INTERNATIONAL LEAGUE AGAINST EPILEPSY
Classification of seizures is aetiological and clinical:

Aetiology:
- Symptomatic causes with underlying pathology evident.
- Idiopathic with no clear cause, often genetic.
- Probably symptomatic or probably idiopathic

The causes of seizures are multifactorial.
The commonest cause of seizures in children is a febrile convulsion but the history, examination and subsequent investigation must be aimed at eliciting/excluding the following differential diagnosis:

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<thead>
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<td>- medicine withdrawal in infancy</td>
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<tr>
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<td>- brain abscess</td>
<td>- environmental toxins</td>
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<tr>
<td>- trauma</td>
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<tr>
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<th>Systemic disorders</th>
<th>Primary cerebral causes</th>
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<tbody>
<tr>
<td>- hypoglycaemia</td>
<td>- vasculitis</td>
<td>- trauma</td>
</tr>
<tr>
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<td>- hypertensive</td>
<td>- haemorrhage</td>
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<td>- thrombosis</td>
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<th>Clinical causes</th>
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<td>- underpinning pathologic</td>
<td>- nature</td>
</tr>
<tr>
<td>- idiopathic</td>
<td>- no clear cause</td>
<td>- epilepsy</td>
</tr>
<tr>
<td>- symptomatic or idiopathic</td>
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<td>- recurrent</td>
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The causes of seizures are multifactorial.
The commonest cause of seizures in children is a febrile convulsion but the history, examination and subsequent investigation must be aimed at eliciting/excluding the following differential diagnosis:

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<td>metabolism</td>
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</tbody>
</table>
Clinical
Within each of the above categories generalised, partial or syndromic seizures occur.

Generalised seizures:
The epileptic focus arises centrally and spreads to the rest of the brain.
Generalised seizures may be:
- tonic-clonic (grand-mal convulsion)
- absence
- clonic
- tonic
- myoclonic
Generalised Tonic Clonic Seizures (GTCS) that continue for more than 30 minutes are called Convulsive Status Epilepticus: See Section 13.4

Partial seizures:
The epileptic activity arises from a particular focus within the brain.
- Simple partial seizure: a focal seizure with retained consciousness.
- Complex partial seizure: a focal seizure with spread of the seizure to involve the whole cerebral cortex, resulting in an altered level of consciousness.

Epileptic Syndromes – See Epilepsy: Section 13.2

DIAGNOSTIC CRITERIA
Clinical
- history:
  - eye witness account, aura
  - perinatal history, developmental history, school record, family history and environment
- examine to exclude obvious aetiology, but in particular look for occult causes:
  - general: skin abnormalities, e.g. Sturge Weber and tuberous sclerosis
  - CNS examination for loss of consciousness, localising signs, head growth, developmental milestones and fundi
  - CVS examination: blood pressure

Investigations
- blood glucose
- electrolytes
- culture
- FBC
- thick/thin film
- serology
- metabolic screen
- toxicology

Always consider hypoglycaemia as a primary or aggravating cause of any seizure.

- urinalysis: blood and protein in renal hypertension, MCS for UTI
- lumbar puncture: if meningitis is suspected and for first febrile generalised tonic clonic seizures in children < 2 years old

Note:
Lumbar puncture is contra indicated in the presence of the following:
- increased intracranial pressure
- GCS < 12/15 (paediatric coma scale reduced by 3 points or more)
or focal neurological signs/seizures. Thus if the seizure has progressed to status (lasted 30 minutes) then lumbar puncture is contraindicated until raised intracranial pressure is excluded.

- CT/MRI scan: if persistently reduced coma score (GCS < 12/15) without known cause, raised intracranial pressure or focal intracranial pathology is suspected
- EEG: is only indicated for recurrent or syndromic seizures where diagnosis cannot be made on clinical grounds alone

The EEG is to be delayed for at least one week after the convulsive episode.

NON-DRUG TREATMENT

- ensure an open airway and administer oxygen, if available
- position to prevent aspiration of vomitus, i.e. head up position
- check glucose during the seizure and blood pressure after the seizure
- obtain intravenous access if seizure duration > 5 minutes
- keep child nil per os and intravenous fluid volumes at maintenance rates
- control fever with tepid sponging
- aetiology will determine further management

DRUG TREATMENT

(Of a first time seizure)

For fever

- paracetamol, oral, 10–15 mg/kg/dose 4–6 hourly as required

Urgent drug treatment is only indicated if the seizure is generalised and lasts more than 5 minutes or is causing systemic compromise.
Treat as for Status Epilepticus: See Section 13.4.

For the management of persistent or recurrent seizures that are not generalised – See Epilepsy: Section 13.2.

13.2 EPILEPSY

DESCRIPTION

A condition characterised by recurrent seizures associated with abnormal paroxysmal neuronal discharges.

See International League Against Epilepsy Classification of Seizures: Section 13.1
When any of the causes and subsequent seizures are recurrent, persistent or syndromic, then the child has epilepsy.

Seizures are managed according to type (i.e. generalised or partial) and also according to specific syndromes.
Epileptic syndromes:

Infantile spasms (West's Syndrome)
- an infantile onset encephalopathy with epileptic spasms associated with hypsarrhythmia on the EEG and developmental regression
- it is a neurological emergency – diagnosis, treatment and referral must not be delayed. Early intervention reduces the subsequent neurodisability.
- clinically, the child appears to stare, give a sudden flexion of the trunk and head, with the limbs either flung in or out but held in this tonic spasm for a few seconds
- events occur in runs and are most common when the infant is going to sleep or rousing
- the episodes are distressing to the infant and he will often appear red in the face and may cry out
- events are often confused with colic

Severe Myoclonic Epilepsy of Infancy (SMEI)
- onset in children under 1 year of age with recurrent clusters of febrile convulsions, severe neuroregression and other non-febrile seizures by 2–3 years

Lennox-Gastaut syndrome (LGS)
- combinations of GTCS, atypical absences, myoclonic seizures, atonic drop attacks and occasionally complex partial seizures
- may occur spontaneously
- onset between 2–3 years of age
- behavioural problems and neuroregression occurs

Benign focal epilepsy of childhood
- sleep related events of hemifacial clonic spasm
- inability to speak but retained awareness
- onset at ± 6–10 years
- usually resolves by late adolescence

Primary generalised absence seizure of childhood (petit mal)
- short spells of motor arrest of maximum 15 seconds duration
- little or no associated movements
- no post-ictal effect
- onset 4–6 years

Generalised epilepsy with febrile seizures plus (GEFS+)
- children with febrile convulsions which persist beyond 6 years
- occasionally associated with afebrile convulsions
- these children have epilepsy triggered by fever and may warrant anticonvulsant intervention
- often family history of febrile convulsions

Note:
Infantile spasms, Severe Myoclonic Epilepsy of Infancy and Lennox-Gastaut syndrome are regarded as malignant forms of epilepsy and are associated with neuroregression and behavioural problems.
DIAGNOSTIC CRITERIA
• a child may be diagnosed:
  ○ with a specific anatomical or systemic cause for the seizure type (see table of possible causes)
  ○ as having an epileptic syndrome, i.e. a specific seizure type associated with a characteristic EEG, natural history, response to therapy and prognosis
  ○ with idiopathic epilepsy

NON-DRUG TREATMENT
ACUTE
• maintain an open airway
• place patient on side at 20–30° head up
• admit to high or intensive care, if possible
• if unconscious, consider catheterisation
• monitor:
  ○ heart rate
  ○ respiratory rate
  ○ blood pressure
  ○ electrolytes
  ○ blood glucose
  ○ anticonvulsant blood levels
  ○ acid–base status
  ○ blood gases
  ○ SaO₂
  ○ neurological status
  ○ fluid balance
  ○ osmolality
• control fever with tepid sponging
• administer oxygen 100 % by facemask, nasal cannula or head box to maintain SaO₂ of ≥ 95%
• cardiovascular and/or respiratory support if the patient is unable to maintain blood gases and blood pressure within the normal physiological range
• ventilation to maintain PaCO₂ in the low normal range, i.e. 4– 4.5 kPa

LONG TERM
• minimise the impact of the epilepsy by obtaining complete seizure control to maximising child’s full potential
• educate the patient and caregiver about epilepsy and associated complications, i.e. learning difficulties and ADHD

DRUG TREATMENT
For acute generalised tonic clonic seizures
See Status Epilepticus: Section 13.4

Maintenance therapy
Monotherapy is preferred but combination therapy may be necessary.
Combination therapy should be specialist initiated.
Drug levels are rarely indicated unless there is concern about toxicity or compliance.
### Maintenance Drug Treatment Choices for Different Types of Epileptic Seizures

<table>
<thead>
<tr>
<th>TREATMENT</th>
<th>SEIZURE TYPE</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Generalised tonic and/or clonic</td>
<td>Partial</td>
<td>Infantile spasms</td>
<td>Absence</td>
<td>Myoclonic</td>
</tr>
<tr>
<td>1st line</td>
<td>sodium valproate <strong>OR</strong> phenobarbital (&lt; 6 months old)</td>
<td>carbamazepine</td>
<td>refer all</td>
<td>sodium valproate</td>
<td>refer all for specialist investigation and initiation of therapy with sodium valproate</td>
</tr>
<tr>
<td>2nd line</td>
<td>carbamazepine</td>
<td>sodium valproate</td>
<td></td>
<td></td>
<td>refer</td>
</tr>
<tr>
<td>3rd line</td>
<td>refer for specialist decision on lamotrigine</td>
<td>refer for specialist decision on lamotrigine</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

- **sodium valproate**, oral, 20–40 mg/kg/24 hours in 2–3 divided doses
  Use under specialist consultation.
  The slow release formulation enable school going children to take medication in a manner such that it does not sedate them with peaks and troughs and can be taken twice a day i.e. not at school.
  Monitor for hepatotoxicity in children under two years of age.

- **carbamazepine**, oral, 15–20 mg/kg/24 hours in 2–3 divided doses
  Initiate slowly over a period of 2–3 weeks.
  Exacerbates myoclonic seizures and absence seizures.

- **lamotrigine**, oral, 0.2 mg/kg/day
  Use as a third line agent, specialist initiated.
  Increase dose incrementing to 5 mg/kg/day slowly in conjunction with valproate.
  Lamotrigine is given as add-on therapy for many seizure types drug-resistant paediatric epileptic syndromes, such as Lennox-Gastaut syndrome.

- **phenobarbital**, oral, 3–5 mg/kg/24 hours as single dose at night
  May be used in children under six months of age.
  Is not recommended as maintenance therapy for children older than 2 years due to undesirable side effects such as sedation, behaviour disturbances, hyperkinesia and dependence, except in situations where there is poor adherence to other drugs.
  Exacerbates absence seizures.
REFERRAL
• suspected secondary cause
• partial seizures for neuroimaging if facilities or expertise not available
• generalised seizures other than typical febrile convulsions in children < 2 years
• seizures that are not controlled within 2 months on one agent with minimal side effects
• neuroregression
• mixed seizure types within one patient.
• all myoclonic seizures and infantile spasms at presentation

13.3 SEIZURES, FEBRILE
R56.0

DESCRIPTION
Seizures occurring in children between the ages of 3 months and 5 years associated with a rapid rise in temperature at the beginning of an extracranial illness. Febrile seizures can be simple or complex febrile seizures.

Simple febrile seizures
• are generalised tonic clonic seizures
• are self limiting, usually less than 5 and always less than 15 minutes
• cause no neurological deficit after the convulsion
• have a good prognosis and very rarely develops into epilepsy
• often consists of only one seizure which needs no specific treatment
• there is often a family history of febrile seizures

Complex febrile seizures
• last longer than 15 minutes
• are recurrent within the same febrile illness
• have a focal (partial) onset
• have postictal, focal neurological abnormalities

Risk factors for recurrent febrile seizures include:
• seizure disorder in a first degree relative
• onset before 12 months of age
• complex initial seizures

DIAGNOSTIC CRITERIA
Clinical
• exclude intracranial, extracranial and biochemical causes
• signs of meningism are unreliable in children under 2 years
• if raised intracranial pressure or meningitis cannot be excluded then the diagnosis of febrile seizures cannot be made. Treat children empirically for meningitis.
Investigations
- a lumbar puncture is indicated in:
  - children under 2 years for exclusion of intracranial infection even when signs of meningism are absent
  - all children who have no focus of infection, particularly those who have received antibiotics prior to the event
    In children older than 2 years, where a focus of extracranial infection is present and intra-cranial infection has been excluded clinically, no further investigation is required.
- all children with complex seizures and persistent lethargy should have a CT scan and then a lumbar puncture if raised intracranial pressure can be reliably excluded
- an EEG is of no value in simple febrile seizures

NON-DRUG TREATMENT
- control fever with tepid sponging
- reassure parents and caregivers
- educate parents and caregivers regarding the management of future episodes of fever

DRUG TREATMENT
For fever: administered by parents
- paracetamol, oral, 10–15 mg/kg/dose 4–6 hourly until fever subsides

If convulsing
See Status Epilepticus: Section 13.4

Continuous prophylactic therapy
Routine daily prophylaxis is not recommended for patients with simple febrile seizures. For children with recurrent complex febrile seizures, prophylactic treatment can be considered, preferably in consultation with a paediatrician.
- phenobarbital, oral, 5 mg/kg/day as a single dose
  OR
  sodium valproate, oral, 20–40 mg/kg/24 hours in 3 divided doses

REFERRAL
- complex febrile seizures for confirmation of diagnosis
- developmental delay/regression

13.4 STATUS EPILEPTICUS (CONVULSIVE)
G41.9

DESCRIPTION
Convulsive status epilepticus is a medical emergency defined as a generalised tonic-clonic convulsion that persists for 30 minutes or longer, or is repeated frequently enough to prevent recovery of consciousness between attacks.

After 30 minutes of generalised tonic-clonic seizures, the brain begins to suffer from hypoxia, acidosis, depletion of local energy stores, cerebral oedema and structural damage.
Complications include:

- hyperpyrexia
- respiratory depression
- cerebral oedema
- blood pressure disturbances
- disturbances of blood glucose
- renal failure
- acidosis
- inappropriate antidiuretic hormone (ADH) secretion
- hypoxic, ischaemic damage to brain, myocardium and muscles

**DIAGNOSTIC CRITERIA**

**Clinical**

- convulsive seizure lasting 30 minutes or longer.
- Convulsive seizures that have lasted for 5 minutes or more should be managed as for Status.
- convulsive status epilepticus may be:
  - idiopathic
  - secondary to an insult to the brain, e.g. encephalitis, hypoxic episode, trauma and complex febrile seizures
  - as a result of non-compliance and changes in anticonvulsant therapy

**NON-DRUG TREATMENT**

- maintain an open airway
- place patient on side at 20–30° head up
- admit to high or intensive care, if possible
- if unconscious, consider catheterisation
- monitor:
  - heart rate
  - respiratory rate
  - blood pressure
  - electrolytes
  - blood glucose
  - anticonvulsant blood levels
  - acid–base status
  - blood gases
  - SaO\(_2\)
  - neurological status
  - fluid balance
  - osmolality
- control fever with tepid sponging
- administer oxygen 100 % by facemask, nasal cannula or head box to maintain SaO\(_2\) of ≥ 95%
- cardiovascular and/or respiratory support if the patient is unable to maintain blood gases and blood pressure within the normal physiological range
- ventilation to maintain PaCO\(_2\) in the low normal range, i.e. 4– 4.5 kPa

**DRUG TREATMENT**

**Status Epilepticus**

Follow ABCD approach.

See flowchart on next page for management of Status Epilepticus.

**Intravenous fluid**

- dextrose 5% in sodium chloride 0.9%, IV
  
  Avoid overhydration-keep fluid volume at maintenance.
  
  Maintain normoglycaemia and electrolytes within the normal range.
Cerebral oedema
Treat when clinically suspected/proven.
If the patient has a serum osmolality < 320
• mannitol, IV, 250 mg/kg administered over 30–60 minutes

Cerebral oedema with associated space occupying lesion
• dexamethasone, IV, 0.5 mg/kg 12 hourly

Other biochemical disorders
Correct abnormalities, if present, i.e. glucose, calcium and sodium

For fever
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required via nasogastric tube
  OR
  paracetamol, rectal, 6 hourly
    3–12 months  60–125 mg
    1–5 years    125–250 mg
    6–12 years   250–500 mg
MANAGEMENT OF STATUS EPILEPTICUS

- **diazepam**, rectal, 0.5 mg/kg
- OR
- **midazolam**, buccal, 0.5 mg/kg

Still convulsing after 10 minutes

- **lorazepam**, IM/IV, 0.1 mg/kg
- OR
- **clonazepam**, IV, 0.5 mg

Still convulsing after 10 minutes

- **phenytoin**, IV, 18 mg/kg infused over 30 minutes in a dextrose-free solution.
  Cardiac monitoring is essential.
  Monitor for bradycardia, arrhythmias, hypotension – if so then stop infusion until stable then restart at \( \frac{2}{3} \) the starting rate.

Still convulsing at the end of phenytoin infusion

- Refer to tertiary level ICU for intubation and **thiopental sodium**.
  Pending referral, start **midazolam**, slow IV infusion, 0.5–5 mcg/kg/minute

Once fits controlled consider maintenance therapy.

**Note:**
Once intravenous access is attained take blood for glucose, blood gases, electrolytes, FBC and culture.

**Monitor carefully for drug related respiratory depression.**

Intubation, ventilation and administration of sodium pentothal infusion should only be performed in a centre with trained anaesthetists and paediatric intensive care unit.
CAUTION
Attempt to control seizures and stabilise the patient before referral.

- failure to control seizures within 1 hour
- where the primary cause is unknown, or if the primary cause itself requires referral

13.5 HEADACHES

DESCRIPTION
Headache is the most common pain syndrome in children of all ages. Recurrent headaches are a health problem. Recurrent headaches can be primary, e.g. migraine or secondary/symptomatic, e.g. raised intracranial pressure.

The actual perception of headache varies according to age and is influenced by factors like experience, memory and cultural environment.

CLASSIFICATION OF HEADACHES

Migraine (without aura)
- 5 or more headaches lasting 1–48 hours fulfilling at least 2 of the following:
  - bilateral or unilateral, frontal or parietal in location
  - pulsating in character
  - moderate or severe
  - aggravated by routine activity
  - nausea and/or vomiting plus photophobia and/or phonophobia during headache

Migraine (with aura)
- at least 2 attacks fulfilling at least 3 of the following:
  - one or more reversible aura symptoms
  - at least one aura developing over > 4 minutes or 2/more successive symptoms
  - no aura lasting > 1 hour
  - headache follows aura in less than 1 hour

Episodic tension-type headache
- at least 10 prior episodes, occurring less than 15 times per month and lasting 30 minutes to 7 days with at least 2 of the following:
  - pressing or tightening quality
  - mild or moderate intensity
  - bilateral location
  - no aggravation by routine physical activity
  - no nausea or vomiting, and photophobia and phonophobia absent during headache

Cluster headache
- severe unilateral sharp headache associated with conjunctival injection and lacrimation
- rare in childhood
Paroxysmal Hemicrania Continua
- cluster headache of shorter duration
- responds well to ibuprofen.

Each of the above can occur in combination in any patient, i.e. mixed/comorbid headache. Headaches can also be sub-classified according to temporal patterns, i.e. acute, acute recurrent, chronic progressive/non-progressive, episodic or constant.

DIAGNOSTIC CRITERIA
- exclude secondary causes of headache, e.g. raised intracranial pressure

NON-DRUG TREATMENT
- environmental and lifestyle changes, e.g. avoid precipitants such as bright lights, sleep deprivation and certain foods
- headache diary

DRUG TREATMENT

 Analgesics
- paracetamol, oral, 10–15 mg/kg/dose 6 hourly as required

 If no response, add
- ibuprofen, oral, 5–10 mg/kg/dose 6 hourly

 Anti-emetic
- metoclopramide, oral, 0.15–0.3 mg/kg as a single dose
  OR
  metoclopramide, IM/IV, 0.1 mg/kg as a single dose

 Migraine prophylaxis
Treat for six months then review.
- propranolol, oral, 0.5–3 mg/kg/day in 2–3 divided doses
  Contraindicated in asthma and heart block.
  OR
  sodium valproate, oral, 10–20 mg/kg/day
  OR
  amitriptyline, oral, 0.25 mg/kg at night starting dose for a 6 week period
  If no response, increase dose slowly to 1 mg/kg at night.
  ECG monitoring prior to dose increases.

REFERRAL
- secondary intracranial cause suspected
- no response to treatment
13.6 NEUROCYSTICERCOSIS
B69.0

DESCRIPTION
Neurocysticercosis is caused by the cysticercal form, i.e. larval form of the pork tapeworm, *T. solium*. The larvae may locate in the brain parenchyma, intraventricular and meningeal areas, spinal canal/cord and eye, or a combination of these regions. Viable cysticerci incite little inflammatory response, but dead cysticerci elicit an increased inflammatory response.

Cysticerci in the brain may remain dormant or may cause complications such as:
- headache
- behavioural disorders
- visual disturbances
- seizures
- meningo-encephalitis
- focal neurological deficits
- increased intracranial pressure
- hydrocephalus
- meningitis
- spinal cord compression

DIAGNOSTIC CRITERIA
Clinical
- location and stage of the life cycle of the parasite in the brain determines the clinical features
- suspect if child from endemic area, i.e. pig farming area presents with neurological abnormalities such as:
  - seizures
  - raised intracranial pressure/hydrocephalus
  - focal neurological deficits
  - cranial nerve palsies
  - meningo-encephalitis
  - meningitis
  - behavioural disorders
  - headache

Investigations
- computed tomography (CT scan) and/or magnetic resonance imaging (MRI scan) of brain showing cysts, peri-lesional oedema or calcification of cysts
- MRI scan may identify more lesions and viable cystic lesions than the CT scan
- soft tissue radiology of muscles of lower limbs may demonstrate calcified cysticerci, i.e. rice grain calcifications in muscles
- follow-up CT scans and/or MRI scans may help to assess the response to therapy

NON-DRUG TREATMENT
- prevention:
  - prolonged freezing or thorough cooking of pork to kill the parasite
  - thorough washing of fresh fruit and vegetables in *T. solium* endemic areas
  - attention to personal hygiene
  - proper sanitation facilities
  - avoid the use of human excreta as fertiliser
DRUG TREATMENT
Calcified cysticerci and a single dying lesion visible on CT scan require no treatment monitoring.

Patients with multiple dying cysts are assumed to have active disease and require treatment.
- albendazole, oral, 5 mg/kg/dose 8 hourly for 5 days

Prevention of neurological manifestations
In massive infestations, cysticidal therapy may trigger an inflammatory response. Delaying therapy and adding corticosteroids may lessen the risk.

24 hours prior to albendazole therapy
- dexamethasone, IM, 0.25–0.5 mg/kg/24 hours
  Continue for the duration of the therapy.
  OR
  - betamethasone, oral, 0.01–0.2 mg/kg/24 hours
    Continue for the duration of the therapy.

Seizure control
See Epilepsy: Section 13.2

REFERRAL
- all patients for CT scan
- neurocysticercosis not responding to adequate therapy
- neurocysticercosis with complications, such as hydrocephalus
- intractable epilepsy

13.7 NEUROMUSCULAR DISORDERS

13.7.1 POLYNEUROPATHY (GUILLAIN-BARRÉ SYNDROME)
G62.9
* Notifiable condition

DESCRIPTION
Guillain-Barré syndrome is an autoimmune-mediated demyelination which is precipitated by a preceding viral or other infection.

It is the most common polyneuropathy in children, characterised mainly by:
- motor weakness,
- areflexia, i.e. absence of tendon reflexes
- distal sensory alteration – “glove and stocking”.
DIAGNOSTIC CRITERIA
Clinical
• preceding respiratory tract or gastrointestinal infection
• symmetrical, flaccid muscle weakness with early areflexia
• the muscle weakness may ascend rapidly upwards to involve the trunk, arms, face and cranial nerves
• bulbar paralysis and respiratory failure may develop
• autonomic dysfunction
• relatively mild, or absence of, sensory signs
• Miller-Fisher variant presents with ataxia and ophthalmoplegia
• exclude the following:
  ○ poliomyelitis – a rare cause of hypotonia with abrupt onset of weakness (often asymmetrical) in association with a febrile illness
  ○ transverse myelitis
    • initial flaccid weakness and areflexia typically involving the lower limbs maximally
    • occasionally with pain at the onset, but rapidly progressing to spasticity and hyperreflexia
    • also a sensory level on trunk
    • bladder and rectal sphincter involvement
  ○ diphtheria
Investigations
• CSF findings after 1 week show elevated protein and no cells or only a few cells, i.e. albumino-cytological dissociation
  CSF Glucose is normal.

NON-DRUG TREATMENT
• admit to high or intensive care unit
• monitor autonomic functions closely
  ○ peak flow
  ○ respiratory rate
  ○ forced vital capacity (FVC)
  ○ arterial blood gases
  ○ blood pressure
  ○ heart rate
  ○ bulbar functions
  ○ bulbar functions

Note:
These changes precede hypoxaemia detected on blood gas analysis, and ventilation should begin before frank hypoxaemia occurs.
Respiratory care must be impeccable.
• ventilation is recommended when:
  ○ PCO₂ levels start rising
  ○ a fall in the peak expiratory flow rate
  ○ tachycardia and sweating occur
  ○ inspiratory efforts are weak
  ○ inability to talk
• shoulder weakness, weak cough and swallowing difficulties are an indication for respiratory support
• to determine fluid losses from autonomic instability monitor urine output and degree of sweating
• provide adequate nutrition
• bladder and bowel care as well as pressure-point care
• routine physiotherapy for chest and lower limbs, keep ankles in dorsiflexion
• protect eyes and keep moist
• communicate with child as awareness is maintained. Staff should remember that children might be very frightened but unable to express their emotions and needs.

DRUG TREATMENT
For rapidly progressive ascending paralysis for respiratory dysfunction or loss of ambulation
• immunoglobulin, IV, 1 g/kg as a single daily dose on 2 consecutive days early in the disease process
  Use under specialist supervision in an intensive care setting.
  OR
immunoglobulin, IV, 0.4 g/kg as a single daily dose on 5 consecutive days early in the disease process
  Use under specialist supervision in an intensive care setting.

REFERRAL
• Guillain-Barré syndrome with bulbar paralysis and/or early signs of respiratory failure
• patients who have lost or are loosing ambulation

13.7.2 MYASTHENIA GRAVIS
G70.0

DESCRIPTION
An autoimmune disorder resulting in muscle fatigue. Mild cases involving the eyes alone, i.e. ptosis and ophthalmoplegia, severe cases involve proximal muscle groups, respiratory and bulbar control.

DIAGNOSTIC CRITERIA
Clinical
• muscle fatigue with exercise and demonstration of this in the clinic setting:
  ○ lid-lag test, i.e. failure to maintain upward gaze for 1 minute
  ○ arm-raising test, i.e. failure to maintain the arms at 90° from the trunk for 1 minute

NON-DRUG TREATMENT
• occupational therapy
DRUG TREATMENT
- pyridostigmine, oral, 1–1.5 mg/kg/day in 4–6 divided doses. Specialist initiated.

REFERRAL
- for confirmation of diagnosis and initiation of treatment

13.8 SYDENHAM’S CHOREA
I102.9

DESCRIPTION
Rapid involuntary jerk affecting any part of the body often incorporated into a voluntary movement in an attempt to mask it. It is an acute post-streptococcal infection movement disorder and constitutes one of the major criteria for the diagnosis of rheumatic fever. Patient has the appearance of being restless with constant movement.

DIAGNOSTIC CRITERIA
Clinical
- exclude drug reactions, hyperthyroidism, systemic lupus erythematosus and neurodegenerative disorders

Investigations
- cardiac screen, i.e. ECG, ECHO
- ASOT, antiDNAse
- Erythrocyte sedimentation rate
- dsDNA, if clinically indicated

NON-DRUG TREATMENT
- emotional support

DRUG TREATMENT
- haloperidol, oral, 0.025 mg/kg/day in 2–3 divided doses. Increase dose slowly and incrementally to 0.05 mg/kg/day.

PLUS
For post-streptococcal infection movement disorders
- phenoxydihydropenicillin, oral, 500 mg twice daily for 10 days

THEN
Until 21 years of age
- benzathine benzylpenicillin (depot formulation), IM, 1.2 million units every 28 days
  OR
  - phenoxydihydropenicillin, oral, 250 mg twice daily

REFERRAL
- all patients for specialist assessment
Psychopharmacology guidelines for children and adolescents

Safe and effective pharmacological management of psychiatric disturbances in children and adolescents should always be part of a comprehensive assessment and management plan by a skilled clinician. There should be awareness of co-existing medical conditions and other medications used.

14.1 ANXIETY DISORDERS

Anxiety disorders with onset in childhood:
- separation anxiety disorder
- selective mutism

Anxiety disorders with onset in childhood and/or adulthood:
- post traumatic stress disorder (PTSD)
- obsessive compulsive disorder (OCD)
- social phobia
- specific phobia
- panic disorder
- generalised anxiety disorder (GAD)

14.1.1 ANXIETY DISORDER, GENERALISED
F41.1

DESCRIPTION
Excessive anxiety or worry occurring on most days for at least 6 months and that interferes with normal daily activities.

DIAGNOSTIC CRITERIA
- presence of 3 of the following:
  - restlessness or a feeling of being ‘on edge’
  - poor concentration or ‘mind going blank’
  - irritability
  - muscle tension
  - sleep disturbance
- causes significant distress and impairment in functioning
- exclude substance abuse or a medical condition

NON-DRUG TREATMENT
- cognitive behavioural therapy (CBT): aimed at changing pessimistic, anxiety- based cognitions and developing strategies to reduce anxieties and avoidant behaviour patterns
- behaviour therapy: relaxation, desensitisation by imagining or exposure to anxiety- provoking situations
- psychodynamic/ supportive psychotherapy: aimed at promoting self esteem, assertiveness and autonomy
CHAPTER 14  PAEDIATRIC PSYCHIATRY

DRUG TREATMENT
• fluoxetine, oral, 0.5 mg/kg/day
  Dose range: 5–40 mg daily.
  Recommended average dose: 10–30 mg/day.

OR
• citalopram, oral, 0.4 mg/kg/day
  Dose range: 10–40 mg daily.

REFERRAL
• failure to respond to an adequate trial of therapy and medication
• separation anxiety disorder
• selective mutism

14.1.2 OBSESSIVE COMPULSIVE DISORDER (OCD)

DESCRIPTION
Obsessions
Persistently recurring thoughts, impulses or images that are experienced as intrusive, inappropriate and that are not simply excessive worries about realistic problems. Children may not experience these as distressing but the obsessions may interfere with day-to-day functioning.

Compulsions
Repetitive behaviours or mental acts that a person feels driven to perform according to a rigidly applied rule in order to reduce distress or to prevent some dreaded outcome.

DIAGNOSTIC CRITERIA
• the most common symptoms of OCD in childhood are:
  ○ contamination fears accompanied by compulsive washing and avoidance of “contaminated” objects
  ○ repetitive checking and counting
  ○ obsessive doubt
  ○ compulsive reading and symmetry concerns
• comorbid conditions:
  ○ rheumatic fever
  ○ streptococcal throat infection (paediatric autoimmune neuropsychiatric disorders associated with streptococcal infections-PANDAS)
  ○ tic disorders
NON-DRUG TREATMENT
• cognitive behavioural therapy is the psychotherapeutic treatment of choice
• exposure-based interventions, e.g. contact with “dirt” in a child with contamination fears, thought stopping techniques, “response prevention”, i.e. blocking of rituals
• family-based interventions are often a very important part of the management, including assisting family members not to “collude” with the rituals

DRUG TREATMENT
• fluoxetine, oral, 0.5 mg/kg/day
  Dose range: 10–40 mg daily.
  Higher dose within the range may be required.
  OR
  citalopram, oral, 0.4 mg/kg/day
  Dose range: 10–40 mg daily.
  Higher dose within the range may be required, maximum dose 60 mg.

If there is a poor response
• clomipramine. Specialist initiated.

REFERRAL
• poor response to adequate trial of therapy and medication
• comorbid conditions

14.2 CHILDHOOD PSYCHOSIS
There are a number of psychiatric conditions in children for which antipsychotic medication is indicated, other than psychosis, such as the pervasive developmental disorders, Tourette’s and tic disorders and conduct disorder, e.g. severe aggression.

14.2.1 PERVASIVE DEVELOPMENTAL DISORDERS (PDDs)
F84
DESCRIPTION
The PDDs are neuropsychiatric disorders characterised by patterns of delay and deviance in the development of social, communication and cognitive skills. Onset is usually during the first few years of life and includes conditions such as:
• Autistic Disorder
• Asperger’s Disorder
• Rett’s disorder
• Childhood Disintegrative Disorder.
NON-DRUG TREATMENT
- social skills, family interventions
- education and social placement
- behaviour modification

DRUG TREATMENT
Not for core autistic symptoms.

For severe aggression and self injurious behaviour
- haloperidol, oral, 0.002–0.12 mg/kg/day. Specialist initiated.
  Dose range: 0.25–1.75 mg twice daily.
  Recommended average dose: 0.5–2 mg/day.
  Monitor for extrapyramidal and anticholinergic side effects.

OR
  chlorpromazine, oral, 2 mg/kg/day
  Dose range: 12.5–50 mg twice daily.
  Recommended average dose: 25–50 mg/day.

OR
  risperidone, oral, 0.125–3 mg twice daily. Specialist initiated.
  Recommended average dose: 0.25–2 mg/day.

Other comorbid conditions
Anxiety disorders: See Section 14.1
ADHD: See section 14.3.2

14.2.2 SCHIZOPHRENIA
F20

DESCRIPTION
Schizophrenia is a chronic psychotic disorder characterised by disturbances in thinking, perceptions, emotions and behaviour associated with significant degrees of impairment in functioning.

DIAGNOSTIC CRITERIA
- presence of two or more of the following symptoms for at least 6 months:
  - delusions
  - hallucinations
  - disorganised speech
  - disorganised behaviour
  - ‘negative’ symptoms i.e. affective flattening and avolition
- delusions are not as bizarre or systematised as in adults
- early history commonly reveals a number of neurodevelopmental difficulties which precede the onset of psychosis, i.e. speech and language, motor and co-ordination problems, cognitive delay and academic difficulties
- significant impairment of functioning
- exclude substance abuse or medical condition
NON-DRUG TREATMENT
• supportive individual and family counselling is an important part of the comprehensive treatment plan
  ○ the aim is to develop healthier coping strategies and defense mechanisms and to provide structure and limit regression.
  ○ family interventions focus on psychoeducation, facilitating acceptance of the diagnosis to ensure adequate compliance and support for the patient

DRUG TREATMENT
• risperidone, oral, 0.5–3 mg/day. Specialist initiated.

If there is a poor response
• haloperidol, oral, 0.002–0.12 mg/kg/day. Specialist initiated.
  Dose range: 0.25–1.25 mg twice daily.
  Recommended average dose: 0.5–2 mg/day.
  Monitor for extrapyramidal and anticholinergic side effects.

REFERRAL
• all for initiation of therapy

14.2.3 TIC DISORDERS
F95.9

DESCRIPTION
A tic is a sudden, rapid, recurrent, nonrhythmic stereotyped motor movement or vocalisation and includes the following subtypes:
• Chronic motor or vocal tic disorder
• Transient tic disorder
• Tourette’s disorder

Tourette’s disorder is a chronic neuropsychiatric disorder that is characterised by vocal and motor tics, and related somatosensory urges.
It is commonly associated with a number of co-morbid conditions such as OCD, ADHD as well as disturbances of mood.

NON-DRUG TREATMENT
• psycho-education of patient, parents, teachers and peers: to reduce the stigma and social consequences of tics
• supportive psychotherapy: to assist the individual cope with the stigma/ teasing, improve self esteem and improve social skills
• family therapy: to assist the family in managing associated symptoms and to reduce stress

DRUG TREATMENT
For severe and frequent tics that seriously impact on child’s development and functioning
• haloperidol, oral, 0.002–0.12 mg/kg/day.
  Dose range: 0.25–1.75 mg twice daily.
  Recommended average dose: 0.5–2 mg/day.
  Monitor for extrapyramidal and anticholinergic side effects.
• risperidone, oral. Specialist initiated.
14.3 DISRUPTIVE BEHAVIOUR DISORDERS

DESCRIPTION
The essential features of conduct disorders are repetitive and persistent patterns of behaviour in which the basic rights of others and major age-related appropriate societal norms and rules are violated.

NON-DRUG TREATMENT
- parenting interventions/family therapy: to reduce harsh, punitive styles of parenting, improving relationship between parent and child
- supportive counseling: anger management and to address hostile/negative assumptions
- group-based interventions: to improve social skills and encourage prosocial behaviour

DRUG TREATMENT
For aggressive conduct disorder unresponsive to other interventions (CD)
- haloperidol, oral, 0.002–0.12 mg/kg/day
  Dose range: 0.25–1.75 mg twice daily.
  Recommended average dose: 0.5–2 mg/day.
  Monitor for extrapyramidal and anticholinergic side effects.
- risperidone, oral. Specialist initiated.

14.3.1 BEHAVIOURAL PROBLEMS ASSOCIATED WITH MENTAL RETARDATION

A significant number of children and adolescents with intellectual disability suffer from a psychiatric disorder. The most common presentation is a constellation of symptoms characterised by impulsivity, irritability, hyperactivity, short attention span and language delay. Autism more commonly co occurs with intellectual disability. Depression occurs at a rate similar to the general population. 25% of children and adolescents with mental retardation suffer significant anxiety. Other problem behaviours are self-injurious behaviour and inappropriate sexual behaviour.

TREATMENT
Although research studies are relatively few, the cautious use of psychotropic medications as part of a multidisciplinary diagnostic and therapeutic intervention programme is recommended.

14.3.2 ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

DESCRIPTION
Children with ADHD display developmentally inappropriate degrees of inattention, impulsiveness and hyperactivity.
• **inattention:**
  - failing to give close attention to details
  - careless mistakes
  - not listening
  - failure to complete tasks
  - losing things
  - distractibility

• **hyperactivity**
  - fidgetiness
  - out of seat
  - running or climbing excessively
  - being “on the go”, or like “driven by a motor”

• **impulsivity:**
  - blurtling out answers
  - difficulty waiting turn
  - interrupting or intruding on others

The 3 subtypes are:
• predominantly inattentive
• predominantly hyperactive-impulsive
• combined

**DIAGNOSTIC CRITERIA**
• symptoms present before age seven
• symptom duration of at least six months
• behaviour is inconsistent with the patient’s developmental level and intellectual ability
• presence of functional impairment in more than one setting
• exclude other mental or physical disorders, e.g.:
  - anxiety disorders
  - psychotic disorders
  - mood disorders
  - hearing impairment

**Note:**
Certain conditions may mimic ADHD, e.g. mental handicap/borderline intellectual functioning, post traumatic and post infectious encephalopathy.

**NON-DRUG TREATMENT**
• parent counseling:
  - rules and limit-setting
  - positive reinforcement of pro-social behaviour
  - consistent routine
Restrictive diets are of no proven value.

• behaviour-based therapy:
  - positive and negative rewarding
  - improve self-image
  - improve social awareness and adjustment
• social skills group
• diagnose and treat educational difficulties – refer remedial teacher
DRUG TREATMENT

Methyphenidate
Do not use in children less than 6 years without subspecialist supervision.

Contraindications:

- absolute:
  - hyperthyroidism
  - cardiac dysrhythmia
  - glaucoma
  - concomitant monoamine oxidase inhibitor therapy
- relative:
  - hypertension
  - tics
  - epilepsy
  - anxiety
  - agitation

Always use the lowest effective dose.
If a paradoxical increase in symptoms occurs, dose should be reduced or the medication withdrawn.
If no objective improvement of symptoms is observed, e.g. Conners’ scales completed by teacher, after appropriate dosage adjustment over a one-month period, discontinue methylphenidate.

For children 6 years or older

- methylphenidate, oral, 0.3–0.5 mg/kg
  - Initial dose: 5 mg 2–3 times daily, before breakfast and lunch, 3rd dose not later than 14h30.
  - Increase the dose at weekly intervals by 5–10 mg until symptoms are controlled.
  - Maximum daily dose: 60 mg. Do not exceed 40 mg/day without subspecialist consultation.

For children who do not respond to methylphenidate or who display marked symptoms of comorbid anxiety

- imipramine, oral, 2–5 mg/kg/day as a single dose at night
  - Average dose range: 10–50 mg.
  - Maximum dose: 75 mg.

REFERRAL

- no response to treatment
- presence of comorbid conditions:
  - conduct disorder
  - oppositional defiant disorder
  - mood disorders
  - tic disorders
- for consideration of long acting methylphenidate
14.4 MOOD DISORDERS
F30–F39

14.4.1 BIPOLAR MOOD DISORDER IN CHILDREN AND ADOLESCENTS
F31

DESCRIPTION

Manic Episode
A distinct period of abnormally and persistently elevated, expansive and/or irritable mood. This should represent a significant change in the patient’s baseline mental status and must last for at least 1 week. During the period of mood disturbance, the patient should display the following symptoms:
- grandiosity
- racing thoughts
- decreased sleep
- pressured speech
- increase in goal-directed or reckless activity

Depressive Episode
Similar to symptoms of major depressive episode except that onset may be more rapid, associated with psychomotor retardation, and/or psychotic symptoms.

Mixed mood states
Presence of both manic and depressive symptoms over a period of 1 week. These are more common in children. Discrete manic and depressive phases are less evident than in adults.

The mood disturbance must cause marked impairment of functioning and should not be due to the direct effects of a substance.

DRUG TREATMENT
Suspicion of a manic episode merits immediate referral for sub-specialist, assessment and admission for containment and further management.

Acute treatment
Sedation before referral
- lorazepam IM/oral, 50–100 mcg/kg, immediately as a single dose
  Dose range: 0.5–4 mg.
  Recommended average dose: 1–2 mg.
  OR
  If lorazepam not available
diazepam, oral/rectal, 0.3 mg/kg, immediately as a single dose
  Dose range: 2.5–10 mg.
  Recommended average dose: 2.5–5 mg.

Maintenance treatment
To be initiated by a specialist.
- neuroleptics, e.g. risperidone
- mood stabilisers, e.g. lithium carbonate, sodium valproate and carbamazepine

REFERRAL
- all
14.4.2 DEPRESSION IN CHILDHOOD AND ADOLESCENCE
F32

DESCRIPTION
The clinical presentation of depression includes:
- symptoms of depressed mood
- decreased pleasure or interest
- neurovegetative symptoms
  - sleep/appetite disturbance
  - fatigue
  - poor concentration
  - psychomotor agitation/retardation
- guilty ruminations
- death thoughts and suicide ideation

Suicide is self-inflicted harm where the intention is to die.
Increased suicide risk is associated with the following:
- male gender
- two peaks-adolescence, elderly
- previous attempts and lethality of method used
- family history of suicide
- presence of a mental illness
- social isolation and poor family support
- associated substance abuse

The clinical picture of child and adolescent major depressive disorder is similar to that of adults except that there are some developmental differences i.e.:
- mood is often irritable rather than sad
- somatic complaints, i.e. headache and abdominal pain
- behavioural and academic problems occur frequently in children
- withdrawal from social activities
- neurovegetative symptoms are less common than in adults
- Suicide attempts increase in number, tend to be more lethal and impairment of functioning worsens with increasing age.

The first episode of bipolar mood disorder can present with depression in adolescents. Bipolar depression is often associated with a more sudden onset, psychomotor retardation and in some instances, psychotic symptoms.

A number of depressed children and adolescents have co-morbid psychiatric disorders. The most frequent co-morbid diagnoses are:
- dysthymic disorder
- anxiety disorders
- disruptive disorders
  - ADHD
  - Oppositional Defiant Disorder
  - Conduct Disorder
- substance abuse
Conduct problems may develop as a complication of the depression and persist after the depression remits. It is important to assess and manage conditions that occur together with depression.

**DIAGNOSTIC CRITERIA**
- presence of at least five of the symptoms of depression for a period of two weeks
- should be of a severity to cause significant functional impairment and feelings of distress
- consider the following:
  - exclude underlying medical conditions
  - infections, e.g. HIV, encephalitis and tuberculosis
  - neurological conditions, e.g. temporal lobe epilepsy
  - endocrine disorders, e.g. thyroid conditions
  - exclude medication-induced mood disturbances, e.g. aminophyllin, corticosteroids and barbiturates
  - exclude substance abuse, alcohol
  - assess for suicide risk

**NON-DRUG TREATMENT**
- psychological interventions
  - cognitive behavioural therapy (CBT): address distorted, negative cognitions, maladaptive patterns of behaviour and communication
  - psychodynamic/play therapy: identify feelings, improve self esteem and improve social interactions
- additional interventions
  - family counselling: to address family disharmony, stressors and provide psycho-education
  - input to school: address academic issues and psycho-education

**DRUG TREATMENT**
Consider a trial of antidepressant medication if there is a failure to respond to psychotherapeutic interventions after three months. Psychotherapy should still be continued. Response to treatment should bring about a meaningful reduction in symptoms and improvement in functioning. Once remission is achieved continue medication therapy for at least a further 6–12 months.

**Note:**
Behavioural complications such as restlessness, social disinhibition, agitation and insomnia may occur. These are usually dose-related and should remit once dosage is lowered. Potential risk of bipolar ‘switch’ or precipitation of mania in patients with a family history of bipolar mood disorder. Tricyclic antidepressants are not recommended as first line agents in children, due to insufficient evidence of efficacy, and potentially adverse cardiovascular side effects. All children/adolescents commenced on an antidepressant, especially SSRI’s, must be monitored with regard to risk for suicide.

- fluoxetine, oral, 0.5 mg/kg/day
  Dose range: 5–30 mg daily.
If there is a poor response to fluoxetine after an adequate trial of treatment, i.e. 4–6 weeks, or if significant symptoms of anxiety are present
- citalopram, oral, 0.4 mg/kg/day. Specialist initiated.
  Dose range: 10–40 mg daily.

REFERRAL
- poor response to an adequate trial of treatment
- presence of comorbid conditions
- psychotic symptoms such as delusions or hallucinations
- suicidal ideation or intent
- existing chronic medical condition with emergence of depressive symptoms

14.5 SEDATION OF ACUTELY DISTURBED CHILD AWAITING PSYCHIATRIC EVALUATION

Exclude organic causes, e.g. encephalopathy, metabolic disease, seizures, intoxication, organ failure and intracranial pathology.

For children under the age of six years
Sedation with psychotropic agents should only be considered in extreme cases and only on the advice of a specialist.

For children over the age of six years
- lorazepam, oral/IM, 0.5–4 mg

If sedation is inadequate
- haloperidol, IM, 0.5–2 mg

In case of an acute dystonic reaction secondary to haloperidol
- biperiden, IM
  6-10 years 3 mg
  >10 years 5 mg
15.1 CHRONIC LUNG INFECTIONS

15.1.1 BRONCHIECTASIS

DESCRIPTION
Recurrent bacterial infections are the result of irreversible dilatation of the subsegmental airways, inflammatory destruction of bronchial and peribronchial tissue and accumulation of exudative material in dependent bronchi. This results in bronchial luminal obstruction; ciliary dyskinesia; thick, tenacious secretions; lung tissue damage; aspiration pneumonia.

Complications include cor pulmonale and respiratory failure.

DIAGNOSTIC CRITERIA
- chronic cough, usually with mucopurulent sputum and occasional haemoptysis
- a bout of coughing on physical activity or change in posture, particularly while reclining
- cyanosis, fever, malaise, anorexia, poor weight gain, halitosis and clubbing
- recurrent and persistent lower respiratory tract infections
- diagnosis can be confirmed by high resolution computed tomography

NON–DRUG TREATMENT
- identify and treat the underlying disorder
- clear secretions effectively with postural drainage and physiotherapy
- eliminate all foci of infection
- nutritional support

SURGICAL TREATMENT
Consider in patients with localised severe disease or progressive disease despite adequate medical treatment.

DRUG TREATMENT
Change antibiotics according to culture and sensitivity results.

Note:
These antibiotic regimens do not apply to children with cystic fibrosis.

For acute lung infections
- ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly for 14 days
  PLUS
- gentamicin, IV, 7.5 mg/kg once daily for at least 14 days
Poor response and no culture to guide antibiotic choice - to cover *S. aureus*
- cloxacillin, IV, 50 mg/kg/dose, every 6 hours 48–72 hours after temperature settles (minimum 5 days)

If there is evidence of good clinical response, change to:
- amoxicillin/clavulanic acid, oral, 30 mg/kg/dose of amoxicillin component 8 hourly
  **PLUS**
  amoxicillin, oral, 30 mg/kg/dose, 8 hourly

**In the acute phase if wheeze is present**
- salbutamol, solution 5 mg/mL, nebulise 4 hourly
  5 mg salbutamol in 2–4 mL sodium chloride 0.9%
  OR
- theophylline, modified release, oral, 12 hourly
  Titrate dose for optimal response.
  Interacts with many other medicines, including antibiotics and quinolones.

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<th>Initial dose mg</th>
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- Influenza vaccine is recommended annually

**REFERRAL**
- poor response to therapy
- for confirmation of the diagnosis
- for early surgical intervention of localised bronchiectasis

**15.1.2 LUNG ABSCESS**

**DESCRIPTION**
A lung abscess is a suppurative process that results from destruction of the pulmonary parenchyma and formation of a cavity containing purulent material. It may be:
- single, e.g. after aspiration
- multiple, e.g. staphylococcal disease and cystic fibrosis.

Lung abscess may follow pneumonia caused by:
- *S. aureus*  *K. pneumoniae*
- *E. histolytica*  *pneumococci*
- *H. influenzae*  *M. tuberculosis*
- anaerobic organisms

Metastatic lung abscesses due to septicaemia or septic emboli may also occur.
Complications include:
- bronchiectasis
- bronchopleural fistula
- brain abscess
- rupture
- empyema
- pulmonary osteo-arthropathy

DIAGNOSTIC CRITERIA
- intermittent or recurrent fever, malaise, weight loss, anorexia and clubbing
- productive purulent cough with halitosis and haemoptysis
- amphorhic breathing over the cavity may be present
- chest X-ray will confirm abscess cavity/cavities with or without an air-fluid level

NON–DRUG TREATMENT
- identify underlying cause, e.g. tuberculosis
- physiotherapy and postural drainage
- correct anaemia
- nutritional support

SURGICAL TREATMENT
Consider surgical drainage of abscess and/or resection if medical treatment fails.

DRUG TREATMENT
Change antibiotics according to culture and sensitivity results.
- ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly for at least 14 days
  PLUS
  - gentamicin, IV, 7.5 mg/kg/day as a single dose for at least 14 days

Poor response and no culture to guide antibiotic choice - to cover S. aureus
- cloxacillin, IV, 50 mg/kg/dose, every 6 hours for at least 14 days

If there is evidence of good clinical response, change to:
- amoxicillin/clavulanic acid, oral, 30 mg/kg/dose of amoxicillin component 8 hourly
  PLUS
  amoxicillin, oral, 30 mg/kg/dose, 8 hourly

REFERRAL
- complicated lung abscess not responding to adequate therapy
- lung abscess where the underlying cause has not been established
15.1.3 TUBERCULOSIS, CONGENITAL

DESCRIPTION
Congenital tuberculosis may be acquired in one of the following ways:
- via through transplacental blood flow
- transmission
- via the passage of swallowed maternal blood or amniotic fluid during delivery
- via inhalation of the bacilli during the neonatal period.
The incidence is increasing.

DIAGNOSTIC CRITERIA
- positive vaginal swabs or sputum for *M. tuberculosis* in the mother
- hepatosplenomegaly in the neonate and one of the following:
  - a suggestive chest X-ray
  - positive gastric aspirate

DRUG TREATMENT
Newborn infantonates born of mother with active tuberculosis and who does not have any proof of active TB
- isoniazid, oral, 5 mg/kg/dose once daily for 5 days a week for 3 months
  
  PLUS
- rifampicin, oral, 10 mg/kg/dose once daily for 5 days a week for 3 months

After 3 months, perform a Mantoux tuberculin skin test.
- if test is negative, give BCG vaccine and discontinue TB treatment
- if test is positive or TB suspected, give full TB treatment
In severely immunosuppressed patients the tuberculin reaction can be negative in the presence of active tuberculosis.

REFERRAL
- patients not responding to adequate therapy

15.1.4 TUBERCULOSIS, PULMONARY

DESCRIPTION
A chronic, granulomatous infection of the lungs caused by *M. tuberculosis*. Most children acquire tuberculosis from infected adults by inhalation.

Malnourished, immunosuppressed (HIV and AIDS) and children under 3 years with pulmonary tuberculosis (PTB) are always regarded as having a very serious disease.
Complications include:
- enlarged hilar and mediastinal lymphadenopathy with obstruction, e.g. tracheal or bronchial airway compression or occlusion with secondary atelectasis or hyperinflation
- local spread of infection, e.g. TB bronchopneumonia, pleural effusion or cavitation
- disseminated disease, e.g. miliary TB, TB meningitis and metastatic extrapulmonary involvement

DIAGNOSTIC CRITERIA
- documented:
  - unexplained weight loss or failure to thrive
  - unexplained fever for $\geq 2$ weeks
  - chronic unremitting cough for more than 14 days
  - exposure to an adult with pulmonary tuberculosis
- Localised lymphadenopathy (especially cervical, often matted), hepatosplenomegaly, consolidation and pleural effusion.
- clubbing in HIV-infected patients does not exclude TB in chronic lung disease which may co-exist
- the following may be evident on chest X-ray:
  - hilar adenopathy with or without parenchymal opacification and/or bronchopneumonia
  - miliary changes
  - pleural effusions
- chest X-rays of HIV infected children with miliary TB may resemble that of LIP
- a positive PPD test, e.g. Mantoux, Tine Test or Monotest.

Tests are regarded as positive if the induration is:

<table>
<thead>
<tr>
<th></th>
<th>Previous BCG</th>
<th>No previous BCG</th>
<th>HIV positive or severely malnourished</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mantoux</td>
<td>$\geq 15$ mm</td>
<td>$\geq 10$ mm</td>
<td>$\geq 5$ mm</td>
</tr>
<tr>
<td>Tine Test</td>
<td>confluent induration of papules</td>
<td>ring of induration</td>
<td>no specific interpretation</td>
</tr>
<tr>
<td>Monotest</td>
<td>$\geq 8$ mm</td>
<td>$\geq 4$ mm</td>
<td>no specific interpretation</td>
</tr>
</tbody>
</table>

The Mantoux skin test may be falsely negative in the presence of:
- malnutrition
- immunodeficiency, e.g. HIV and AIDS
- immunosuppression, e.g. steroid therapy, cancer chemotherapy
- following overwhelming viral infection, e.g. measles or vaccination

In these circumstances a Mantoux induration of $\geq 5$ mm may be regarded as positive.
Mycobacterium tuberculosis is suggested by positive microscopy (acid fast bacilli on Ziehl-Neelsen stain) and confirmed by culture on:

- early morning gastric aspirate
- sputum (older children)
- induced sputum
- CSF
- pleural and ascitic fluids
- fine needle aspirate biopsies of LN
- ear swabs for culture in chronic otorrhea

NON-DRUG TREATMENT

- identify and treat the source or index case
- In case of known adult MDR TB contact, the child requires referral for appropriate MDR TB prophylaxis or treatment.
- screen all contacts for tuberculous infection
- monitor the nutritional status of the child to assess response to treatment
- only drain symptomatic pleural effusions

DRUG TREATMENT

TUBERCULOSIS CONTROL PROGRAMME DRUG REGIMENS (2003)

Directly observed therapy (DOT), short-course, using fixed medicine combinations is recommended to avoid the development of antimicrobial resistance.

Treatment should be given five times per week in both the intensive (initial) and the continuation phases.

In special circumstances, where people stay far away from health facilities and have no DOT supporter, treatment may be given three times per week in the continuation phase only.

HIV infected children with tuberculosis should be treated according to the standard treatment protocol with clinical, radiologic and microbiologic follow-up for response to treatment

<table>
<thead>
<tr>
<th>Fixed dose combination tablets available for children up to 8 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>RHZ (60,30,150 mg)</td>
</tr>
<tr>
<td>RH (60,30 mg)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Fixed dose combination tablets available for children 8 years older</th>
</tr>
</thead>
<tbody>
<tr>
<td>RHZE (150,75,400,275 mg)</td>
</tr>
<tr>
<td>RH (300,150 mg)</td>
</tr>
</tbody>
</table>

R Rifampicin
H Isoniazid
Z Pyrazinamide
E Ethambutol
### Regimen 3: Children with Tuberculosis up to the Age of 8 Years

<table>
<thead>
<tr>
<th>Pretreatment body weight</th>
<th>Two months initial phase treatment given five times per week</th>
<th>Four months continuation phase</th>
<th>When given five times a week</th>
<th>When given three times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZ (60, 30, 150)</td>
<td>RH (60, 30)</td>
<td>RH (60, 60)</td>
<td></td>
</tr>
<tr>
<td>3 to 4 kg</td>
<td>½ tab</td>
<td>½ tab</td>
<td>½ tab</td>
<td></td>
</tr>
<tr>
<td>5 to 7 kg</td>
<td>1 tab</td>
<td>1 tab</td>
<td>1 tab</td>
<td></td>
</tr>
<tr>
<td>8 to 9 kg</td>
<td>1½ tabs</td>
<td>1½ tabs</td>
<td>1½ tabs</td>
<td></td>
</tr>
<tr>
<td>10 to 14 kg</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td></td>
</tr>
<tr>
<td>15 to 19 kg</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td></td>
</tr>
<tr>
<td>20 to 24 kg</td>
<td>4 tabs</td>
<td>4 tabs</td>
<td>4 tabs</td>
<td></td>
</tr>
<tr>
<td>25 to 29 kg</td>
<td>5 tabs</td>
<td>5 tabs</td>
<td>5 tabs</td>
<td></td>
</tr>
<tr>
<td>30 to 35 kg</td>
<td>6 tabs</td>
<td>6 tabs</td>
<td>6 tabs</td>
<td></td>
</tr>
</tbody>
</table>

* RH (60, 60) should only be used when treatment is given THREE times weekly in the continuation phase only.

### Regimen 1: New Cases – Children > 8 Years and Adolescents

**New smear positive patients, new smear negative patients and extra-pulmonary TB**

<table>
<thead>
<tr>
<th>Pre treatment body weight</th>
<th>Two months initial phase given FIVE times a week</th>
<th>Four months continuation phase</th>
<th>When given FIVE times a week</th>
<th>When given THREE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZE (150, 75, 400, 275)</td>
<td>RH (150, 75)</td>
<td>RH (300, 150)</td>
<td>RH** (150, 150)</td>
</tr>
<tr>
<td>30–37 kg</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td></td>
</tr>
<tr>
<td>38–54 kg</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td></td>
</tr>
<tr>
<td>55–70 kg</td>
<td>4 tabs</td>
<td>2 tabs</td>
<td>3 tabs</td>
<td></td>
</tr>
<tr>
<td>&gt; 71 kg</td>
<td>5 tabs</td>
<td>2 tabs</td>
<td>3 tabs</td>
<td></td>
</tr>
</tbody>
</table>
**Regimen 2: Retreatment Cases – Children > 8 Years and Adolescents**

When given five times a week in the *continuation phase*:

<table>
<thead>
<tr>
<th>Pre-treatment body weight</th>
<th>Two months initial phase</th>
<th>3rd month initial phase</th>
<th>Five months continuation phase</th>
<th>When given FIVE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZE (150,75, 400,275)</td>
<td>RHZE (150,75, 400,275)</td>
<td>RH (150, 75)</td>
<td>E (400)</td>
</tr>
<tr>
<td>30–37 kg</td>
<td>2 tabs 0.5</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td>2 tabs</td>
</tr>
<tr>
<td>38–54 kg</td>
<td>3 tabs 0.75</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td>2 tabs</td>
</tr>
<tr>
<td>55–70 kg</td>
<td>4 tabs 1.0</td>
<td>4 tabs</td>
<td></td>
<td>2 tabs 3 tabs</td>
</tr>
<tr>
<td>&gt;71 kg</td>
<td>5 tabs 1.0</td>
<td>5 tabs</td>
<td></td>
<td>2 tabs 3 tabs</td>
</tr>
</tbody>
</table>

When given three times a week in the *continuation phase*:

<table>
<thead>
<tr>
<th>Pre-treatment body weight</th>
<th>Two months initial phase</th>
<th>3rd month initial phase</th>
<th>Five months continuation phase</th>
<th>When given FIVE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZE (150,75, 400,275)</td>
<td>RHZE (150,75, 400,275)</td>
<td>RH (150, 75)</td>
<td>E (400)</td>
</tr>
<tr>
<td>30–37 kg</td>
<td>2 tabs 0.5</td>
<td>2 tabs</td>
<td>2 tabs</td>
<td>2 tabs</td>
</tr>
<tr>
<td>38–54 kg</td>
<td>3 tabs 0.75</td>
<td>3 tabs</td>
<td>3 tabs</td>
<td>2 tabs</td>
</tr>
<tr>
<td>55–70 kg</td>
<td>4 tabs 1.0</td>
<td>4 tabs</td>
<td></td>
<td>3 tabs 4 tabs</td>
</tr>
<tr>
<td>&gt;71 kg</td>
<td>5 tabs 1.0</td>
<td>5 tabs</td>
<td></td>
<td>3 tabs 4 tabs</td>
</tr>
</tbody>
</table>

* Streptomycin should not be given during pregnancy

** RH (150,150) should only be used when treatment is given THREE times weekly in the continuation phase only.

Adjust treatment dosages to body weight. When calculating dosages, rather give ½ tablet more than ½ tablet less.

**Complicated Intrathoracic and Extrapulmonary TB**

**Miliary TB**

Treat for full duration of 9 months with:
- isoniazid, oral, 20 mg/kg/day to a maximum dose of 400 mg/day
- rifampicin, oral, 20 mg/kg/day to a maximum dose of 600 mg/day
- ethionamide, oral, 20 mg/kg/day to a maximum dose of 750 mg/day
- pyrazinamide, oral, 40 mg/kg/day to a maximum dose of 2 g/day
**All other forms of severe TB: extensive pulmonary TB, spinal or osteo-articular TB and abdominal TB**

For children under 8 three or four drug therapy

Initial phase standard dose for 2 months

4–drug therapy daily (isoniazid + rifampicin + pyrizinamide + ethambutol)

For children < 8 years

<table>
<thead>
<tr>
<th>Pretreatment body weight</th>
<th>Two months initial phase treatment given five times per week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZ (60,30,150)</td>
</tr>
<tr>
<td>3–4 kg</td>
<td>½ tab</td>
</tr>
<tr>
<td>5–7 kg</td>
<td>1 tab</td>
</tr>
<tr>
<td>8–9 kg</td>
<td>1½ tabs</td>
</tr>
<tr>
<td>10–14 kg</td>
<td>2 tabs</td>
</tr>
<tr>
<td>15–19 kg</td>
<td>3 tabs</td>
</tr>
<tr>
<td>20–24 kg</td>
<td>4 tabs</td>
</tr>
<tr>
<td>25–29 kg</td>
<td>5 tabs</td>
</tr>
<tr>
<td>30–35 kg</td>
<td>6 tabs</td>
</tr>
</tbody>
</table>

For children > 8 years

<table>
<thead>
<tr>
<th>Pretreatment body weight</th>
<th>Two months initial phase treatment given FIVE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RHZE (150,75,400,275)</td>
</tr>
<tr>
<td>30–37 kg</td>
<td>2 tabs</td>
</tr>
<tr>
<td>38–54 kg</td>
<td>3 tabs</td>
</tr>
<tr>
<td>55–70 kg</td>
<td>4 tabs</td>
</tr>
<tr>
<td>&gt; 71 kg</td>
<td>5 tabs</td>
</tr>
</tbody>
</table>

**AND**

Continuation phase standard dose for 4 months

isoniazid + rifampicin daily 5 times per week

**Spinal TB**

continuation phase standard dose for 7 months

isoniazid + rifampicin daily 5 times per week
For children < 8 years

<table>
<thead>
<tr>
<th>Pretreatment body weight</th>
<th>Four months continuation phase When given FIVE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RH (60,30)</td>
</tr>
<tr>
<td>3–4 kg</td>
<td>½ tab</td>
</tr>
<tr>
<td>5–7 kg</td>
<td>1 tab</td>
</tr>
<tr>
<td>8–9 kg</td>
<td>1½ tabs</td>
</tr>
<tr>
<td>10–14 kg</td>
<td>2 tabs</td>
</tr>
<tr>
<td>15–19 kg</td>
<td>3 tabs</td>
</tr>
<tr>
<td>20–24 kg</td>
<td>4 tabs</td>
</tr>
<tr>
<td>25–29 kg</td>
<td>5 tabs</td>
</tr>
<tr>
<td>30–35 kg</td>
<td>6 tabs</td>
</tr>
</tbody>
</table>

For children > 8 years and adolescents

<table>
<thead>
<tr>
<th>Pretreatment body weight</th>
<th>Four months continuation phase When given FIVE times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RH (150,75) E (400) RH (300,150) E (400)</td>
</tr>
<tr>
<td>30–37 kg</td>
<td>2 tabs 2 tabs</td>
</tr>
<tr>
<td>38–54 kg</td>
<td>3 tabs 2 tabs</td>
</tr>
<tr>
<td>55–70 kg</td>
<td>2 tabs 3 tabs</td>
</tr>
<tr>
<td>&gt;71 kg</td>
<td>2 tabs 3 tabs</td>
</tr>
</tbody>
</table>

Ethionamide is preferred to ethambutol for children under 8 years of age.

**Treatment of Latent TB (Chemoprophylaxis)**

Active case finding is necessary for all children under the age of five years who are in close contact with an infectious TB case.

If the diagnosis of active TB is excluded, these children should be given prophylaxis with:

- rifampicin/isoniazid 60/30, oral for three months
  **OR**
  isoniazid, oral, 5 mg/kg daily for six months

All HIV positive children of any age in contact with an adult who is TB infected should be screened for tuberculosis. If negative, the child should receive chemoprophylaxis.

**REFERRAL**

- MDR TB – resistance to both rifampicin and isoniazid
- poor response to standard TB treatment
- failed therapeutic trial of tuberculosis therapy
- adverse drug reactions (ADR) requiring single drug combinations
15.2 CONDITIONS WITH PREDOMINANT WHEEZE

15.2.1 ASTHMA ATTACK, ACUTE

DESCRIPTION
Acute exacerbation of wheezing, unresponsive to bronchodilator therapy that is usually effective in a child who had been previously diagnosed with asthma.

DIAGNOSTIC CRITERIA
Cough after exercise, or nocturnal cough.
Clinical signs include:
- intense wheezing
- hyperinflation
- tachypnoea
- hypoxaemia
- restlessness
- difficulty or inability to talk or feed
- decreased air entry
- dyspnoea
- tachycardia
- anxiety
- palpable pulsus paradoxus
- reduced peak flow rate

The following are danger signs in acute, severe asthma and require referral:
- restlessness
- rising PaCO₂
- silent chest with auscultation
- PEFR < 60% of predicted
- disturbance in level of consciousness
- decreasing oxygen saturation
- palpable pulsus paradoxus
- chest pain (air leaks)

CLASSIFICATION OF SEVERITY OF ACUTE ASTHMA EXACERBATIONS

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>arterial PaO₂</td>
<td>&gt; 95%</td>
<td>90–95%</td>
<td>&lt; 90%</td>
</tr>
<tr>
<td>PEFR*</td>
<td>70–90%</td>
<td>50–70%</td>
<td>&lt; 50%</td>
</tr>
<tr>
<td>arterial PaCO₂</td>
<td>&lt; 35 mmHg</td>
<td>&lt; 40 mmHg</td>
<td>&gt; 40 mmHg</td>
</tr>
<tr>
<td>pulsus paradoxus</td>
<td>&lt; 10 mmHg</td>
<td>10–20 mmHg</td>
<td>20–40 mmHg</td>
</tr>
<tr>
<td></td>
<td>expiratory</td>
<td>expiratory and inspiratory</td>
<td>breath sounds soft</td>
</tr>
<tr>
<td>respiratory rate</td>
<td>&lt; 40</td>
<td>&gt; 40</td>
<td>&gt; 40</td>
</tr>
<tr>
<td>additional signs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Peak expiratory flow rate – patient’s best as percentage of predicted value of patient’s best
**NON-DRUG TREATMENT**
- admit child to a high care unit, if available
- monitor:
  - heart rate
  - respiratory rate
  - PEFR
  - pulse oximetry
  - blood pressure
  - acid–base status
  - blood gases
- ensure adequate hydration
  - encourage intake of normal maintenance oral fluids
  - do not overhydrate
  - if unable to drink IV fluid is deemed necessary, give:
- paediatric maintenance solution, IV ≤ 50 mL/kg over 24 hours. Give 60% of maintenance requirements.

**Note:**
Physiotherapy, antihistamines, antibiotics and sedation is not beneficial in the acute setting. Agitation or restlessness may be signs of severe hypoxia.

**DRUG TREATMENT**

**Step 1:**
To maintain arterial oxygen saturation ≥ 95% administer
- oxygen, 100%, at least 4–6 L/minute by facemask or 3–4 L/minute by nasal cannula or by head box

**PLUS**
Bronchodilator, i.e. short-acting β₂ agonist
- salbutamol, inhalation, 400–600 mcg (4–6 puffs) using a metered-dose inhaler with a spacer device
  - OR
  - salbutamol, solution, 0.15–0.3 mg/kg/dose (maximum 5 mg/dose), nebulise at 20 minute intervals for 3 doses
  - salbutamol 5 mg in 2–4 mL sodium chloride 0.9% delivered at a flow of 5 L/minute with oxygen

**PLUS**
Corticosteroids early in the attack
- prednisone, oral, 1–2 mg/kg, immediately up to a maximum of:
  - children < 2 years  20 mg
  - children 2–5 years  30 mg
  - children 6–12 years  40 mg

If salbutamol is not available
or patient is unable to inhale medication
or in a life-threatening situation not responding to other modalities of treatment
- adrenaline 1:1000, subcutaneous, 0.01 mL/kg (maximum 0.3 mL)
**Step 2:**
Assess response to treatment in step 1 by using the following table:

<table>
<thead>
<tr>
<th></th>
<th>Responder</th>
<th>Non-responder</th>
</tr>
</thead>
<tbody>
<tr>
<td>PEFR</td>
<td>improvement &gt;20% OR &gt; 80% (best/predicted)</td>
<td>improvement &lt; 20% OR &lt; 80% (best/predicted)</td>
</tr>
<tr>
<td>respiratory rate</td>
<td>&lt; 40/minute</td>
<td>&gt; 40/minute</td>
</tr>
<tr>
<td>retraction</td>
<td>absent</td>
<td>present</td>
</tr>
<tr>
<td>speech</td>
<td>normal</td>
<td>impaired</td>
</tr>
<tr>
<td>feeding</td>
<td>normal</td>
<td>impaired</td>
</tr>
</tbody>
</table>

**Responder:** patient who maintains an adequate response for at least 1 hour

**Non-Responder:** patient who fails to respond adequately to treatment in Step 1

**Step 3:**

**Responder**
Review current treatment, possible precipitating or aggravating factors and follow up.
- prednisone, oral, 2 mg/kg as a single daily dose for 7 days

if oral corticosteroids are not available
- dexamethasone, IV, 0.4 mg/kg, as a single dose. Not to be repeated.

**Non-responder**
Maintain hydration.
Intensify treatment as follows:
 Bronchodilator, i.e. short-acting $\beta_2$ agonist
- salbutamol, solution, 0.15–0.3 mg/kg/dose (maximum 5 mg/dose), nebulise at 20 minute intervals for 3 doses
  - 5 mg salbutamol in 2–4mL sodium chloride 0.9% delivered at a flow of 5 L/minute with oxygen

If no improvement after 30 minutes of continuous inhalation

**ADD**
- ipratropium bromide, solution, 0.25mg, nebulise 4 hourly
  - 0.25mg (2mL) ipratropium bromide in 2 mL sodium chloride 0.9%
  - Ipratropium bromide may be mixed with a $\beta_2$ agonist.

**PLUS**
Continue corticosteroid
- prednisone, oral, 2 mg/kg as a single daily dose for 7 days

If oral corticosteroids are not available
- dexamethasone, IV, 0.4 mg/kg, as a single dose. Not to be repeated.
Note:
Salbutamol, IV, can be used under supervision of a paediatrician.
• salbutamol, IV, loading dose 0.5 mcg/kg, followed by 0.2 mcg/kg/minute.
  The dose may be increased by 0.1 mcg/kg every 15 minutes to a maximum of
  4 mcg/kg/minute.

Step 4
Assess response to treatment in Step 3.
If non-responsive, admit to intensive care unit where aminophylline may be used under
specialist supervision.

REFERRAL
• acute exacerbation not responding to nebulised β2 agonists and/or corticosteroids

15.2.2 ASTHMA, CHRONIC

DESCRIPTION
Asthma is a chronic inflammatory disorder of the airways in which many cells and
cellular elements play a role. In susceptible individuals, this inflammation causes
recurrent episodes of wheezing, breathlessness, chest tightness and cough particularly
at night and in the morning. These episodes are usually associated with widespread
but variable airflow obstruction that is reversible either spontaneously or with treatment.
The inflammation also causes an associated increase in the existing bronchial
hyperresponsiveness to a variety of stimuli, e.g. allergens, viral infections, weather
changes, emotional upsets or other irritants.

DIAGNOSTIC CRITERIA
• chronic, persistent or recurrent coughing and/or wheezing that responds to a bronchodilator
• objective evidence of reversible airway obstruction, measured by > 12% improvement
  of the peak flow or FEV1, 20 minutes after administration of an inhaled bronchodilator
  confirms the diagnosis (FEV1 = forced expiratory volume in 1 second)
• a family history of atopy, night or exercise-induced coughing and/or wheezing supports
  the diagnosis

Assessment of Severity and Classification of Chronic Asthma
Before initiating treatment, classify patient according to clinical features (assign to the most
severe grade). Asthma can vary with time and regular reassessment is essential (at least
every 3 months).

Infrequent asthma: less than one acute exacerbation in 4–6 months
Persistent asthma: mild, moderate or severe

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>day time symptoms</td>
<td>2–4/week</td>
<td>&gt; 4/week</td>
<td>continuous</td>
</tr>
<tr>
<td>night time symptoms</td>
<td>2–4/month</td>
<td>&gt; 4/month</td>
<td>frequent</td>
</tr>
<tr>
<td>prior admission to hospital for asthma</td>
<td>none</td>
<td>one previous admission</td>
<td>&gt; one previous admission or admission to ICU</td>
</tr>
<tr>
<td>PEFR*</td>
<td>&gt; 80</td>
<td>60–80</td>
<td>&lt; 60</td>
</tr>
</tbody>
</table>

* Peak expiratory flow rate – patient’s best as percentage of predicted value of patient’s best.
NON-DRUG TREATMENT

- environmental control. If house dust mites are identified as a trigger, take measures such as:
  - use plastic mattress covers
  - remove bedroom carpets
  - wash blankets in hot water (> 70 °C)
- avoid triggers, e.g.:
  - exposure to cigarette smoke
  - preservatives such as sulphites and benzoates
  - house pets such as cats and dogs
- educate children, parents, caregivers and teachers

DRUG TREATMENT

Drug delivery systems

Spacer devices are recommended when a metered dose inhaler is used. All spacers should be primed with 2 doses of inhaled medication prior to first use. The size of the spacer is dependent on tidal volume of the child:

<table>
<thead>
<tr>
<th>Spacer volume</th>
<th>Face mask</th>
</tr>
</thead>
<tbody>
<tr>
<td>infants</td>
<td>150–250 mL</td>
</tr>
<tr>
<td>children</td>
<td>500 mL</td>
</tr>
<tr>
<td>adolescents</td>
<td>750 mL</td>
</tr>
</tbody>
</table>

Inhaled corticosteroid

The efficacy of inhaled corticosteroids is increased by the use of a spacer device. Rinse the mouth after inhalation of inhaled corticosteroids to reduce systemic absorption and adverse effects. The lowest possible effective dose of steroids should be used. Doses ≤ 400 mcg per day are associated with minimal side effects.

15.2.2.1 Asthma, Infrequent

Bronchodilator, i.e. short-acting β₂ agonist, when needed to relieve symptoms (inhalation therapy preferred).
- salbutamol, inhalation, 100–200 mcg, 3–4 times daily, using a metered-dose inhaler with a spacer device, until symptoms are relieved
  OR
  - salbutamol, oral, 0.15 mg/kg/dose, 4 times daily

Note:
Failure to respond to 2 doses of an inhaled bronchodilator given 20 minutes apart is a serious exacerbation of asthma. See Asthma, Acute Attack: Section 15.2.
### 15.2.2.2 Persistent Asthma

#### Mild persistent asthma
Bronchodilator, i.e. short-acting $\beta_2$ agonist, when needed for acute exacerbations.
- salbutamol, inhalation, 100–200 mcg, 3–4 times daily, using a metered-dose inhaler with a spacer device, until symptoms are relieved
  **OR**
  salbutamol, oral, 0.15 mg/kg/dose, 4 times daily
**PLUS**
Low dose inhaled corticosteroids
- beclomethasone or budesonide, inhalation, 50–100 mcg, 12 hourly using a metered-dose inhaler with a spacer device

#### Moderate persistent asthma
Bronchodilator, i.e. short-acting $\beta_2$ agonist, when needed to relieve symptoms
- salbutamol, inhalation, 100–200 mcg, 3–4 times daily, using a metered-dose inhaler with a spacer device, until symptoms are relieved
  **OR**
  salbutamol, oral, 0.15 mg/kg/dose, 4 times daily
**PLUS**
Regular anti-inflammatory treatment with medium dose inhaled corticosteroids, $\leq$ 400 mcg/day
- beclomethasone, inhalation, 100–200 mcg, 12 hourly, using a metered-dose inhaler with a spacer device, according to response

In children > 6 years (preferred option)
- budesonide, 100–200 mcg, 12 hourly using a metered-dose inhaler with a spacer device

In children > 4 years or children with multiple allergies on other steroid formulations
Low dose inhaled corticosteroids
- beclomethasone or budesonide, inhalation, 50–100 mcg, 12 hourly using a metered-dose inhaler with a spacer device

**PLUS**
Long acting $\beta_2$ agonist to allow the lower dose of corticosteroids e.g.:
- salmeterol or formoterol, inhalation, 12 hourly

#### Severe persistent asthma
Bronchodilator, i.e. short-acting $\beta_2$ agonist, when needed to relieve symptoms
- salbutamol, inhalation, 100–200 mcg, 3–4 times daily, using a metered-dose inhaler with a spacer device, until symptoms are relieved
  **OR**
  salbutamol, oral, 0.15 mg/kg/dose, 4 times daily

**PLUS**
Regular anti-inflammatory treatment with high dose inhaled corticosteroids
- beclomethasone or budesonide, inhalation, 200–400mcg, 12 hourly using a metered-dose inhaler with a spacer device
PLUS
Long acting $\beta_2$ agonist, e.g. salmeterol or formoterol, 12 hourly using a metered-dose inhaler with a spacer device.

REFERRAL
- after a life-threatening episode
- unstable asthma
- asthma interfering with normal life, despite treatment
- severe persistent asthma not responding to therapy

15.2.3 BRONCHIOLITIS

DESCRIPTION
Bronchiolitis is a viral infection of acute onset of the lower respiratory tract. It involves the small airways and causes varying degrees of wheeze.
Mainly occurs in autumn and winter and affects children between 4 months to 2 years of age.
The most common pathogen is the respiratory syncytial virus.
Recurrent episodes of wheeze may occur for months after an acute attack but in the vast majority the wheeze stops. Some children may develop asthma.

Assessment of Severity
Signs of severe disease include:
- infants under 3 months of age, especially premature babies
- inability to feed
- lower chest wall indrawing
- grunting
- discomfort in breathing
- hypoxia
- pneumothorax
- central cyanosis
- respiratory failure
- nasal flaring
- distress when speaking or crying
- apnoea
- convulsions and decreased level of consciousness
Mild cases are managed as outpatients.

DIAGNOSTIC CRITERIA
- prodrome of viral infection, irritability and feeding difficulties
- a wheeze that is slowly responsive or non-responsive to bronchodilators
- crepitations and signs of hyperinflation of the chest
A chest X-ray is useful in confirming hyperinflation and associated segmental atelectasis.

NON-DRUG TREATMENT
- isolate from other infants, if possible
- patients with signs of severe disease or associated complications or underlying cardiorespiratory disorders should be hospitalised for monitoring of:
  - breathing pattern (apnoea monitoring if < 3 months of age)
  - signs of respiratory failure
  - heart rate and respiratory rate
  - temperature
  - $\text{SaO}_2$
  - hydration and nutrition. IV maintenance fluid according to age, if oral/nasogastric feeds/fluids are not tolerated. Avoid overhydration.
DRUG TREATMENT
For all hospitalised patients
- oxygen, humidified, 1–2 L/minute via nasal prongs or nasal cannula or 4–6 L/minute via headbox
Oxygen therapy should be utilised to maintain a saturation of > 92%. Discontinue if this saturation is obtained on room air.
Ensure clear nasal passages and correct position of the nasal prongs.

Antibiotic therapy
Routine antibiotic therapy is not indicated. Only use if bacterial bronchopneumonia is suspected i.e.:
- raised leucocyte count
- persistent fever of ≥ 38.5°C
- and/or a chest X-ray showing opacification suggestive of pneumonia

In children < 20 kg
- amoxicillin, oral, 30 mg/kg/dose, 8 hourly for 7 days

Bronchodilators
There is no clear evidence of efficacy.
Evaluate response and discontinue treatment if no benefit is evident.
- ipratropium bromide, 0.25mg/2mL solution, nebulise 6–8 hourly
  0.5–1 mL ipratropium bromide diluted to 2–4 mL with sodium chloride 0.9% solution
AND/OR
- short-acting β₂ agonist, e.g. salbutamol, 0.5% solution, nebulise 4–6 hourly
  0.5–1 mL salbutamol diluted to 2–4 mL with sodium chloride 0.9% solution

Corticosteroids
There is no clear evidence of efficacy.
Use only in severe disease.
- prednisone, oral, 2 mg/kg/day, oral, as a single daily dose for 5 days.

REFERRAL
- bronchiolitis with signs of respiratory failure

15.3 COUGH WITH PREDOMINANT FEVER AND TACHYPOENOEA

15.3.1 PNEUMONIA
J18.9

DESCRIPTION
Infection of the lung parenchyma caused by bacteria and viruses. Pneumonia is classified as non-severe, severe or very severe based on clinical features.

Empiric antibiotics are indicated in all cases of pneumonia, as delay in treatment is associated with poor outcome. Antibiotic choice is based on an assessment of severity and likely aetiology.
Common bacterial causes of pneumonia include:

Neonates:
- Group B ß-haemolytic Streptococci
- *Klebsiella*
- *E. coli*
- *Chlamydia*
- *S. aureus*

Children:
- *S. pneumoniae*
- *H. influenzae*
- *S. aureus*
- *M. catarrhalis*
- *M. pneumoniae*

Common viral causes in infancy and early childhood include:
- influenza virus
- measles virus
- respiratory syncytial virus
- para-influenza virus
- cytomegalovirus
- adenovirus

Predisposing factors for pneumonia include:
- aspiration
- septicaemia
- measles
- cardiac disease
- presence of abnormalities in clearance of mucus/secretions (e.g. cystic fibrosis, foreign body and ciliary dysfunction)

Complications of pneumonia include:
- pleuritis
- empyema
- bronchiectasis
- respiratory failure
- pleural effusion
- pneumothorax
- lung abscess

**DIAGNOSTIC CRITERIA**

**Non-severe pneumonia**
- cough and fast breathing
- tachypnoea: age dependent:

<table>
<thead>
<tr>
<th>Age</th>
<th>Respiratory rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 60 days</td>
<td>&gt; 60/minute</td>
</tr>
<tr>
<td>2–12 months</td>
<td>&gt; 50/minute</td>
</tr>
<tr>
<td>1–5 years</td>
<td>&gt; 40/minute</td>
</tr>
</tbody>
</table>
Severe pneumonia
Above plus one of the following:
• lower chest wall indrawing
• auscultatory signs i.e. decreased breath sounds, bronchial breathing, crackles, increased vocal resonance or pleural rub
• dullness to percussion

Very severe pneumonia
Above plus at least one of the following
• central cyanosis
• inability to feed
• convulsions, lethargy or decreased level of consciousness
• grunting
• nasal flaring
• < 60 days old

Note:
All infants aged up to 60 days with pneumonia must be considered as having very severe disease.
Consider HIV infection and *S. aureus* in children with very severe pneumonia.

Investigations
• a chest X-ray should be performed when there is failure to respond to therapy, in severe and very severe disease where complications or the diagnosis of TB is suspected. A lateral and posterior-anterior view should be performed. A chest X-ray is not essential in non-severe pneumonia.
• PPD skin test
• if facilities are available, blood, induced sputum, nasopharyngeal aspirates (viruses and PCP) and gastric aspirate (TB) should be sent for microscopy and culture, preferably before initiating antibiotics

NON-DRUG TREATMENT
• bed rest
• clear nasal and oral passages of thick secretions
• neonates should be nursed in a neutral thermal environment
• monitor:
  • respiratory rate
  • blood pressure
  • acid–base status
  • temperature
  • heart rate
  • blood gases
  • SaO₂
  • hydration
  Hypercapnia and/or hypoxia are indications for ventilatory support.
• maintain nutrition - continue breast and oral feeds
  • consider small frequent feeds by oro/nasogastric tube or IV fluids if respiratory rate > 60/minutes or enteral feeds are not tolerated
SURGICAL TREATMENT
- chest tube drainage
  - large or symptomatic pneumothorax
  - empyema
  - most large pleural effusions
- needle aspiration
  - to relieve a tension pneumothorax, followed by chest tube placement
- small or asymptomatic pneumothoraces in infants and children (excluding neonates) usually do not require treatment other than close observation

DRUG TREATMENT
- oxygen, humidified, by nasal prongs is preferred
  - Oxygen should be continued until respiratory rate is < 60/minute.

Until fever has subsided
- paracetamol, oral or by nasogastric tube, 10 mg/kg, 6 hourly as required

If significant degree of wheezing is present
- salbutamol 1–2 puffs, 100–200 mcg, as required

Antibiotics, empirical
Choice of antibiotic depends on the severity of the condition and predisposing factors. Reconsider choice of antibiotic when the results of cultures become available or the child does not improve.

Non severe pneumonia
- amoxicillin, oral, 30 mg/kg/dose, 8 hourly for at least 3 days

Severe pneumonia
- benzylpenicillin (Penicillin G), IV, 50,000 units/kg/dose, 6 hourly for at least 2 days
When child improves follow with
- amoxicillin, oral, 30 mg/kg/dose, 8 hourly for 3 days
  OR
  - If unable to hospitalise
    amoxicillin, oral, 30 mg/kg/dose, 8 hourly for 5 days

Very severe pneumonia, including infants up to 60 days
- ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly for 5–10 days
  - children > 20 kg
    - ampicillin, IV, 250–500 mg, 6 hourly for 7 days
PLUS
- gentamicin, IV, 7.5 mg/kg as a single daily dose for 5–10 days
15.3.2 PREDISPOSING CONDITIONS AND MODIFICATION OF ANTIMICROBIAL THERAPY

15.3.2.1 Fungal infection

DESCRIPTION
May occur in immunosuppressed patients and present with deep draining sinuses or associated fungal lesions in the larynx, trachea or mouth.

DRUG TREATMENT
- amphotericin B, IV, 0.6–1.0 mg/kg as a single daily dose for at least 14 days
  OR
  fluconazole, IV/oral, 6–12 mg/kg as a single daily dose for at least 14 days

Neonates
< 2 weeks
- amphotericin B, IV, 0.6–1.0 mg/kg every 72 hours for at least 14 days
  OR
  fluconazole, IV/oral, 6–12 mg/kg every 72 hours for at least 14 days

2–4 weeks
- amphotericin B, IV, 0.6–1.0 mg/kg every 48 hours for at least 14 days
  OR
  fluconazole, IV/oral, 6–12 mg/kg every 48 hours for at least 14 days

15.3.2.2 Pneumonia due to anaerobic infection

DESCRIPTION
Often seen in comatose patients with aspiration syndromes.

DRUG TREATMENT
- metronidazole, oral, 7.5 mg/kg/dose, 8 hourly for 5 days
PLUS
- ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly for 5 days
  children > 20 kg
  - ampicillin, IV, 250–500 mg, 6 hourly for 5 days
PLUS
- gentamicin, IV, 7.5 mg/kg as a single daily dose for 5 days

15.3.2.3 Pneumonia, atypical due to Mycoplasma or Chlamydial infection
J15.7/J16.0

DESCRIPTION
Seen in school going children.
Presents with fever, arthralgia, headache, cough and crepitations.
In the neonatal period, chlamydial pneumonia presents with a staccato cough and sticky eyes.
Chest X-ray show interstitial infiltrates, lobar consolidation and hilar adenopathy.
**15.3.2.4 Pneumonia in HIV exposed or infected children**

**DESCRIPTION**
Due to additional unusual micro-organisms or dual infections, these children may fail to respond to the standard treatment for pneumonia. Micro-organisms more commonly involved are:

- *Pneumocystis jiroveci* (PCP)
- Cytomegalovirus
- Mycobacteria, e.g. *M. tuberculosis*
- *Salmonella*
- *E. coli*
- *Klebsiella*
- Pneumococci
- Fungi

PCP is common in infants from 2–6 months. It is suggested by an acute onset of respiratory distress with minimal/absent chest signs in a child who is HIV exposed. Hypoxaemia and cyanosis are common features and a chest X-ray shows bilateral perihilar interstitial changes. CMV commonly co-exists with PCP.

*S. pneumoniae* and gram negative bacteria also cause a significant proportion of pneumonia in early childhood.

Tuberculosis commonly affects children after infancy but can occur at any age. The diagnosis is difficult to confirm. A Mantoux test of \( \geq 5 \) mm induration is regarded as indicative of tuberculosis.

HIV positive children often acquire lymphoid interstitial pneumonitis and bronchiectasis. Secondary infection with bacteria similar to those found in acute pneumonia is common in these children.

**NON-DRUG TREATMENT**
- Avoid exposure to infectious agents
- Adequate nutrition

**DRUG TREATMENT**
For all severities of pneumonia in infants

**ADD**
- Trimethoprim/sulfamethoxazole, IV/oral, 5/25 mg/kg/dose, 6 hourly for 21 days
  - Continue trimethoprim/sulfamethoxazole prophylaxis for PCP and other bacterial infections after treatment. Primary prophylaxis is required in all HIV exposed children up to 12 months of age.
  - Secondary prophylaxis should be for life in any child who has been diagnosed with PCP
  - See *Pneumocystis jiroveci* Pneumonia (PCP): Section 8.16

Children who remain hypoxic on oxygen with proven or highly suspected PCP
- Prednisone, oral, 1–2 mg, daily for 7 days, then taper dose over 7 days
Children > 2 months with HIV associated pneumonia
Treat as above for pneumonia.

Any child failing standard therapy:

- exclude tuberculosis
- empiric treatment for \textit{S. aureus} and gram negative bacteria
- vancomycin, IV, 10 mg/kg/dose, 6 hourly, infused over 1 hour

\textbf{PLUS}

- amikacin, IV, 15–20 mg/kg once daily

Children with acute pneumonia or chronic lung disease
Treat as above for pneumonia.

Any child failing standard therapy, consider:

- tuberculosis
- fungi
- gram negative bacterial pathogens

\textbf{15.3.2.5 Pneumonia, nosocomial}

\textbf{DESCRIPTION}

Pneumonia developing 48–72 hours after hospitalisation.

The common pathogens are:

- extended spectrum ß-lactamase producing \textit{Klebsiella pneumoniae}
- \textit{Multi Resistant Staphylococcus Aureus}
- \textit{P. aeruginosa}
- Acinetobacter species
- respiratory viruses

\textbf{DRUG TREATMENT}

\textbf{Empiric treatment}

Broad spectrum antibiotics according to resident pathogens.

Children with underlying predisposing factors should be managed according to the susceptibility of the most likely pathogen

Review antibiotic choice once culture and sensitivity results become available.

\textbf{Modifications of therapy for antimicrobial allergy or resistance}

\textbf{Penicillin allergy}

- erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 7 days
  \textbf{OR}
  - clindamycin, IV, 10 mg/kg/dose, 8 hourly

\textbf{ß-lactamase producing pathogens}

- amoxicillin/clavulanic acid, oral, 30 mg/kg/dose of amoxicillin component, 8 hourly
15.3.2.6 Pneumonia, staphylococcal

DESCRIPTION
Presents as a toxic child with tachypnoea, chest wall indrawing chest wall and cough. White blood cells are usually markedly elevated. Chest X-ray shows a destructive pneumonia with pneumatocele, pleural effusion or empyema.

DRUG TREATMENT
- cloxacillin, IV, 50 mg/kg/dose, every 6 hours

if there is evidence of good clinical response, change to:
- flucloxacillin, oral, 12.5–25 mg/kg/dose, 6 hourly for at least 21 days

MRSA pneumonia
- fucidic acid, oral, 20 mg/kg/dose 8 hourly for 48 hours then stop.

PLUS
- clindamycin IV, 10 mg/kg/dose, 8 hourly for 7 days
  OR
  vancomycin, IV, 10 mg/kg/dose, 6 hourly infused over 1 hour

15.4 PLEURAL DISEASE

15.4.1 EFFUSION AND EMPYEMA

DESCRIPTION
Pleural effusion is an accumulation of fluid between the visceral and parietal pleura which may be an exudate or transudate. Common causes for exudates are infections, inflammation and malignancy. Common causes of a transudate are cardiac failure, renal failure and hepatic failure. A straw coloured or haemorrhagic effusion is indicative of tuberculosis. A cloudy or frankly purulent fluid indicates an empyema.

DIAGNOSTIC CRITERIA
- decreased breath sounds and stony dull on percussion
- pleural rub early in disease
- chest X-ray shows uniform opaque opacities in a lamellar distribution at the costophrenic angles

NON–DRUG TREATMENT
- small effusions should be treated conservatively
- other effusions should be drained by either chest drain or needle aspiration
  Samples should be sent for protein, glucose, cytology, microscopy and culture. If pus is identified or pH < 7.2, insert chest drain.
- transudates do not require drainage
- more aggressive surgical procedures such as open drainage or decortication are rarely indicated in children
DRUG TREATMENT
For purulent effusion
• cloxacillin, IV, 50 mg/kg/dose, every 6 hours
PLUS
• gentamicin, IV, 7.5 mg/kg as a single daily dose for 10 days

If there is evidence of good clinical response, change to:
• flucloxacillin, oral, 12.5–25 mg/kg/dose, 6 hourly for a total of 21 days

If pathogens are cultured, treat according to sensitivity for prolonged period of 21–42 days.

For straw coloured or haemorrhagic effusion
• start antituberculosis therapy
If no response, consider fungal infection.

15.5 UPPER AIRWAY DISEASES

15.5.1 EPIGLOTTITIS
J05

DESCRIPTION
Life-threatening upper airway obstruction at the level of the supraglottic structures (epiglottis and arytenoids).
The condition is rare since due to H. influenzae type B vaccinations has been introduced.

DIAGNOSTIC CRITERIA
• acute onset, high fever, sore throat, dysphagia, refusal to eat or swallow, drooling and muffled voice
• position of comfort to protect the upper airway: sitting upright, head forward, open mouth, neck in extension

NON–DRUG TREATMENT
• provide supplemental humidified oxygen
• monitor oxygen saturation (pulse oximeter)
• do not interfere with the protective mechanism of the patient
  ○ allow the child to remain sitting up
• avoid all measures that could agitate the patient
  ○ make no attempt to see the epiglottis
  ○ do not perform X-rays of neck and chest routinely
  ○ delay blood sampling and IV line insertion until after airway is secured

Acute airway obstruction

CAUTION
Epiglottitis is an upper airway emergency.
Total upper airway obstruction is imminent by the time stridor appears.
Prepare equipment for bag-mask ventilation, endotracheal intubation, needle cricothyroidotomy and tracheostomy.
If airway obstructs completely or respiratory arrest occurs, attempt to establish an airway.
• ventilate with bag and mask
If unable to ventilate
• intubate
If unable to intubate
• perform needle or surgical cricothyroidotomy

Total airway obstruction may occur suddenly and quite unpredictably, the patient should ideally be intubated before referral. Intubation should preferably be performed under general anaesthesia in an operating theatre.

If intubation before referral is not possible, transport patient immediately to a centre. Inform the receiving hospital before departure.

During transport, if the child decompensates, attempt bag and mask ventilation. Inform the receiving hospital before departure.

After an open airway has been secured:
• take blood for cultures
• swab epiglottis for microscopy, culture and sensitivity
• monitor heart rate, respiratory rate, blood pressure and $\text{SaO}_2$
• ensure adequate nutrition and hydration

**DRUG TREATMENT**
• cefotaxime, IV, 50 mg/kg/dose, 8 hourly for 7 days

**Penicillin allergy**
• chloramphenicol, IV, 25 mg/kg/dose, 6 hourly for 7 days

**REFERRAL CRITERIA**
• all, once airway is secured

**15.5.2 LARYNGOTRACHEOBRONCHITIS, ACUTE VIRAL (CROUP)**

**DESCRIPTION**
Potentially life-threatening airway obstruction in children and one of the most common causes of stridor in children aged between 6 months and 2 years.
The most common viruses causing laryngotracheobronchitis (LTB) include:
• para-influenza virus (most common)
• measles
• herpes simplex
• adenovirus

**DIAGNOSTIC CRITERIA**
**Clinical**
• a previously healthy child who, a day or two after the onset of an upper respiratory tract infection, develops progressive airway obstruction with a barking cough and stridor
• a mild fever may be present
• stridor becomes softer as airway obstruction becomes more severe
The following features suggest a different diagnosis:

- acute onset of obstruction without prodromal features (foreign body or angioneurotic oedema)
- incomplete immunisation and a membrane in the upper airway (diphtheria)
- high fever, dysphagia, drooling or sitting position (epiglottitis, retropharyngeal abscess, bacterial tracheitis)
- recurrent upper airways obstruction (laryngeal papilloma)

**Assessment of Severity of Airway Obstruction in LTB**

<table>
<thead>
<tr>
<th>Severity</th>
<th>Inspiratory obstruction (Stridor)</th>
<th>Expiratory Obstruction (Stridor)</th>
<th>Pulsus paradoxus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 2</td>
<td>+</td>
<td>+ passive expiration</td>
<td></td>
</tr>
<tr>
<td>Grade 3</td>
<td>+</td>
<td>+ active expiration using abdominal muscles</td>
<td></td>
</tr>
<tr>
<td>Grade 4</td>
<td>cyanosis, apathy, marked retractions, impending apnoea</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Non-Drug Treatment**

- monitor the nutritional status and fluid requirements
- monitor oxygen saturation, heart rate and respiratory rate
  - Avoid arterial blood gas estimations – clinical criteria are more effective in determining severity.
- depending on severity, admit child to high care or intensive care ward

**Drug Treatment**

**Grade 1 Obstruction**

*Note:*

Do not give corticosteroids to patients with measles or herpes infection.

- prednisone, oral, 2 mg/kg as a single dose
  - OR
dexamethasone, IV/IM, 0.5 mg/kg as a single dose

**Grade 2 Obstruction**

As above

**Plus**

- adrenaline, 1:1000, nebulise with oxygen, every 15–30 minutes until expiratory obstruction is abolished
  - 1 mL adrenaline 1:1 000 diluted in 1 mL sodium chloride 0.9%

**Grade 3 Obstruction**

As above

- if improvement, treat as in grade 2
- if no improvement within 1 hour, intubate, preferably under general anaesthetic
- refer
**Grade 4 obstruction**

As above and:
- give steroids
- continue with adrenaline nebulised with 100% warm humidified oxygen
- emergency intubation or intubation under general anaesthesia if circumstances permit
  - if unable to intubate, bag and mask ventilate
- refer urgently

For fever
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly until fever subsides

For suspected herpes
- aciclovir IV, 10–15 mg/kg/dose 8 hourly for 5–7 days
- ampicillin, IV, 12.5–25 mg/kg/dose, 6 hourly for 5–10 days
  - children > 20 kg
    - ampicillin, IV, 250–500 mg, 6 hourly for 7 days
- PLUS
  - cloxacillin, IV, 50 mg/kg/dose 6 hourly for 7 days

**REFERRAL**

**Note:**
Patient should be intubated before referral.
- all grade 4 airway obstruction
- grade 3 not responding within one hour to adrenaline nebulisations
- children where features suggest a different diagnosis
Note:
Use only preservative free sterile eye drops if there is a possibility of an open eye injury.

16.1 EYE INFECTION, COMPLICATED (SEVERE EYE INFECTION)
H44

DESCRIPTION
Intensely painful, red eye with or without a discharge.

DIAGNOSTIC CRITERIA
Clinical
Intensely painful, red eye with any of the following:
• reduced vision
• a cloudy cornea
• a corneal opacity or a staining ulcer
• pus level in the anterior chamber
• blood in the anterior chamber
• cloudiness in the anterior chamber (poor view of iris details)
• a fixed semi dilated pupil (which hasn’t been atropinised)
• a cloudy fundal view or poor (greyish ) red reflex
• proptosis
• restricted ocular movements
• vomiting

Investigations
• pus samples or scrapings for microscopy and culture

DRUG TREATMENT
If associated with purulent discharge
• chloramphenicol 0.5%, ophthalmic drops, 1 drop instilled hourly for the first 24 hours,
  then 1 drop 2 hourly for the next 24 hours thereafter 4 hourly for 7–10 days

URGENT REFERRAL
Urgent – to an ophthalmologist
• any of the above clinical findings with an intensely painful red eye

REFERRAL
Within 24 hours
• any red eye not responding to treatment
16.2 EYE INJURY, CHEMICAL BURN
T26.9

DESCRIPTION
Damage to one or both eyes caused by contact with irritating chemical substances e.g. alkali or acid, presenting with:
- pain
- inability to open eye
- blurred vision
- excessive teary and watery eye

CAUTION
do not neutralise acid with alkaline and vice versa

NON-DRUG TREATMENT
- irrigate affected eye/s immediately and continuously with copious amounts of sterile sodium chloride 0.9% solution or sterile water (at least 2L) using an eye speculum and an IV fluid giving set
- in severe alkaline burn cases, irrigation should be prolonged further
- for severe chemical burns check pH of conjunctival sac with litmus. If alkaline, irrigation should be prolonged.
- stain with fluorescein to assess extent of epithelial loss

DRUG TREATMENT
For pain
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

Anaesthetise affected eye/s with benoxinate or amethocaine drops.

CAUTION
Do not attempt to neutralise alkali with acid or vice versa.

REFERRAL
- any severe chemical burn producing epithelial loss or cloudiness of the cornea - immediately to the nearest ophthalmologist

16.3 EYE INJURY WITH FOREIGN BODY
S05.5

DESCRIPTION
Foreign body on or embedded in the cornea or conjunctiva.
Penetration through the cornea or sclera to deeper structures.

Complications range from a corneal scar to intraocular infection and loss of the eyeball depending upon where the foreign body is situated.

DIAGNOSTIC CRITERIA
Examine the cornea and anterior chamber, ideally with a slit lamp or by any means of magnification, to verify the position of the foreign body and ascertain the damage. Look for signs of ocular penetration in cases with high velocity injuries, e.g. from a hammer and chisel.
Investigations
If a foreign body is not visible, corneal abrasions can be diagnosed by fluorescein stain, which fluoresces bright green when a blue light is applied. An orbital X-ray may reveal a retained intraocular foreign body following a high velocity injury.

SURGICAL TREATMENT
Removal of a deeply embedded or full thickness corneal foreign body or any intraocular foreign body should be done with an operating microscope in a specialist ophthalmic unit.

DRUG TREATMENT
To remove a superficial corneal foreign body with a bud or hypodermic needle
Anaesthetise the cornea with amethocaine or benoxinate drops.

To relieve ciliary spasm which causes much of the discomfort
• homatropine 2%, ophthalmic drops, 1 drop instilled immediately
  OR
  cyclopentolate 0.5–1%, ophthalmic drops, 1 drop instilled immediately

Cover affected eye firmly with eye pad for 12 hours.

Until epithelialisation is complete
• chloramphenicol 1%, ophthalmic ointment, applied three times daily, for 5–10 days

REFERRAL
Within 12 hours
• any deeply embedded corneal foreign body
• any eye with suspected intraocular penetration

16.4 EYE INJURY WITHOUT A FOREIGN BODY
S05.6

DESCRIPTION
A blunt injury causing closed ocular contusion.
A penetrating eye injury with or without prolapse of intracocular contents.
Complications depend on the severity and type of injury.

DIAGNOSTIC CRITERIA
A penetrating eye injury requires recognition and urgent referral to the nearest ophthalmic specialist to avoid endophthalmitis and loss of the eyeball. A severely contused eye also needs specialist attention.

SURGICAL TREATMENT
Should be done by a specialist ophthalmologist with an operating microscope.
DRUG TREATMENT
To prevent infection prior to repair or referral
• chloramphenicol 0.5%, ophthalmic drops, instil 1 drop, immediately
  Apply a clean sterile eye pad and transfer to the nearest specialist eye unit.

URGENT REFERRAL
• any severely traumatised eye
• a penetrating eye injury
• corneal or scleral laceration
• distorted pupil
• flat or very shallow anterior chamber (compare with other eye)
• blood inside the eye
17.1 ABSCESS, RETROPHARYNGEAL

DESCRIPTION
An infective process of the retropharyngeal space either due to:
- lymphatic spread
- extension of infection from surrounding tissues
- local injury.
Consider cold abscess of TB as a possible cause.

DIAGNOSTIC CRITERIA
Clinical
- stridor and difficulty in breathing
- dysphagia, drooling
- extension of the neck
- swelling on one side of posterior pharyngeal wall

Investigations
- lateral X-ray of the neck may show the retropharyngeal space to be wider than the C4 vertebral body when a retropharyngeal abscess is present

NON-DRUG TREATMENT
- surgical drainage of abscesses
- protect the airway
- ensure adequate hydration by providing fluids intravenously or by nasogastric tube

DRUG TREATMENT
Empirical antibiotic therapy
Antibiotic treatment should be initiated immediately even if transfer of the patient is anticipated.
Antibiotic therapy should be adjusted if cultures are available.
Early complications may be treated with antibiotic therapy alone.

Third generation cephalosporin, e.g.
- ceftriaxone, IM/IV, 80–100 mg/kg/dose as a single daily dose
  OR
  cefotaxime, IV, 25–50 mg/kg/dose, 6-8 hourly
PLUS
- metronidazole, IV, 7.5 mg/kg/dose, 8 hourly
Change to oral medication as soon as there is a response and patient is able to swallow
- amoxicillin/clavulanic acid, oral, 25–30 mg/kg/dose, 8 hourly
PLUS
- metronidazole, oral, 7.5 mg/kg/dose, 8 hourly
Penicillin allergy
- clindamycin, IV, 10–15 mg/kg/dose 6–8 hourly. (Max adult dose 900 mg/dose)

Analgesia and antipyretic
- paracetamol 10 mg/kg/dose 4–6 hourly as required

REFERRAL
- all children with suspected retropharyngeal abscess

17.2 TONSILLITIS, COMPLICATED (PERITONSILLAR CELLULITIS, PERITONSILLAR ABSCESSES)

DESCRIPTION
An infective process involving the tonsils with spread of infection into the adjacent tissue. Local complications include peritonsillar cellulitis and abscess (quinsy), and suppurative cervical adenitis.
Systemic complications include glomerulonephritis, rheumatic fever and bacterial endocarditis.

DIAGNOSTIC CRITERIA
- pyrexia, malaise
- sore throat, dysphagia, drooling, trismus
- earache (referred otalgia)
- tender and enlarged cervical lymph nodes

Signs of peritonsillar abscess/cellulitis:
- usually unilateral
- soft palate and uvula on the infected side are oedematous and displaced medially towards the uninvolved side

NON-DRUG TREATMENT
- drain abscesses surgically
- if necessary, maintain the airway

DRUG TREATMENT
Empiric antibiotic therapy
Antibiotic treatment should be initiated immediately even if transfer of the patient is anticipated.
Antibiotic therapy should be adjusted if cultures are available.
Early complications may be treated with antibiotic therapy alone.

Third generation cephalosporin, e.g.
- ceftriaxone, IM/IV, 80–100mg/kg as a single daily dose
  OR
  cefotaxime, IV, 25–50mg/kg/dose, 6–8 hourly
PLUS
- metronidazole, IV, 7.5mg/kg/dose, 8 hourly
Change to oral medication as soon as there is a response and patient is able to swallow
  • amoxicillin/clavulanic acid, oral, 25–30mg/kg/dose of amoxicillin component, 8 hourly
  PLUS
  • metronidazole, oral, 7.5 mg/kg/dose, 8 hourly

Penicillin allergy
  • clindamycin, IV, 10–15 mg/kg/dose 6–8 hourly
    OR
    erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 7 days
  PLUS
  • metronidazole, oral, 7.5 mg/kg/dose, 8 hourly

Analgesia and antipyretic
  • paracetamol 10–15 mg/kg/dose 4–6 hourly as required

REFERRAL
  • tonsillitis with local complications not responding to adequate treatment
  • all cases where surgery may be required and is not available locally

17.3 EPISTAXIS (Nose bleed)
R04.0

DESCRIPTION
Nose bleed may be caused by local or systemic diseases, or local trauma, especially nose
picking and occurs from an area anterior and inferiorly on the nasal septum. Consider
other conditions associated with nosebleeds, especially if recurrent, e.g. hypertension and
bleeding tendency. Persistent or severe bleeds may require hospital care.

Complications include anaemia and hypovolaemic shock.

DIAGNOSTIC CRITERIA
  • history of spontaneous nose bleeds
  • recurrent nose bleeds
  • underlying problems include bleeding disorders and local intranasal pathology. The
    child should be examined for nasal lesions and signs of haematological disease and
    coagulopathies.

NON-DRUG TREATMENT
  • digital pressure
    ○ squeeze the nasal wings (alae) of the nose between the thumb and forefinger to
      apply pressure to the nasal septum and maintain pressure for about 10 minutes
    ○ the child should sit up and lean forward so as not to swallow the blood, and should
      breathe through the mouth.
    ○ if digital pressure fails blood clots should be removed from the nose. The child may
      be able to do this by blowing his nose.
DRUG TREATMENT

Vasoconstrictor

If digital pressure fails
- oxymetazoline 0.025%, nose drops, 1–2 drops instilled into the affected nostril(s) and repeat digital pressure as above

Anterior nasal pack

If bleeding continues and appears to originate from the anterior nasal cavity, pack the cavity with cotton gauze tape impregnated with bismuth iodine paste.
Lidocaine spray may be used for topical anaesthesia prior to packing.

Anaemia

Packed cells, IV, 10–15 mL/kg, if:
- there is symptomatic anaemia
- the haemoglobin is less than 7 g/dL with ongoing epistaxis
- there is an underlying disorder in which severe re-bleeding is likely

Treat the underlying disorder appropriately.

REFERRAL

- epistaxis caused by a serious underlying disorder
- epistaxis that is not controlled by the above measures
- recurrent epistaxis

17.4 MASTOIDITIS

H70.9

DIAGNOSTIC CRITERIA

Clinical
- fever, severe pain, increasing hearing impairment, tenderness over mastoid antrum
- swelling in post-auricular area. Pinna is pushed down and forward.
- tympanic membrane is usually perforated with otorrhoea
- occasionally, pus breaks through the mastoid tip and forms an abscess in the neck (Bezold’s abscess)

Investigations
- mastoid X-rays show opacity and air-cell coalescence
- a CT scan can confirm the diagnosis
- collect blood and pus for Gram stain, microscopy, culture and sensitivity tests before initiation of antibiotic therapy

NON-DRUG TREATMENT

- dry mopping of the external auditory canal
Antibiotic therapy
Reassess antibiotic therapy as soon as culture results become available or if response to antibiotic therapy is unsatisfactory. Oral antibiotics may be considered after clinical improvement and should be determined by culture and sensitivity. Total duration of therapy of at least 14 days is recommended.

- ceftriaxone, IV, 80–100 mg/kg as a single daily dose.

If improvement
- amoxicillin/clavulanic acid, oral, 25–30 mg/kg/dose of amoxicillin component, 8 hourly

For pain
- paracetamol, oral, 10–15 mg/kg/dose 6 hourly as required

URGENT REFERRAL
- urgently to ENT surgeon after initiation of antibiotics

### 17.5 OTITIS EXTERNA
H60.9

**DESCRIPTION**
Inflammation of the external ear.

**NON-DRUG TREATMENT**
- exclude underlying chronic otitis media prior to treatment
- keep the ear clean and dry using a wick of rolled absorbent cloth

**DRUG TREATMENT:**
- acetic acid 2% in alcohol, instil 3–4 drops 4 times daily into the cleaned and dried ear

### 17.6 OTITIS MEDIA, ACUTE
H66.9

**DESCRIPTION**
Inflammation of the middle ear that may be complicated by perforation and a purulent ear discharge.

**NON-DRUG TREATMENT**
- avoid getting the inside of the ear wet

**DRUG TREATMENT**
- amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5–10 days

### 17.7 OTITIS MEDIA, CHRONIC, SUPPURATIVE
H66.3

**DIAGNOSTIC CRITERIA**
A purulent discharge from the ear for more than 2 weeks.
Note:
TB is an important cause of a chronic discharge from the ear. Chronic otitis media is also associated with HIV.

NON-DRUG TREATMENT
• dry mopping is the most important part of the treatment. It should be demonstrated to the child’s caregiver or patient if old enough.
• Continue with dry mopping for 4 weeks
  ○ roll a piece of clean absorbent cloth into a wick
  ○ carefully insert the wick into the ear with twisting action
  ○ remove the wick and replace with a clean dry wick
  ○ repeat this until the wick is dry when removed
  ○ soak a clean wick in acetic acid 1% in sodium chloride 0.9%
  ○ insert carefully into the ear
  ○ leave in place for 1 minute
  ○ remove the wick and replace with a clean dry wick
  ○ watch the patient or caregiver repeat this until the wick is dry when removed
  ○ dry the ear by wicking at home three to four times daily until the wick stays dry
  ○ if bleeding occurs, drying the ear should be stopped temporarily
• do not leave anything in the ear
• do not instil anything else in the ear
• avoid getting the inside of the ear wet, e.g. swimming and bathing

DRUG TREATMENT
• fluoroquinolone, eardrops, e.g. ofloxacin drops, 2 drops 8 hourly instilled in affected ear after dry mopping for 4 weeks.

URGENT REFERRAL
• all with suspected intracranial complication

ELECTIVE REFERRAL
• large central perforation
• no improvement after 4 weeks

17.8 RHINITIS, ALLERGIC
J30.4

DESCRIPTION
Recurrent inflammation of the nasal mucosa due to hypersensitivity to inhaled allergens.

NON-DRUG TREATMENT
• avoid allergens and irritants

DRUG TREATMENT
• chlorpheniramine, oral, 0.1 mg/kg/dose three times daily
• corticosteroid aqueous nasal solution, 2 sprays into each nostril twice daily
17.9 SINUSITIS, ACUTE
J01.9

DESCRIPTION
Inflammation of one or more sinuses that occurs most often after a viral nasal infection or with allergic rhinitis.

NON-DRUG TREATMENT
• steam inhalation may be effective in liquefying and removing secretions blocking the nose

DRUG TREATMENT
• amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5 days
• paracetamol, oral, 10–15 mg/kg/dose 6 hourly as required
• oxymetazoline 0.025%, nose drops, 2 drops instilled into each nostril, 6–8 hourly for not more than 5 days continuously.

17.10 SINUSITIS, CHRONIC
J32.9

DIAGNOSTIC CRITERIA
Clinical
• chronic purulent postnasal drip for more than two weeks
• nasal congestion, headache, facial pain or percussion tenderness

Investigations
• X-ray or CT scan may show opacities and fluid levels

NON-DRUG TREATMENT
• identify and treat the underlying cause, e.g. nasal allergy
• hypertonic sodium chloride, 3.5% drops, may improve outcome

DRUG TREATMENT
There is no clear evidence that antibiotics improve the outcome.
If non-medicine treatment fails, a trial of antibiotics may be tried in unresponsive cases
• amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5 days

Analgesia
• paracetamol, oral, 10–15 mg/kg/dose 6 hourly as required

REFERRAL
• failure to achieve progressive improvement
17.11 SINUSITIS, COMPLICATED

DIAGNOSTIC CRITERIA

Clinical
• signs and symptoms of complications:
  ○ peri-orbital swelling and fever
• signs of meningeal irritation:
  ○ neck stiffness, positive Kernig’s and Brudzinski’s signs
• signs of increased intracranial pressure:
  ○ hypertension, bradycardia, papilloedema, headache
• signs of involvement of orbital structures:
  ○ periorbital oedema, erythema, chemosis, proptosis, vision loss, ophthalmoplegia
• signs of brain involvement:
  ○ neurological signs, ataxia, paresis, paralysis, convulsions, altered level of consciousness

Investigations
• X-ray or CT scan may show opacities and fluid levels
• CT scan will show if there is involvement of intracranial structures, e.g. brain abscess.
• pus, cerebrospinal fluid (CSF) and blood for culture and sensitivity tests. Microscopy and Gram-staining of pus and CSF specimens may give some indication of the microorganism/s involved.

DRUG TREATMENT

Empiric antibiotic therapy
Initiate empiric antibiotic therapy and reassess as soon as culture and sensitivity results become available or if there is no improvement within 48–72 hours.
Total duration of therapy of 14 days is recommended.

• ceftriaxone, IV, 80–100 mg/kg as a single daily dose

Once improvement
• amoxicillin/clavulanic acid, oral, 25–30mg/kg/dose of amoxicillin component, 8 hourly

Penicillin allergy
• clindamycin, IV, 10 mg/kg/dose, 8 hourly
  OR
  erythromycin, oral, 6.25–12.5 mg/kg/dose, 6 hourly for 7 days

For pain
• paracetamol, oral, 10–15 mg/kg/dose 6 hourly as required

URGENT REFERRAL
• spread of infection to:
  ○ eye/orbital structures
  ○ intracranial structures/brain
CHAPTER 18
POISONING

18.1 POISONING

DESCRIPTION
Frequently encountered poisonings in children include:

- analgesics
- vitamins and minerals
- hydrocarbons
- anticonvulsants
- pesticides
- phenothiazines
- plant material
- sedatives and antidepressants

DIAGNOSTIC CRITERIA

Clinical
- can be divided into ‘toxidromes’:
  **Cholinergic:**
  - salivation
  - lacrimation
  - urination
  - defaecation

  - diarrhoea
  - vomiting
  - bronchorrhoea
  - bradycardia

  **Salicylism:**
  - tachypnoea
  - metabolic acidosis
  - seizures

  - agitation
  - coma

  **Anticholinergic:**
  - fever
  - ileus
  - flushing
  - tachycardia
  - urination

  - dry/warm skin
  - blurred vision
  - mydriasis (dilated pupil)
  - coma
  - hallucinations and seizures

  **Sedative-hypnotic:**
  - obtundation or coma, with normal vital signs

  **Opiates:**
  - miosis
  - respiratory depression
  - bradycardia

  - decreased bowel sounds
  - hypothermia
  - altered (decreased) mental status

  **Dystonic reaction:**
  - torticollis
  - opisthotonus
  - intermittent spasms and tongue thrusting
**Sympathomimetic:**
- hypertension
- agitation
- tachycardia
- diaphoretic skin
- hyperthermia
- dilated pupils

Sympathomimetic toxidrome resembles anticholinergic toxidrome, i.e. fight, flight and fright response.

**Toxic alcohols:**
- metabolic acidosis
- hypoglycaemia
- increased osmolar gap
- convulsions
- visual disturbances (methanol)
- renal failure (ethylene glycol)
- depressed level of consciousness (alcohol)

**TREATMENT**
If the ingestion has definitely occurred, establish whether toxicity is expected and act accordingly.

If the possibility of ingestion was remote, only observation is necessary.

- stabilise patient, if necessary
- if there is the risk of toxicity, decontaminate patient

**Gastric lavage**
- contraindications
  - if a corrosive substance or volatile hydrocarbon has been ingested
  - if patient is unconscious unless the airway is protected
- indicated only if patient has ingested a potentially life-threatening poison and the procedure can be undertaken within 60 minutes of ingestion

**Use of adsorbents, i.e. activated charcoal**
- the following substances are NOT adsorbed by activated charcoal
  - all alcohols
  - hydrocarbons
  - metals, e.g. lead
  - minerals, e.g. sodium
- administer only if patient has ingested a potentially toxic amount of a poison which is known to be adsorbed by charcoal up to one hour previously. There is insufficient data to support or exclude its use after one hour of ingestion.
- dose of activated charcoal given as a slurry:
  - < 6 years 10 g in 50–100 mL water
  - > 6 years 20–50 g in 100–300 mL water
- Placement of a nasogastric tube may be necessary for its prompt administration.

**Whole bowel irrigation**
- use only for poison due to iron, lithium, lead, or sustained release or enteric-coated medicines
- polyethylene glycol solution, oral, 30 mL/kg/hour equivalent to:
  - children 0.5 L/hour
  - adolescents 2 L/hour
- Continue until rectal effluent is clear.

- antidote, if available
- enhance elimination, if possible
REFERRAL
- severely ill patient for ventilatory/circulatory support
- where relevant diagnostic testing is not available, e.g. paracetamol levels
- where relevant medication/antidotes is not available
- where dialysis/haemoperfusion is required
- for psychiatric evaluation

18.1.1 ANTICHOLINERGIC POISONING

DESCRIPTION
Various plant species and pharmaceutical preparations can cause anti-cholinergic toxicity. Plants: *Datura stramonium*, e.g. “stinkblaar and malpitte”
Drugs including atropine, diphenoxylate with atropine and diphenhydramine.
Other classes of drugs include Antiparkinsonian agents, antispasmodics, antipsychotics and tricyclic antidepressants.

DIAGNOSTIC CRITERIA
Clinical
- alteration of mental status, including delirium, hallucinations, agitation and seizures
- peripheral anticholinergic effects include:
  - mydriasis
  - urinary retention
  - tachycardia
  - decreased GIT motility
  - flushing
  - dry skin and mucous membranes

Investigations
- continuous cardiac monitoring
- pulse oximetry

NON-DRUG TREATMENT
- stabilise patient, i.e. airway, breathing and circulation
- cooling for hyperthermia
- perform decontamination depending on route of exposure

DRUG TREATMENT
- activated charcoal

For agitation
- diazepam, IV/oral, 0.1–0.2 mg/kg
  Maximum dose: 10 mg.

For seizures
See Status Epilepticus: Section 13.4

REFERRAL
- cardiac dysrhythmia
- no response to treatment
18.1.2 ANTICOAGULANT POISONING

**DESCRIPTION**
Poisoning with warfarin and super warfarin, marketed as pellets. Over the counter pesticides containing warfarin may be accidentally ingested by toddlers or young children.

**DIAGNOSTIC CRITERIA**
**Clinical**
- signs and symptoms depend on the potency
- symptoms may range from the asymptomatic child, e.g. a small child who has tasted a rodenticide, to significant cases which present with bruising or bleeding, e.g. if the child has ingested "super-warfarin" containing pesticides

**Investigations**
- measure prothrombin time

**NON-DRUG TREATMENT**
- observe asymptomatic child

**DRUG TREATMENT**
- consider gastric decontamination
- vitamin K\(_1\) (phytomenadione), IV/oral, 1–5 mg/dose 6 hourly
  Repeat if large doses were administered.

Ingestion of super warfarin may be refractory to large doses of vitamin K\(_1\). These cases may be candidates for repeated doses of fresh frozen plasma.

18.1.3 ANTIDEPRESSANT (TRICYCLIC) POISONING

**DESCRIPTION**
Poisoning with tricyclic antidepressants represent a large portion of poisoning fatalities. There is a high risk of tricyclic antidepressant toxicity in children because of its narrow therapeutic index.

**DIAGNOSTIC CRITERIA**
- 10–20 mg/kg of tricyclic antidepressant medication will cause significant toxicity in most children
- causes anticholinergic syndromes
- mainly affect the cardiovascular system, autonomic nervous system, and CNS, leading to:
  - conduction delays
  - dysrhythmias
  - hypotension
  - altered mental status
  - seizures
NON–DRUG TREATMENT
- gastric lavage for large ingestions or patient presenting within 1 hour, except if patient is unconscious
- circulatory and respiratory support
- cardiac and ECG monitoring for 48 hours

DRUG TREATMENT
- activated charcoal, oral, 10–20 g every 2 hours until charcoal appears in the stool

For cardiac arrythmias
- antiarrhythmic agents. Only under specialist supervision

Alkalisation
Alkalisation up to an arterial pH of 7.45 – 7.5 has been shown to reduce the toxic effects on the heart.
- sodium bicarbonate 4.2%, IV, 1–2 mmol/kg as a bolus
  May be repeated.
  Follow with a continuous infusion in consultation with senior/poison centre.

For hypotension
- sodium chloride 0.9% or Ringer–Lactate, IV bolus, 20 mL/kg

For circulatory and respiratory support
See Cardiorespiratory Arrest: Section 1.1.3

REFERRAL
- any cardiac arrhythmia

18.1.4 CAUSTIC OR CORROSIVE AGENTS, INGESTION
Y19

DESCRIPTION
Caustic agents, e.g. sodium hydroxide or potassium permanganate, corrosive agents, e.g. hydrochloric acid.
Acids and alkali do not differ in their severity.

Note:
Battery acid causes significant corrosive damage, whereas bleach seldom has a corrosive effect.

DIAGNOSTIC CRITERIA
Clinical
- chief symptom is pain
- young children may present with:
  - crying
  - drooling
  - refusal to swallow
  - vomiting
- stridor or hoarseness indicate laryngeal injury
- the presence of oral or pharyngeal burns does not predict the presence of oesophageal or gastric injury
- oesophageal or gastric injury can cause perforation or subsequent fistula formation
- patients with no clinical signs or symptoms are unlikely to have significant oesophageal or other organ injury
NON- DRUG TREATMENT

Asymptomatic
• monitor for development of symptoms
  ○ a 12 hour symptom free period usually indicates that no intervention is necessary

Symptomatic
• gastric decontamination is contraindicated in all cases
• keep patient nil per mouth
• airway injury may necessitate endotracheal intubation
• endoscopic evaluation for patient with caustic injury

DRUG TREATMENT
Prophylactic antibiotics are not indicated.
Steroid therapy to reduce oedema and fibrosis, preferably within 24 hours of ingestion
• methylprednisolone 2 mg/kg/day
  OR
dexamethsone 1 mg/kg/day

For pain control
See Pain Syndromes: Section 20.2

REFERRAL
• all symptomatic cases for endoscopic evaluation

18.1.5 INHALANT INGESTION

DESCRIPTION
Inhalants include: spray paints, glues and paint thinners which may contain toluene and or n-Hexane.

DIAGNOSTIC CRITERIA
• distinctive odour
• discolouration around mouth/nose
• palpitations
• dizziness
• cardiac arrhythmias
• mucous membrane irritation, i.e. sneezing coughing and tearing
• GIT complaints, i.e. nausea, vomiting and abdominal pain
• distal renal tubular acidosis, i.e. hyperchloeraemic metabolic acidosis with a normal anion gap
• peripheral neuropathy and hepatotoxicity may be complications

NON-DRUG TREATMENT
• stabilise airway, breathing and circulation
• correct fluid and electrolyte abnormalities
DRUG TREATMENT
For agitation
• diazepam, IV/oral, 0.1–0.2 mg/kg.
  Maximum dose: 10 mg.

For cardiac dysrhythmias, e.g.: ventricular fibrillation
See Arrhythmias: Section 4.1

REFERRAL
• cardiac arrhythmia

18.1.6 ETHANOL POISONING

DESCRIPTION
Ethanol is a selective CNS depressant at low concentrations, and a generalized depressant at high concentrations.

DIAGNOSTIC CRITERIA
Clinical
• lack of coordination
• ataxia
• slurred speech
• gait disturbances
• drowsiness
• stupor
• coma
• hypoglycaemia
• convulsions

Investigations
• monitor blood glucose levels

DRUG TREATMENT
Obtunded patients
• dextrose 10%, IV, 1–2 mL/kg
  If patients respond to glucose administration, serial glucose levels should be done to detect recurrent hypoglycaemia.

18.1.7 IRON POISONING

DESCRIPTION
Iron is widely available as an over the counter product and is commonly ingested accidentally by toddlers.

DIAGNOSTIC CRITERIA
Clinical
• toxicity is related to ingested dose of elemental iron
• doses of elemental iron > 40 mg/kg in a child or 1.5 g in adolescents require hospital assessment and management
**categories of iron toxicity:**

<table>
<thead>
<tr>
<th>Low risk</th>
<th>Medium risk</th>
<th>High risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>o no history of:</td>
<td>o minimal gastrointestinal symptoms</td>
<td>o lethargic</td>
</tr>
<tr>
<td>▪ abdominal pain</td>
<td>o normal physical examination</td>
<td>o acidicotic</td>
</tr>
<tr>
<td>▪ nausea</td>
<td></td>
<td>o shocked</td>
</tr>
<tr>
<td>▪ vomiting, or diarrohea</td>
<td></td>
<td>o may have evidence of haematemesis or melaena</td>
</tr>
<tr>
<td>o asymptomatic for 6 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o &lt; 20 mg/kg of elemental iron ingested</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- low risk patients are unlikely to have ingested enough iron to lead to serious poisoning and can be discharged.
- high and medium risk patients must be admitted

**Investigations**

<table>
<thead>
<tr>
<th>Medium risk</th>
<th>High risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>o abdominal X-ray</td>
<td>o arterial blood gas</td>
</tr>
<tr>
<td>o arterial blood gas</td>
<td>o electrolytes</td>
</tr>
<tr>
<td>o electrolytes</td>
<td>o serum iron levels within 2–6 hours after ingestion</td>
</tr>
<tr>
<td>o serum iron levels within 2–6 hours after ingestion</td>
<td>▪ if no clinical features are present and serum iron &lt; 500 mcg/dL – patient is low risk</td>
</tr>
</tbody>
</table>

**NON-DRUG TREATMENT**

**Medium risk**
- if more than mild gastrointestinal symptoms or altered mental state, shock, or acidosis, refer for chelation therapy

**High risk**
- manage airway
- refer for chelation therapy

**DRUG TREATMENT**

**Medium and high risk**

Fluid resuscitation
- sodium chloride 0.9%, IV, 20 mL/kg as an initial bolus followed with maintenance therapy

Whole bowel irrigation.

**Chelation therapy**
For iron ingestion > 60 mg/kg of elemental iron
- desferrioxamine, IV, 15 mg/kg/hour as a continuous infusion until urine is no longer pink
  Beware of hypotension.
18.1.8 NEUROLEPTIC POISONING

DESCRIPTION
Acute dystonic reactions / extrapyramidal symptoms are distressing adverse reactions (sustained muscle spasms) occurring after overdose or during chronic therapy with neuroleptics. A typical dystonic reaction includes overextension or overflexion of the limbs with abnormal posturing of the trunk. Other extrapyramidal symptoms may occur.

Neuroleptic malignant syndrome is an idiosyncratic life threatening reaction presenting with:
- temperature dysregulation
- autonomic instability
- altered mental state
- diaphoresis
- musculoskeletal effects (pipe like rigidity)

DIAGNOSTIC CRITERIA
- dystonic reactions
- other extrapyramidal symptoms

NON-DRUG TREATMENT
- observe asymptomatic patients for a minimum of 6 hours
- admit all symptomatic patients for continuous cardiac monitoring

DRUG TREATMENT
- activated charcoal

For acute dystonic reactions
- biperidine, IV, slow injection
  - < 1 year 1 mg
  - 1–6 years 2 mg
  - 6–10 years 3 mg
  - If concomitant significant anticholinergic findings are present, such as fever and dry skin and mucous membranes, a benzodiazepine is preferred.

REFERRAL
- patients with neuroleptic malignant syndrome
- patient with conduction abnormalities (prolonged QT)

18.1.9 ORGANOPHOSPHATE POISONING

DESCRIPTION
Organophosphates are potent inhibitors of pseudocholinesterase. Poisoning due to organophosphates is notifiable.
DIAGNOSTIC CRITERIA
Clinical
- cholinergic toxidrome
- cholinergic symptoms include:
  - muscarinic symptoms:
    - diarrhoea
    - urination
    - miosis
    - secretions
  - central nicotinic effects
    - confusion
    - coma
    - convulsions
- cardiac effects include bradycardia or tachycardia depending on whether muscarinic or nicotinic effects predominate
- signs depend on dose and route of exposure (vapour of liquid) as well as the time exposed (vapour)

Investigations
- decreased levels of pseudocholinesterase in plasma and red cells
  Use for confirmation only.
  Do not wait for levels before treating.

NON-DRUG TREATMENT
- ventilate, if necessary
- wash affected skin with soap and water
- remove all clothing and wash clothes thoroughly
- suction secretions frequently
- monitor respiratory function closely, as well as heart rate, pupillary size and level of consciousness

DRUG TREATMENT
For bronchorrhoea or bronchospasm
- atropine, IV, 0.02–0.05 mg/kg.
  Repeat every 10–15 minutes until bronchial secretions are controlled.
  Titrate dose against the secretions.
  The therapeutic endpoint is clearing of secretions and resolution of bronchospasm

Note:
Atropine may need to be continued for prolonged periods.
Many repeated doses of atropine may be required and large quantities may be needed. Beware of relapses.
Tachycardia and mydriasis are not contraindications for atropine

Treat convulsions: See Section 13.4.

REFERRAL
- where ICU facilities are not available
18.1.10 OPIOID POISONING

DESCRIPTION
Codeine is a common drug of abuse.

The duration of action of morphine lasts 3–6 hours. Other oral agents, e.g. codeine and long acting morphine, demonstrate a delayed effect of up to 4–12 hours.

DIAGNOSTIC CRITERIA
• altered level of consciousness
• classic triad of CNS depression, respiratory depression and pupillary constriction
• hypotension, hypothermia, bradycardia and hyporeflexia
• vomiting is common and exposes the patient to the risk of aspiration especially with depressed consciousness
• early symptoms: awake and alert presenting within 1–2 hours of ingestion
• late symptoms: classic triad of coma, respiratory depression and miosis

NON-DRUG TREATMENT
• supportive care, ventilate with bag-mask device.
• monitor oxygen saturation constantly
• observe for urinary retention

DRUG TREATMENT
• activated charcoal: See Section 18.1

• naloxone, IV, 0.1 mg/kg – 2 mg
  If no response after 5 minutes, repeat dose and titrate according to response.
  Duration of action of naloxone is 20–30 minutes.
  If repeated doses are naloxone are necessary, a continuous IV infusion of naloxone can be instituted.

CAUTION
All patients treated with naloxone should be observed for at least 12 hours for relapse, especially if a long acting opioid has been ingested.

REFERRAL
• patients requiring multiple doses of naloxone

18.1.11 PARACETAMOL POISONING

DESCRIPTION
Paracetamol poisoning in childhood is almost always intentional. The accidental ingestion of paediatric paracetamol elixir preparations by the toddler very rarely achieves toxicity. Adolescents are often not aware that paracetamol ingestion can be lethal and may unknowingly take a lethal dose as a suicidal gesture.
### DIAGNOSTIC CRITERIA

- Dose in excess of 150 mg/kg in healthy children is potentially toxic.
- Serum paracetamol concentration must be measured at least four hours following ingestion.
- Use nomogram to assess risk of toxicity.

### SEMILOGARITHMIC PLOT OF PLASMA PARACETAMOL LEVELS VERSUS TIME

![Graph showing semi-logarithmic plot of plasma paracetamol levels versus time.](image)

- Probable hepatic toxicity
- No hepatic toxicity
- Possible hepatic toxicity
- 25%

Modified and reproduced from Rumack BH, Matthew H: Acetaminophen poisoning and toxicity. Pediatrics 1975; 55:871

- Cautions for use of this chart:
  - The time co-ordinates refer to time of ingestion.
  - Serum levels drawn before 4 hours may not represent peak levels.
  - The graph should be used only in relation to a single acute ingestion.
  - The lower solid line 25% below the standard nomogram is included to allow for possible errors in paracetamol plasma assays and estimated time from ingestion of an overdose.

- If patients present > 8 hours post-ingestion, start on treatment without waiting for the paracetamol levels.

- If the time of ingestion is unknown, start treatment for any detectable level of paracetamol or any elevation of AST or ALT.

- Patients with normal LFT’s and undetectable paracetamol levels four hours after ingestion do not require treatment.

- Baseline urine and electrolytes
- Liver enzymes
- Coagulation profile
- Normal results at 48 hours excludes hepatic damage.
NON-DRUG TREATMENT
• gastric lavage if patient presents within one hour of ingestion

DRUG TREATMENT
Only if patients presents within 1 hour of ingestion
• activated charcoal
• acetylcysteine, IV,
  First 24 hours
  150 mg/kg in dextrose 5% 5 mL/kg given over 15 minutes loading dose
  50 mg/kg in dextrose 5% 5 mL/kg over the next 4 hours, then
  100 mg/kg in dextrose 5% 10 mL/kg over 16 hours
  Second 24 hours
  100 mg/kg in dextrose 5% 10 mL/kg over 24 hours

REFERRAL
• patients with severe hepatocellular damage

18.1.12 PETROCHEMICAL POISONING

DESCRIPTION
Accidental ingestion of paraffin, particularly by toddlers, is common in South Africa.

DIAGNOSTIC CRITERIA
Clinical
• paraffin is volatile and inhalation of the fumes can cause serious chemical pneumonitis
• respiratory distress
• CNS symptoms
Investigations
• chest X-ray

NON-DRUG TREATMENT

CAUTION
Do not attempt gastric lavage.

• observe patient for up to 12–24 hours if asymptomatic
• administer oxygen, if necessary
• education and counseling regarding prevention

DRUG TREATMENT
If infection develops after 48 hours after ingestion
• amoxicillin, oral, 30 mg/kg/dose 8 hourly for 5 days

REFERRAL
• for ventilatory support
18.1.13 SALICYLATE POISONING

DESCRIPTION
Salicylate poisoning may result from oral and/or topical exposure. Salicylate products vary widely in concentration e.g. oil of wintergreen is 100% methylsalicylate. As little as 4 mL of oil of wintergreen may be fatal in a child.

DIAGNOSTIC CRITERIA

Clinical
- doses less than 150 mg/kg will not cause toxicity except in a child with hepatic or renal disease
- ingestion of 150–300 mg/kg may result in mild to moderate toxicity
- ingestion of > 500 mg/kg should be considered a potentially lethal dose
- features include:
  - fever
  - epigastric pain
  - CNS depression
  - hyperventilation
  - renal failure
  - respiratory alkalosis (initially) followed by metabolic acidosis
- monitor blood gases, urine output and urine and electrolytes
- monitor salicylate level: toxic > 30 mg/dL
  - Serial monitoring until declining levels are documented.
- monitor and treat hypoglycaemia

NON-DRUG TREATMENT
- gastric lavage.
- correct hydration

DRUG TREATMENT

After gastric lavage
- activated charcoal

Urine alkalinisation
If metabolic disturbances present
- sodium bicarbonate, IV, 1–2 mmol/kg/day in maintenance fluid
  - Increase if necessary to maintain urine pH above 7.5.

For hydration
- Darrows half strength in dextrose 5%, IV
- vitamin K₁ (phytomenadione), IM, 5 mg as a single dose

Antacid
- magnesium trisilicate, oral, 5–10 mL as required
18.1.14 SEDATIVE-HYPNOTIC POISONING

DESCRIPTION
Young children or toddlers are typically involved in accidental exposure and ingest small amounts of sedatives. Adolescents may ingest large amounts during suicide, suicidal gesture or for recreational use. Examples of sedative-hypnotics include: benzodiazepines and diphenhydramine.

DIAGNOSTIC CRITERIA
Clinical
• cardiorespiratory depression
• decreased level of consciousness
Investigations
• serum drug levels: of no value in the acute treatment phase
• urine test: may have medico-legal implications

NON-DRUG TREATMENT
• if there is respiratory depression, intubate, ventilate and transfer
• gastric lavage
• supportive treatment only is necessary in most patients

DRUG TREATMENT
If significant overdose is suspected
• activated charcoal

REFERRAL
• respiratory depression

18.1.15 SULFONYLUREA

DESCRIPTION
First generation sulfonylureas include chlorpropamide, which is excreted renally. Second generation agents include glimepiride and glipizide and are excreted in the faeces.

DIAGNOSTIC CRITERIA
Clinical
• coma and seizures
• profound hypoglycaemia, usually within 4 hours of ingestion
Investigations
• glucose monitoring is the mainstay of diagnostic testing

NON-DRUG TREATMENT
• observe for 24 hours even if a single tablet is ingested
• glucose containing fluid orally
DRUG TREATMENT
- activated charcoal
- dextrose, IV. Titrate until blood glucose is controlled.

Note:
Glucagon and corticosteroids are contraindicated.

REFERRAL
- patients not responding to intravenous glucose

18.1.16 SYMPATHOMIMETIC AGENT POISONING

DESCRIPTION
Pseudoephedrine in decongestants, methylphenidate and illicit drugs such as cocaine and amphetamines (Tik) are sympathomimetic agents. These agents are frequently abused, especially as recreational drugs.

DIAGNOSTIC CRITERIA
Clinical
- hypertension
- tachycardia
- tachypnoea
- agitation
- hyperthermia: effects of sympathomimetics that predispose to hyperthermia include:
  - peripheral vasoconstriction and impaired cutaneous heat loss
  - agitation
  - seizures
  - increased muscle activity
  - impaired behavioral responses
- with cocaine toxicity, cardiovascular manifestations predominate including:
  - supraventricular and ventricular dysrhythmias
  - myocardial ischaemia
- neonates of mothers addicted to cocaine may present with withdrawal signs manifested by jitteriness

Investigations
- ECG monitoring to evaluate dysrhythmias

NON-DRUG TREATMENT
- admit all seriously ill children to ICU
- maintain hydration
- cooling for hyperthermia
- mildly toxic patients require no specific treatment

DRUG TREATMENT
- activated charcoal – See Section 18.1
For agitation and tachycardia
• diazepam, IV/oral, 0.1–0.2 mg/kg
  Maximum dose of 10 mg.

For severe hypertension
See Acute Severe Hypertension: Section 4.9.1

For seizures
See Status Epilepticus: Section 13.4

REFERRAL
• status epilepticus requiring ICU
• hypertensive crisis

18.2 ENVENOMATION

18.2.1 SCORPION BITES

DESCRIPTION
Some scorpion species can cause serious systemic toxicity.

DIAGNOSTIC CRITERIA
• pain and paraesthesia occur immediately after envenomation
  Autonomic and motor findings may differentiate scorpion bites from other causes of pain.
• the pain can be exquisitely accentuated by tapping on the affected region, i.e. “tap test”
• in severe cases cranial nerve dysfunction, blurred vision, pharyngeal muscle incoordination, drooling and respiratory compromise can occur
• excessive motor activity may present as restlessness, or uncontrollable jerking of extremities.
• other serious effects include cardiac dysfunction, pulmonary oedema, pancreatitis, bleeding disorders and skin necrosis
• nausea, vomiting, tachycardia and severe agitation can also occur

NON-DRUG TREATMENT
• general supportive care
• monitor airway, breathing and circulation

DRUG TREATMENT
For pain
• paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

Antivenom therapy
Routine antivenom therapy is recommended only in severe cases with systemic signs
• scorpion antivenom, slow IV, 10 mL administered over 3–5 minutes
  OR
  scorpion antivenom, IV infusion, 10 mL diluted in sodium chloride 0.9% or dextrose 5%, 50–100 mL administered over 5–10 minutes
REFERRAL
• severe cases requiring intensive care

18.2.2 SNAKEBITE
T63.0

DESCRIPTION
The effects of snakebites may be cytotoxic, neurotoxic and/or haemotoxic. The overall effect is determined by the predominant toxin in the snake venom.
In the majority of cases, the species of snake is unknown. The patients can divided into:
• no evidence of bite, no envenomation
• evidence of bite, minor envenomation, i.e. fang marks, minimal pain, minimal swelling and no systemic signs
• evidence of serious envenomation

DIAGNOSTIC CRITERIA
• cytotoxic venom: puff adder, spitting cobra
  ○ venom causes severe local damage to tissues and vascular endothelium
  ○ severe swelling and local necrosis occurs
• neurotoxic venom: mamba, non-spitting cobra, rinkhals, berg adder
  ○ venom causes a paresis and paralysis of skeletal muscles
  ○ paralysis of respiratory muscles with respiratory failure may occur
  ○ preceded by severe pain and paraesthesias
  ○ ophthalmoplegia occurs when ocular muscles become paralysed
  ○ speech and swallowing may be affected
  ○ signs and symptoms start within 15–30 minutes
• haemotoxic venom: boomslang, vine snake
  Venom may cause:
  ○ haemolysis of red blood cells
  ○ anaemia
  ○ consumptive coagulopathy
  ○ bruises
  ○ ecchymosis
  ○ epistaxis
  ○ haemoptysis
  ○ haematuria may occur

NON-DRUG TREATMENT
• patients with no evidence of bite and patients with evidence of bite but only minor envenomation should be admitted for observation. No anti-venom is indicated.
• sucking/cutting the wound has not been found to be of any benefit
• do not apply tourniquet
• where serious envenomation is suspected, immediate treatment includes:
  ○ minimise movement of affected limb
  ○ emergency treatment by bandaging affected limb with crepe bandage without compromising blood supply
  ○ rapid transportation to a facility with available antivenom is the most important principle of pre-hospital care
  ○ optimal therapy consists of placing the patient at rest with the affected body part raised to the level of the heart
  ○ stabilise circulation and blood pressure
• for cytotoxic envenomation, surgical intervention, i.e. decompression surgery for established compartment syndrome and debridement of necrotic tissue should only be done when absolutely necessary and as conservatively as possible
• fasciotomy may be necessary if compartment syndrome is suspected
• for neurotoxic envenomation, ventilatory and cardiovascular support may be needed in an Intensive Care Unit

**DRUG TREATMENT**

**All patients**

• tetanus toxoid vaccine (TT), IM, 0.5 mL

If child with penetrating wound not completely immunised

• tetanus immunoglobulin, IM
  
<table>
<thead>
<tr>
<th>Age Group</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5 years</td>
<td>75 IU</td>
</tr>
<tr>
<td>5–10 years</td>
<td>125 IU</td>
</tr>
<tr>
<td>&gt; 10 years</td>
<td>250 IU</td>
</tr>
</tbody>
</table>

Cleanse wound

• chlorhexidine 0.05% solution in water

**Antivenom therapy**

Indications:

• any evidence of envenomation, i.e. severe or burning pain and/or local swelling, bleeding from puncture marks
• bite in close proximity to airway structures
• platelet count less than $100 \times 10^9$ L
• fibrinogen less than 100 mg/dL
• presence of neurotoxic symptoms

The dose of antivenom is the same for adult and children.

**CAUTION**

Never administer antivenom without being fully prepared to manage acute anaphylaxis.

For cobras, mambas, rinkhals, puff adders and Gaboon viper

• polyvalent snake antivenom, IV
  
  Dilute 10 mL in sodium chloride 0.9% 50 mL.
  
  Administer slowly over 15 minutes.
  
  If no reaction occurs, 60–120 mL antivenom diluted in sodium chloride 0.9%, 200 mL administered slowly over 30 minutes.

For boomslang bites

• boomslang antivenom, slow IV, 10 mL administered over 3–5 minutes
  
  OR
  
  boomslang antivenom, IV infusion, 10–20 mL diluted in sodium chloride 0.9% or dextrose 5%, 50–100 mL administered over 5–10 minutes
  
  After administration, observe patient.

Correct anaemia and bleeding tendency.
REFERRAL
• snakebite with neurotoxic or haemotoxic manifestations may need intensive care

18.2.3 SPIDER BITES (WIDOW SPIDERS)

DESCRIPTION
The vast majority of spiders are not harmful to humans. Widow spiders (*Lactrodectus*) are found in dark confined areas and the female can produce a potent venom that acts through a calcium mediated mechanism leading to the release of acetylcholine and noradrenaline from nerve terminals.

DIAGNOSTIC CRITERIA
• bites are felt immediately as pinprick sensation, followed by increasing local pain that may spread to include the entire extremity
• typical target lesions, i.e. erythematous ring surrounding a pale center
• cramp like spasms in large muscle groups, abdominal pain or rigidity, progressing to generalised pain involving the trunk and abdomen have been described

NON-DRUG TREATMENT
• supportive care of airway, breathing and circulation

DRUG TREATMENT
To control pain and muscle spasm
• morphine, oral
  Short acting: for children over 6 months
  Starting dose: 0.2–0.5 mg/kg/dose 4 – 6 hourly

AND
• diazepam, oral, 0.1–0.2 mg/kg/dose once daily
  Maximum dose: 10 mg

For severe envenomation to resolve symptoms and shorten duration of illness
• spider antivenom, IV infusion, 5–10 mL diluted in sodium chloride 0.9% or dextrose 5%, 50–100 mL administered over 5–10 minutes

18.2.3.1 Spider Bites: Necrotic Arachnidism

DESCRIPTION
*Loxosceles* spiders can produce local necrotic skin lesions that are mediated by enzymes.

DIAGNOSTIC CRITERIA
• bites are initially painless
• skin lesions can vary from mildly erythematous lesions to severe local reaction, i.e. blistering, bluish discolouration progressing to frank necrosis.
• systemic effects include nausea, vomiting, fever, chills, arthralgia, haemolysis, thrombocytopenia, haemoglobinuria and renal failure
NON-DRUG TREATMENT

- supportive care
- surgical debridement once the clear margins around the necrotic lesions are established

DRUG TREATMENT

For pain
- paracetamol, oral, 10–15 mg/kg/dose, 6 hourly as required

Antibiotic therapy for septic lesions.
CHAPTER 19
PREMATURITY AND NEONATAL CONDITIONS

19.1 APNOEA, NEONATAL
P28.4

DESCRIPTION
A neonate presenting with episodes of cessation of breathing.

DIAGNOSTIC CRITERIA
• cessation of respiration for longer than 20 seconds, with or without cyanosis, pallor or bradycardia
• cessation of respiration for less than 20 seconds with cyanosis, pallor and/or bradycardia

Causes
• apnoea episodes in a previously asymptomatic well neonate may be the first indication of a serious underlying disease
• apnoea episodes in an already unwell neonate indicate deterioration in the condition of the neonate

Central apnoea
Causes include:
• prematurity
• hypoxia
• sepsis
• acidosis
• meningitis
• temperature disturbances
• rough handling
• intraventricular haemorrhage
• patent ductus arteriosus
• hypoglycaemia
• hypermagnesaemia
• sedatives
• atypical convulsions

Obstructive apnoea
Neonates are obligatory nose breathers and if their nares are obstructed, they are prone to apnoea.
Causes include:
• choanal atresia
• micrognathia
• secretions (milk, meconium, blood, mucus) lodged in the upper airway
• gastro-oesophageal reflux
• macro glossia

Reflex apnoea or vagally mediated apnoea
Is due to:
• endotracheal intubation
• gastro-oesophageal reflux
• suction of the pharynx or stomach
• passage of a nasogastric tube
• overfeeding

Mixed apnoea
Apnoea caused by a combination of the above causes.
NON-DRUG TREATMENT
For all forms of neonatal apnoea
• identify and treat the underlying cause
• frequent gentle physical stimulation e.g. rubbing of soles of feet
• prematurity – nurse in the prone position
• ambient temperature at lower range of neutral thermal environment
• axillary temperature or anterior abdominal wall temperature at 36.2–36.8°C
• oxygen via headbox, nasal cannula or mask to maintain oxygen/haemoglobin saturation of 90–92% or an oxygen tension in the blood at 60–80 mmHg
• maintain haematocrit at 40%
• nasal CPAP (continuous positive airway pressure) of 3–5 cm water
(Nasal CPAP - not for central apnoea except for apnoea of prematurity.)
• monitor:
  o heart rate
  o respiratory rate
  o haematocrit
  o acid base status
  o oxygen/haemoglobin saturation

DRUG TREATMENT
Only for apnoea of prematurity
• caffeine base, oral
  Loading dose: 10 mg/kg, followed by
  Maintenance dose: 2.5–4 mg/kg/24 hours.
  Maintain blood levels of 10–20 mcg/mL.
  (Caffeine citrate 20 mg = caffeine base 10 mg)
  OR
  aminophyline, IV/oral
  Loading dose: 5–6 mg/kg, followed by
  Maintenance dose: 1–2 mg/kg, 8 hourly.
  Maintain blood levels at 10–12 mcg/mL.
If neonate responds favourably to caffeine/aminophyline continue until neonate is apnoea free for 7 days or until the baby weighs 1.8 kg.

REFERRAL
• underlying cause of apnoea unclear and/or neonate requiring ventilatory support
• recurrent life-threatening episodes of apnoea, not responding to adequate treatment.

19.2 CYANOTIC HEART DISEASE IN THE NEWBORN
Q24.9

DESCRIPTION
Blue or grey discoloration of skin and tongue in the presence of a cardiac lesion, in room air, with an oxygen saturation of less than 85%.
Note:
Strongly suspect cyanotic cardiac disease if centrally cyanosed, not in respiratory distress and normotensive.
DIAGNOSTIC CRITERIA

- Rule out non-cardiac causes of central cyanosis:
  - respiratory conditions, e.g. hyaline membrane disease, pneumonia and pneumothorax. Signs of respiratory distress usually improve with oxygen administration. Chest X-ray may be helpful.
  - central nervous system involvement, e.g. sedation and asphyxia, which usually improves with oxygen administration
  - PaCO<sub>2</sub> may be increased in respiratory and central nervous system causes of cyanosis
  - methaemoglobinaemia

- Confirm cardiac cause:
  - little or no improvement with oxygen administration – see hyperoxia test
  - tachypnoea, but usually no retraction
  - heart murmur (may be absent)
  - chest X-ray may show cardiomegaly or abnormal cardiac silhouette and/or reduced pulmonary blood flow
  - echocardiography will confirm the diagnosis

- Hyperoxia Test
  - give 100% oxygen via a head box for 10 minutes
    - Unnecessary if saturation under 85% in 100% head box.
  - obtain arterial blood from the right radial artery (preductal flow)

<table>
<thead>
<tr>
<th>PaO&lt;sub&gt;2&lt;/sub&gt; mmHg</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 100</td>
<td>Most likely to be a cyanotic heart lesion, persistent fetal circulation or severe lung disease. PaCO&lt;sub&gt;2&lt;/sub&gt; will be increased with severe lung disease.</td>
</tr>
<tr>
<td>≥ 100–200</td>
<td>Unlikely to be cyanotic heart lesion.</td>
</tr>
<tr>
<td>≥ 200</td>
<td>Excludes cyanotic heart lesion.</td>
</tr>
</tbody>
</table>

NON-DRUG TREATMENT

- neutral thermal environment
- monitor and maintain within physiological range for age:
  - heart rate
  - minerals
  - respiration
  - blood glucose
  - blood pressure
  - blood gases
  - body temperature
  - acid-base status
  - electrolytes
- provide adequate hydration and nutrition

DRUG TREATMENT

To keep ductus arteriosus open if a cyanotic heart lesion is suspected, prostaglandin therapy, i.e.:

- alprostadil, IV, 0.05–0.1 mcg/kg/minute, initial dose
  - Maintenance dose 0.01–0.1 mcg/kg/minute.

OR
- dinoprostone, via naso/orogastric tube, 0,125mg every 30 minutes
  - 1/4 tablet suspended in 1 mL sterile water
Continue with prostaglandin therapy until corrective or palliative surgery can be done or until patency of the duct is not deemed essential for survival of the infant. Babies on prostaglandin therapy: inspiratory oxygen not more than 40%. An oxygen saturation of haemoglobin > 75% is acceptable.

Side effects of prostaglandin therapy:
- apnoea
- jitteriness
- fever
- diarrhoea

if pH ≤ 7.2, correct metabolic acidosis
- sodium bicarbonate 4.2%, IV
  \[
  \text{HCO}_3^- \text{ needed (mmol) = base excess \times 0.3 \times body mass (kg)}
  \]
  \[
  2 \text{ mL sodium bicarbonate 4.2% = 1 mmol HCO}_3^-
  \]

SURGICAL TREATMENT
- corrective or palliative surgery

REFERRAL
- all cyanotic infants with an underlying cardiac cause for central cyanosis

19.3 ENTEROCOLITIS, NECROTISING

DESCRIPTION
Neonate presenting with the consequences of bowel wall injury or necrosis. Risk factors include:
- prematurity
- sepsis
- early formula feedings
- patent ductus arteriosus
- perinatal asphyxia (hypoxia)
- hypotension/shock
- lack of early maternal contact
- high feeding volumes
- polycythaemia

DIAGNOSTIC CRITERIA
- early signs are often non-specific
  - feeding intolerance
  - gastric aspirates
  - vomiting
  - body temperature instability
  - apnoea and lethargy
- non-specific signs may progress to more specific signs including:
  - abdominal distention with ileus
  - bloody stools
  - peritonitis
  - red-purple discolouration of the abdominal wall with abdominal wall cellulitis and bowel perforation.
• X-ray of abdomen may show:
  ○ distended loops of intestines
  ○ bowel-wall thickening
  ○ pneumatosis intestinalis
  ○ hepatic portal venous gas and free intra peritoneal air due to perforation

NON-DRUG TREATMENT
• admit to neonatal high-care unit
• nurse in neutral thermal environment
• insert oro/nasogastric tube and apply free drainage
• IV fluids, neonatal maintenance solution, volume according to age, weight and hydration status. Add volume of gastric aspirates to daily maintenance fluid volume.
• suspected cases should be nil per mouth for 72 hours
• confirmed cases should be nil per mouth for at least 7 days
• provide adequate IV nutrition (hyperalimentation) as soon as diagnosis is confirmed
• if coagulopathy or septic shock is present, plasma, lyophilised, IV, 20 mL/kg over 2 hours
• if haematocrit < 40%, packed red cells, IV, 10 mL/kg
• provide cardiovascular and ventilatory support, if necessary
• send blood samples for culture and sensitivity testing before starting antibiotic therapy

DRUG TREATMENT
• dopamine, IV, 5–15 mcg/kg/minute until blood pressure is stabilised

Antibiotics, empirical
Reassess choice of antibiotics when the culture and sensitivity results become available.
First line:
• ampicillin, IV, 50 mg/kg for 10 days
  < 7 days 50 mg/kg 12 hourly
  7 days – 3 weeks 50 mg/kg 8 hourly
  > 3 weeks 50 mg/kg 6 hourly
• amikacin, IV, 15 mg/kg once daily for 10 days
  OR
gentamicin, IV, 5 mg/kg once daily for 10 days
PLUS
• metronidazole, IV, 7.5 mg/kg/dose, 8 hourly for 7 days
Second line (if already on antibiotics):
PLUS
Third generation cephalosporin, e.g.
• cefotaxime, IV, 50 mg/kg for 10 days
  < 7 days 50 mg/kg 12 hourly
  7 days – 3 weeks 50 mg/kg 8 hourly
  > 3 weeks 50 mg/kg 6 hourly
PLUS
• amikacin, IV, 15 mg/kg once daily for 10 days
  OR
gentamicin, IV, 5 mg/kg once daily for 10 days
PLUS
• metronidazole, IV, 7.5 mg/kg/dose, 8 hourly for 7 days
SURGICAL TREATMENT
Surgical intervention is required when there is progressive deterioration of the clinical condition despite maximal medical support and/or bowel necrosis with or without bowel perforation.

REFERRAL
- all confirmed cases for specialist care
- deterioration of clinical condition, despite adequate treatment
- signs and symptoms of intestinal perforation and peritonitis requiring surgical intervention
- recurrent apnoea episodes and/or signs of respiratory failure, requiring respiratory support

19.4 HAEMORRHAGIC DISEASE OF THE NEWBORN
P53

DESCRIPTION
This is due to a deficiency of vitamin K dependent clotting factors II, VII, IX and X. All newborns who did not receive vitamin K, at birth, especially premature babies and breastfed babies, are at risk.
Spontaneous bleeding from any site usually gastro-intestinal producing haematemesis or melaena. Bleeding from umbilical stump, epistaxis and a cephalohaematoma/subgaleal haemorrhage are also relatively common.
Complications may include anaemia, hypovolaemic shock and intracranial haemorrhage with neurological damage.

There are three forms of the disorder:
**Early form:** presents within 24 hours of birth in newborns of mothers on treatment with anticonvulsants, e.g. phenytoin and phenobarbital, or oral anticoagulants.
**Classical form:** presents during the first week of life usually on the second to seventh day
**Late form:** presents during the first to fourth month of life usually with intracranial haemorrhage in exclusively breastfed babies who did not receive vitamin K prophylaxis at birth

DIAGNOSTIC CRITERIA
Special investigations
- prolonged prothrombin time (PT)
- partial prothrombin time (PTT), and
- international normalized ratio (INR) with a normal platelet count
- normal fibrinogen levels and
- normal thrombin time

**Note:**
Exclude other causes of bleeding in the neonate.
NON-DRUG TREATMENT
• neutral thermal environment
• fresh frozen plasma/reconstituted lyophilized plasma powder, IV, 20 mL/kg over one hour
• If anaemic (haematocrit < 40% or Hb < 13 g/dL):
  packed red cells 10 mL/kg IV over 1 hour. May be repeated if necessary.
• oxygen, if needed
• monitor:
  o blood pressure
  o heart rate
  o respiratory rate
  o body temperature
  o coagulation parameters
• provide adequate nutrition

DRUG TREATMENT
• vitamin K₁, IM, 1 mg as a single dose

Prophylaxis
• vitamin K₁, IM, single dose at birth
  Full term newborns: 1 mg
  Preterm newborns: 0.5 mg
Prophylaxis with oral vitamin K formulation is not recommended.

REFERRAL
• deterioration of clinical condition despite adequate treatment
• suspected intracranial haemorrhage

19.5 HEART FAILURE IN NEONATES
P29.0

DESCRIPTION
Clinical syndrome reflecting the inability of the myocardium to meet the oxygen and nutritional/metabolic requirements of the body. Heart failure may be acute or chronic.

The main causes of heart failure are:
• congenital heart abnormalities
  o left-sided outflow obstruction, e.g. interrupted aortic arch, coarctation of the aorta, aortic valve stenosis
  o left to right shunts, VSD and PDA
  o hypoplastic left heart
  o complex congenital heart lesions
• acquired conditions
  o fluid overload
  o hypoglycaemia
  o acidosis
  o dysrhythmias
  o myocarditis
  o pneumopericardium
  o hypertension
  o sepsis
  o hypoxia
  o severe anaemia
  o cardiomyopathy
  o cardiac tamponade
  o hyperthyroidism
DIAGNOSTIC CRITERIA
Diagnosis relies on history, physical examination and a chest X-ray.

Clinical
- acute cardiac failure may present with shock, i.e. cardiogenic shock
- cardiac failure is usually associated with fluid retention and congestion
- history of recent onset of:
  - poor feeding
  - tachypnoea, > 60/minute
  - sweating
  - poor or excessive weight gain in excess of 30 g/24hours
- physical findings
  - tachycardia (>180/minute)
  - gallop rhythm (with or without a cardiac murmur)
  - cardiomegaly
  - cold wet skin
  - weak pulses
  - hypotension
  - reduced urinary output
  - pulmonary venous congestion and fluid retention
    - tachypnoea
    - central cyanosis
    - coarse crepitations
    - recession
  - systemic venous congestion
    - hepatomegaly
    - abnormal weight gain
    - periorbital oedema
- always check the femoral pulses

Special Investigations
- radiology: cardiomegaly is almost always present, cardiothoracic ratio > 60%
- electrocardiogram may show evidence of hypertrophy of one or more heart chambers and/or dysrhythmias

NON-DRUG TREATMENT
- oxygen via face mask, nasal cannula or head box to prevent hypoxia
  - treat shock first, if present
  - treat the underlying condition, e.g. sepsis and cardiac tamponade
- restrict fluids but ensure adequate nutrition
  - administer 75% of daily requirements
  - use breast milk or low-salt milk formulae
  - tube feeding may be necessary
- maintain a thermoneutral environment

DRUG TREATMENT
Combination medicine therapy is usually indicated,
e.g. digoxin AND a diuretic, WITH or WITHOUT an ACE inhibitor
Digoxin
Monitor digoxin blood levels and ECG.
Digoxin is contraindicated in bradycardia, heart block, cardiac tamponade or hypertrophic cardiomyopathy.
- digoxin, IV, 75–80% of oral dose
  OR
digoxin, oral, 0.01 mg/kg/dose 8 hourly for 3 doses, and then
  Maintenance: oral, 0.005 mg/kg/dose 12 hourly for as long as needed to control the cardiac failure.

Diuretics
Continue diuretic therapy as long as needed to control heart failure.
Monitor blood potassium levels.
Potassium supplements may be necessary if furosemide is used without spironolactone.
Hypokalaemia and hypochloraemic alkalosis may increase digitalis toxicity.
- furosemide, IV/oral, 1–3 mg/kg/24 hours as a single daily dose, or in 4 divided oral doses
  WITH or WITHOUT
  - spironolactone, oral, 1–2 mg/kg/dose, 12 hourly

Inotropic support
May help to stabilise patients with severe myocardial dysfunction, hypotension or low cardiac output.
May be lifesaving in severe myocarditis or cardiogenic shock.
- dobutamine, IV infusion, 2.5–15 mcg/kg/minute
  Continue until myocardial function and blood pressure improve.

Afterload reduction: ACE inhibitor or vasodilator
Monitor blood potassium levels and stop potassium supplements while patient is on an ACE inhibitor.
ACE inhibitors are contraindicated in bilateral renal artery stenosis or a single functioning kidney.
Consider in persistent heart failure where left sided outflow obstruction has been excluded, other measures have failed and only after consultation with a paediatrician or paediatric cardiologist.
- captopril, oral, 0.01–0.05 mg/kg/dose, 8–12 hourly, initially
  Continue as long as needed to control the cardiac failure.

Acute left-heart failure: acute pulmonary oedema/ pulmonary venous congestion
- administer 100% oxygen via face mask or nasal cannula
- furosemide, IV, 1–3 mg/kg, immediately

For patients not responding to furosemide
- morphine, IV, 0.1 mg/kg
For patients not already on digoxin treatment
- digoxin, IV, as above
- inotropic support, as above
- afterload reduction, as above
- intubation with intermittent positive ventilation – to raise the alveolar pressure above pulmonary capillary pressure

SURGICAL TREATMENT
Palliative or corrective surgery for certain congenital heart lesions.

REFERRAL
- deterioration despite adequate treatment
- for determination of the underlying cause, initiation of treatment and stabilisation

19.6 HYPOCALCAEMIA, NEONATAL
P71.1

DESCRIPTION
Acute symptomatic hypocalcaemia which presents with seizures or prolonged QT interval on ECG, may be due to:
- maternal factors:
  - diabetes
  - toxaemia
  - severe dietary calcium deficiency
- intrapartum factors:
  - asphyxia
  - prematurity
  - maternal magnesium administration
- postnatal factors:
  - hypoxia
  - shock
  - asphyxia
  - poor intake
  - sepsis
  - exchange transfusion
  - respiratory metabolic acidosis

Neonatal hypocalcaemia usually resolves in 2 to 3 days.

Three days after birth, other causes may be:
- high phosphate diet
- Mg deficiency
- renal disease
- hypoparathyroidism
DIAGNOSTIC CRITERIA
- serum calcium < 2.2 mmol/L, or
- ionised calcium < 1.2 mmol, equivalent to <3.8 mEq/L, or
- ionized calcium < 4.0 mg/dL

DRUG TREATMENT
Symptomatic hypocalcaemia
- calcium gluconate 10%, IV/oral, 1–2 mL/kg 6–8 hourly
  1 mL of calcium gluconate 10% = 100 mg calcium gluconate
  = 9 mg elemental calcium
  = 0.45 mEq/mL

- Correct hypomagnesaemia.

Acute hypocalcaemia with seizures
- calcium gluconate 10%, IV, 1–1.5 mL/kg over 5–10 minutes
  Administer slowly at a rate of 1 mL/minute.
  Rapid infusion causes bradycardia/arrhythmias.
  Repeat in 15 minutes.
  Electrocardiographic monitoring is advised.
  Monitor the heart rate.

CAUTION
Extravasations of calcium can cause tissue necrosis.

REFERRAL
- persisting or recurrent unexplained hypocalcaemia

19.7 HYPOGLYCAEMIA, NEONATAL

DESCRIPTION
Neonate presenting with a low blood glucose.
Risk factors include:
- prematurity
- small for gestational age
- baby of diabetic mother
- sepsis
- hypothermia/ hyperthermia
- birth asphyxia
- hereditary defects in carbohydrate or aminoacid metabolism
DIAGNOSTIC CRITERIA

Clinical
Asymptomatic: Hypoglycaemia detected when screening neonates at risk.
Symptomatic:
- lethargy
- hypotonia
- apnoea
- jitteriness
- irritability
- coma

Investigations
- whole blood glucose (heel prick) < 2.6 mmol/L

The blood glucose of all neonates who are at risk of hypoglycaemia should be monitored regularly, at least 3 hourly, to prevent the development of hypoglycaemia.

NON-DRUG TREATMENT
- determine and treat the underlying cause
- enteral feeding, oral or via oro/nasogastric tube, after exclusion of vomiting, ileus or obstruction

DRUG TREATMENT
- dextrose 10%, IV
  Dilute dextrose 50% solution before use.
  250 mg/kg = 0.5 mL/kg of dextrose 50%

Add more dextrose if necessary, to deliver dextrose at 6–12 mg/kg/minute or more, in order to raise heel prick blood glucose to a level of 2.6 mmol/L or more.

Dose (mg/kg/min) = (% dextrose solution x rate) ÷ (weight x 6)

OR
Glucose infusion rate (mg/kg/min) = % glucose given x total ml/kg/d x 0.007

If heel prick blood glucose is above 2.6 mmol/L after IV infusion has been started
- continue infusion at maintenance rate
If heel prick blood glucose remains below 2.6 mmol/L
- dextrose 10%, IV, 500 mg/kg as bolus (5 mL/kg of 10% dextrose).

Do not repeat.
Monitor blood glucose at least 2 hourly until blood glucose level stabilises at 2.6 mmol/L or above, before the IV dextrose infusion is gradually reduced.
Before the IV infusion is finally discontinued, give neonate all his/her milk feeds orally or via nasogastric tube.

If the neonate requires > 12 mg/kg/min of dextrose to maintain heel prick blood glucose > 2.6 mmol/L, other serious underlying metabolic or biochemical abnormality should be suspected.
For high concentrations of dextrose use a central venous line.

Prior to referral give the following if available:
- glucagon, IM, 0.1 mg/kg (one dose)
REFERRAL
• hypoglycaemia not responding to adequate treatment
• recurrent or persistent hypoglycaemia

19.8 HYPOXIA/ISCHAEMIA OF THE NEWBORN
(PERINATAL HYPOXIA/HYPOXIC-ISCHAEMIC ENCEPHALOPATHY)

DESCRIPTION
Ischaemia and decreased oxygen delivery to the fetus/baby during the prepartum, intrapartum or immediate postpartum period, with hypoxic-ischaemic damage to the central nervous system and to other body systems.

Complications
- **Cardiovascular**: heart rate and rhythm disturbances, cardiac failure and hypotension.
- **Pulmonary**: respiratory distress/respiratory failure, pulmonary hypertension and pulmonary haemorrhage.
- **Renal**: renal failure, acute tubular/cortical necrosis and urinary retention.
- **Gastrointestinal tract**: ileus and necrotizing enterocolitis.
- **Central nervous system**: increased intracranial pressure, cerebral oedema, encephalopathy, seizures, inappropriate antidiuretic hormone (ADH) secretion, hypotonia and apnoea.
- **Metabolic**: hypoglycaemia, hyperglycaemia, hypocalcaemia, hypomagnesaemia and metabolic acidosis.
- **Body temperature**: hypo/hyperthermia
- **Other**: disseminated intravascular coagulation

DIAGNOSTIC CRITERIA
• history of fetal distress and/or meconium stained amniotic fluid
• Apgar scores:
  - one-minute Apgar score ≤ 3
  - five-minute Apgar score of ≤ 6
• arterial blood lactate > 5 mmol/L
• severe mixed acidosis
  - pH < 7.2
  - base excess > -10
  - PaCO₂ > 55 mmHg
# Stages of Hypoxic-Ischaemic Encephalopathy (HIE)

<table>
<thead>
<tr>
<th>Stage</th>
<th>Stage 1 mild</th>
<th>Stage 2 moderate</th>
<th>Stage 3 severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prognosis</td>
<td>good</td>
<td>guarded ± 50% may have varying degree of neurological sequelae.</td>
<td>poor ≥ 90% mortality with major neurological sequelae in survivors</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>hyperalert, irritable</td>
<td>lethargic or obtunded</td>
<td>stuporous, comatose.</td>
</tr>
<tr>
<td>Neuromuscular control</td>
<td>uninhibited, over-reactive</td>
<td>diminished spontaneous movement</td>
<td>diminished or absent spontaneous movement</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>normal</td>
<td>mild hypotonia</td>
<td>flaccid</td>
</tr>
<tr>
<td>Posture</td>
<td>mild distal flexion</td>
<td>strong distal flexion</td>
<td>intermittent decerebration</td>
</tr>
<tr>
<td>Tendon reflexes</td>
<td>overactive</td>
<td>overactive</td>
<td>decreased or absent</td>
</tr>
<tr>
<td>Complex reflexes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suck</td>
<td>weak</td>
<td>weak or absent</td>
<td>absent</td>
</tr>
<tr>
<td>Moro</td>
<td>strong</td>
<td>weak</td>
<td>absent</td>
</tr>
<tr>
<td>Autonomic function</td>
<td>general sympathetic</td>
<td>general parasympathetic</td>
<td>both systems depressed</td>
</tr>
<tr>
<td>Pupils</td>
<td>mydriasis</td>
<td>miosis</td>
<td>mid-position, often unequal; poor light reflex</td>
</tr>
<tr>
<td>Respirations</td>
<td>spontaneous</td>
<td>spontaneous; occasional apnoea episodes</td>
<td>periodic; apnoea episodes</td>
</tr>
<tr>
<td>Heart rate</td>
<td>tachycardia</td>
<td>bradycardia</td>
<td>variable, usually bradycardia</td>
</tr>
<tr>
<td>Bronchial and salivary secretions</td>
<td>sparse</td>
<td>profuse</td>
<td>variable</td>
</tr>
<tr>
<td>Gastrointestinal motility</td>
<td>normal or decreased</td>
<td>increased</td>
<td>variable, ileus</td>
</tr>
<tr>
<td>Seizures</td>
<td>none</td>
<td>common</td>
<td>uncommon, decerebrate</td>
</tr>
</tbody>
</table>
NON-DRUG TREATMENT

• resuscitate
• admit to neonatal high care or intensive care facility if available
• ambient temperature at lower range of neutral thermal environment
• oxygen to keep $\text{PaO}_2$ between 60 and 80 mmHg and sats 88–92% (normal range)
• ventilatory support if $\text{PaO}_2 < 60$ mmHg and /or $\text{PaO}_2 > 55$ mmHg in newborns with stage 2 (moderate) asphyxia

**Note:**
Newborns with stage 3 Hypoxic Ischaemic Encephalopathy should not be ventilated.

• maintain:
  • blood glucose at 2.6–6mmol/L
  • haematocrit at ≥ 40% – packed red cells, IV, 10mL/kg
  • blood pressure at $70/35$ in a term infant and $50/35$ in a pre term infant.
  
  Mean blood pressure at least 5–10 more than the gestational age in mmHg.
• IV Fluids
  • frequent assessment of fluid balance, i.e. intake and output
  • restrict fluids to 50–60 mL/kg in the first 24–48 hours
  • use dextrose water 10% or a neonatal maintenance solution potassium-free until the possibility of renal failure has been excluded
• maintain serum electrolytes, calcium, magnesium and acid-base status within normal physiological range
• nutrition
  • no enteral feeds for at least the first 12–24 hours
  • enteral milk feeds only after ileus has been excluded
  • consider IV alimentation if enteral feeds are still not possible after 72 hours
• monitor:
  • neurological status
  • vital signs
  • acid-base status
  • blood gases
  • $\text{SaO}_2$
  • blood pressure
  • fluid balance
  • temperature
  • blood glucose
  • electrolytes
  • minerals
  • renal function

DRUG TREATMENT

• vitamin $K_1$, IM
  • pre-term infants 0.5 mg
  • full term infants 1 mg

If infection is suspected or confirmed
• cefotaxime, IV, 25–50 mg/kg/dose, 12 hourly for 7–10 days
  
  If infection is excluded, antibiotics can be stopped in 72 hours.

Hypotension

• sodium chloride 0.9% IV, 20 mL/kg over 1 hour

AND

• dopamine, IV, 5–15 mcg/kg/minute

AND/OR

• dobutamine, IV, 5–15 mcg/kg/minute until blood pressure is stabilised
Convulsions
- phenytoin, IV
  Loading dose: 15 mg/kg diluted in 3 mL sodium chloride 0.9% given over 30 minutes by slow IV infusion preferably under ECG control.
  Flush IV line with sodium chloride 0.9% before and after administration of the phenytoin.
  Maintenance: IV/oral, 5–10 mg/kg/24 hours as a single dose or 2 divided doses.

If response is unsatisfactory, consider use of other anticonvulsants, e.g. lorazepam.

**Note:**
Phenytoin must not be given in glucose/dextrose-containing solutions. To minimise risk of precipitation administer phenytoin in 0.9% sodium chloride solution.

Do not administer phenytoin intramuscularly.

Cardiac failure
Restrict fluid.
- furosemide, IV/oral/nasogastric tube, 1 mg/kg/24 hours as a single daily dose

Hypocalcaemia
Serum total calcium < 1.7 mmol/L or ionized calcium < 0.7 mmol/L
- calcium gluconate 10%, slow IV, 1–2 mL/kg over 15 minutes under ECG control

Hypomagnesaemia
Serum magnesium < 0.7 mmol/L:
- magnesium sulphate 50%, IV, 0.2 mL/kg as a single dose

Hypoglycaemia
Blood glucose < 2.6 mmol/L
- dextrose, IV as bolus, 250–500 mg/kg
  Do not repeat.
  Dilute dextrose 50% solution before use to 10% strength.
  0.5–1 mL of dextrose 50% = 250–500 mg
  OR
  2.5 mL of dextrose 10% = 250 mg

**Inappropriate ADH: Cerebral oedema/raised intracranial pressure**
Moderate fluid restriction of 50–60 mL/kg/24 hours for the first 24–48 hours.
Raise head of cot by 10–15 cm.
Moderate hyperventilation to lower PaCO₂ to 30–35 mmHg, if ventilation facilities are available.
Steroids are not considered to be of value.

**REFERRAL**
- neurological assessment of survivors at 3 months
19.9 JAUNDICE, NEONATAL

DESCRIPTION
Yellow staining of the skin and mucous membranes due to hyperbilirubinaemia. Bilirubin is formed mainly from haem catabolism, and jaundice develops when there is an over production of bilirubin, defective bilirubin metabolism and/or defective excretion of bilirubin from the body.

DIAGNOSTIC CRITERIA
Jaundice may have a physiological and a pathological component.

Physiological jaundice
- seldom appears before 24–36 hours after birth
- rarely lasts more than 10 days in the full term infant and 14 days in the pre-term infant
- only the unconjugated bilirubin fraction is increased
- total peak serum bilirubin concentration is usually below 275 micromol/L in the term infant
- total bilirubin concentration does not rise by more than 85 micromol/L/24 hours
- the baby thrives and shows no signs of illness or anaemia
- treatment is unnecessary

Pathological jaundice
- appears within the first 24 hours of birth but may also appear at any other time after birth
- persists for longer than 10 days in the full term infant or 14 days in the pre-term infant
- the unconjugated and/or conjugated fractions of bilirubin are increased
- the conjugated bilirubin level exceeds 10% of the total bilirubin value, or the conjugated bilirubin fraction is 30 micromol/L or more
- total bilirubin concentration rises by more than 85 micromol/L/24 hours
- the total serum bilirubin level is above physiological level
- there are signs and symptoms of illness in the baby
- stools are pale in conjugated hyperbilirubinaemia (obstructive jaundice)

19.9.1 HYPERBILIRUBINAEMIA, UNCONJUGATED

<table>
<thead>
<tr>
<th>Excessive haemolysis</th>
<th>Defective conjugation</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABO incompatability</td>
<td>premature</td>
</tr>
<tr>
<td>Rhesus disease</td>
<td>infection</td>
</tr>
<tr>
<td>enclosed haemorrhages</td>
<td>hypoxia</td>
</tr>
<tr>
<td>polycythaemia</td>
<td>hypoglycaemia</td>
</tr>
<tr>
<td>infections*</td>
<td>hypothyroidism*</td>
</tr>
<tr>
<td>spherocytosis</td>
<td>breast milk jaundice*</td>
</tr>
<tr>
<td>G6PD deficiency</td>
<td></td>
</tr>
</tbody>
</table>

* may cause prolonged neonatal jaundice
NON-DRUG TREATMENT

- treat the underlying cause
- monitor the infant’s body temperature
- maintain adequate nutrition and hydration
- Correct factors known to increase the risk of brain damage in babies with jaundice e.g.:
  - hypoxia
  - prematurity
  - hypoglycaemia
  - hypothermia
  - acidosis
  - hypoalbuminaemia and haemolysis
- phototherapy

GUIDELINE FOR INITIATING PHOTOTHERAPY:
(See also appendix for alternative guideline for phototherapy)

<table>
<thead>
<tr>
<th>Body mass</th>
<th>Unconjugated bilirubin (micromol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 000 g or less</td>
<td>85–100</td>
</tr>
<tr>
<td>&gt; 1 000–1 500 g</td>
<td>&gt; 100–150</td>
</tr>
<tr>
<td>&gt; 1 500–2 000 g</td>
<td>&gt; 150–200</td>
</tr>
<tr>
<td>&gt; 2 000–2 500 g</td>
<td>&gt; 200–250</td>
</tr>
<tr>
<td>&gt; 2 500–3 000 g</td>
<td>&gt; 250–275</td>
</tr>
<tr>
<td>&gt; 3 000 g with jaundice caused by haemolysis or an identifiable serious disease process, e.g. sepsis)</td>
<td>&gt; 275</td>
</tr>
<tr>
<td>&gt; 3 000 g without any identifiable cause for the jaundice</td>
<td>300</td>
</tr>
</tbody>
</table>

After exchange transfusion irrespective of body mass and unconjugated bilirubin level.

- terminate phototherapy when the unconjugated bilirubin level is lower than the recommended phototherapy initiating level, and the cause of the jaundice has been determined and adequately addressed
- the skin colour of a baby receiving phototherapy does not reflect the degree of jaundice (bilirubin blood level) or the efficacy of the phototherapy
- undress the baby and cover the eyes with gauze pad
- position the phototherapy unit (fluorescent light bulbs of 400-500nm wavelength) not higher than 45 cm above the baby
- check spectral irradiance of the fluorescent lights after every 200–300 hours of use to ensure that they are still effective (use radiometer if available).
- the spectral irradiance should be above 10 microwatt/cm²/nanometer of wavelength. If spectral irradiance cannot be checked regularly, replace fluorescent light bulbs after 1 000 hours of continuous use.
- a quartz halogen light source (400–500 nanometer wavelength) can also be used for phototherapy
- a rebound increase in bilirubin may follow termination of phototherapy. Monitor bilirubin levels ± 6 hourly after phototherapy has been stopped.
exchange transfusion is indicated when the risk of bilirubin encephalopathy and kernicterus is significant

<table>
<thead>
<tr>
<th>At birth</th>
<th>history of Rh incompatibility</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>cord unconjugated bilirubin level &gt; 85 micromol/L</td>
</tr>
<tr>
<td></td>
<td>cord haemoglobin level 10 g/dL or lower</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Within 24 hours</th>
<th>A rise in the serum unconjugated bilirubin level exceeding 20 micromol/L/hour despite phototherapy</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>After 24 hours</th>
<th>Body mass</th>
<th>Unconjugated bilirubin (micromol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 000 g or less</td>
<td>200</td>
</tr>
<tr>
<td></td>
<td>&gt;1 000–1 500 g</td>
<td>250</td>
</tr>
<tr>
<td></td>
<td>&gt;1 500–2 500 g</td>
<td>300</td>
</tr>
<tr>
<td></td>
<td>&gt;2 500–3 000 g</td>
<td>340</td>
</tr>
<tr>
<td></td>
<td>&gt; 3 000 g with jaundice caused by haemolysis or an indentifiable serious disease process, e.g. sepsis</td>
<td>340</td>
</tr>
<tr>
<td></td>
<td>&gt; 3 000 g without any indentifiable cause of jaundice</td>
<td>425</td>
</tr>
</tbody>
</table>

**DRUG TREATMENT**
As soon as the diagnosis is confirmed
- gammaglobulin, IV, 500 mg/kg over 1 hour
  For ABO incompatibility, repeat once after 6–8 hours.
Mothers of babies with Rh incompatibility as soon as possible after birth but within 72 hours of birth
- anti D immunoglobulin, IM, 100 mcg

**19.9.2 HYPERBILIRUBINAEMIA, CONJUGATED**

<table>
<thead>
<tr>
<th>Hepatocellular disease</th>
<th>Bile duct obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>• hepatitis*</td>
<td>• bile duct hypoplasia/atroresia*</td>
</tr>
<tr>
<td>• total parenteral nutrition*</td>
<td>• choledochal cyst</td>
</tr>
<tr>
<td>• syphilis</td>
<td>• cystic fibrosis</td>
</tr>
<tr>
<td>• other congenital infections</td>
<td></td>
</tr>
<tr>
<td>• galactosaemia*</td>
<td></td>
</tr>
</tbody>
</table>

* may cause prolonged neonatal jaundice
Conjugated hyperbilirubinaemia is due to intra/extrahepatic obstruction of bile ducts (cholestasis) and usually presents in the second week of life or later. The baby has a green yellow skin discolouration, dark bile stained urine and pale acholic stools. Hepatomegaly is commonly present and the infant often fails to thrive. Neonatal hepatitis, prolonged total parenteral nutrition and biliary atresia or hypoplasia accounts for the majority of cases of conjugated hyperbilirubinaemia.

**NON-DRUG TREATMENT**
- treat the underlying cause
- dietary modifications to counteract the malabsorption of fat and fat soluble vitamins (A,D,E,K) that may occur in patients with a prolonged conjugated hyperbilirubinaemia
- avoid lactose containing feeds, i.e. breast milk and lactose containing formula, when galactosaemia is suspected

**DRUG TREATMENT**
- fat soluble vitamins A, D, E and K

**SURGICAL TREATMENT**
Conditions amenable to surgery e.g. biliary artresia. Hepatoporto-enterostomy for biliary atresia should be done before 60 days of age for optimal outcome.

**19.10 JAUNDICE, NEONATAL, PROLONGED**

**DESCRIPTION**
Jaundice for more than 10 days in a term infant and 14 days in a preterm infant. (Static or rising bilirubin). The usual causes are:
- breast milk jaundice
- hypothyroidism
- hepatitis
- galactosaemia, and
- infections, e.g. UTI's

Breast milk jaundice may be confirmed by substituting breast-feeding with formula feeds for 24–48 hours. The bilirubin level will always drop to a lower level and increase again when breastfeeding is resumed. Breast milk jaundice is an unconjugated hyperbilirubinaemia and the infant is always well and thriving.

Abnormal thyroid functions, increased TSH and decreased $T_3$ and $T_4$, indicates hypothyroidism. Unconjugated bilirubin fraction is raised and the infant may have clinical signs of hypothyroidism e.g.:
- lethargy
- feeding difficulties
- poor cry
- nasal obstruction
- bradycardia
- constipation
- hypotonia
- umbilical hernia
- hypothermia
Infants with galactosaemia usually present with:
- a conjugated hyperbilirubinaemia
- vomiting
- refusal to feed
- hepatomegaly
- failure to thrive
- encephalopathy and later cataracts

Suspect galactosaemia if urine is positive for reducing substances but negative to glucose. A galactose-1-phosphate uridyl transferase assay will confirm the diagnosis.

**DIAGNOSTIC CRITERIA**
- hepatitis may be confirmed by abnormal liver function tests, i.e. raised values of:
  - AST
  - alkaline phosphatase
  - ALT
  - bilirubin, mainly the conjugated fraction
  - gamma GT
- hepatomegaly and/or hepatosplenomegaly
- if conjugated hyperbiliruniaemia – See above

**NON-DRUG TREATMENT**
- monitor bilirubin levels
- treat the underlying cause
- dietary adjustment for prolonged conjugated hyperbilirubinaemia to counteract the malabsorption of fat and fat soluble vitamins (A,D,E,K)
- avoid lactose containing feeds, i.e. breast milk and lactose containing formulae, when galactosaemia is suspected.
- regular follow up until the underlying condition has been resolved

**DRUG TREATMENT**
- fat soluble vitamins, A, D, E and K

**REFERRAL**
- pathological jaundice, unconjugated and/or conjugated, where the underlying cause cannot be identified
- serum unconjugated bilirubin at exchange transfusion level
- jaundice, unconjugated and/or conjugated, not improving on adequate treatment
- conjugated hyperbilirubinaemia due to conditions requiring surgical intervention e.g. biliary atresia
- prolonged neonatal jaundice, excluding breast milk jaundice
PHOTOTHERAPY

In presence of risk factors use one line lower (gestation below) until < 1 000 g
If gestational age is accurate, rather use gestational age (weeks) than body weight

Infants > 12 hours old with TSB level below threshold, repeat TSB level as follows:
1 - 20 micromol/L below line: repeat TSB in 6 hours or start phototherapy and rept TSB in 12–24 hours.
21-50 micromol/L below line: repeat TSB in 12–24 hours,
> 50 micromol/L below line: rept TSB until it is falling and/or until jaundice is clinically resolving

Infants under phototherapy:
Check the TSB 12–24 hourly but if TSB > 30 micromol/L above the line, check TSB 4–6 hourly.
STOP phototherapy:
If TSB > 50 micromol/L below the line. Recheck TSB in 12–24 hours.

Start intensive phototherapy when the TSB is > the line according to gestation or weight

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EXCHANGE TRANSFUSION

In presence of sepsis, haemolysis, acidosis, or asphyxia, use one line lower (gestation below) until < 1000 g
If gestational age is accurate, rather use gestational age (weeks) than body weight

Note:
1. Infants who resent with TSB above the threshold should have Exchange done if the TSB is not expected to be below the threshold after 6 hours of intensive phototherapy.
2. Immediate Exchange is recommended if signs of bilirubin encephalopathy or TSB > 85 micromol/L above threshold
3. Also exchange if TSB continues to rise >17 micromol/L/hour
19.11 MENINGITIS BACTERIAL, NEONATAL

DESCRIPTION
A bacterial infection of the meninges in the first month of life.

Meningitis should be considered in any neonate being evaluated for sepsis or infection as most organisms implicated in neonatal sepsis also cause neonatal meningitis. The most common causative organisms are Group B ß-haemolytic streptococcus type III and Gram-negative organisms such as *E. Coli* with K1 antigen. *S. epidermidis* and *S. aureus* as causative organisms are to be considered with central nervous system anomalies such as open defects or with indwelling devices such as VP shunts.

DIAGNOSTIC CRITERIA

Clinical
- the clinical presentation is usually with one or more non-specific signs such as:
  - temperature disturbances
  - lethargy
  - irritability
  - vomiting
  - feeding problems
  - vasomotor changes
  - altered level of consciousness
  - blood glucose disturbances
  - bulging/full fontanel
  - convulsions
  - apnoea

- complications include:
  - cerebral oedema
  - raised intracranial pressure
  - vasculitis, with haemorrhage
  - ventriculitis
  - ischaemia and infarctions of the brain
  - inappropriate antidiuretic hormone (ADH) secretion
  - convulsions
  - hydrocephalus
  - subdural effusion
  - brain abscess

- late complications include:
  - neurological sequelae
  - blindness
  - deafness
  - mental retardation

Special Investigations
- lumbar puncture
  - the CSF appears opalescent to purulent
  - protein concentration is increased
  - leucocyte count is increased with a predominance of polymorphonuclear leucocytes
  - glucose concentration is low, $< \frac{2}{3}$ of blood glucose
- Gram stain, microscopy, culture and sensitivity of CSF. Rapid antigen tests on the CSF.
- blood cultures for microscopy, culture and sensitivity
NON-DRUG TREATMENT

• admit to high or intensive care unit, if available
• maintain a neutral thermal environment
• monitor, where indicated:
  ○ neurological status
  ○ vital signs
  ○ electrolytes
  ○ haematocrit
  ○ fluid balance (hydration)
  ○ minerals
  ○ acid-base status
  ○ blood glucose
  ○ serum and urine osmolality
  ○ blood gases
• ensure adequate nutrition
  ○ enteral feeding where possible, use nasogastric tube, if necessary
  ○ if enteral feeding is not possible, IV fluids, e.g. neonatal maintenance solution and
    parenteral nutrition under supervision by paediatrician
• limit total daily fluid intake, IV and oral,
  ○ do not exceed the daily requirements for age
  ○ prevent fluid overload

DRUG TREATMENT

Antibiotics, empirical

Reconsider choice of antibiotic when the results of blood and CSF cultures become available or the child does not improve within 72–96 hours.

Discontinue cefotaxime or ceftriaxone if Group B β-haemolytic streptococci or L. monocytogenes is cultured.

• cefotaxime, IV, 50 mg/kg over 30 minutes, for 14–21 days
  < 7 days 50 mg/kg, 12 hourly
  7 days – 3 weeks 50 mg/kg, 8 hourly
  > 3 weeks 50 mg/kg, 6 hourly

  OR
  ceftriaxone , IV, 100 mg/kg loading dose then 80 mg/kg/24 hours 12–24 hourly
  Gram positive organisms: 14 days
  Gram negative organisms: 21 days

PLUS

• ampicillin, IV, for 14 days
  < 7 days 50–100 mg/kg, 12 hourly
  > 7 days 50 mg/kg, 6–8 hourly

No response or intolerant to cephalosporins or ampicillin

For patients not responding to adequate antibiotic therapy where no organisms were identified or cultured, or patients intolerant of ampicillin and cephalosporins, consider:

Anaerobic bacteria

• metronidazole, IV, 7.5 mg/kg for 14 days
  < 7 days 7.5 mg/kg, 12 hourly
  > 7 days 7.5 mg/kg, 8 hourly
Methicillin resistant staphylococci
- vancomycin, IV, 15 mg/kg loading dose followed by 10 mg/kg for 14 days
  ≤ 7 days  10 mg/kg, 12 hourly
  > 7 days  10 mg/kg, 8 hourly

Sensitive staphylococci
- cloxacillin, IV, 50–100 mg/kg/dose for 14 days
  ≤ 7 days  50–100 mg/kg, 12 hourly
  > 7 days  50–100 mg/kg, 6 hourly

Pseudomonas aeruginosa
- ceftazidime, IV, 30 mg/kg/dose for 14-21 days
  ≤ 7 days  30 mg/kg/dose, 12 hourly
  > 7 days  30 mg/kg/dose, 8 hourly

AND
- amikacin, IV, 15 mg/kg/dose, once daily for 7–10 days

For fever
- paracetamol, oral, 10 mg/kg/dose, 6 hourly when needed until fever subsides

Convulsions
See Neonatal Seizures: Section 19.16.

Raised intracranial pressure or cerebral oedema
Avoid fluid overload.
Limit total daily intake, IV and oral.
Do not exceed the maintenance requirements for age.

REFERRAL
- meningitis not responding to adequate treatment
- meningitis with complications

19.12 PATENT DUCTUS ARTERIOSUS (PDA) IN THE NEWBORN
Q25.0

DESCRIPTION
PDA (Patent ductus arteriosus) is the persistence of the normal fetal vessel that joins the pulmonary artery to the aorta extra-uterine.

DIAGNOSTIC CRITERIA
Clinical
depending on size of PDA
- systolic or continuous murmur at left heart base
- hyperactive precordium with easily palpable bounding peripheral pulses

Investigations
- echocardiography will confirm the diagnosis
Risk factors include:
- prematurity
- hypoxia
- fluid overload
- anaemia
- pulmonary hypertension
- sepsis
- lung disease
- congenital cardiac abnormalities

Complications include cardiac failure, systemic hypotension and pulmonary haemorrhage.

NON-DRUG TREATMENT

Preterm Infants
- identify and treat underlying risk factors
- restrict fluid intake to 80–120 mL/kg/24 hours. Individualise volume.
- maintain haematocrit at ≥ 40% and Hb ≥ 13 g/dL
- monitor cardiac function, renal function and urinary output
- provide adequate nutrition
- nurse in neutral thermal environment

DRUG TREATMENT

Cardiac failure
Diuretics
- furosemide, IV/oral, 1 mg/kg/24 hours

Short term
- digoxin, IV/oral, 0.005 mg/kg/dose 12 hourly

Closure of PDA in preterm infant less than 14 days of age
- ibuprofen, oral
  First dose: 10 mg/kg followed by 2 additional doses after 24 hours.
  Additional doses: 5 mg/kg each 12–24 hours apart.

Note:
Contraindications to ibuprofen therapy
- thrombocytopenia (<50 000/mm$^3$)
- bleeding disorders
- impaired renal function
- jaundice approaching exchange transfusion levels

SURGICAL TREATMENT
- if medicine treatment is contraindicated or fails

REFERRAL
- patients with complications, e.g. cardiac failure, pulmonary haemorrhage, ventilator dependence
- PDA which remained patent despite adequate treatment
- term babies with symptomatic or persistent PDA
19.13 PREMATURITY/PRETERM NEONATE

DESCRIPTION
Neonate born before 37 completed weeks of pregnancy. Newborns with birth weight under 2.5kg are often premature.

NON-DRUG TREATMENT
- admit unwell/unstable infants to neonatal high/intensive care facility
- temperature control
  - Kangaroo mother care: Initiate if baby is well and vital signs are stable
  - provide a neutral thermal environment (incubator or infant crib with overhead heater) and keep ambient temperature at 26–28°C
  - keep infants temperature, axilla or skin of anterior abdominal wall, at 36.2–36.8°C
  - see table for neutral thermal environment for age and body mass

<table>
<thead>
<tr>
<th>Neutral Thermal Environment</th>
<th>Temperature for body mass range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>&lt; 1 200 g ± 0.5°C</td>
</tr>
<tr>
<td>0–12 hours</td>
<td>35</td>
</tr>
<tr>
<td>12–24 hours</td>
<td>34.5</td>
</tr>
<tr>
<td>2–4 days</td>
<td>34.5</td>
</tr>
<tr>
<td>4–14 days</td>
<td>33.5</td>
</tr>
<tr>
<td>2–3 weeks</td>
<td>33.1</td>
</tr>
<tr>
<td>3–4 weeks</td>
<td>32.6</td>
</tr>
<tr>
<td>4–5 weeks</td>
<td>32.0</td>
</tr>
<tr>
<td>5–6 weeks</td>
<td>31.4</td>
</tr>
</tbody>
</table>
- monitor to prevent or detect early the diseases/complications of prematurity:
  - respiratory rate
  - haematocrit
  - blood pressure
  - bilirubin
  - blood gasses
  - blood glucose
  - acid-base status
  - electrolytes
  - minerals
  - hydration status
  - growth parameters
- nutritional support
  - give naso/orogastric tube feedings to infants with audible bowel sounds and no complications/diseases of prematurity
  - preferably use own mother’s expressed breast milk or pre-term formula. Give small frequent bolus feeds, 1, 2 or 3 hourly, or continuous naso/orogastric tube feeds.
  - monitor gastric emptying by aspirating the stomach before each feed
  - reconsider enteral feeding if:
    - aspiration of 3 mL or more of gastric contents before the next feed
    - vomiting
    - abdominal distension
    - diarrhoea
  - IV alimentation if enteral feeds are contraindicated or not tolerated
• IV fluids to ensure adequate hydration, electrolyte and mineral intake, and normoglycaemia (blood glucose ≥ 2.6 mmol/L) until enteral (tube or oral) intake is satisfactory.
  ○ discontinue IV fluids gradually to avoid reactive hypoglycaemia
  ○ discontinue the infusion when several oral feedings have been retained
  ○ if renal function is compromised, the potassium-free solution should be used

<table>
<thead>
<tr>
<th>Fluid requirements for a healthy premature infant</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day of birth</td>
</tr>
<tr>
<td>---------------</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>4</td>
</tr>
<tr>
<td>5</td>
</tr>
<tr>
<td>6 and onwards</td>
</tr>
</tbody>
</table>

Some infants may require fluid volumes up to 200 mL/kg/24 hours after day 6
• packed red cells, IV, 10 mL/kg, to maintain haematocrit at 40% or ±13 g/dL Hb for the first 2 weeks of life
• oxygen, humidified via head box, to maintain oxygen tension in the blood at 60–80 mmHg. Oxygen therapy should be utilised to maintain oxygen saturation of haemoglobin at 90–92%. Use pulse oximeter.
• Hospital discharge if:
  ○ clinically well
  ○ able to breast feed or formula feed
  ○ able to maintain body temperature
  ○ usually greater than 1.8 kg

Follow-up visits to assess growth parameters, neurodevelopment, hearing and vision.

**DRUG TREATMENT**

**At birth**
• vitamin K₁, IM, 0.5–1 mg
• immunise according to EPI schedule
• iron and multivitamin supplementation from the third week of life

**Prophylaxis**
• iron, oral, 2–4 mg of elemental iron/kg/24 hours
  ferrous lactate 1 mL = 25 mg elemental iron
• multivitamin, oral, providing per 24 hours at least:
  vitamin D:  400–800 IU
  vitamin A:  1 250–5 000 IU

Continue with iron and vitamin supplementation until the infant is on a balanced diet.
REFERRAL
Presence of one or more of the following complications that cannot be managed at the facility:
- respiratory distress and/or apnoea attacks requiring ventilatory support
- PDA with cardiac failure not responding to medical management
- necrotising enterocolitis requiring surgical intervention
- jaundice with serum unconjugated bilirubin level in the exchange transfusion zone
- septic/infected infants or infants with infections not responding to therapy
- pulmonary and/or intraventricular haemorrhage
- feeding difficulties where the underlying cause is unclear
- infants requiring hyperalimentation
- convulsions not responding to treatment
- congenital abnormalities requiring surgical intervention
- hypoglycaemia not responding to treatment
- infants less than 1.5 kg for eye examination

19.14 RESPIRATORY DISTRESS IN THE NEWBORN
P22.9

DESCRIPTION
Newborn experiencing difficulty with breathing.
Causes of respiratory distress include:

<table>
<thead>
<tr>
<th>Pulmonary Causes</th>
<th>Extrapulmonary Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• hyaline membrane disease (surfactant deficiency)</td>
<td>• sepsis</td>
</tr>
<tr>
<td>• meconium aspiration</td>
<td>• cardiac failure irrespective of cause</td>
</tr>
<tr>
<td>• pneumonia</td>
<td>• pulmonary hypertension</td>
</tr>
<tr>
<td>• pneumothorax</td>
<td>• hypothermia/hyperthermia</td>
</tr>
<tr>
<td>• wet lung syndrome</td>
<td>• hypoglycaemia</td>
</tr>
<tr>
<td>• pulmonary haemorrhage</td>
<td>• anaemia</td>
</tr>
<tr>
<td>• hypoplastic lungs</td>
<td>• polycythaemia</td>
</tr>
<tr>
<td>• diaphragmatic hernia</td>
<td>• hypovolaemic shock</td>
</tr>
<tr>
<td></td>
<td>• perinatal hypoxia</td>
</tr>
</tbody>
</table>

DIAGNOSTIC CRITERIA
Clinical
- Pulmonary and/or extra pulmonary disorders presenting with two or more of the following signs in a newborn baby:
  - tachypnoea (60 breaths/minute)
  - expiratory grunting
  - intercostal and sternal retractions (recession)
  - central cyanosis while breathing room air

Investigations
- a chest X-ray should be performed to determine underlying pathology
- echocardiography, if available, to exclude cardiac causes of respiratory distress
- haematocrit, blood glucose and temperature
• shake test to assess risk for hyaline membrane disease
  ○ within 15 minutes after birth place 0.5 mL gastric aspirate in a clean dry test tube
  ○ add 0.5 mL of sodium chloride 0.9% and replace the cap
  ○ shake well for 15 seconds
  ○ add 1 mL 95% alcohol to the 1 mL mixture of gastric aspirate and sodium chloride 0.9%
  ○ replace cap and shake well for 15 seconds
  ○ read at 15 minutes
  ○ interpretation of test

<table>
<thead>
<tr>
<th>Observation</th>
<th>Result</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>No bubbles on surface</td>
<td>Negative</td>
<td>High</td>
</tr>
<tr>
<td>Incomplete ring of bubbles on surface</td>
<td>Intermediate</td>
<td>Possible</td>
</tr>
<tr>
<td>Complete ring of bubbles or bubbles covering the entire surface</td>
<td>Positive</td>
<td>Very low</td>
</tr>
</tbody>
</table>

**NON-DRUG TREATMENT**

• identify and treat underlying cause, e.g.:
  ○ chest tube and underwater drainage of pneumothorax,
  ○ isovolaemic dilutional exchange transfusion for symptomatic polycythaemia,
  ○ antibiotics for bronchopneumonia, etc.

• admit to neonatal high care/intensive care facility, if available

• handle neonate as little as possible

• nurse non-intubated infant in the prone position

• nurse in a neutral thermal environment (incubator or infant crib with overhead heater)
  Keep ambient temperature within thermoneutral range, at 26–28°C, or anterior abdominal wall skin temperature at 36.2–36.8°C.

• monitor
  blood pressure  respiratory rate
  peripheral perfusion  heart/pulse rate
  haematocrit  acid-base status
  blood glucose  body temperature
  blood gases  SaO₂
  minerals and electrolytes  fluid balance

• nutrition
  ○ provide adequate IV dextrose to maintain blood glucose ≥ 2.6 mmol/L
  ○ commence orogastric feeding after 12–24 hours if bowel sounds are audible and meconium has been passed
  ○ if enteral feeding is still not possible on day 3 after birth, start IV hyperalimentation

• oxygen, humidified via head box, to eliminate central cyanosis
  ○ if a pulse oximeter or facility for blood gas analysis is not available, regulate the inspired oxygen concentration in such a way that the least amount of oxygen that will prevent central cyanosis is used
  ○ oxygen, humidified via head box, to maintain oxygen tension in the blood at 60–80 mmHg. Oxygen therapy should be utilised to maintain oxygen saturation of haemoglobin at 90–92%. Use pulse oximeter.
  ○ keep PaO₂ at 60–80 mmHg and PaCO₂ at 35–45 mmHg (arterial blood gas analysis)
• nasal CPAP is needed if:
  the neonate has a good respiratory drive
  a PCO\textsubscript{2} of ≤ 55 mmHg but unable to maintain a SaO\textsubscript{2} of 90–92% on an inspiratory oxygen concentration of ≥ 60% (F\textsubscript{1}O\textsubscript{2})
  pneumothorax has been excluded
  ○ administer nasal CPAP at 4–7 cm H\textsubscript{2}O and monitor SaO\textsubscript{2}, blood gas and acid base status

• ventilation is needed if:
  ○ an oxygen saturation of at least 90% or PaO\textsubscript{2} of at least 60mmHg cannot be maintained with an inspiratory oxygen concentration of ≥ 80% with or without nasal CPAP
  ○ the PaCO\textsubscript{2} rises to > 55 mmHg with uncompensated respiratory acidosis (pH ≤ 7.2), irrespective of oxygen saturation or PaO\textsubscript{2}
    \((1\text{kPa} = 7.5 \text{ mmHg}; 1 \text{ mmHg} \times 0.133 = 1 \text{kPa})\)

**DRUG TREATMENT**

**Stabilise circulation and blood pressure**

• neonatal maintenance solution, IV infusion, 60–80 mL/kg/24 hours (day of birth) and adapt to daily maintenance requirements

**AND/OR**

• sodium chloride 0.9%, 10–20 mL/kg over 1–2 hours
  For premature infants restrict to 10 mL/kg.

**AND/OR**

• plasma, lyophilised, 10–20 mL/kg over 1–2 hours.

**Inotropic support**

• dopamine, IV, 5–15 mcg/kg/minute, continued until blood pressure has stabilised
  Response to inotropic support will be unsatisfactory if the circulating volume is not corrected.

**Anaemia**

If anaemia is present, Hct < 40 % and Hb <13 g/dL

• red cells, packed, IV, 10mL/kg over 1–2 hours

**Metabolic acidosis**

If pH ≤ 7.2 and the metabolic acidosis does not respond to normalisation of PaO\textsubscript{2}, PaCO\textsubscript{2}, blood pressure, volume expansion (hydration) and correction of anaemia

• sodium bicarbonate, 4.2 %, IV, given slowly
  \(1 \text{ mmol} = 2 \text{ mL}\)
  \[\text{HCO}_3^- \text{ needed (mmol)} = \text{base excess} \times 0.3 \times \text{body mass (kg)}\]
  (½ correct base deficit initially)

If blood gas and acid base analysis is not available and metabolic acidosis is suspected

• sodium bicarbonate, 4%, slow IV, 2mL

**CAUTION**

Do not administer Ca++ containing infusions with sodium bicarbonate solution
Polycythaemia
Treat with isovolaemic dilutional exchange transfusion using sodium chloride 0.9% if the venous haematocrit is Hct > 65%: Hb >22 g/dL and the baby is symptomatic. Perform under paediatrician’s supervision
Formula taking desired Hct = 50:
Volume to be exchanged (mL)
= [Baby’s Hct – desired Hct (i.e. 50) x body mass (kg)] x 90 ÷ Baby’s Hct

Hyaline membrane disease (Surfactant deficiency)
Restricted to supervision by paediatrician.
Shake test to assess risk for hyaline membrane disease – see above.
If surfactant deficiency is suspected or present the baby must be intubated and ventilated before administration of the first dose of surfactant.
Semi-synthetic surfactant preparation, e.g. poractant and beractant are recommended while 100% synthetic preparations are not recommended.

Infection
If infection, e.g. bronchopneumonia, is present or suspected, give antibiotics after blood cultures have been taken.

Aminoglycoside, e.g.:
• amikacin, IV, 15 mg/kg as single daily dose for 7–10 days
  OR
  gentamicin, IV, in the first week of life
  < 33 weeks gestation  5 mg/kg/48 hours
  < 38 weeks gestation  4 mg/kg/36 hours
  ≥ 38 weeks gestation  4 mg/kg/24 hours

PLUS
Penicillin, e.g.:
• benzylpenicillin (penicillin G), IV, 25 000–50 000 units/kg/dose, 12 hourly for 10 days
  OR
  ampicillin, IV, 50–100 mg/kg,
  < 7 days        50–100  mg/kg, 12 hourly
  7 days – 3 weeks 50–100  mg/kg, 8 hourly
  > 3 weeks      50–100  mg/kg, 6 hourly

Review after 72 hours. If infection is confirmed or very strongly suspected continue for 7–10 days.

REFERRAL
• no improvement or deterioration despite adequate treatment
• development of respiratory failure and need for ventilatory support
19.15 RESUSCITATION OF THE NEWBORN

Be prepared!
Be at the delivery!
Check the equipment and emergency medicines!

Ask 3 questions to evaluate the infant:
1. Is the baby breathing adequately and not just gasping?
2. Is the baby’s heart rate (HR) above 100 beats per minute?
3. Is the baby centrally pink, i.e. no central cyanosis?

- If the answer to all three questions is “yes”, the baby does not need resuscitation.
- If the answer to all three questions is “no” the baby needs resuscitation.

- Assess the infant using the above 3 questions every 30 seconds during resuscitation.
  - If the baby is improving, then the intervention e.g. bagging can be stopped.
  - Only if the baby is not responding or getting worse, is further intervention needed e.g. chest compressions (see algorithm).

- Check that each step has been effectively applied before proceeding to the next step. The algorithm follows the assumption that the previous step was unsuccessful and the newborn is deteriorating.

- Use the lowest inspiratory oxygen concentration to alleviate central cyanosis and restore a heart rate above 100 beats per minute. There is some evidence that resuscitation with 100% oxygen may be harmful to the baby.

- An unsatisfactory response to resuscitation includes:
  - a sustained slow heart rate, usually less than, or equal to, 60/minute or a progressive decrease in heart rate until cardiac arrest occurs
  - episodes of cardiac arrest, with a progressively weaker response to chest compressions, positive pressure ventilation and medicines
  - a decreasing blood pressure, increasing acidosis, severe hypotonia with central cyanosis or intense pallor
  - apnoea or weak, irregular and inefficient respiratory efforts

- Consider discontinuation of resuscitation if the unsatisfactory response to resuscitation persists for > 20 minutes and underlying conditions e.g. pneumothorax, diaphragmatic hernia have been excluded or > 10 minutes of unresponsive cardiac arrest (asystole) and/or > 20 minutes of unsustainable respiration.

- Newborns with a favourable response to resuscitation should be admitted to a neonatal high or intensive care unit, if available, for post resuscitation care –See Hypoxia/Ischaemia of the Newborn: Section 19.8
### Drugs Used During Neonatal Resuscitation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indications</th>
<th>Dosage</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>adrenaline</td>
<td>• asystole&lt;br&gt;• heart rate &lt; 60 per minute</td>
<td>IV, 0.01 mg/kg/dose (0.1 mL/kg of a 1:10 000 dilution)&lt;br&gt;ET, 0.1 mg/kg/dose (0.1 mL/kg of a 1:1 000 solution)</td>
<td>↑ Heart rate&lt;br&gt;↑ Myocardial contractility&lt;br&gt;↑ Arterial pressure</td>
</tr>
<tr>
<td>sodium bicarbonate (4%)</td>
<td>• life threatening metabolic acidosis&lt;br&gt;• pH &lt; 7.2&lt;br&gt;• BE &gt; –10 mmol/L&lt;br&gt;• PaCO₂ &lt; 55 mmHg</td>
<td>IV, 1–2 mmol/kg very slowly</td>
<td>Corrects metabolic acidosis&lt;br&gt;Improves cardiac output and peripheral perfusion</td>
</tr>
<tr>
<td>naloxone</td>
<td>• maternal administration of opiates + apnoeic infant</td>
<td>ET/IV/SC/IM, 0.1 mg/kg</td>
<td>Corrects apnoea and/or hypoventilation</td>
</tr>
<tr>
<td>Fluids sodium chloride 0.9%</td>
<td>• hypovolaemia</td>
<td>slow IV, 10–20 mL/kg</td>
<td>↑ Blood pressure and improves tissue perfusion</td>
</tr>
<tr>
<td>dextrose</td>
<td>• hypoglycaemia</td>
<td>IV, 250 mg–500 mg/kg (2.5–5 mL/kg of 10% dextrose water)</td>
<td>Corrects hypoglycaemia</td>
</tr>
</tbody>
</table>
A

AIRWAY
Remove MECONIUM or BLOOD if present before stimulating

Warm, position, Clear Airway, Dry and Stimulate

breathing, blue and HR < 100

breathing, pink and HR > 100

Assess:
Breathing, colour and heart rate

Supportive care

Apnoea or blue or HR < 100

Administer oxygen

B

BREATHE
Ventilation Rate 40–60/minute

Assess:
Breathing, colour and heart rate

HR < 60

HR > 60

C

CHEST COMPRESSIONS
Rate: 120/minute
Ratio: 3 compressions: 1 ventilation until intubated

Assess:
Breathing, colour and heart rate

HR < 60

HR > 60

D

DRUGS
Adrenaline 0.01 mg/kg, IV/ET, every 3–5 minutes as required
Neonatal seizures are usually secondary to a serum biochemical disorder or an underlying brain disturbance/injury/malformation. They may be subtle due to the relatively underdeveloped cortex, and do not stop when limbs are flexed (as opposed to jitteriness). The most likely causes are:

- perinatal asphyxia
- birth trauma
- intracranial haemorrhage
- meningitis
- narcotic or alcohol withdrawal syndrome
- hypocalcaemia
- hypomagnesaemia
- hyponatraemia
- hypoglycaemia

**DIAGNOSTIC CRITERIA**

**Categories of convulsions**

- subtle seizures
  - tonic deviation of the eyes
  - ‘swimming’ movements of the arms
  - fluttering of the eyelids
  - ‘cycling’ movements of the legs
  - sucking and chewing movements
  - vasomotor changes
- tonic clonic movements
- focal clonic movements
- myoclonic movements
- tonic movements/posturing

**NON-DRUG TREATMENT**

- identify and treat the underlying cause, e.g. meningitis and hypoxic-ischaemic encephalopathy
- ensure an open airway and administer oxygen if necessary
- nurse in neutral thermal environment
- ensure adequate nutrition and hydration
- monitor and maintain within accepted physiological range:
  - respiration
  - acid-base status
  - heart rate
  - electrolytes
  - blood pressure
  - minerals
  - blood gases
  - blood glucose
  - SaO₂
  - haematocrit
  - body temperature
DRUG TREATMENT

Seizure control
Administer phenytoin and phenobarbitone with monitoring of cardiorespiratory function.

Option 1:
- phenytoin, IV
  Loading dose: 15 mg/kg diluted in 3 mL 0.9 % sodium chloride given over 30 minutes by slow IV infusion preferably under ECG control.
  Flush IV line with sodium chloride 0.9% before and after administration of the phenytoin.
  Maintenance: IV/oral, 5–8 mg/kg/24 hours in 3 divided doses.

  Note:
  Phenytoin must not be given in glucose/dextrose- containing solutions. To minimise risk of precipitation administer phenytoin in 0.9% sodium chloride solution.
  Do not administer phenytoin intramuscularly.

AND
- phenobarbital, oral
  Loading dose of 20–40 mg/kg via oro/nasogastric tube.
  Maintenance: 5 mg/kg/day in two divided dosages.

Option 2:
- clonazepam, IV
  Loading dose 0.1–0.15 mg/kg administered by slow IV injection.
  Maintenance: 0.1 mg/kg/day.

AND
- phenobarbital, oral
  Loading dose of 20–40 mg/kg via oro/nasogastric tube.
  Maintenance: 5 mg/kg/day in two divided dosages.

Option 3:
- midazolam, IV bolus, 0.5 mg/kg

AND
- phenobarbital, oral
  Loading dose of 20–40 mg/kg via oro/nasogastric tube.
  Maintenance: 5 mg/kg/day in two divided dosages.
  Monitor cardiorespiratory function.

Seizures refractory to the above mentioned treatment.
Cardiorespiratory support is usually required in this category of infants.

- midazolam as continuous infusion at 60 mcg/kg/hour.
  May be increased every 5–10 minutes by 25 mcg/kg/hour to maximum of 100 mcg/kg/hour in preterm and 1 000 mcg/kg/hour in term infants.

OR
- lorazepam, IV/IM, 0.1 mg/kg/dose. Repeat once if necessary.
Maintenance anticonvulsant therapy
Maintenance anticonvulsant therapy is usually considered for neonates with underlying brain damage due to hypoxic ischaemic encephalopathy, meningitis, intracranial haemorrhage or birth trauma.
Continue until neonate is seizure-free for 2 weeks, then slowly taper to stop.
If seizures recur during tapering of anticonvulsant, continue with maintenance therapy.
Follow-up after discharge by medical practitioner or at clinic/hospital.

Underlying biochemical disorders
Hypocalcaemia
Serum total calcium $\leq 1.7$ mmol/L, or ionised calcium $< 0.7$ mmol/L.
- calcium gluconate 10%, IV, 1–2 mL/kg
  Dilute 1:4 with dextrose 5% water.
  Give preferably under ECG control over 5 minutes or until seizure ceases.
  Repeat if necessary.

Hypoglycaemia
Serum glucose $< 2.6$ mmol/L
- dextrose, IV as bolus, 250–500 mg/kg, followed by 8–10 mg/kg/minute or more until blood glucose is within the physiological range
  Dilute dextrose 50% solution before use to 10% strength.
  0.5–1 mL of dextrose 50% = 250–500 mg
  OR
  2.5 mL of dextrose 10% = 250 mg

Hypomagnesaemia
Serum magnesium $< 0.6$ mmol/L
- magnesium sulphate 50%, IV, 0.25 mL/kg slowly over 3 minutes as a single dose

REFERRAL
- seizures not responding to adequate therapy
- seizures where the underlying cause is unclear

19.17 SEPTICAEMIA OF THE NEWBORN
P36.9

DESCRIPTION
Bacterial or fungal invasion of blood before or after birth, which may spread to involve other organs/systems, e.g. meninges (meningitis), lungs, (pneumonia), bone (osteomyelitis), and kidneys (pyelonephritis).
DIAGNOSTIC CRITERIA

Clinical
- the baby usually presents with one or more non-specific clinical sign e.g.:
  - vasomotor changes
  - feeding problems
  - lethargy
  - jaundice
  - diarrhoea
  - tachypnoea
  - temperature disturbances
  - apnoea attacks
  - sclerema
  - acidosis
- complications include:
  - septic shock
  - hypoglycaemia
  - apnoea
  - convulsions
  - anaemia
  - bronchopneumonia
  - cardiac failure
  - dehydration

Investigations
- blood and cerebrospinal fluid cultures
- blood count and differential count
- C-reactive protein and procalcitonin, if available

NON-DRUG TREATMENT
- admit to neonatal high or intensive care facility, if available
- ensure a neutral thermal environment
- start IV infusion with appropriate IV fluid, e.g. neonatal maintenance solution
- ensure adequate nutrition
  - enteral feeding where possible, via oro/nasogastric tube after ileus, obstruction, or other contraindications to enteral feeding have been excluded
  - if enteral feeding is not possible, IV fluids, e.g. neonatal maintenance solution and parenteral nutrition under supervision by paediatrician
- insert naso/orogastric tube
- oxygen to maintain PaO₂ at 60–80 mmHg or oxygen saturation of haemoglobin at 88–92%
- ventilatory support if PaCO₂ exceeds 55 mmHg
- monitor:
  - body temperature 36.2–36.8°C (axillary or anterior abdominal wall)
  - maintain blood glucose level of 2.6–6.8 mmol/L
  - acid-base status and maintain blood pH of 7.35–7.45
  - maintain a haematocrit of 40–45%
  - vital signs and respiration, and maintain blood electrolytes and minerals within their normal physiological ranges
  - clinical progress and for the emergence of complications
DRUG TREATMENT

Antibiotic therapy
Reconsider choice of antibiotic when the results of blood and CSF cultures become available or the child does not improve within 72–96 hours.
Be aware of the antibiotic sensitivity/resistance profile of bacterial pathogens in your hospital/community.

- amikacin, IV, 15 mg/kg/dose, once daily for 7–10 days. Monitor blood levels.
  OR
  gentamicin, IV, 5 mg/kg/dose, once daily for 7–10 days. Monitor blood levels.

PLUS

- cefotaxime, IV, 50 mg/kg over 30 minutes, for 7–10 days
  < 7 days  50 mg/kg, 12 hourly
  > 7 days  50 mg/kg, 8 hourly
  OR
  benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 12 hourly, for 7–10 days
  OR
  ampicillin, IV, 50 mg/kg for 7–10 days
  < 7 days  50 mg/kg, 12 hourly
  > 7 days  25 mg/kg, 6 hourly

Fungal infections

- fluconazole, IV, 6–12 mg/kg as a single dose infused over 60 minutes
  ≤ 2 weeks  6–12 mg/kg, every 72 hours
  > 2 weeks  6–12 mg/kg, every 48 hours
  OR
  amphotericin B, IV, 0.5–1 mg/kg/24 hours infused over 2 hours for 14 days. Monitor renal function.

Anaerobic infections

- metronidazole, oral/IV, 7.5 mg/kg for 7–10 days
  ≤ 7 days  7.5 mg/kg, 12 hourly
  > 7 days  7.5 mg/kg, 8 hourly

Inotropic support
Mean blood pressure should not be less than the gestational age (weeks) of the infant plus 5–10 mmHg.
If blood pressure is:
  < \frac{60}{40} \text{ mmHg in term infant}
  < \frac{50}{35} \text{ mmHg in pre-term infant}

- dopamine, IV, 5–15 mcg/kg/minute as a continuous infusion
  Continue with dopamine as long as it is necessary to maintain the blood pressure.

REFERRAL

- septicaemia with complications
- septicaemia not responding to treatment
**19.18 SYPHILIS, EARLY CONGENITAL**

**DESCRIPTION**
Multi-organ infection caused by *Treponema pallidum* and acquired by vertical transmission via the transplacental route during pregnancy.

**DIAGNOSTIC CRITERIA**

**Clinical**
- suspect if mother has syphilis or positive serology for syphilis and the baby a positive non-treponemal serological test at birth with a titre significantly higher than that of the mother
- the following signs may be present at birth or will develop within the first 3 months of life:
  - hydrops fetalis
  - anaemia
  - hepatosplenomegaly
  - oedema
  - condylomata
  - hepatitis
  - nephrosis/nephritis
  - transient bullous lesions, commonly on the hands and feet with later desquamation and an erythematous appearance of palms and soles
  - a generalised reddish maculopapular rash which may also desquamate
  - rhinitis with mucopurulent bloodstained discharge excoriating the upper lip
  - other mucocutaneous lesions of the mouth, anus and genitalia, healing with scars, especially the corners of the mouth and on the chin
  - involvement of long bones with/without pseudoparalysis of one or more limbs and radiological findings

**Investigations**
- X-ray of long bones:
  - translucent metaphyseal bands
  - osteochondritis
  - osteitis
  - metaphysitis and periostitis
- positive non-treponemal serological tests, i.e. RPR or VDRL
- positive anti-treponemal IgM test is of limited value

Do not use umbilical cord blood at delivery for laboratory investigations.

**NON-DRUG TREATMENT**
- nurse infant in a neutral thermal environment
- maintain adequate nutrition and hydration
- monitor and maintain within physiological range for age:
  - albumin
  - pH
  - blood pressure
  - electrolytes
  - monitor hepatic and renal function

A50.9
*Notifiable condition.*
Pneumonia
To maintain oxygen saturation at 90–92% or PaO₂ at 60–80 mmHg
• oxygen via a head box or nasal cannula
  1 kPa = 7.5 mmHg
  1 mmHg x 0.133 = 1 kPa

Anaemia
• packed red cells, 10 mL/kg over 3 hours if haemoglobin < 10 g/dL

DRUG TREATMENT
Asymptomatic, well baby
mother seropositive or result unknown, and mother has not been treated or was only partially treated:
• benzathine benzylpenicillin (depot formulation), IM, 50 000 units/kg as a single dose into the antero-lateral thigh

Symptomatic baby
• procaine penicillin (depot formulation), IM, 50 000 units/kg daily for 10–14 days (not for IV use)
OR
benzylpenicillin (Penicillin G), IV, 50 000 units/kg, 12 hourly for 10–14 days

CAUTION
Procaine penicillin and benzathine benzylpenicillin (depot formulation) should not be given intravenously.

Prevention
Screen pregnant women for syphilis at first visit and repeat during the second and/or third trimester.
Investigate and treat parents, if necessary.

REFERRAL
• symptomatic infant with complications, e.g. respiratory failure, hepatic failure, nephrotic syndrome and meningitis

19.19 TETANUS, NEONATAL
A33

DESCRIPTION
Tetanus is an acute spastic paralytic illness caused by tetanospasmin, the neurotoxin produced by Clostridium tetani. Neonatal tetanus is the most common form of the disease, usually caused by umbilical stump infections.

The disease only occurs in infants of non-immunised mothers or mothers with insufficient levels of protecting antibody to tetanus toxin.
DIAGNOSTIC CRITERIA

Clinical signs
- presents with difficulty in sucking and swallowing due to masseter spasm, i.e. trismus, usually on day three with associated hunger and crying
- temperature of 40–41°C
- tenseness and rigidity of all muscles, including paraspinal and abdominal muscles
- fists clenched and the toes fanned
- opisthotonic spasms and clonic jerks following sudden stimulation by touch and noise
  - spasms are painful
  - not true seizures
  - there is no loss of consciousness
  - laryngeal spasms may result in respiratory distress
- umbilicus may appear normal but there may be discharge from, or dirt/dung on umbilicus

complications include:

ACUTE
- aspiration pneumonia
- pulmonary haemorrhage
- respiratory failure
- CNS haemorrhage
- urinary retention
- rhabdomyolysis
- cardiac arrhythmias
- unstable blood pressure
- asystole
- starvation
- bleeding into muscles
- iatrogenic paralytic ileus

CHRONIC
- contractures
- myositis ossificans
- secondary neurologic sequelae due to hypoxic cerebral injury, including mental retardation, cerebral palsy etc

Special Investigations
- Gram stain of infected umbilical stump may reveal typical Gram positive bacilli
- anaerobic cultures are not necessary as attempts to culture C. tetani have a poor yield
- Gram stain of cerebrospinal fluid may be required to rule out meningitis

NON-DRUG TREATMENT
- majority of cases may require admission to neonatal ICU, full intermittent positive pressure ventilation, muscle relaxation and sedation
  - if not available, nurse in quiet, cool and dark environment
- if not intubated, suction the mouth and turn infant 30 minutes after each dose of sedative
- insert a nasogastric tube 30 minutes after sedative was given
  - start nasogastric tube feeds preferably after 24 hours of admission
  - give expressed breast milk in small feeds and augment with IV neonatal maintenance solution as required
- cut off umbilical stump if present and clean with solution of chlorhexidine and water 2 hours after tetanus immunoglobulin (TIG) was given
- physiotherapy is important to prevent muscle atrophy and contractures
  - limit unnecessary stimulation, i.e. sound, touch and therapeutic manipulation
- monitor and maintain body temperature
- cardiorespiratory monitoring is important due to involvement of respiratory muscles and sympathetic over activity, i.e. hypertension and tachycardia
• careful nursing attention to bladder and bowel function
  ○ bladder may successfully be emptied using Credé’s method
  ○ urine retention may occasionally require bladder catheterisation
  ○ constipation can be prevented by giving expressed breast milk
  ○ if necessary glycerine suppositories may be used, once muscle spasms become less frequent and always with prior treatment with sedatives and muscle relaxant (see drug treatment)
• place small balls of cotton wool in clenched fists and put splints on feet when muscle relaxants are given
  ○ remove them daily to check for pressure sores

**DRUG TREATMENT**
• tetanus immunoglobulin, IM, 500 units
  Give at 2 sites, as volume is too large for one site.
**PLUS**
• benzylpenicillin (Penicillin G), IV, 50 000 units/kg/dose, 12 hourly for 10 days
**PLUS**
• tetanus toxoid vaccine, IM, 0.5 mL into arm

For non-ventilated patient sedate with:
• chlorpromazine, oral, 1 mg/kg/dose, 8 hourly via nasogastric tube
**AND**
• phenobarbital, oral, 3 mg/kg once daily

For intubated and ventilated patients
• diazepam, IV, 0.1 mg/kg/dose, 2–4 hourly, as necessary to control spasms in first few days.
  Treatment is sustained for 2–4 weeks and frequency of administration is decreased as patient improves.
  Titrate dose according to response.
  Change to oral as intravenous preparation can cause thrombophlebitis.
**AND**
• pancuronium, IV, 0.1–0.2 mg/kg, 2-4 hourly as necessary
  Decrease frequency of administration as spasms become less frequent and less forceful, usually within 7–10 days.
**AND**
• morphine, IV, 0.05–0.1 mg slowly, every 4–6 hours
  Once infant has improved, replace with
  • paracetamol, oral, 10 mg/kg/dose 4-6 hourly

**Constipation**
• glycerine, rectal, ¼ suppository every 2nd day

**Aspiration pneumonia**
Treat as for nosocomial pneumonia – See chapter 12
Preventive management
Prevention of neonatal tetanus can be accomplished by prenatal immunisation of the previously unimmunized mother.
Pregnant women who have not completed their primary series should do so before delivery, if possible.

All pregnant women:
- First pregnancy – three doses:
  - first dose on first contact
  - second dose 4 weeks later
  - third dose 6 months later even if it is given in the post natal period (after birth)
- Subsequent pregnancy:
  - one dose during the antenatal period (up to a total of 5 recorded doses)

Active immunisation of the infant against tetanus should always be undertaken during convalescence, because the disease does not confer immunity.

REFERRAL
- all infants with complicated neonatal tetanus
- onset of neonatal tetanus within the first 3 days of life

19.20 PARENTERAL NUTRITION (PN)

DESCRIPTION
Parenteral nutrition is the administration of amino acids (proteins), lipids, carbohydrates, electrolytes, minerals, vitamins and trace elements via the intravenous route where enteral feeds are not tolerated or indicated.

Total parenteral nutrition is administered via a central venous line in infants intolerant to enteral feeds due to medical or surgical conditions, e.g. NEC and post intestinal surgery, that will require TPN for 14 days or longer.

Partial parenteral nutrition (peripheral) is administered via a peripheral vein to infants intolerant to enteral feeds due to medical, e.g. very low birth weight infant, or surgical conditions reversible within 14 days.

Parenteral nutrition should be prescribed and administered under the supervision of a paediatrician and dietician.

FORMULATIONS
- use standard TPN formulations that are ready to hang and gamma irradiated
  Do not decant contents or add to bag as stability has not been established and risk of contamination is increased.
- bags should be hung in an aseptic manner and used for 24 hours
- remove all outside bags so that the contents can be seen
- administer TPN through a dedicated line. Do not administer medication, blood, etc, through this line.
Commercially available fluids have the following average constituents per 100 mL:

<table>
<thead>
<tr>
<th>Constituents</th>
<th>Glucose–electrolyte solution with/without potassium</th>
<th>3-IN-1 TPN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogen (g)</td>
<td>0</td>
<td>0.33</td>
</tr>
<tr>
<td>Dextrose (g)</td>
<td>10</td>
<td>10.5</td>
</tr>
<tr>
<td>Lipid (g)</td>
<td>–</td>
<td>1.7</td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>40</td>
<td>60</td>
</tr>
<tr>
<td>Sodium (mmol)</td>
<td>2</td>
<td>2.2</td>
</tr>
<tr>
<td>Potassium (mmol)</td>
<td>1.5 or 0</td>
<td>1.75</td>
</tr>
<tr>
<td>Chloride (mmol)</td>
<td>2.0</td>
<td>4.3</td>
</tr>
<tr>
<td>Calcium (mmol)</td>
<td>0.25</td>
<td>1.16</td>
</tr>
<tr>
<td>Magnesium (mmol)</td>
<td>0.05</td>
<td>0.19</td>
</tr>
<tr>
<td>Phosphate (mmol)</td>
<td>0.37</td>
<td>0.87</td>
</tr>
</tbody>
</table>

- do not exceed 48 hours of only an electrolyte/glucose solution in babies without enteral feeding

**Upward Titrations of PN Volumes**

<table>
<thead>
<tr>
<th>Volume</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>mL/kg</td>
<td>55</td>
<td>84</td>
<td>111</td>
<td>139</td>
<td>167</td>
</tr>
<tr>
<td>mL/kg/hour</td>
<td>2.3</td>
<td>3.5</td>
<td>4.6</td>
<td>5.8</td>
<td>7.0</td>
</tr>
<tr>
<td>Protein g/kg</td>
<td>1.09</td>
<td>1.64</td>
<td>2.19</td>
<td>2.7</td>
<td>3.29</td>
</tr>
<tr>
<td>Lipid g/kg</td>
<td>1</td>
<td>1.5</td>
<td>2</td>
<td>2.5</td>
<td>3</td>
</tr>
<tr>
<td>Kcal/kg</td>
<td>38</td>
<td>57</td>
<td>75</td>
<td>93</td>
<td>112</td>
</tr>
<tr>
<td>mg/kg/min glucose</td>
<td>4</td>
<td>6</td>
<td>8</td>
<td>10</td>
<td>12.3</td>
</tr>
</tbody>
</table>

- duration of infusion: between 6 and 24 hours per day depending on condition of infant and volume to be administered.
- remainder of daily fluid requirements to be made up by neonatal maintenance solution
- tapering of parenteral nutrition should commence when the infant tolerates equal to or more than 75% of enteral feeds

**CAUTION**

Extravasation of TPN causes severe tissue damage and necrosis.
Do not infuse TPN into poorly running IV lines.
Monitoring
- vital signs and hydration
- infusion site and patency of the catheter: regularly, at least hourly
- blood glucose: at least 6 hourly
- electrolytes, minerals and acid base: on a daily basis
- growth parameters, especially weight: on a weekly basis
- infection markers: at least once weekly
- liver function, ammonia, renal function and lipids: once weekly or more frequently as indicated by the condition of the infant

Daily requirements
- proteins: up to 3 g/kg/day
- lipids: up to 2.5 g/kg/day
- glucose: 10–15 g/kg/day. Maintain blood glucose at 2.6–6 mmol/L
- average energy requirements: 120 kcal/kg/day
Some infants may be intolerant to the total daily requirements of the different nutrients and the nutrients may require slow upward titration.

Complications
- line complications, e.g. extravasation and blockage
- metabolic complications, e.g. hyperglycaemia, high ammonia, metabolic acidosis, electrolyte and mineral disturbances and hyperlipidaemia
- infections/sepsis
- cholestatic hepatitis

REFERRAL
- no progress with the introduction of enteral feeds
- recurrent/serious complications
20.1 PALLIATIVE CARE

DESCRIPTION
Palliative care for children and young people with end of life conditions is an active and total approach to care, embracing physical, emotional, social and spiritual elements. It focuses on enhancement of quality of life for the child and support for the family, including the management of distressing symptoms, provision of respite and care through death and bereavement.

The decision to palliate is made when further invasive or life-supporting measures are considered to be futile. It should be a team decision and should involve caregivers. Where appropriate the older child may be part of that decision.

Pain management in children is equally important in non-terminal care.

DIAGNOSTIC CRITERIA
Clinical features in terminally ill children
• pain is the most common and also the most feared of all symptoms
  Pain needs to be recognised and assessed before it can be managed appropriately. It is more challenging to assess pain in the non- or pre-verbal child. Assess other physical signs of pain.
  For children over 4 years useful pain scales exist, e.g. visual analogue scale/ faces pain scale.

Self-report of pain is the gold standard of pain assessment in older children. Parental report gives valuable information.
Children in acute pain react more vocally and demonstratively.
Children with chronic pain appear quiet, withdrawn, lack interest in activities or surroundings, may be reluctant to move, have clinging behaviour or have sleep disturbances.
• physiologic features of pain and anxiety include tachycardia, hypertension and sweating
• behavioural features are: crying, irritability, apathy, disinterest, depression and decreased activity level
• other important symptoms to address in the holistic management include:
  ○ GIT: anorexia, nausea and vomiting, constipation, dysphagia
  ○ pruritus
  ○ bleeding
  ○ dyspnoea
  ○ organ failure

**Pruritus**

Special investigations
• limited to those required to specifically guide palliative care

**NON-DRUG TREATMENT**
• discuss the management with the family, including the child as appropriate for development
• children should preferably be nursed at home/hospice where parents can be present at all times
• address all factors that may contribute to pain and associated symptoms, e.g. family stress, anxiety and sleep deprivation. Address parental anxiety.
• use therapies, e.g. massage, splints, music or play therapy and storybook reading, where appropriate
• give small regular feeds as required, preferably orally and not via naso-gastric tube

**DRUG TREATMENT**
Children receiving properly titrated doses of analgesics, including opioids, do not become addicted. There is a difference between tolerance, which is a need for escalating doses to achieve the same therapeutic effect, and addiction.

Most pain syndromes in children are responsive to timeous treatment. Utilise the least invasive route of medication administration, preferably orally. There is little place for intramuscular injections.

**Analgesics**
Analgesics must be administered regularly.
Give immediate release drugs 30 minute prior to pain inducing activity.
Plan ahead for exacerbations and crises, e.g. when wound care is done.
Monitor response to treatment and seek advice if pain is not quickly controlled.
THREE STEP ANALGESIC LADDER
Proceed up the ladder once any one step is ineffective.

<table>
<thead>
<tr>
<th>Mild pain</th>
<th>Moderate pain</th>
<th>Severe pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Step 1:</strong> non-opioid, e.g. paracetamol or ibuprofen where anti-inflammatory effect is required</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 2:</strong> weak opioid, e.g. codeine ± non opioid ± adjuvant drugs</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Step 3:</strong> strong opioid e.g. morphine ± non opioid ± adjuvant drugs</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Non-opioid drugs
- paracetamol, oral
  - Loading dose 20 mg/kg/dose, then 15 mg/kg/dose 4–6 hourly.
  - OR
    Where oral medication cannot be used
    paracetamol, rectal, 6 hourly
      - 3–12 months 60–125 mg
      - 1–5 years 125–250 mg
      - 6–12 years 250–500 mg

Non-steroidal anti-inflammatory drugs (NSAIDS)
Can be used in combination with paracetamol or opioids
- ibuprofen, oral, 4–10 mg/kg/dose 6–8 hourly

Opioid drugs
Increase doses of opioids according to the individual need. Take into account the development of tolerance.
The correct dose is that which relieves patient’s symptoms and may exceed the recommended dose used in other pain relief settings.
Assess child frequently.
Laxatives should be used prophylactically.
- lactulose, oral, 2.5–10 mL twice daily
- tilidine, oral, 1 mg/kg/dose, 4 hourly
  - 1 drop = 2.5 mg
  - Number of drops = body weight / 2.5
• codeine phosphate syrup, oral, 0.5 – 1 mg/kg/dose 4 hourly
  Syrup = 25 mg/5 mL.

• morphine, oral
  Short acting: for children over 6 months.
  Starting dose: 0.2–0.5 mg/kg/dose 4 – 6 hourly.
  Increase dose by 30–50 % every 12 hours if pain control is sub-optimal.
  Longer acting: where a practical dose is available and where stable total daily
  dose has been determined.
  Dose: half total daily dose of short acting morphine. Give 12 hourly.

  **Note:**
  Use in infants less than six months requires specialist supervision.
  If intravenous treatment is required, administer under specialist supervision.

Opioids and other analgesics should not be withdrawn in terminally ill patients unless a
specific indication for withdrawal is present. Replacement analgesia should be used in
such instances.

**Adjuvant therapy to analgesia**
Adjuvant agents can enhance pain control by targeting specific pain mechanisms
Steroids may be used as an adjuvant in:
• infiltration of bone/meninges
• compression of nerves and spinal cord
• visceromegaly,
• tumour invasion of organs
• stretching of periosteum
• elevated intracranial pressure

**Stretching of periosteum**
• prednisone, oral, 1–2 mg/kg/dose as single dose or in 2 divided doses

**Elevated intracranial pressure**
• betamethasone, oral, 0.5 mg/kg/dose once daily

Depending on the underlying condition, drug combinations to give analgesia, anxiolysis
and sedation may be much more effective.
Examples of medicine combinations:
• paracetamol + ibuprofen + lorazepam
• ibuprofen + carbamazepine + lorazepam
• morphine + promethazine + lorazepam

**REFERRAL**
• pain resistant to medical management in children who are possible candidates for
  invasive pain therapies.
  There is no place for invasive/life supporting measures in terminal patients. Discuss
  end of life events with parents to relieve their anxiety and avoid unnecessary referrals.
20.2 PAIN SYNDROMES

DESCRIPTION
• burning paresthesia
• neuropathic pain
• nerve root compression
• HIV neuropathy
• chemotherapeutic nerve injuries

DRUG TREATMENT
In addition to analgesia
• carbamazepine, oral, 5 mg/kg/dose twice daily
  Maximum dose: 100 mg/dose.

For nausea and vomiting
• cyclizine, oral, 1 mg/kg/dose 8 hourly
  OR
  metoclopramide, oral, 0.1 mg/kg/dose, 6–12 hourly
  OR
  metoclopramide, IV, 0.05 mg/kg/dose, 6–12 hourly
  Maximum dose: 2.5 mg/dose.

For pruritus
• hydroxyzine, oral, 0.5–1 mg/kg/dose, 8–12 hourly
  OR
  promethazine, IV/oral, 0.1 mg/kg/dose 6 hourly

For anxiety
• hydroxyzine, oral, 0.5–1 mg/kg/dose, 8–12 hourly
  OR
  lorazepam, oral, 0.1 mg/kg/dose once daily

For spasmodic abdominal pain
• hyoscine butylbromide, IV/oral, 0.5 mg/kg/dose 6–8 hourly

For lips and mouth care
• zinc and castor oil, topical, applied to lips every 2 hours
• sodium chloride solution, gargle, to rinse mouth
  5 g sodium chloride in 1 L of water
  OR
  thymol glycerine compound, gargle, to rinse mouth

For painful oral mucositis
• chohexidine/benzydamine, oral rinse, rinse or gargle 6–8 hourly

Candida
See Candidiasis, Systemic And Other: Section 8.4
For perineal mucositis/nappy rash
- zinc and castor oil, topical, applied as needed
  if pain is a feature: mix with 2% lidocaine hydrochloride gel.

For dyspnoea
- oxygen 2L/minute via nasal prongs
- morphine, oral, for children over 6 months
  Starting dose: 0.2–0.5 mg/kg/dose, every 4 hours.

### 20.2.1 TREATMENT OF PAIN IN CHILDREN

#### DESCRIPTION
Pain is a subjective unpleasant experience comprising sensory and emotional components. The inability of the child to communicate a painful experience has led to serious misconceptions, e.g. that they have higher tolerance, decreased perception or little/no memory of a painful experience.

#### DIAGNOSTIC CRITERIA
**Clinical features of pain:** See Section 20.1
- assessment of pain in neonates and infants is necessarily indirect. Facial expression is the most consistently valid indicator of pain.
- autonomic responses: increased blood pressure, heart rate, pulmonary vascular resistance, intracranial pressure, palmar sweat and decrease in oxygenation
- hormonal responses result in marked hyperglycaemia and prolonged state of catabolism

#### NON-DRUG TREATMENT
- where possible, allow a parent to room-in or stay with the child as long as possible
- make child comfortable, clean and dry nappy
- pacifier/dummy moistened with dextrose water 25% given during painful procedures may provide some comfort

#### DRUG TREATMENT

**FOLLOW THE THREE STEP ANALGESIC LADDER – See Section 20.1**

**Pain relief for painful procedures of short duration**
For some procedures both local anaesthetic and systemic treatment is necessary to relieve anxiety and pain e.g. insertion of arterial line, placement of central venous line/intercostal drainage tube, needle pricks, etc.
For some procedures, e.g. to remove an intercostal drain or deep wound drain or stitches, it is necessary to give sedation in combination with systemic pain treatment.

**For needle prick site**
- lidocaine/prilocaine cream, topical, applied 30 minutes before procedure
  Apply 1–1.5 cm length of cream over the needle puncture site. Spread cream thinly over 1 cm radius on skin and cover with polyurethane film dressing.
For lumbar puncture/insertion of intercostal drainage tube
• lidocaine 1%, SC
  First infiltrate the surrounding superficial skin and subcutaneous tissue with 1 mL of lidocaine 1% and then deeper tissue with additional 1 mL.
  In lumbar puncture preparation do not go deeper than the interspinous ligament. Allow sufficient time (2 minutes) for the anaesthetic to work before commencing with the procedure.

Drug combination options for short procedures
  For systemic analgesia the same drug combinations are used as for pain management with change of dressings depending on the severity of pain.
  Change of dressing medications: See Burns: Section 1.2.1

Short acting sedatives
The intravenous formulations of ketamine and midazolam can be given orally.
• midazolam, oral, 0.5 mg/kg/dose  (anxiolysis only)
• ketamine, oral, 2–5 mg/kg/dose at least 30 minutes before procedure

Before administering a sedative/anxiolytic drug
Withhold food for 4 hours before planned procedure.
Oxygen and resuscitation equipment should be available.
Put up an intravenous line with heparin lock should an unexpected complication arise.
Vital signs should be monitored during the procedure and until effects of sedative has worn off.

Withdrawal from opioids and midazolam
This must be done for any child who has had these drugs for more than 5–7 days. Wean by decreasing the daily dose by one third for three days.
### 21.1 Anaesthesia Drugs

#### Premedication
- midazolam
- promethazine
- tramadol

#### Induction of anaesthesia
- ketamine
- propofol
- thiopental sodium

#### Muscle relaxation
- alcuronium chloride
- pancuronium bromide
- suxamethonium chloride
- vecuronium bromide

#### Maintenance of anaesthesia
- fentanyl
- halothane
- isoflurane
- morphine

#### Reversal
- atropine
- neostigmine bromide

#### Regional anaesthesia
- bupivacaine 0.5% without adrenaline
- bupivacaine 0.5% with dextrose
- lidocaine 2%
- lidocaine 2% with dextrose
- lidocaine 5% with dextrose
- lidocaine topical

### 21.2 Emergency/Resuscitation Drugs

- calcium chloride
- phenylephrine
- doxapram

### 21.3 Miscellaneous Drugs to Manage Intra-Operative Emergencies/Other

- dantrolene
- ergometrine
- glyceryl trinitrate
- heparin sodium
- lanolin eye ointment, liquid
- oxytocin
- protamine sulfate
- verapamil

### 21.4 Peri- and Post Operative Analgesic Drugs

- diclofenac, rectal 25 mg
- fentanyl
21.5 INTENSIVE CARE DRUGS

- diclofenac, rectal 25 mg
- isosorbide dinitrate
- isosorbide mononitrate
- sodium thiosulphate
- sotalol
- tetracaine (amethocaine)
The disease reporting system in South Africa is based on government law (Health Act, Act 63 of 1977) and regulations where specific infectious diseases (see list of notifiable medical conditions below) must be reported to the Provincial Health Departments, who then report to the National Department of Health (see flow chart of data below). Disease surveillance comprises mainly four types: Notifiable disease-reporting system, Laboratory-based surveillance, Hospital-based surveillance and Population based surveillance.

**Notifiable Disease reporting**
A notification serves as the first step in a surveillance cycle, namely for data-capturing or data collection. Notification can be done via the mail, fax or telephone to the local authority concerned. Any person (not necessarily a health worker) can notify a notifiable medical condition (see the Health Act regulations - legal obligations). The list of notifiable medical conditions at the moment determines that 40 different diseases are notifiable (see list below).

**Process**

**Forms involved**
- GW17/5: initial diagnosis (complete immediately)
- GW17/3: line list of cases (complete weekly)
- GW17/4: line list of deaths (complete weekly)

The initial diagnosis of a notifiable medical condition are done on a case-based form with the relevant address and fine details on it, to make tracing of the case as easy as possible, since a disease notification demands action (follow-up) at the lowest level (GW17/5 - for cases and deaths).

In South Africa it is required by law that completed weekly disease notification forms are submitted for all notifiable diseases from each local authority or district office to the provincial office. These should be completed and sent by all reporting units e.g. hospitals, health centres, health posts, clinics, private practitioners, private nurses, to the district public health office. The initial diagnosis forms are summarised weekly on separate line list forms for cases (GW17/3) and for deaths (GW17/4).

**To ensure complete reporting of all EPI diseases, a zero report should be sent if no cases of a notifiable disease were seen for the reporting period.**

**Reporting**
- from reporting units to district office within 9 days
- reporting week is Sunday to Saturday

All the reporting units should submit their disease notifications to reach the district no later than 9 days after the end of the reporting week. A reporting week is normally taken from Sunday to Saturday. Thus, the weekly notifications are normally expected by the following Monday.

All reports received within that period are considered to be **on time.** After that period
has passed, any reports received is considered **late**. Some diseases can be monitored more accurately through the laboratory because of the nonspecificity of the clinical syndrome e.g. most types of food poisoning. For other diseases, laboratory data acts only as a confirmation of the clinical diagnosis. These include Rabies, Cholera and Crimean Congo Haemorrhagic fever

**Hospital-based surveillance**
Hospital discharge information as well as mortality data can be used to monitor disease trends and disease burden in a particular area served by the hospital.

**Population-based surveillance**
A population-based surveillance system collects and analyses medical information in a well-defined population.

Complete reporting is needed when doing surveillance on rarely occurring diseases as well as for the elimination of diseases (e.g. polio eradication in SA by 2000 - surveillance of Acute Flaccid Paralysis).
FLOW CHART

Procedure to follow with notifiable medical conditions

**Diagnosis**
can be any health worker, not necessarily a Doctor

↓
GW 17/5
immediately

↓

**Local authority / Hospital / District**
whoever is responsible for disease containment

↓
GW17/3 (cases)
GW 17/4 (deaths)
weekly

↓

**Regional office**
Health Information Unit
if data entry is done at regional level -
province specific

↓
computer disks
e-mail
weekly

↓

**Provincial office**
Health Information Unit
if data entry is done at provincial level -
province specific

↓
computer disks
e-mail
weekly

↓

**National Department**
Directorate HSR & Epidemiology
Private Bag X828, Pretoria 0001
Notifiable Medical Conditions

- Acute flaccid paralysis
- Anthrax
- Brucellosis
- Cholera
- Congenital syphilis
- Crimean-Congo haemorrhagic fever
- Other haemorrhagic fevers of Africa
- Diphtheria
- Food poisoning
- Haemophilus Influenza type B
- Lead poisoning
- Legionellosis
- Leprosy
- Malaria
- Measles
- Meningococcal infection
- Paratyphoid fever
- Plague
- Poisoning agricultural stock remedies
- Poliomyelitis
- Rabies
- Rheumatic fever
- Tetanus
- Tetanus neonatorum
- Trachoma
- Tuberculosis primary
- Tuberculosis pulmonary
- Tuberculosis of other respiratory organs
- Tuberculosis of meninges
- Tuberculosis of intestines, peritoneum
- Tuberculosis of bones and joints
- Tuberculosis of genito-urinary system
- Tuberculosis of other organs
- Tuberculosis miliary
- Tuberculosis total
- Typhoid fever
- Typhus fever (lice-borne)
- Typhus fever (rattle-borne)
- Viral hepatitis type A
- Viral hepatitis type B
- Viral hepatitis non-A non-B
- Viral hepatitis unspecified
- Viral hepatitis total
- Whooping cough
- Yellow fever
GUIDELINES FOR ADVERSE DRUG REACTION REPORTING

National Pharmacovigilance Programme
The Medicines Control Council (MCC) has a responsibility to ensure the safety, efficacy and quality of all medicines used by the South African public. The National Pharmacovigilance Programme is coordinated by the MCC and has two dedicated Units responsible for the monitoring of the safety of medicines. The National Adverse Drug Event Monitoring Centre (NADEMC) in Cape Town monitors the safety of all registered medicines in South Africa. In addition, a focused surveillance unit at MEDUNSA is responsible for monitoring the safety of anti-retroviral (ARV) medicines and complementary medicines. The unit at MEDUNSA is also responsible for monitoring the safety of unregistered medicines used during clinical trials.

What is Pharmacovigilance?
Pharmacovigilance is defined as the science and activities concerned with the detection, assessment, understanding and prevention of adverse reactions to medicines (i.e. adverse drug reactions or ADRs). The ultimate goal of this activity is to improve the safe and rational use of medicines, thereby improving patient care and public health.

What is an Adverse Drug Reaction (ADR)?
The Medicines Control Council (MCC) defines an Adverse Drug Reaction (ADR) or adverse reaction as a response to a medicine which is noxious and unintended, including lack of efficacy, and which occurs at any dosage and can also result from overdose, misuse or abuse of a medicine.

Who should report Adverse Drug Reactions?
All health care workers, including doctors, dentists, pharmacists, nurses and other health professionals are encouraged to report all suspected adverse reactions to medicines (including vaccines, X-ray contrast media, traditional and herbal remedies), especially when the reaction is not in the package insert, potentially serious or clinically significant.

What happens to a report?
All ADR reports are entered into a national ADR database. Each report is evaluated to assess the causal relationship between the event and the medicine. A well-completed adverse drug reaction/product quality form submitted could result in any of the following:

- Additional investigations into the use of the medicine in South Africa
- Educational initiatives to improve the safe use of the medicine
- Appropriate package insert changes to include the potential for the reaction
- Changes in the scheduling or manufacture of the medicine to make it safer

The purpose of ADR reporting is to reduce the risks associated with the use of medicines and to ultimately improve patient care.

Will reporting have any negative consequences on the health worker or the patient?
An adverse drug reaction report does not constitute an admission of liability or that the health professional contributed to the event in any way. The outcome of a report, together with any important or relevant information relating to the reaction, will be sent back to the
reporter as appropriate. The details of a report are stored in a confidential database. The names of the reporter or any other health professionals named on a report and the patient will be removed before any details about a specific adverse drug reaction are used or communicated to others. The information is only meant to improve the understanding of the medicines used in the country.

Is the event possibly an ADR?
The following factors should be considered when an adverse drug reaction is suspected:

1. What exactly is the nature of the reaction? (describe the reaction as clearly as possible and where possible provide an accurate diagnosis)

2. Did the reaction occur within a reasonable time relationship to starting treatment with the suspected medicine? (some reactions occur immediately after administration of a medicine while others take time to develop)

3. Is the reaction known to occur with the particular medicine as stated in the package insert or other reference? (If the reaction is not documented in the package insert, it does not mean that the reaction cannot occur with that particular medicine)

4. Did the patient recover when the suspected medicine was stopped? (some reactions can cause permanent damage, but most reactions are reversible if the medication is stopped)

5. Did the patient take the medicine again after the reaction abated (i.e. rechallenge). If so, did the same reaction occur again? (In most situations it is not possible or ethical to rechallenge the patient with the same medicine. If such information is available or if such a rechallenge is necessary, recurrence of the event it is a strong indicator that the medicine is may be responsible

6. Can this reaction be explained by other causes (e.g. underlying disease/s; other medicine/s; toxins or foods)? (It is essential that the patient is thoroughly investigated to decide what the actual cause of any new medical problem is. A medicine-related cause should be considered, when other causes do not explain the patient’s condition)

What types of reactions should be reported?
The following adverse drug reactions should be reported:

- All ADRs to newly marketed drugs or new drugs added to the EDL
- All serious reactions and interactions
- ADRs that are not clearly stated in the package insert.
- All adverse reactions or poisonings to traditional or herbal remedies

Report even if you are not certain the medicine caused the event.
What Product Quality Problems should be reported?
The following product quality problems should be reported:
• Suspected contamination
• Questionable stability
• Defective components
• Poor packaging or labeling
• Therapeutic failures

How can ADRs be prevented from occurring?
Some ADRs are unavoidable and cannot be prevented. However, most ADRs can be prevented by following the basic principles of rational use of medicines

How are adverse drug reactions reported?
An Adverse Drug Reaction/Product Quality Report Form is enclosed in this book and should be completed in as much detail as possible before returning it by fax or post to any of the addresses provided below. Additional forms can be obtained by contacting the MCC at these addresses. Report forms may also be accessed via the following website:
http://www.mccza.com

1. The Registrar of Medicines
Medicines Control Council, Department of Health, Private Bag X828
Pretoria, 0001
Tel: (021) 312 0295; Fax: (021) 3123106

2. The National Adverse Drug Event Monitoring Centre (NADEMC)
C/o Division of Pharmacology, University of Cape Town,
Observatory, 7925
Tel: (021) 447 1618; Fax: (021) 448 6181

3. MEDUNSA Pharmacovigilance Unit
Fax (012) 521 4335
ADVERSE DRUG REACTION AND PRODUCT QUALITY PROBLEM REPORT FORM

(Identity of reporter and patient will remain strictly confidential)

NATIONAL ADVERSE DRUG EVENT MONITORING CENTRE

Department of Health
Logo Here

In collaboration with the WHO International Drug Monitoring Programme

PATIENT INFORMATION

Name (or initials): .................................................. Age: ..........................................
Sex: M F
DOB: yyyy / yyyy / yyyy
Weight (kg): .............................................
Height (cm): .............................................

ADVERSE REACTION/PRODUCT QUALITY PROBLEM

Adverse reaction [ ] and/or Product Quality problem [ ] Date of onset of reaction: yyyy / yyyy / yyyy
Time of onset of reaction: hh:mm

Description of reaction or problem (Include relevant tests/lab data, including dates):

1. MEDICINES/VACCINES/DEVICES (include all concomitant medicines)

<table>
<thead>
<tr>
<th>Trade Name &amp; Batch No, (Asterisk Suspected Product)</th>
<th>Daily Dosage</th>
<th>Route</th>
<th>Date Started</th>
<th>Date Stopped</th>
<th>Reasons for use</th>
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ADVERSE REACTION OUTCOME (Check all that apply)

- death [ ]
- disability [ ]
- life-threatening [ ]
- hospitalisation [ ]
- congenital anomaly [ ]
- Other .........
- required intervention to prevent permanent impairment/damage [ ]
- event reappeared on rechallenge: [ ]
- sequela: [ ]
- recovered: Y N
- rechallenge not done: Y N
- treatment (of reaction):
- describe sequelae: .........................

COMMENTS: (e.g. Relevant history, Allergies, Previous exposure, Baseline test results/lab data)

2. PRODUCT QUALITY PROBLEM:

<table>
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<tr>
<th>Trade Name</th>
<th>Batch No</th>
<th>Registration No</th>
<th>Dosage form &amp; strength</th>
<th>Expiry Date</th>
<th>Size/Type of container</th>
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Product available for evaluation?: Y N

REPORTING DOCTOR/PHARMACIST Etc:

NAME: .......................................................... QUALIFICATIONS: ..................................
ADDRESS: ..........................................................
TEL: (.....) ..........................................

Signature: ............................................. Date: ..........................................

This report does not constitute an admission that medical personnel or the product caused or contributed to the event.
ADVICE ABOUT VOLUNTARY REPORTING

Report adverse experiences with:
- medications (drugs, vaccines and biologicals)
- medical devices (including in-vitro diagnostics)
- traditional and herbal remedies
- For Adverse Events Following Immunisation (AEFI), please follow the reporting procedure recommended by the Expanded Programme in Immunisation (EPI)

Please report:
- adverse drug reactions to recently marketed products
- serious reactions and interactions with all products
- adverse drug reactions which are not clearly reflected in the package insert.

Report even if:
- you're not certain the product caused the event
- you don't have all the details

Confidentiality: Identities of the reporter and patient will remain strictly confidential.

Important numbers:
- Investigational Products and Product Quality Problems:
  - (012) 326-4344 to fax a report
  - (012) 312-0000 to report by phone
- Registered Medicines and Traditional and Herbal remedies:
  - (021) 448-6181 to fax a report
  - (021) 447-1618 to report by phone
- Adverse Events Following Immunisation:
  - (012) 312 0110 to phone for information
  - (012) 321 9882 to fax a report

Your support of the Medicine Control Council’s adverse drug reaction monitoring programme is much appreciated. Information supplied by you will contribute to the improvement of drug safety and therapy in South Africa.

PLEASE USE ADDRESS PROVIDED BELOW- JUST FOLD IN THIRDS, TAPE and MAIL

BUSINESS REPLY SERVICE
BESIGHEIDSANTWOORDDIENS
Free Mail Number: BNT 178
Vryposnommer: BNT 178

DEPARTMENT OF HEALTH
DEPARTEMENT VAN GESONDHEID
REGISTRAR OF MEDICINES
REGISTRATEUR VAN MEDISYNE
PRIVATE BAG/PRIVAATSAK X828
PRETORIA
0001

Postage will be paid by Addressee
Postgeld sal deur die geadresseerde betaal word

No postage stamp necessary if posted in the Republic of South Africa
Geen posseel is nodig nie indien in die Republiek van Suid Afrika gepoch
GUIDELINE ON EDL REVIEW PROCESS AND SUBMISSION FOR AMENDMENTS

The National Essential Drugs selection process is based upon a well-developed network of provincial, district and institutional Pharmacy and Therapeutics committees.

Motivations for inclusion in the list will only be considered if:
- The prescribed form has been fully completed.
- The motivators’ contact details are complete.
- The drug name has been stated
- The submission has been evaluated and approved by the provincial Pharmacy and Therapeutics Committee (PTC).
- The indication has been clearly stated.
- All relevant comparator drug/s have been listed.
- There is sufficient evidence to support the proposed amendment.

Motivations may address major or minor amendments.

Major amendments include:
- new indications
- new therapeutic entities
- new therapeutic classes

All major amendments must be supported by evidence reflecting safety, efficacy and cost of the medicine compared to an already listed drug for the same indication.

A major amendment may also include motivations for drugs not listed and for conditions not addressed in the EDL. In such cases submissions must be supported by demographic data.

Minor amendments include:
- new formulations
- combination therapies of existing essential drugs

For minor amendments the supporting evidence should be relevant to the nature of amendment.

Screening
Motivations are screened by the Rational Selection Group (RSG) at the National Department of Health to ensure that:
- the submission has been approved by the provincial PTC
- the motivators’ contact details are included
- the drug can be identified in terms of the INN
- an indication has been included
- relevant comparator drug/s have been identified with their corresponding dosing regimens
- there are supporting references to substantiate the request

RSG will compile a review of the prevailing cost of therapy.
GUIDELINE ON EDL REVIEW PROCESS AND SUBMISSION FOR AMENDMENTS

Submissions that have been accepted by RSG are tabled at the relevant technical subcommittee for allocation to a suitably qualified reviewer who compiles a technical report. This technical report summarizes a review of the submitted data in terms of the following:

- relative safety
- relative efficacy
- practice environment - the focus here being efficacy relative to current EDL drugs
- pharmacoeconomic evaluation

The report is then presented to the technical subcommittee. The committee may request further information from the applicant through the province or commission a literature search and review.

The technical subcommittee will make recommendations to the National Essential Drug List Committee (NEDLC) for approval or rejection. Where the NEDLC is of the opinion that further review is required the decision will be sent back to the technical subcommittee for further review.

THE DATA ELEMENTS OF THE SUBMISSION FORM

The motivation form is divided into 5 sections. (Annexure 1)

Section 1: Proposal

The proposal consists of:

a) The International Nonproprietary Name (INN) of the medicine – this identifies a pharmaceutical substance or active pharmaceutical ingredient by a unique name that is globally recognized and is public property. A nonproprietary name is also known as a generic name.

b) Level of Care - indicate whether the proposed medicine should be listed for use at primary care (PHC) or hospital level (Note drugs at PHC level are automatically included at the hospital level).

c) Prescriber level - indicate the level of competency required to prescribe the drug.

Section 2: Motivators’ Details

The NEDLC will acknowledge all submissions and communicate decisions with supporting arguments where appropriate. This section therefore forms a vital link between the motivator and the decision making process.

Section 3: Proposed Indications

a) Indication

Points to consider:

- The EDL targets those conditions that are the most prevalent in South Africa. Where the motivator suggests an indication not currently reflected in the EDL, a brief motivation based upon South African epidemiological data must be included as an annexure.

- The indication allows for the identification of the appropriate comparator in the current EDL.

- Many drugs have multiple indications. However, not all are equally cost effective.
b) Proposed Regimen
This data will be used for cost comparison and is very important for pharmacoeconomic evaluation.

c) Cost assessment
The information is necessary for the determination of affordability. It is expected that the provincial PTC will deliberate about the affordability during their review prior to submission to NEDLC. For this reason, this data is considered mandatory at the national level.

Section 4: Drugs on the current EDL for the same indication

As a principle, the addition of an EDL item should replace an existing item. This is of particular importance when safety and economic implications are taken into account.

Evidence
Evidence is a vital component of the submission and review process. Evidence does not constitute a drug decision and merely informs the strength of the argument. It forms the basis upon which the decision is made and allows for transparent scrutiny of the decision as well as facilitating the review.

Evidence is required in support of:
- relative efficacy
- relative safety
- pharmacoeconomic benefits

Note:
Evidence needs to be relevant to the South African context. Multinational or foreign studies must be supported by a motivation of the relevance of both the outcome measures as well as socio-economic facets to the South African context.

The inclusion of at least one relevant reference is mandatory. A copy of the full journal article should be included in order to expedite the review process.

Section 5: For use at national level only

This section is intended to ensure that the submissions have followed the proper process.
Motivation Form for the Inclusion of a Drug on the National Essential Drugs List

Please complete Sections 1 to 4 in full

**SECTION 1**

**NB - Only use INN (International Nonproprietary Name/Generic names) on this form**

**Proposed Drug**

For Inclusion on the Essential Drug List for  
PHC Hospital  
Check all appropriate blocks

Prescriber Level  
Primary Health Care - 1  
Medical Officer -2  
Specialist -3  
Designated Specialist - 4

Submission Date  
PTC Title

**SECTION 3**

**Proposed Indication**

See reverse side for the level of evidence schedule

<table>
<thead>
<tr>
<th>Indication</th>
<th>Proposed Regimen</th>
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<tr>
<td></td>
<td>Dose</td>
</tr>
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<td>1</td>
<td>2</td>
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</table>

**Level Of Evidence**

1a Meta-analysis  
Ib Randomized Controlled Trial  
II Controlled study with no randomization  
III Comparative, correlation or case control study  
IV Expert committee  
V Clinical experience

**SECTION 4**

**Drugs on the Current EDL for the Same Indication**

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**SECTION 5**

FOR NATIONAL USE ONLY

Correspondence  
Date received  
Acknowledged  
No of articles submitted:

For National Evaluation  
Yes No Further evidence  
Motivation New Drug

Decision  
Accepted Rejected
## Motivation Form for the Inclusion of a Drug on the National Essential Drugs List

### Title

### Name

### Tel No

### Code Number

### Fax No

### Code Number

### Postal Address

### Proposed Drug

For Inclusion on the Essential Drug List for

### PHC Hospital

Check all appropriate boxes:

- 1 Medical Officer
- 2 Specialist
- 3 Designated Specialist

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### Indication

### Proposed Regimen

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### Evidence

**NB** The literature review on the reverse side must support this.

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### Current regimen

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### New Drug

### Standard Therapeutic Guideline

### New/Change

### Prescriber level

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### Level of Evidence

- Ia Meta-analysis
- Ib Randomized Controlled Trial
- II Controlled study with no randomization
- III Comparative, correlation or case control study
- IV Expert committee
- V Clinical experience

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###SECTION 2

Motivator’s Details

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### SECTION 3

Proposed Indication

### SECTION 4

Cost Assessment

### SECTION 5

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### No of articles submitted:

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### SECTION 2

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### SECTION 2

Level of Evidence

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### SECTION 2

Proposed Drug

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### SECTION 2

Can Be Replaced by Proposed Drug

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### SECTION 2

New Drug

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### SECTION 2

Standard Therapeutic Guideline

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### SECTION 2

New/Change

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### SECTION 2

Prescriber level

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### SECTION 2

For National Use Only

---

### SECTION 2

Date received / Acknowledged / Request for more evidence / Evidence

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### SECTION 2

Yes / No

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### SECTION 2

Motivation

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### SECTION 2

New Drug

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### SECTION 2

Standard Therapeutic Guideline

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### SECTION 2

New/Change

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### SECTION 2

Prescriber level

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### SECTION 2

For National Use Only

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### SECTION 2

Date received / Acknowledged / Request for more evidence / Evidence

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### SECTION 2

Yes / No

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### SECTION 2

Motivation

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### SECTION 2

New Drug

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### SECTION 2

Standard Therapeutic Guideline

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### SECTION 2

New/Change

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### SECTION 2

Prescriber level
**Levels of Evidence**  

Ia Meta-analysis  

Ib Randomized Controlled Trial  

II Controlled study with no randomization.  

III Comparative correlation or case study  

IV Expert committee  

V Clinical experience

**Evidences (articles or abstracts) included with your submission**

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**Comments**  

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<tr>
<td>maintenance, neonatal, potassium free solution</td>
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<td>omeprazole</td>
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<td>oxybutinin</td>
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<td>ABCD</td>
<td>Airways, Breathing, Circulation, Drip/Doctor/Drugs</td>
</tr>
<tr>
<td>ABO</td>
<td>blood group system A, AB, B and O</td>
</tr>
<tr>
<td>ACE</td>
<td>angiotensin-converting enzyme</td>
</tr>
<tr>
<td>ADH</td>
<td>antidiuretic hormone</td>
</tr>
<tr>
<td>ADR</td>
<td>adverse drug reaction</td>
</tr>
<tr>
<td>ACEI</td>
<td>angiotensin-converting enzyme inhibitor</td>
</tr>
<tr>
<td>AIDS</td>
<td>acquired immunodeficiency syndrome</td>
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<tr>
<td>ALP</td>
<td>alkaline phosphatase</td>
</tr>
<tr>
<td>ALT</td>
<td>alanine amino transferase</td>
</tr>
<tr>
<td>ARDS</td>
<td>acute respiratory disease syndrome</td>
</tr>
<tr>
<td>ART</td>
<td>antiretroviral therapy</td>
</tr>
<tr>
<td>ARV</td>
<td>anti-retroviral</td>
</tr>
<tr>
<td>ASO</td>
<td>arteriosclerosis obliterans</td>
</tr>
<tr>
<td>ASOT</td>
<td>antistreptolysin-O test</td>
</tr>
<tr>
<td>AST</td>
<td>aspartate amino transferase</td>
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<tr>
<td>AV</td>
<td>atrioventricular</td>
</tr>
<tr>
<td>BCG</td>
<td>Bacillus Calmette-Guerin vaccine</td>
</tr>
<tr>
<td>BE</td>
<td>base excess</td>
</tr>
<tr>
<td>BMI</td>
<td>body mass index</td>
</tr>
<tr>
<td>BP</td>
<td>blood pressure</td>
</tr>
<tr>
<td>BSA</td>
<td>body surface area</td>
</tr>
<tr>
<td>ºC</td>
<td>degree Celsius</td>
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<tr>
<td>C\textsubscript{3}</td>
<td>third component of compliment</td>
</tr>
<tr>
<td>C\textsubscript{4}</td>
<td>fourth component of compliment</td>
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<tr>
<td>CCB</td>
<td>calcium-channel blockers</td>
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<tr>
<td>CD4</td>
<td>cluster designation 4</td>
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<tr>
<td>Cl</td>
<td>chloride</td>
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<tr>
<td>CMV</td>
<td>cytomegalovirus</td>
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<tr>
<td>CNS</td>
<td>central nervous system</td>
</tr>
<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
</tr>
<tr>
<td>CRP</td>
<td>C-reactive protein</td>
</tr>
<tr>
<td>CSF</td>
<td>cerebro-spinal fluid</td>
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<tr>
<td>CT</td>
<td>computed tomography</td>
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<tr>
<td>CuSO\textsubscript{4}</td>
<td>copper sulphate</td>
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<td>CVP</td>
<td>central venous pressure</td>
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<tr>
<td>DC</td>
<td>direct current</td>
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<tr>
<td>DIC</td>
<td>diffuse intravascular coagulation</td>
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<tr>
<td>DKA</td>
<td>diabetic ketoacidosis</td>
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<tr>
<td>dL</td>
<td>decilitre</td>
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<tr>
<td>DNA</td>
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<td>deoxyribonuclease</td>
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<td>DOTS</td>
<td>directly observed therapy short-term</td>
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<td>DPT</td>
<td>diphtheria, pertussis and tetanus vaccine</td>
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<tr>
<td>DsDNA</td>
<td>double-stranded DNA</td>
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<tr>
<td>DT</td>
<td>diphtheria and tetanus vaccine</td>
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<td>E</td>
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<tr>
<td>F</td>
<td>female</td>
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<tr>
<td>ECG</td>
<td>electrocardiogram</td>
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<tr>
<td>ECHO</td>
<td>echo cardiogram</td>
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<tr>
<td>EEG</td>
<td>electroencephalogram</td>
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<tr>
<td>ELISA</td>
<td>enzyme-linked immunosorbent assay</td>
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<tr>
<td>EPI</td>
<td>Expanded Programme on Immunisation</td>
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<tr>
<td>ESR</td>
<td>erythrocyte sedimentation rate</td>
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<tr>
<td>ET</td>
<td>endotracheal tube</td>
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<tr>
<td>ETT</td>
<td>endo-tracheal tube</td>
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<tr>
<td>$F_{O_2}$</td>
<td>inspiratory oxygen concentration</td>
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<tr>
<td>FBC</td>
<td>full blood count</td>
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<tr>
<td>FeNa</td>
<td>fractional excretion of sodium</td>
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<tr>
<td>FEV$_1$</td>
<td>forced expiratory volume in 1 second</td>
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<tr>
<td>FH</td>
<td>familial hypercholesterolaemia</td>
</tr>
<tr>
<td>FNA</td>
<td>fine needle aspiration</td>
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<tr>
<td>g</td>
<td>gram</td>
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<tr>
<td>GCS</td>
<td>Glasgow coma scale</td>
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<tr>
<td>GFR</td>
<td>glomerular filtration rate</td>
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<tr>
<td>GIT</td>
<td>gastro intestinal tract</td>
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<tr>
<td>G6PD</td>
<td>glucose-6-phosphate dehydrogenase</td>
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<tr>
<td>H</td>
<td>isoniazid</td>
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<td>HAART</td>
<td>highly active antiretroviral therapy</td>
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<tr>
<td>HACEK</td>
<td><em>Haemophilus actinobacillus cardiobacterium eikenella and kingella</em></td>
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<tr>
<td>Hb</td>
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<tr>
<td>HbA1c</td>
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<td>HCO$_3^-$</td>
<td>bicarbonate</td>
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<td>Hct</td>
<td>hematocrit</td>
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<tr>
<td>HIV</td>
<td>human immunodeficiency virus</td>
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<tr>
<td>HR</td>
<td>heart rate</td>
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<td>ICP</td>
<td>intracranial pressure</td>
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<tr>
<td>ICU</td>
<td>intensive care unit</td>
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<tr>
<td>IE</td>
<td>infective endocarditis</td>
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<tr>
<td>IgA</td>
<td>immunoglobulin A</td>
</tr>
<tr>
<td>IgG</td>
<td>immunoglobulin Gamma</td>
</tr>
<tr>
<td>IgM</td>
<td>immunoglobulin M antibodies</td>
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<tr>
<td>IM</td>
<td>intramuscular</td>
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<tr>
<td>IMT</td>
<td>intima-media thickness</td>
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<td>INH</td>
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<td>INR</td>
<td>international normalised ratio</td>
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<td>IO</td>
<td>intra ossius</td>
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<td>ITP</td>
<td>idiopathic thrombocytopenic purpura</td>
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<td>international units</td>
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<td>IV</td>
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<tr>
<td>JVP</td>
<td>jugular venous pressure</td>
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<td>K</td>
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<tr>
<td>kcal</td>
<td>kilocalorie</td>
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<tr>
<td>kg</td>
<td>kilogram</td>
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<td>kilojoule</td>
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<td>Description</td>
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<tr>
<td>kPa</td>
<td>kiloPascal</td>
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<tr>
<td>L</td>
<td>Litre</td>
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<tr>
<td>LDL</td>
<td>low density lipoprotein</td>
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<tr>
<td>LFT</td>
<td>liver function test</td>
</tr>
<tr>
<td>LN</td>
<td>lymph node</td>
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<tr>
<td>LOC</td>
<td>level of consciousness</td>
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<tr>
<td>Lp(a)</td>
<td>lipoprotein a</td>
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<tr>
<td>LTB</td>
<td>laryngotracheobronchitis</td>
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<tr>
<td>M</td>
<td>male</td>
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<td>m²</td>
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<tr>
<td>mcg</td>
<td>microgram</td>
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<tr>
<td>mcmol</td>
<td>micromol</td>
</tr>
<tr>
<td>MCS</td>
<td>micro culture and sensitivity</td>
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<td>MCNS</td>
<td>minimal change nephritic syndrome</td>
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<tr>
<td>MCUG</td>
<td>micturating cysto-urethrogram</td>
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<tr>
<td>MDR TB</td>
<td>multiple drug resistant tuberculosis</td>
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<tr>
<td>mEq</td>
<td>milliequivalent</td>
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<tr>
<td>mg</td>
<td>milligram</td>
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<tr>
<td>MgSO₄</td>
<td>magnesium sulphate</td>
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<td>mL</td>
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<td>mmol</td>
<td>millimol</td>
</tr>
<tr>
<td>mOsm</td>
<td>milliosmole</td>
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<td>MRI</td>
<td>magnetic resonance imaging</td>
</tr>
<tr>
<td>MRSA</td>
<td>methicillin resistant <em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>MU</td>
<td>million units</td>
</tr>
<tr>
<td>Na</td>
<td>sodium</td>
</tr>
<tr>
<td>NaCl</td>
<td>sodium chloride</td>
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<tr>
<td>NSAID</td>
<td>non-steroidal anti-inflammatory</td>
</tr>
<tr>
<td>ORS</td>
<td>oral rehydration solution</td>
</tr>
<tr>
<td>ORT</td>
<td>oral rehydration therapy</td>
</tr>
<tr>
<td>PaO₂</td>
<td>Partial pressure of oxygen in arterial blood</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>partial pressure of carbon dioxide in arterial blood</td>
</tr>
<tr>
<td>PCO₂</td>
<td>partial pressure of carbon dioxide</td>
</tr>
<tr>
<td>PCP</td>
<td>Pneumocystis carinii pneumonia</td>
</tr>
<tr>
<td>PCR</td>
<td>polymerase chain reaction</td>
</tr>
<tr>
<td>PDA</td>
<td>patent ductus arteriosus</td>
</tr>
<tr>
<td>PEFR</td>
<td>peak expiratory flow rate</td>
</tr>
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<td>PEP</td>
<td>post exposure prophylaxis</td>
</tr>
<tr>
<td>pH</td>
<td>acidity ((partial pressure of hydrogen)</td>
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<tr>
<td>PPD</td>
<td>purified protein derivative</td>
</tr>
<tr>
<td>PT</td>
<td>prothrombin time</td>
</tr>
<tr>
<td>PTT</td>
<td>partial prothrombin time</td>
</tr>
<tr>
<td>PTTT</td>
<td>partial thromboplastin time</td>
</tr>
<tr>
<td>PUVA</td>
<td>psoralen plus ultraviolet A (therapy)</td>
</tr>
<tr>
<td>PZA</td>
<td>pyrazinamide</td>
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</tbody>
</table>
ABBREVIATIONS

R rifampicin
RBC red blood cell
RDW red cell distribution width
Rh Rhesus
RH rifampicin, isoniazid combination
RHZ rifampicin, isoniazid pyrazinamide combination
RHZE rifampicin, isoniazid pyrazinamide ethambutol combination
RIG human anti- rabies immunoglobin
RPR/VDRL rapid plasma reagent test/venereal disease research laboratory test
SaO2 arterial oxygen concentration
SC subcutaneous
Se-K serum potassium
SIADH secretion of inappropriate antidiuretic hormone
SLE systemic lupus erythematosus
SSS sugar and salt solution
STI sexually transmitted infections
STS serological testing for syphilis
TB tuberculosis
T3 triiodothyronine
T4 thyroxine
TSH thyroid-stimulating hormone
TT tetanus vaccine
TV television
UTI urinary tract infection
UVB ultraviolet B
VL viral load
VLDL very low density lipoproteins
VSD ventricular septal defect
WCC white cell count
WHO World Health Organisation
Z pyrazinamide
ZnSO4 zinc sulphate