POCKET BOOK OF HOSPITAL CARE FOR NEONATES AND INFANTS

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The authors welcome any suggestions for the next edition and also any corrections. These will be addressed in future printed and on-line versions.

This pocketbook is a summary of the emergency components of basic neonatal and older infants hospital care from our textbook "International Maternal & Childhealth Care. A practical manual for hospitals worldwide". The reader is referred to the textbook when more details on the medical problem under consideration are required.

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Training

Members of the clinical team must know their roles. They will ideally have trained together in:

- clinical situations and their diagnoses and treatments
- drugs and their use, administration and side effects
- emergency equipment and how it functions.

The ability of a facility to deal with emergencies should be assessed and reinforced by the frequent practice of emergency drills.

Initial management

1. Stay calm.
2. Do not leave the infant unattended.
3. Have a team leader in charge to avoid confusion.
4. Shout for help. Ask one person to go for help and another to get emergency equipment and supplies (e.g. oxygen cylinder and emergency kit). Ideally resuscitation equipment and drugs should be available on one dedicated trolley.
5. Assess and resuscitate in sequence using the structured approach – Airway, Breathing, Circulation, Disability (Neurological Status) (see below).
6. Constantly reassess the patient, particularly after any intervention.

Structured approach to any infant presenting as an emergency

Approach emergencies using the structured ABCD (Airway, Breathing, Circulation, Disability) approach, which ensures that all patients with a life-threatening or potentially life-threatening problem are identified and managed in an effective and efficient way whatever their diagnosis or pathology.

The structured approach is outlined here, allows the health worker to focus on the appropriate level of diagnosis and treatment during the first hours of care. Primary assessment and resuscitation are concerned with the maintenance of vital functions and the administration of life-saving treatments, whereas secondary assessment and emergency treatment allow more specific urgent therapies to be started.

Secondary assessment and emergency care require a system-by-system approach in order to minimise the risk of significant conditions being missed.

Following cardiac and/or respiratory arrest, the outcome is poor. Earlier recognition and management of potential respiratory, circulatory or central neurological failure which may progress rapidly to cardiac and/or respiratory arrest will reduce mortality and secondary morbidity. The following section outlines the physical signs that should be used

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for the rapid primary assessment, resuscitation, secondary assessment and emergency treatment of newborn and older infants..

Primary assessment and resuscitation involves sequential assessment and resuscitation of vital functions.

Airway, Breathing and Circulation.
If there are no life-threatening signs, the primary assessment can be completed within about 1 minute. If life-threatening signs are identified, resuscitation procedures are immediately required.

If you are working on your own and have been unable to summon help, you must resuscitate Airway before Breathing, and Breathing before Circulation. This is because oxygen cannot be carried around in the blood to the vital organs if the blood is not oxygenated first, and the lungs cannot oxygenate the blood if there is no airway to allow air containing oxygen to enter the lungs.

If assistance is available, one person can deal with Airway, another with Breathing and a third with Circulation, all working simultaneously, but there must be a 'team leader' to take overall control.

During resuscitation, interventions that are either life-saving or designed to prevent the patient reaching a near-death situation are performed (see below). These include such procedures as basic airway opening procedures, suction, oropharyngeal airway insertion, intubation, assisted ventilation, venous cannulation and fluid resuscitation (when safe and appropriate). At the same time, oxygen is provided to all patients with life-threatening Airway, Breathing or Circulatory problems, vital signs are recorded, and essential monitoring is established.

This sequential primary assessment and any necessary resuscitation occur before any illness-specific diagnostic assessment or treatment takes place. Once the patient's vital functions are working safely, secondary assessment and emergency treatment can begin.

After each intervention, its effects should be tested by reassessment. Regular reassessments are a key component of the structured approach.

During secondary assessment, illness-specific pathophysiology is sought and emergency treatments are instituted. Before embarking on this phase, it is important that the resuscitative measures are fully under way. During the secondary assessment, vital signs should be checked frequently to detect any change in the patient's condition. If there is deterioration, primary assessment and

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resuscitation should be repeated in the in the 'Airway, Breathing, Circulation' sequence.

Primary assessment and resuscitation

Assessment and resuscitation occur at the same time. The order of assessment and resuscitation enables identification of immediately life-threatening problems, which are treated as they are found.

A rapid examination of vital ABC functions is required. If at any stage a life-threatening A, B, or C problem is identified:
 CALL FOR HELP.
After ABC, always assess for neurological problems, and resuscitate their components (sometimes referred to as 'D' for disability of the ABC approach).

Primary assessment and resuscitation of airway

The first priority is establishment or maintenance of airway opening. If there is a need for resuscitation in a patient who is bleeding (e.g. in cases of massive postpartum haemorrhage or trauma), try to stop this at the same time as you are opening the airway.

> **PRIMARY ASSESSMENT**
> LOOK – for chest or abdominal movement. LISTEN –
> for breath sounds.
> FEEL – for breath.
> An Infant who can cry has a clear airway.

Signs associated with airway obstruction may include any of the following:
- an absence of breathing
- stridor, snoring, or gurgling in the throat
- cyanosis
- chest wall recession
- agitation, reduced consciousness, or coma.

Airway obstruction is most commonly due to obstruction by the tongue in an unconscious patient.

Resuscitation

Open the airway and keep it open.

If there is no evidence of air movement, **open the airway** using the following:

– a head tilt, chin lift or jaw thrust manoeuvre (see Section 2 on basic life support). If this opens the airway and breathing starts, keep the airway open manually until it can be secured.
– suction/removal of blood, vomit or a foreign body.

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If there is no improvement after adjusting the airway manually and trying different techniques, place an oropharyngeal airway, which may be helpful if the patient is unconscious and has no gag reflex. Avoid using a nasopharyngeal airway if there is any suspicion of base of skull injury.

If the airway is still obstructed, a definitive airway by intubation or surgical airway may be needed.

Give oxygen to all patients.

Identify the 'at-risk' airway

Reassess the airway after any airway-opening manoeuvres. If there continues to be no evidence of air movement, then airway opening can be assessed by performing an airway-opening manoeuvre while giving rescue breaths. Proceed to Breathing (see below).

Advanced airway management

Advanced airway management techniques for securing the airway by intubation may be required in patients with any of the following:

- persistent airway obstruction
- altered level of consciousness, with failure to protect the airway, especially from vomiting
- facial trauma, including burns, penetrating neck trauma with expanding haematoma, and severe head injury

This should be performed by a skilled professional such as an anaesthetist (if available). The following sequence should be followed:

1. pre-oxygenation with 100% oxygen with manual lung inflation if required
2. administration of a carefully judged, reduced dose of an anaesthetic induction agent
3. application of cricoid pressure
4. suxamethonium 1–2 mg/kg
5. intubation with a correctly sized tracheal tube.

Confirmation of correct placement of the tube

Signs such as chest movement and auscultation remain helpful, but are occasionally misleading, especially in inexperienced hands. The most important point is to see the tube pass through the vocal cords. The correct size is a tube that can be placed easily through the cords with only a small leak. Intubation of the right main bronchus is best avoided by carefully placing the tube only 2–3 cm below the cords and noting the length at the teeth before checking by auscultation (best in the left and right lower axillae). Capnography, if available, is a useful adjunct for helping to confirm correct tube placement.

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If it is not possible to provide an airway using intubation, a surgical airway may be required.

NOTE: It is extremely risky to proceed to Circulation (and IV/IO cannulation) when partial upper airway obstruction is present as invasive procedures can precipitate complete airway closure. Stabilise the airway first. This will require help from an anaesthetist.

Emergency treatment situations

If the infant has severe bronchiolitis, clear the nasal airways by using gentle suction.

Primary assessment and resuscitation of breathing

An open airway does not guarantee adequate ventilation. The latter requires an intact respiratory centre and adequate pulmonary function augmented by coordinated movement of the diaphragm and chest wall.

Primary assessment

Assess whether breathing is adequate by:

assessing effort:
- recession
- rate
- added noises
- accessory muscles
- alar flaring

assessing efficacy:
- listening for reduced or absent breath sounds, or any wheezing, with a stethoscope or ear on chest wall
- chest and/or abdominal expansion (symmetrical or asymmetrical)
- abdominal excursion
- SaO_2 if available

assessing effects on heart rate
assessing effects on skin colour (check for cyanosis)
assessing effects on mental status.

Evidence of life-threatening respiratory difficulty

This includes the following:

1. absence of breathing (apnoea)
2. very high or very low respiratory rates
3. gasping, which is a sign of severe hypoxaemia, and may indicate impending respiratory arrest and death
4. severe chest wall recession, usually with increased respiratory rate, but pre-terminally with a fall in rate
5. severe hypoxaemia (cyanosis)
6. signs of tension pneumothorax (respiratory distress with hyper-resonant percussion)
7. major trauma to the chest (e.g. tension pneumothorax, haemothorax, flail chest) (see Textbook)

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Evidence of respiratory difficulty which can progress if not treated

This includes the following:

1 increased respiratory rate
2 inspiratory stridor
3 reduced or absent breath sounds on auscultation
4 expiratory wheezing
5 chest expansion (most important), and reduced abdominal excursion
6 pulse oximetry showing oxygen saturation (SaO_2) of less than 94% (normal SaO_2 in a patient at sea level is 94–100% in air).

Fast breathing can be caused by either an airway problem, lung disease or metabolic acidosis.

Care should be taken when interpreting single measurements. Infants can show rates of between 30 and 90 breaths/minute depending on their state of activity. It is more useful to use trends in measurements as an indicator of improvement or deterioration.

WHO definitions of fast breathing in infancy

< 2 months	≥ 60 breaths/minute
2-11 momths	≥ 50 breaths/minute

Slow breathing rates may result from fatigue or raised intracranial pressure, or may immediately precede a respiratory arrest due to severe hypoxaemia.

Other signs of breathing difficulty

Chest wall recession

- Intercostal, subcostal or sternal recession reflects increased effort of breathing, which is seen in particular in infants, who have more compliant chest walls.
- The degree of recession indicates the severity of respiratory difficulty.
- In the patient with exhaustion, chest movement and recession will decrease.

Inspiratory or expiratory noises

- Stridor, usually inspiratory, indicates laryngeal or tracheal obstruction.
- Wheeze, predominantly expiratory, indicates lower airway obstruction.
- Volume of noise is not an indicator of severity.

Grunting

- This is observed in infants with stiff lungs in an attempt to prevent airway

collapse (it represents the noise made by closure of the larynx during expiration).

- It is a sign of severe respiratory distress.

Accessory muscle use

- In infants, the use of the sternocleidomastoid muscle creates 'head bobbing' and does not help ventilation.
- Flaring of the alae nasi is also seen in infants with respiratory distress.

Exceptions

Increased effort of breathing does not occur in three circumstances:

1 exhaustion
2 central respiratory depression (e.g. from raised intracranial pressure, poisoning or encephalopathy)
3 neuromuscular disease.

Effects of breathing failure on other physiology

Heart rate: this is increased with hypoxia, but decreases when hypoxia is severe, when bradycardia is a sign of impending cardiorespiratory arrest.

Skin colour: hypoxia first causes vasoconstriction and pallor. Cyanosis is a late sign and may indicate impending cardiorespiratory arrest. In an anaemic patient it may never be seen, however hypoxic the patient is.

Mental status: hypoxia causes initial agitation, then drowsiness, followed by loss of consciousness.

Resuscitation of breathing

In the patient with absent or inadequate breathing, it is essential to breathe for the patient using:

- mouth-to-mouth or mouth-to-mouth-and-nose ventilation, or
- bag-valve-mask ventilation: if using oxygen, add a reservoir to increase the oxygen concentration.

Intubate (if skilled professionals are available) and provide assisted ventilation through the tube if long-term ventilation is needed or bag–mask ventilation is ineffective.

However, do not persist with intubation attempts without ventilating the patient intermittently with a bag and mask as necessary to prevent hypoxaemia during the intubation process.

Give high-flow oxygen to all patients with respiratory difficulty.

Give as much oxygen as possible through a mask with a reservoir bag to any patient who is breathing but has respiratory difficulty or the other signs of hypoxia (e.g. cyanosis).

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Situations in which emergency treatment is given

1 Perform needle thoracocentesis if the diagnosis is tension pneumothorax (see Section 31). This should be followed by a chest drain.
2 Consider inserting a chest drain if there is major trauma to the chest (see Section 31).
3 Give nasal continuous positive airway pressure (CPAP) if a neonate has severe respiratory distress (see Section 35).

Primary assessment and resuscitation of circulation
Primary assessment

The circulatory system is more difficult to assess than airway and breathing, and individual measurements must not be over-interpreted.

If there is no palpable pulse, a very slow heart rate < 60 beats/minute in an infant or no 'signs of life' (e.g. movements, coughing, normal breathing), cardiac arrest or near-cardiac arrest is likely, and basic life support must be started (see Section 2).

Agonal gasps (irregular, infrequent breaths) do not provide adequate oxygenation and are not for these purposes a 'sign of life'.

In addition to cardiac arrest or near-arrest, shock and heart failure are additional life-threatening issues that it is important to identify.

Shock

The following clinical signs can help to identify shock (inadequate circulation) (see Section 27).

Heart rate
- Heart rate increases in shock and heart failure.
- Severe bradycardia due to hypoxaemia may be a sign of near cardiorespiratory arrest.

The WHO definition of tachycardia is a heart rate of > 160 beats/min in infants

Pulse volume
Absent peripheral pulses or reduced strength of central pulses can signify shock.

Capillary refill time (CRT)
- Pressure on the centre of the sternum for 5 seconds should be followed by return of the circulation to the skin within 3 seconds or less. CRT may be prolonged by shock, cold environment, or the vasoconstriction that occurs as a fever develops.
- Prolonged CRT is not a specific or sensitive sign of shock, and should not be used alone as a guide to the need for or the response to treatment.

Blood pressure
- The cuff should cover at least 80% of the length of the upper arm, and the bladder

should be more than two-thirds of the arm's circumference.

- Measuring blood pressure in the neonate is very difficult
- Korotkoff phase 5 (K5, disappearance of sound) should be used to measure diastolic pressure. Korotkoff phase 5 (K5A, muffling or softening of sound) should only be used if the sound does not disappear until near to zero cuff pressure.
- Hypotension is a late sign of circulatory failure in both children and pregnant mothers, and will be rapidly followed by cardiorespiratory arrest unless it is treated urgently.

TABLE 1.1 Systolic blood pressure in infancy

Age (years)	Systolic blood pressure (mmHg) 5th centile	Systolic blood pressure (mmHg) 50th centile
< 1	65–75	80–90

The cardiovascular system in infants compensates well initially in shock. Hypotension is a late and often sudden sign of decompensation and, if not reversed, will be rapidly followed by death. Serial measurements of blood pressure should be performed frequently.

Effects of circulatory failure on other organs
Respiratory system: tachypnoea and hyperventilation occur as a result of the acidosis caused by poor tissue perfusion.

Skin: pale or mottled skin indicates poor perfusion.

Mental status: circulatory failure causes initial agitation, then drowsiness, followed by unconsciousness.

Urine output: a reduction in urine output to < 2 mL/kg/ hour in infants indicates inadequate renal perfusion.

The WHO definition of shock is cold hands, plus CRT of > 3 seconds, plus a weak and rapid pulse.
Life-threatening shock is usually associated with:

- severe tachycardia
- a weak-volume pulse (ideally assess centrally: brachial, femoral or carotid arteries)
- low blood pressure (this is a late sign, and difficult to measure in infants)
- extreme central pallor (if due to severe anaemia)
- raised respiratory rate (due to acidosis)
- poor skin circulation, with a CRT of > 3 seconds
- reduced conscious level.

Anaphylaxis is one cause of shock, and typically there is a relevant history and other signs such as angio-oedema and urticaria.

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If shock is due to heart failure, fluid overload will be fatal (for information on how to recognise and manage shock caused by heart failure, see Section 27).

Resuscitation in shock

For cardiac arrest or near arrest, chest compressions should be undertaken.

Ensure that there is an open and secure airway.

Give high-flow oxygen to any infant who has an inadequate circulation (whether due to shock or to heart failure). This should be administered via a face mask with a reservoir bag (or an endotracheal tube if intubation has been necessary).

Venous or intra-osseous access should be obtained and blood for essential tests taken (haemoglobin, cross-matching, blood clotting factors, and urea and electrolytes if possible).

Fluids in shock

In most cases of shock, if obvious bleeding is the cause then the first priority must be to stop this. IV or IO fluids are then required as the immediate resuscitation treatment, once the airway has been opened and secured and oxygen is being given. However, different causes of shock require different approaches to treatment, as described below.

- If loss of fluid causing hypovolaemia is the cause of shock: for infants give an immediate IV/IO bolus of 10 mL/kg of crystalloid (usually Ringer-Lactate or Hartmann's solution) as appropriate for weight, provided that heart failure is not present (see above).

- If the loss of fluid causing shock is due to severe gastroenteritis, there will usually be evidence of severe dehydration and a history of profound or long-standing diarrhoea. Give 10 mL/kg of Ringer-lactate or Hartmann's solution as an initial IV or IO bolus as rapidly as possible, reassess, and then repeat if necessary. In cases of cholera, up to 60 mL/kg might be required. Additional potassium will usually be required (see Section 28).

- If the loss of fluid causing shock is due to bleeding, give crystalloid immediately and then try to obtain blood for transfusion as rapidly as possible, ideally fresh blood. Give O-negative blood if this is available and if shock is due to haemorrhage.

- Adequate perfusion of vital organs in neonates may best be indicated by a decrease in tachycardia and improved level of consciousness.

- If shock is due to septicaemia with purpura (meningococcus), give IV or IO boluses of Ringer-lactate or Hartmann's or 0.9% saline as fast as possible, 10-20 mL/ kg in the

infant, and then reassess. Usually at least 40 mL/kg in infants will be required to overcome shock (see Section 27). In this situation, inotropes may be valuable if they are available and safe to use (see Section 27).

- If shock is due to anaphylaxis, give adrenaline, 10 micrograms/kg (0.1 mL/kg of 1 in 10 000) IM in addition to IV or IO fluid.

- If shock is due to severe anaemia, IV crystalloid boluses such as Ringer-Lactate or Hartmann's solution must be given with extreme care (due to the risk of heart failure). As soon as possible, give blood carefully (10 mL/kg in infants over 15 minutes) and then reassess and repeat if it is safe to do so.

Partial exchange transfusion may be helpful in this situation. Successively remove 20-mL aliquots of the patient's blood and replace each 20 mL with 40 mL of packed donor red blood cells until shock has resolved.

Heart failure
This life-threatening situation can be seen in severe anaemia and after fluid overload and in the presence of structural heart disease. It is important to distinguish heart failure from shock, as the resuscitation required is different. Some of the following signs will be present in infants with heart failure:
- tachycardia out of proportion to respiratory difficulty
- severe palmar pallor (if anaemia is the cause)
- some heart murmurs (if structural heart defect is responsible)
- an enlarged, sometimes tender, liver
- crepitations on listening to the lung bases
- cyanosis that does not respond to oxygen in the case of infants with cyanotic congenital heart disease.

Resuscitation of infants with heart failure
1 Tilt the patient head-up.
2 Give oxygen.
3 Give furosemide 1–2 mg/kg by IV/IO injection.
4 If the patient has severe anaemia, careful blood transfusion and consider exchange transfusion.

Situations where emergency treatment is given in heart failure with shock
1 Supraventricular tachycardia can cause both shock and heart failure. The heart rate in infants can reach > 220 beats/ minute. If available, ECG will confirm tachycardia. Treat by vagal manoeuvres, defibrillation if available, or adenosine if rapid IV access is available (see Textbook).
2 In ventricular tachycardia, defibrillation is needed if shock is present (see Section 3).
3 If congenital or rheumatic heart disease or cardiomyopathy is the cause of heart

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failure, inotropes or digoxin may be appropriate, but specialist advice will be needed.

4   If cyanotic congenital heart disease in the newborn is the cause of shock, give prostaglandin E2, but specialist paediatric advice will be necessary (see Section 25).

Primary assessment and resuscitation of neurological failure (disability)
Always assess and treat Airway, Breathing and Circulation problems before undertaking neurological assessment.

Primary assessment
Conscious level: AVPU
Alert is the normal state for an awake infant. If the infant does not respond to gentle shaking to wake them up, it is important that assessment of the response to Pain is undertaken next. A painful central stimulus can be delivered by supra- orbital ridge pressure or by pulling frontal hair. An infant who is Unresponsive or who only responds to pain has a significant degree of coma which can seriously interfere with vital Airway and Breathing functions.

Fits (see Section 21)
Generalised convulsions, also known as 'fits' or 'seizures', can seriously interfere with vital Airway and Breathing functions, both during the fit itself and immediately afterwards, when lowered levels of consciousness may be present.

Posture
Many patients who have a serious illness in any system are hypotonic. Stiff posturing, such as that shown by decorticate (flexed arms, extended legs) or decerebrate (extended arms, extended legs) posturing, is a sign of serious brain dysfunction. These postures can be mistaken for the tonic phase of a convulsion. Alternatively, a painful stimulus may be necessary to elicit these postures.

Severe extension of the neck due to upper airway obstruction can mimic the opisthotonus that occurs with meningeal irritation. In infants, a stiff neck and full fontanel are signs that suggest meningitis.

Pupils
Many drugs and cerebral lesions have effects on pupil size and reactions. However, the most important pupillary signs to seek are dilatation, un-reactivity and inequality, which suggest possible serious brain disorders.

Always check blood glucose levels or suspect **hypoglycaemia** in any unwell infant, especially if they have impaired consciousness. Hypoglycaemia with a blood glucose level of less than 2.5 mmol/L (45 mg/dL) can cause impaired consciousness, coma or fits.

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Respiratory effects of central neurological failure
The presence of any abnormal respiratory pattern in a patient with coma suggests mid- or hindbrain dysfunction.

Circulatory effects of central neurological failure Systemic hypertension with sinus bradycardia (Cushing's response) indicates compression of the medulla oblongata caused by herniation of the cerebellar tonsils through the foramen magnum. This is a late and pre-terminal sign.

Raised intracranial pressure (ICP) may cause:
- hyperventilation
- slow sighing respirations
- apnoea
- hypertension
- bradycardia.

Resuscitation for neurological problems
1. If the patient is unconscious (P or U on the AVPU scale) but their airway and breathing are adequate, place them in the recovery position, so that if they vomit there is less likelihood of aspiration because when unconscious, the gag reflex may not be operative.
2. If the patient is unconscious or fitting, always give oxygen.
3. If hypoglycaemia is a cause of reduced consciousness (or a suspected cause, but immediate blood glucose measurements are not possible), treatment with glucose is urgently required. Give 2.5 mL/kg of 10% glucose IV or IO in the infant and repeat if required. Recheck the blood glucose level after 20 minutes, and if the level is low (< 2.5 mmol/litre or < 45 mg/ dL), repeat the IV/IO glucose (2.5 mL/kg)..
4. If fitting occurs in an infant and continues in your presence for more than 5 minutes and there is no hypoglycaemia, give IV or rectal anticonvulsants. **Always make sure that a bag and mask are available** in case the patient stops breathing, which is possible if not likely. Commonly used anticonvulsants in this situation are diazepam or, if there is no IV access, rectal diazepam, rectal paraldehyde or buccal midazolam (see Section 21).
   - IV or IO diazepam: 250 micrograms/kg IV over 5 minutes
   - rectal diazepam: 500 micrograms/kg
   - rectal paraldehyde: 0.4 mL/kg
   - buccal midazolam: 300 micrograms/kg.
5. To gain time in acutely raised intracranial pressure (e.g. in cases of head injury), consider the use of IV mannitol, 250 –500 mg/kg, which will draw fluid out of the brain for a short while, thereby temporarily reducing the ICP. Because the effect of mannitol is only short-lived (a matter of hours), it is used to gain time while definitive care is being set up (e.g. surgical intervention to drain an extradural or subdural haematoma).

6   In any case where meningitis or encephalitis is suspected, it is vital that suitable antibiotics and/or antiviral drugs are started IV or IO as soon as the condition is suspected. Antibiotic choices might include cefotaxime, penicillin, w i t h amoxicillin and gentamicin in the newborn. Consider adjunctive treatment with dexamethasone 150 micrograms/kg every 6 hours for 4 days starting before or with the first antibiotic dose. Do not use dexamethasone in cases where there is also septic shock (e.g. in meningococcal disease).

Secondary assessment and emergency treatments

The secondary assessment takes place once vital functions have been assessed and the initial resuscitation of those vital functions has been started. Primary assessment and resuscitation can usually be undertaken in less than 1 minute if the patient does not have a life-threatening airway, breathing, circulation or neurological problem.

Secondary assessment includes a focused medical history, a focused clinical examination and specific investigations. It differs from a standard medical history and examination in that it is designed to establish which emergency treatments might benefit the patient. Time is limited, and a focused approach is essential. At the end of secondary assessment, the practitioner should have a better understanding of the illness or component of injury likely to be affecting the patient, and may have formulated a differential diagnosis. Emergency treatments will be appropriate at this stage – to treat either specific disorders e.g. meningitis or conditions (e.g. respiratory failure). Emergency treatments will be undertaken at this stage in addition to those given as part of resuscitation/life-saving treatments, in order to manage specific components of serious illnesses or injuries (e.g. IV antibiotics for neonatal sepsis). The establishment of a definite diagnosis is part of definitive care.

The history often provides the vital clues. In the case of infants, the history is often obtained from an accompanying parent. Do not forget to ask any health worker who has seen the patient about the initial condition and about treatments and the response to treatments that have already been given.

Some patients will present with an acute exacerbation/ complication of a known condition, such as epilepsy. Such information is helpful in focusing attention on the appropriate system, but the practitioner should be wary of dismissing new pathologies in such patients. The structured approach avoids this problem. Unlike trauma, illness affects systems rather than anatomical areas. The secondary assessment must reflect this, and the history of the complaint should be sought with special attention to the presenting system or systems involved. After the presenting system has been dealt with, all of the other systems should be assessed and any additional emergency treatments commenced as appropriate.

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The secondary assessment is not intended to complete the diagnostic process, but rather it aims to identify any problems that require emergency treatment.

The symptoms, signs and treatments relevant to each emergency condition in the neonate are elaborated further in the relevant sections of this pocketbook.

Basic Life Support for infants

Introduction

Basic life support (BLS) is a technique that can be employed by one or more rescuers to support the respiratory and circulatory functions of a collapsed patient using no or minimum equipment.

The international guidelines for resuscitation from cardiac arrest (European Resuscitation Council, 2010) detail two approaches to basic life support. The sequence of actions in the 'child' programme is predicated on a hypoxic event (including any respiratory failure or obstruction, or hypoxia at a cellular level as seen in shock). In this type, re-establishing oxygenation is of prime importance, and moving the oxygenated blood to the coronary and cerebral arteries is the second step. Therefore the rescuer's sequence of actions after assessment starts with rescue breaths and then moves on to chest compressions. The 'child'-type cardiac arrest is seen in almost all infants (excluding those rare arrhythmic events in infants with congenital or acquired heart disease and those in whom sudden, unexpected collapse is preceded by apparent normal respiratory and circulatory function. This includes patients who have had convulsions, trauma (including drowning), poisoning, bleeding, sepsis, etc.

The sequence taught in the post neonatal infant therefore includes five preliminary rescue breaths and a subsequent ratio of 15 chest compressions to 2 lung inflations. In the neonatal period the ratio of compressions to ventilations is 3:1.

The initial approach: the three S's

Safety: it is essential that the rescuer does not become a second victim. They must approach the patient with care, and remove the patient from any continuing source of danger if necessary.

Stimulate: ask the question 'Are you all right?' in order to establish the state of consciousness of the patient.

Shout: this is essential because help will be needed.

If more than one rescuer is present, one person should start basic life support. The second person should activate the Emergency Medical Services (EMS) system and then returns to assist in the basic life support effort.

If the patient is an infant, and there is only one rescuer and no help has arrived, the rescuer should open the airway, deliver the five rescue breaths and give 1 minute of cardiopulmonary resuscitation (CPR), and then activate the EMS system (if one is available) using a mobile phone if available so as to continue CPR. If a mobile is not available the rescuer will probably be able to carry the infant to a telephone while continuing CPR.
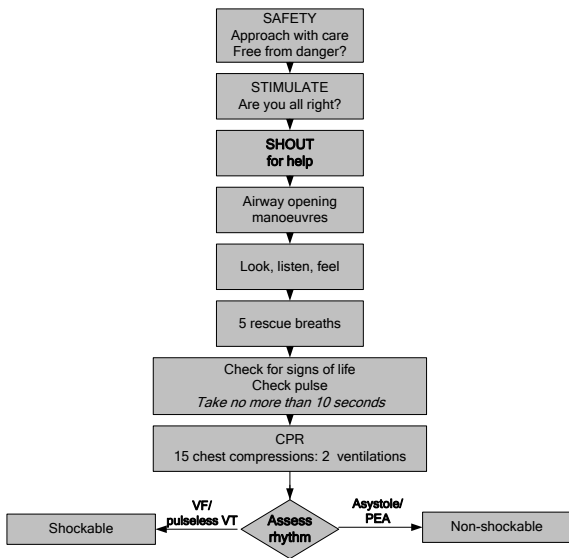
FIGURE 2.1 Algorithm for basic life support in infants. CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; VT, ventricular tachycardia; PEA, pulseless electrical activity.

'Are you all right?'

An initial simple assessment of responsiveness consists of gently shaking the infant by the shoulder resulting in some vocalisation or opening their eyes.

In cases associated with trauma, or possible trauma, the cervical spine should be immobilised during this procedure by placing one hand firmly on the forehead while one of the patient's shoulders is shaken.

Airway-opening actions

An obstructed airway may be the primary problem, and correction of the obstruction can result in recovery without the need for further intervention. If the patient is unconscious but breathing, the recovery position should be used.

FIGURE 2.2 Head tilt with chin lift in neutral position for the infant.

If the patient is not breathing, this may be because the airway is blocked by the tongue falling back and obstructing the pharynx. Attempt to open the airway using the head tilt/chin lift manoeuvre. The rescuer places their nearest hand on the patient's forehead, and applies pressure to tilt the head back gently. The correct position is 'neutral' in the infant (0–1 year of age) (see Figure 2.2).

The fingers of the other hand should then be placed under the chin, and the chin of the supine patient should be lifted upwards. As this action may close the patient's mouth, it may be necessary to use the thumb of the same hand to part the lips slightly.

As an alternative to the head tilt/chin lift, the jaw thrust manoeuvre can be very effective, but requires more training and experience.

FIGURE 2.3 Jaw thrust to open airway.

Jaw thrust is achieved by placing two or three fingers under the angle of the mandible bilaterally, and lifting the jaw upward (see Figure 2.3). This is potentially safer than the head tilt/chin lift if there is a history of major trauma, as the latter manoeuvre may exacerbate a

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cervical spine injury. BUT airway opening is always the most important action which must be achieved, and should always take precedence over concerns about a possible cervical spine injury.

The openness of the airway should then be assessed by:

- looking for adequate chest movements
- listening for breath sounds
- feeling for breaths.

This is best achieved by the rescuer placing their face above that of the patient, with the ear over the nose, the cheek over the mouth, and the eyes looking along the line of the chest. They should take no longer than 10 seconds to assess breathing.

If there is any object obvious in the mouth and it is easy to reach, remove it. Do not perform a blind finger sweep in the mouth. A blind finger sweep can damage the soft palate, and foreign bodies may be forced further down the airway and become lodged below the vocal cords.

Breathing actions

If airway-opening techniques do not result in the resumption of adequate breathing within 10 seconds, and a self-inflating bag–mask system is not available, then the rescuer should commence mouth-to-mouth-and-nose exhaled air resuscitation.

Definition of adequate breathing

A patient may have very slow or shallow breathing, or take infrequent, noisy, agonal gasps. Do not confuse this with normal breathing.

Rescue breaths

If in doubt about the adequacy of breathing, five initial rescue breaths should be given. While the airway is held open, the rescuer breathes in and seals their mouth around the patient's mouth and nose (in the case of infants) (see Figures 2.4). Slow exhalation, 1–2 seconds, by the rescuer should result in the patient's chest rising. The rescuer should take a further breath him- or herself before the next rescue breath.

FIGURE 2.4 Mouth-to-mouth and nose breaths in the neutral position for an infant.

If the chest does not rise, the airway is not clear. The usual cause is failure to correctly apply the airway-opening techniques discussed earlier. The first step is to readjust the head tilt/chin lift position and try again. If this is not successful, jaw thrust should be tried. If two rescuers are present, one should maintain the airway while the other breathes for the patient.

Failure of both head tilt/chin lift and jaw thrust should lead to suspicion that a foreign body is causing the obstruction (see below).

While performing rescue breaths, the presence of a gag reflex or coughing is a positive sign of life (see below).

Circulation actions
Once the initial five breaths have been given successfully, circulation should be assessed and managed.

Check signs of life and/or pulse (take no more than 10 seconds).

Even experienced health professionals can find it difficult to be certain that the pulse is absent within 10 seconds, so the absence of 'signs of life' is the best indication for starting chest compressions, especially in an infant. 'Signs of life' include movement, coughing, gagging or normal breathing (but not agonal gasps, which are irregular, infrequent breaths). Thus the absence of evidence of normal breathing, coughing or gagging (which may be noticed during rescue breaths) or any spontaneous movement is an indication for chest compressions.

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Inadequacy of circulation is also indicated by the absence of a central pulse for up to 10 seconds, but it can be difficult and therefore time wasting to be certain about this – hence the current emphasis on assessing the presence of 'signs of life'.

In infants, if a slow pulse (less than 60 beats/minute) is felt or heard by stethoscope, this is also an indication for chest compressions. Infants have a short fat neck, so the carotid pulse may be difficult to identify. The brachial artery in the medial aspect of the ante-cubital fossa or the femoral artery in the groin can more easily be felt in infants. If there are no signs of life and/or a pulse is absent for up to 10 seconds, start chest compressions. Compressions should also be started if in an infant there is an inadequate heart rate (less than 60 beats/minute), but only if this is accompanied by signs of poor perfusion, which include pallor, lack of responsiveness and poor muscle tone.

Start chest compressions if:

- there are no signs of life or
- there is no pulse or
- there is a slow pulse or heart rate (less than 60 beats/minute in an unconscious infant with poor perfusion).

Unnecessary' chest compressions are almost never damaging. It is important not to waste vital seconds before starting chest compressions after oxygenating the patient with the rescue breaths. If there are signs of life and the pulse is present (and has an adequate rate, with good perfusion), but apnoea persists, exhaled air resuscitation must be continued until spontaneous breathing resumes.

Chest compressions
For the best output, the patient must be placed on their back, on a hard surface. The chest should be compressed by a third of its depth.

Position for chest compressions
Chest compressions should compress the lower half of the sternum.

Infants: Infant chest compression can be more effectively achieved using the hand-encircling technique: the infant is held with both the rescuer's hands encircling or partially encircling the chest. The thumbs are placed over the lower half of the sternum and compression is carried out as shown in Figure 2.5.
This method is only possible when there are two rescuers, as the time needed to reposition the airway precludes the use of the technique by a single rescuer if the recommended rates of compression and ventilation are to be achieved.

The single rescuer should use the two-finger method as shown in Figure 2.6, employing the other hand to maintain the airway position.

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Once the correct technique has been chosen and the area for compression identified, 15 compressions should be given to 2 ventilations.

FIGURE 2.5 Two-thumb method for chest compressions in an infant (two rescuers).

FIGURE 2.6 Two-finger method for chest compressions in an infant (one rescuer).

Continuing cardiopulmonary resuscitation

The compression rate is 100–120 compressions per minute. A ratio of 15 compressions to 2 ventilations is maintained irrespective of the number of rescuers. With pauses for ventilation there will be less than 100–120 compressions per minute, although the rate is 100–120 per minute. Compressions can be recommenced at the end of inspiration and may augment exhalation.

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If no help has arrived, the emergency services must be contacted after 1 minute of cardiopulmonary resuscitation. Apart from this interruption to summon help, basic life support must not be interrupted unless the patient moves or takes a breath.

Effective chest compressions are tiring for the rescuer. Continually check that the compressions and ventilations are satisfactory and, if possible, alternate the rescuers involved in this task. Any time spent readjusting the airway or re-establishing the correct position for compressions will seriously decrease the number of cycles given per minute. This can be a real problem for the solo rescuer, and there is no easy solution. In infants the free hand can maintain the head position. The correct position for compressions does not need to be measured after each set of ventilations.

The cardiopulmonary resuscitation manoeuvres recommended for infants is summarised in Table 2.1

TABLE 2.1 Summary of basic life support techniques in infants

	Infants (< 1 year)
Airway	
Head-tilt position	Neutral
Breathing	
Initial slow breaths	Five
Circulation	
Pulse check	Brachial or femoral
Landmark	Lower half of sternum
Technique	Two fingers or two thumbs
CPR ratio	15:2

If recovery occurs and signs of life return, place the patient in the recovery position and continue to reassess them and ensure that specialist help arrives.

Chest-compression-only CPR.

If you either unable or unwilling to give rescue breaths, give chest compressions only. This is particularly relevant in countries where there is a high prevalence of HIV, hepatitis or TB (see below).

If chest compressions only are given, these should be continuous at a rate of 100 compressions per minute.
Stop to recheck the patient only if they start to breathe normally; otherwise do not interrupt resuscitation.

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Continue resuscitation until:

- qualified help arrives and takes over or
- the patient starts breathing normally or
- you become exhausted.

Basic life support and infection risk

Few cases have been reported. The most serious concerns are meningococcus and TB. In the case of meningococcus, rescuers involved in the resuscitation of the airway in such patients should take standard prophylactic antibiotics. There have been no reported cases of transmission of either hepatitis B or HIV infection through mouth-to-mouth ventilation. Blood-to-blood contact is the single most important route of transmission of these viruses, and in non-trauma resuscitation the risks are negligible. Sputum, saliva, sweat, tears, urine and vomit are low-risk fluids. Precautions should be taken, if possible, in cases where there might be contact with blood or amniotic fluid. Devices that prevent direct contact between the rescuer and the patient (such as resuscitation masks) can be used to lower the risk. Gauze swabs or any other porous material placed over the patient's mouth is of no benefit in this regard.

Infection rates vary from country to country, and rescuers must be aware of the local risk. In countries where HIV/ AIDS is more prevalent, the risk to the rescuer will be greater.

If available, bag-valve-mask ventilation is preferable to   mouth-to-mouth ventilation.

The recovery position

The patient should be placed in a stable, lateral position that ensures maintenance of an open airway with free drainage of fluid from the mouth, ability to monitor and gain access to the patient, security of the cervical spine and attention to pressure points (see Figure 2.7). The Resuscitation Council (UK) recommends the following sequence of actions when placing a patient in the recovery position:

- Kneel beside the patient and make sure that both of their legs are straight.
- Place the arm nearest to you out at right angles to their body, elbow bent with the hand palm uppermost.
- Bring the far arm across the chest, and hold the back of the hand against the patient's cheek nearest to you.
- With your other hand, grasp the far leg just above the knee and pull it up, keeping the foot on the ground.
- Keeping their hand pressed against their cheek, pull on the far leg to roll the patient towards you on to their side.
- Adjust the upper leg so that both the hip and knee are bent at right angles.
- Tilt the head back to make sure the airway remains open.
- Adjust the hand under the cheek, if necessary, to keep the head tilted.

- Check the patient's breathing regularly.

If the patient has to be kept in the recovery position for more than 30 minutes, turn them to the opposite side in order to relieve the pressure on the lower arm.

FIGURE 2.7 The semi-prone or recovery position.

Hand supports
head

Knee stops body
rolling onto abdomen

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Airway and breathing

Management of the airway (A) and breathing (B) components of the ABC must take priority in all situations. Resuscitation will fail if effective ventilation does not occur.

Before effective resuscitation techniques can be applied, it is essential that the operator is able to:

1 understand the airway equipment available and how to use it
2 recognise respiratory failure and when it may occur
3 perform a systematic and prioritised approach (the structured ABC approach) to the management of the infant who has a problem of the airway or breathing (see Section 1).

Airway: equipment and skills for opening and maintaining the airway

Essential airway and breathing equipment includes the following:

- face masks (ideally with reservoirs)
- airways, including laryngeal mask airways (LMAs) if anaesthetic skills are available
- self-inflating bag-valve-mask devices
- tracheal tubes, introducers and connectors
- laryngoscopes
- Magill's forceps
- suction devices
- surgical airway packs for performing an emergency surgical airway.

This equipment should be available in all resuscitation areas, ideally on a resuscitation trolley. It is crucial to gain familiarity with it before an emergency situation occurs.

Pharyngeal airways

There are two main types of pharyngeal airway, namely oropharyngeal (see Figures 3.1 and 3.2) and nasopharyngeal.

FIGURE 3.1 Oropharyngeal airway, showing position when inserted.
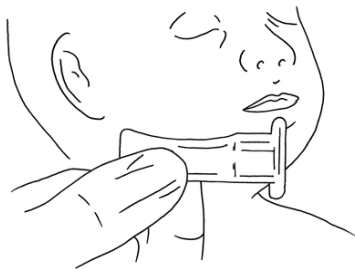
FIGURE 3.2 Oropharyngeal airway, showing sizing technique.

Oropharyngeal airways

The oropharyngeal or Guedel airway is used in the unconscious or obtunded patient to provide an open airway channel between the tongue and the posterior pharyngeal wall In the patient with an intact gag reflex, it may not be tolerated and may induce vomiting.

The oropharyngeal airway is available in a variety of sizes. A correctly sized airway when placed with its flange at the centre of the incisors, then curved around the face, will reach the angle of the mandible. Too small an airway may be ineffective, and too large an airway may cause laryngospasm. Either may cause mucosal trauma or may worsen airway

obstruction. Reassessment following placement is therefore a vital part of safe insertion of an airway device.

The important point is not to push the tongue back by inserting the airway carelessly.

In infants as the tongue is larger relative to the size of the mouth, the airway cannot be rotated in the mouth without causing trauma. Therefore, the tongue is depressed with the airway or a spatula and not by the convex side of the airway (see Figure 3.3).
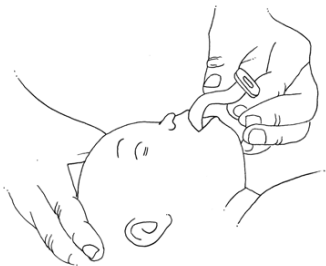
FIGURE 3.3 When inserting the airway without rotation, a tongue depressor can be helpful (not shown).

Nasopharyngeal airways

The nasopharyngeal airway is often better tolerated than the Guedel airway. It is contraindicated in fractures of the base of the skull. It may also cause significant haemorrhage from the vascular nasal mucosa if it is not inserted with care, preferably with lubrication. A suitable length can be estimated by measuring from the lateral edge of the nostril to the tragus of the ear. An appropriate diameter is one that just fits into the nostril without causing sustained blanching of the alae nasi. If small-sized nasopharyngeal airways are not available, shortened endotracheal tubes may be used.

Ensure that insertion of one or other of these devices results in an improvement in the patient's airway and breathing. It if does not improve the airway as shown by improved breathing, then a reappraisal of the choice or size of airway is urgently required.

Laryngoscopes

There are two principal designs of laryngoscope, namely straight bladed and curved bladed.

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The straight-bladed laryngoscope is usually employed to directly lift the epiglottis, thereby uncovering the vocal folds. The advantage of this approach is that the epiglottis is moved sufficiently so that it does not obscure the cords. The potential disadvantage is that vagal stimulation may cause laryngospasm or bradycardia.

The curved-bladed laryngoscope is designed to move the epiglottis forward by lifting it from in front. The tip of the blade is inserted into the mucosal pocket, known as the vallecula, anterior to the epiglottis, and the epiglottis is then moved forward by pressure in the vallecula. This may be equally effective for obtaining a view of the cords, and it has the advantage that less vagal stimulation ensues, as the mucosa of the vallecula is innervated by the glossopharyngeal nerve instead.

A laryngoscope blade appropriate for the age of the patient should be chosen; **in infants a straight blade is usually used**. It is possible to intubate with a blade that is too long, but not with one that is too short.

Laryngoscopes are notoriously unreliable pieces of equipment which may develop flat batteries and unserviceable bulbs very quickly between uses. **Therefore, it is vital that a spare is available at all times, and equipment must be regularly checked to ensure that it is in good working order.**

Tracheal tubes

Un-cuffed tubes should be used during resuscitation, by operators who do not have paediatric anaesthetic experience. If the operator is familiar with cuffed tube placement, both cuffed and uncuffed tubes are acceptable for infants but the youngest infants will usually need an uncuffed tube.

In infants the larynx is circular in cross section and the narrowest part of it is at the cricoid ring, rather than the vocal cords. An appropriately sized tube should give a relatively gas-tight fit in the larynx, but the fit should not be so tight that no leak is audible when the bag is compressed. Failure to observe this condition may lead to damage to the mucosa at the level of the cricoid ring, and to subsequent oedema following extubation.

Neonates usually require a tube of internal diameter 3–3.5 mm, although preterm infants may need one of diameter 2.5mm. Cuffed tubes are generally not used in neonates.

For infants of weight over 3 kg and up to 1 year in age a size 3 cuffed tube maybe acceptable.

The size of tracheal tubes is measured in terms of their internal diameter in millimetres. They are available in whole- and half-millimetre sizes. The clinician should select a tube of appropriate size, but also have ready one a size smaller and one a size larger.

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In the case of resuscitation in an infant where the lungs are very 'stiff' (e.g. in a cardiac arrest from severe bronchiolitis), a cuffed tube rather than an uncuffed tube may be used by a non-expert, but the risk of airway damage from the cuff must be balanced against the risk of failure to inflate the lungs.

Tracheal tube introducers

Intubation can be facilitated by the use of a stylet or introducer, which is placed through the lumen of the tracheal tube. There are two types – either soft and flexible or firm and malleable.

The soft and flexible type can be allowed to project beyond the tip of the tube, so long as it is handled very gently. **The firm type is used to alter the shape of the tube, but can easily damage the tissues if allowed to protrude from the end of the tracheal tube.** Tracheal tube introducers should not be used to force a tracheal tube into position.

Bougies, which are flexible, deformable, blunt-ended gum elastic rods of different sizes, can be used to help to introduce a tracheal tube when access is difficult. A Seldinger-type technique is used. The bougie is introduced into the trachea using the laryngoscope, the endotracheal tube is then passed over it into the trachea, and finally the bougie is removed.

Tracheal tube connectors

The proximal end of the tube connectors is of standard size, based on the 15-mm/22-mm system, which means that they can be connected to a standard self-inflating bag.

Magill's forceps

Magill's forceps (see Figure 3.4) are angled to allow a view around the forceps when they are in the mouth. They may be useful to help to position a tube through the cords by lifting it anteriorly, or to remove pharyngeal or supra-glottic foreign bodies.
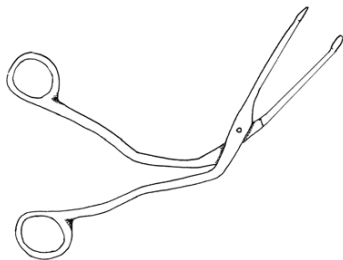
FIGURE 3.4 Magill's forceps.

Suction devices

These are used to remove blood, vomit and secretions from the mouth and throat, usually with a rigid suction tube (Yankauer suction tube; see below). In resuscitation areas, ideally the suction device should be connected to a central vacuum unit. This consists of a suction hose inserted into a wall terminal outlet, a controller (to adjust the vacuum pressure), a reservoir jar, suction tubing and a suitable sucker nozzle or catheter. In order to aspirate vomit effectively, it should be capable of producing a high negative pressure and a high flow rate, although these can be reduced in non-urgent situations, so as not to cause mucosal injury.

Portable suction devices are required for resuscitation when central suction is not available (as is the case in most resource-limited hospitals), and for transport to and from the resuscitation room. These are either manual, mains electrical or battery powered. A manual or battery-operated suction system must be available at all sites where resuscitation may be needed.

To clear the oropharynx of debris (e.g. vomit), a rigid sucker (e.g. Yankauer sucker) should be used with care not to damage delicate tissue or induce vomiting. The Yankauer sucker is available in both adult and paediatric sizes. It may have a side hole, which can be occluded by a finger, allowing greater control over vacuum pressure.

Tracheal suction catheters (see Figure 3.5) These may be required after intubation to remove bronchial secretions or aspirated fluids. In general, the appropriate size in French gauge is numerically twice the internal diameter in millimetres (e.g. for a 3-mm tube the correct suction catheter is a French gauge 6).
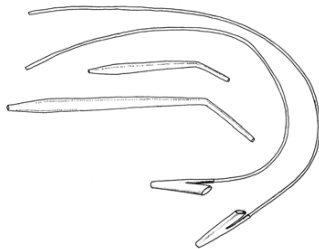
FIGURE 3.5 Tracheal and oral or nasal suction catheters.

Advanced airway techniques

Advanced airway techniques are used when the above techniques fail to maintain and protect an airway over the longer term, particularly if there is potential for it to become obstructed and thus prevent accurate control of oxygenation and ventilation. Advanced airway techniques (tracheal intubation, and surgical cricothyroidotomy) are described in Sections 32-33.

Breathing: equipment and skills for helping the infant to breathe

The following equipment for oxygenation and ventilation should be readily available:

- an oxygen source
- masks for those who are spontaneously breathing
- close-fitting face masks (for artificial ventilation)
- self-inflating bag-valve systems to be used with close- fitting face masks
- T-piece and open-ended bag systems (only to be used by those with anaesthetic skills)
- mechanical ventilators
- chest tubes
- gastric tubes.

Oxygen treatment
Indications

Give oxygen to infants:

- with respiratory distress (severe in-drawing of the lower chest wall, also known as recessions, raised respiratory rate, gasping, grunting with each breath, nasal flaring, head bobbing, etc.)
- with cyanosis (blueness) that is central (around the lips and tongue, or inside the mouth in babies with dark skin)
- who are shocked

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- who are fitting
- who are unconscious, with abnormal reduced oxygen saturation (SaO_2) on a pulse oximeter.

Ideally, where the resources for this are available, oxygen therapy should be guided by pulse oximetry (see below). Give oxygen to full term infants with a SaO_2 of < 94%, and aim to keep SaO_2 at 94–98% (except at high altitude, where normal oxygen saturation levels are lower). For SaO2 levels needed in preterm infants see Section 7).

If pulse oximeters are not available, the need for oxygen therapy has to be guided by clinical signs, which are less reliable.

Provision of oxygen

Oxygen must be available at all times. The two main sources of oxygen are cylinders and oxygen concentrators.

Oxygen cylinders contain compressed gas. A flow meter needs to be fitted to regulate flow. A hissing noise can be heard if gas is being delivered. Flow meters are used to ascertain how much oxygen is being delivered. Take the reading of flow rate from the middle of the ball. Always switch off the flow when the source is not in use (ensure that the indicator ball is at the bottom of the flow meter and not moving).

Do not leave anything inflammable near to the oxygen supply. Do not allow smoking near to the oxygen supply.

At least once a day, check that an adequate oxygen supply is available (use a signed logbook). If a gauge indicating the amount left in the cylinder is not available, switch on the flow and listen for a hissing noise. Replace empty cylinders promptly. Ensure that cylinders are stored and secured in an upright position in suitable containers so that they cannot fall over and cause injury. Cylinder keys to permit changes of regulator should be tied to each cylinder.

Oxygen concentrators produce more than 95% oxygen with a flow of 1–8 litres/ minute but, unlike cylinders, they require a continuous electricity supply. **It is also advisable to use an oxygen sensor to regularly check the concentration of oxygen being produced by each concentrator**. For this reason, all areas where patients might need oxygen must have both cylinders and concentrators.

There are now small oxygen plants available that can provide oxygen for a defined area or even for the whole of a hospital or health facility. Some of them can be used to fill oxygen cylinders as well, thus providing a constant back-up see Textbook).

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Oxygen delivery

A mask with a reservoir bag (see Figure 3.6) allows up to 100% oxygen to be delivered. Without a reservoir, it is only possible to deliver around 40% oxygen. If only low flow rates of oxygen are available, do not use a reservoir bag.

If an oxygen mask is being used, ensure that the mask is large enough to cover the mouth and nose. Both low- and high-flow oxygen (with a delivery rate of up to 15 litres/minute) can be given. Hold the mask in place using the elastic strap around the back of the head and/or ask the mother to hold it as close as possible to the infant's face.
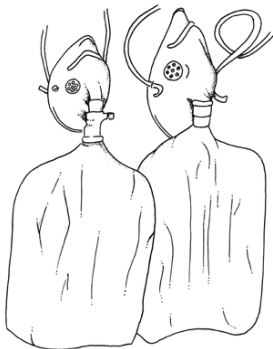
FIGURE 3.6 Reservoir bags

Nasal cannulae (also known as nasal prongs) (see Figure 3.7) are the preferred method of delivery in most circumstances, as they are safe, non-invasive, reliable and do not obstruct the nasal airway. Head boxes are not recommended in low resource settings, as they use up too much oxygen and deliver a low concentration.

Face masks can be used for resuscitation purposes, ideally with a reservoir attached to deliver 100% oxygen.

Monitoring of oxygenation

Nursing staff must know how to place and secure the nasal cannulae correctly. Check regularly that the equipment is working properly, and remove and clean the cannulae at least twice a day.

Monitor the patient at least every 3 hours to identify and correct any problems, including:

- SaO_2 values measured by pulse oximeter
- nasal cannulae out of position
- leaks in the oxygen delivery system
- incorrect oxygen flow rate
- airway obstructed by mucus (clear the nose with a moist wick or by gentle suction).

Pulse oximetry

Normal oxygen saturation at sea level in a full term born infant is 95–100%. Oxygen is ideally given to maintain oxygen saturation at 94–98%. Different cut-off values might be used at high altitude or if oxygen is scarce. Aim for values of 86–92% in preterm infants. The response to oxygen therapy in lung disease can be measured with the pulse oximeter, as the patient's SaO_2 should increase (in patients with cyanotic heart disease, SaO_2 may not change significantly when oxygen is given). The oxygen flow can be titrated using the pulse oximeter as a monitor to obtain a stable SaO_2 without giving too much oxygen. This is especially important in pre-term babies with respiratory disease (see Sections 7 and 14).

FIGURE 3.7 Nasal cannulae delivering oxygen and taped in place.

Assessment of oxygenation at and above sea level

A systematic review in 2009 found an SaO_2 of 90% is the 2.5th centile for a population of healthy children living at an altitude of approximately 2500 m above sea level. This decreases to 85% at an altitude of approximately 3200 m.

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TABLE 3.1 SaO$_2$ levels at different altitudes[a]

Altitude	Location	n	Age	SpO$_2$ (%)	Author	Year
Sea level	UK	70	2–16 (mean, 8) years	Range, 95.8–100 Median, 99.5	Poets et al.	1993
Sea level	Peru	189	2 months to 5 years	Range, 96–100 Mean, 98.7	Reuland et al.	1991
1610 m	Colorado	150	<48 hours 3 months	95% CI, 88–97 Mean, 93 95% CI, 86–97 Mean, 92.2	Thilo et al.	1991
1670 m	Nairobi	87	7 days to 3 years	Range, 89.3–99.3 Mean, 95.7	Onyango et al.	1993
2640	Bogota	189	5 days to 2 years	Range, 84–100 Mean, 93.3	Lozano et al.	1992
2800	Colorado	72	3–670 days	Range, 88–97 Mean, 91.7	Nicholas et al.	1993
3100	Colorado	14	6 hours to 4 months 1 week to 4 months	Range, 81–91 Mean, 80.6±5.3 Mean, 86.1±4.6	Niemeyer et al.	1993
3658	Tibet[b]	15	6 hours to 4 months	Immigrant, 76¯90 Indigenous, 86–94	Niemeyer et al.	1995
3750	Peru	153	2 –60 months	Range, 81–97 Mean, 88.9	Reuland et al.	1991

[a]Values given are those in quiet sleep.

[b]Ranges refer to those born to immigrant Chinese mothers and to those indigenous babies whose families have lived at that altitude for innumerable generations.

Duration of oxygen therapy

Continue giving oxygen continuously until the full term born infant is able to maintain an SaO$_2$ of 94% or higher in room air. When the patient is stable and improving, take them off oxygen for a few minutes. If the SaO$_2$ remains in the range 94–98%, discontinue oxygen, but check again 30 minutes later, and 3-hourly thereafter on the first day off oxygen to ensure that the patient is stable. Where pulse oximetry is not available, the duration of oxygen therapy has to be guided by clinical signs, which are less sensitive.

Breathing for the patient
Face masks with seal over nose and mouth for positive pressure ventilation (see Figure 3.8)

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These face masks are used for either mouth-to-mask or, more commonly, bag–mask ventilation. Masks are available in various sizes, and the appropriate size to cover the mouth and nose should be chosen.

Face masks for mouth-to-mouth or bag-valve-mask ventilation in infants are of two main designs. Some masks conform to the anatomy of the patient's face and have a low dead space. Circular soft plastic masks give an excellent seal and are often preferred. Masks should ideally be clear so that the infant's colour or the presence of vomit can be seen.

A pocket mask is a single-size clear plastic mask with an air-filled cushion rim designed for mouth-to-mask resuscitation. It can be used upside down to ventilate infants.

FIGURE 3.8 Face masks with cushioned rim for a leak-proof fit, and round shape for infants.

Self-inflating bags *(see Figure 3.9)*
This is one of the most important pieces of equipment, allowing hand ventilation by face mask without a supply of gas. The 500 mL bags are appropriate for infants under 1 year of age. There is also a 250-mL version for small premature babies. These bags have pressure-limiting valves that operate at 30–45 cm H_2O. Test the valve by placing the mask on a surface and pressing the bag and ensuring that the valve opens. It can be over-ridden if necessary for stiff, poorly compliant lungs.

The bag connects to the patient through a one-way valve to direct exhaled air to the atmosphere. The other end connects to the oxygen supply and can attach to a reservoir bag which allows high concentrations (up to near 100%) of oxygen to be delivered. Without the reservoir bag, only concentrations of up to 40% can be delivered. The bag itself is easily dismantled and reassembled. It is important to realise that this system will operate without an attached oxygen supply, allowing resuscitation to be initiated before oxygen is available. However, if resuscitation is failing, check that oxygen is being delivered into the bag and to the patient and that the oxygen supply has not been disconnected.

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Always use high-flow oxygen (if available) and a reservoir bag during resuscitation apart from at birth where room air is satisfactory for almost all babies (see Section 6).

It is important to clean the system after each patient.
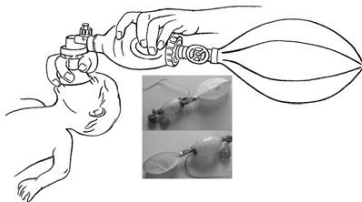
FIGURE 3.9 Two sizes of self-inflating bags and masks.

It is essential that the mask is properly sized and correctly placed over the mouth and nose of the infant (see Figures 3.10 and 3.11).
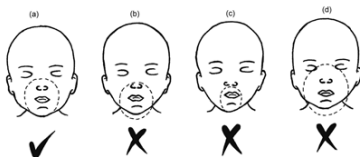
FIGURE 3.10 (a) Correct placement of infant mask. (b), (c) and (d) Incorrect placement of infant mask.
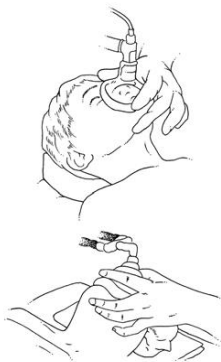
FIGURE 3.11 Two views of single-handed grip on mask.

FIGURE 3.12 Two-handed grip on mask incorporating jaw thrust.

If the chest does not rise, the airway is not clear. The usual cause is failure to correctly apply the airway-opening techniques discussed previously. The first step to try is to readjust the head-tilt/chin-lift position and try again. If this is not successful, the jaw-thrust manoeuvre should be tried (see Figure 2.3). Failure of both the head-tilt/chin-lift and jaw-thrust manoeuvres should lead to suspicion that a foreign body is causing the obstruction.

Once breathing has restarted, replace the bag-valve-mask system with a simple face mask and reservoir. Because of the internal valves it is not possible to spontaneously breathe through the bag-valve-mask system.

Chest tubes

In cases with a significant haemothorax or pneumothorax (particularly tension pneumothorax), ventilation may be compromised and insertion of a chest drain is mandatory (see Section 34).

Gastric tubes (see Section 44)

Insertion of a gastric tube is essential after intubation, and may also relieve respiratory distress in spontaneously breathing patients with abdominal emergencies or gastric stasis. It allows decompression of a stomach full of air from both bag and mask ventilation as well as air swallowed by a distressed patient. Without a gastric tube, the patient may vomit or there may be aspiration of stomach contents. In addition, venting of stomach gas will avoid diaphragmatic splinting. A nasogastric tube will increase airway resistance through the nose, which in a spontaneously breathing infant with respiratory failure can be significant. An orogastric tube has less effect on ventilation, but is less readily tolerated and less easily fixed in position.

Further information

Additional breathing procedures are described in Sections 32-35

Circulation: equipment and skills for maintaining the circulation

Details of how to undertake the following procedures are covered in Sections 36-43.:

- peripheral venous cannulation
- blood sampling from an IV cannula
- intraosseous cannulation and infusion
- cut-down long saphenous venous cannulation
- insertion of central venous catheters

Management of cardiac arrest

Cardiac arrest occurs when there is no effective cardiac output. Before any specific therapy is started, effective basic life support must be established.

Four cardiac arrest rhythms can occur:

1 asystole
2 pulseless electrical activity (including electromechanical dissociation)
3 ventricular fibrillation
4 pulseless ventricular tachycardia.

These are divided into two groups.

1. Asystole and pulseless electrical activity, which do not require defibrillation, are called 'non-shockable' rhythms.
2. Ventricular fibrillation and pulseless ventricular tachycardia, which do require defibrillation, are called 'shockable' rhythms.

Section 3 Advanced Life Support for the infant

Reversible causes of cardiac arrest

The causes of cardiac arrest in infancy are multifactorial, but the two commonest final pathways are through hypoxia and hypovolaemia. All reversible factors are conveniently remembered as the 4Hs and 4Ts (see below). Sometimes cardiac arrest is due to an identifiable and reversible cause, such as shock due to massive haemorrhage, septicaemia or severe diarrhoea. In the trauma setting, cardiac arrest may be caused by severe hypovolaemia or tension pneumothorax or pericardial tamponade (see Textbook).

It is often appropriate to give an early IV bolus of Ringer-lactate or Hartmann's solution (10 mL/kg in an infant), as this will be supportive in cases related to severe hypovolaemia. In addition, however, a tension pneumothorax requires definitive treatment. Continuing blood replacement and the prevention of haemorrhage may also be required.

Rapid identification and treatment of reversible causes such as hypovolaemic shock, hypothermia, electrolyte and acid–base disturbance, tension pneumothorax and pericardial tamponade are vital.

During CPR it is important to continually consider and correct reversible causes of the cardiac arrest based on the history of the event and any clues that are found during resuscitation.

The 4Hs and 4Ts

1 Hypoxia is a prime cause of cardiac arrest in infancy, and its reversal is key to successful resuscitation.
2 Hypovolaemia may be significant in arrests associated with trauma, gastroenteritis and sepsis. It requires infusion of crystalloid, and in the case of haemorrhage, blood should be given.
3 Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic abnormalities may be suggested by the patient's underlying condition (e.g. renal failure), tests taken during the resuscitation or clues from the ECG. Intravenous calcium (0.2 mL/kg of 10% calcium gluconate) is indicated in infants with hyperkalaemia and hypocalcaemia.
4 Hypothermia requires particular care. A low-reading thermometer must be used to detect it.
5 Tension pneumothorax and cardiac tamponade are especially associated with Pulseless Electrical Activity (PEA) and are often found in trauma cases.
6 Toxic substances, resulting either from accidental or deliberate overdose or from an iatrogenic mistake, may require specific antidotes.

Non-shockable cardiac arrest
Asystole

This is the most common cardiac arrest rhythm in infants. The response of the heart to prolonged severe hypoxia and shock (which are the usual pathologies in these groups) is progressive bradycardia leading to asystole.

The ECG will distinguish asystole from ventricular fibrillation, ventricular tachycardia and pulseless electrical activity. The ECG appearance of ventricular asystole is an almost straight line; occasionally P-waves are seen (see Figure 3.13). Check that the appearance is not caused by an artefact (e.g. a loose wire or disconnected electrode). Turn up the gain on the ECG monitor.

FIGURE 3.13 ECG appearance of asystole.

Pulseless electrical activity (PEA)

This is the absence of a palpable pulse or other signs of life despite the presence on the ECG monitor of recognisable ECG complexes that normally produce a pulse (see Figure 3.14). PEA is treated in the same way as asystole, and is often a pre-asystolic state.

PEA can occur with major trauma, often with an identifiable and reversible cause such as severe hypovolaemia, tension pneumothorax or pericardial tamponade. PEA is also seen in hypothermic patients and in those with electrolyte abnormalities.
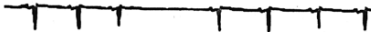
FIGURE 3.14 Pulseless electrical activity (PEA) in an infant with no pulse or signs of life.

Management of asystole/PEA in infants

The first essential step is to establish ventilations and chest compressions effectively. Ensure an open airway, initially using an airway manoeuvre to open the airway and stabilising it with an airway adjunct.

Ventilations are provided initially by bag and mask with high-concentration oxygen.

Provide effective chest compressions at a rate of 100–120 per minute with a compression : ventilation ratio of 15:2. The depth of compression should be at least one-third of the antero-posterior diameter of the chest, and compressions should be given in the middle of the lower half of the sternum. Ideally a cardiac monitor is attached. Properly performed basic life support is key to any chance of

Section 3 Advanced Life Support for the infant

successful resuscitation from cardiac arrest. Ensure that the person performing chest compressions is keeping the correct rate and depth of compression, and if possible change operator every 2 to 3 minutes, to avoid fatigue causing poor performance.

If asystole or PEA is identified, give adrenaline 10 micrograms/kilogram (0.1 mL of 1:10 000 solution/ kg) intravenously or intra-osseously (IO) in infants. Adrenaline increases coronary artery perfusion, enhances the contractile state of the heart and stimulates spontaneous contractions. The drug is best given through a central line, but if one is not in place it may be given through a peripheral line. Where there is no existing IV access, the IO route is recommended as the route of choice, as it is rapid and effective. In each case, the adrenaline is followed by a normal crystalloid flush (2 mL).

If available, and as soon as is feasible, a skilled and experienced operator (usually an anaesthetist) should intubate the infant's airway. This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Once the patient has been intubated and compressions are uninterrupted, the ventilation rate should be around 20-30 breaths per minute. It is important for the team leader to check that the ventilations remain adequate when chest compressions are continuous. An algorithm for non-shockable rhythms is shown in Figure 3.15.

During and following adrenaline treatment, chest compressions and ventilation should continue. It is vital that chest compressions and ventilations continue uninterrupted during advanced life support, as they form the basis of the resuscitative effort. The only reason for interrupting compressions and ventilation is to shock the patient if necessary (see below), and to check the rhythm. A brief interruption may be necessary during difficult intubation. Giving chest compressions is tiring for the operator, so if enough personnel are available, change the operator frequently and ensure that they are achieving the recommended rate of 100–120 compressions per minute together with a depression of the chest wall by at least one-third of the antero-posterior diameter of the chest.

At intervals of about 2 minutes during the delivery of chest compressions, pause briefly to assess the rhythm on the monitor. If asystole persists, continue CPR while again checking the electrode position and contact.

- If there is an organised rhythm, check for a pulse and signs of life.
- If there is a return of spontaneous circulation (ROSC), continue post-resuscitation care, increasing the ventilation rate to 20 -30 breaths per minute.
- If there is no pulse and no signs of life, continue the protocol.
- Give adrenaline about every 4 minutes at a dose of 10 micrograms/kg IV/IO in infants.
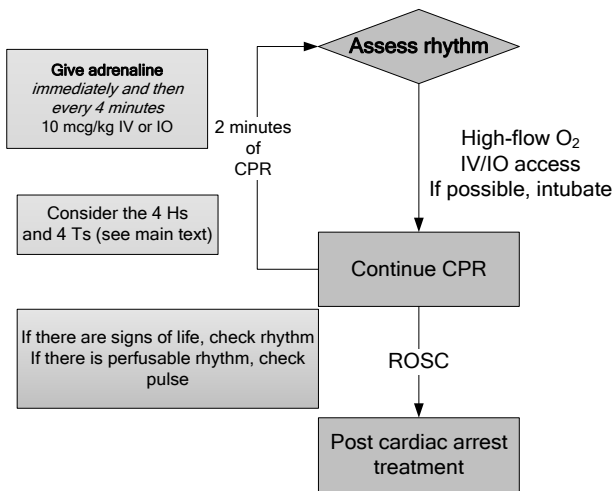
FIGURE 3.15 Algorithm for the treatment of non-shockable (asystole and PEA) rhythms in infants..CPR, cardiopulmonary resuscitation; IV, intravenous; IO, intra-osseous; ROSC, return of spontaneous circulation

Shockable cardiac arrest

These arrhythmias are rare in infants but either of them may be expected in patients with sudden collapse, hypothermia, poisoning by tricyclic antidepressants, or cardiac disease. The protocol for ventricular fibrillation (VF) (see Figure 3.16) and pulseless ventricular tachycardia (pVT) (see Figure 3.17) is the same, and is shown in Figure 3.18.
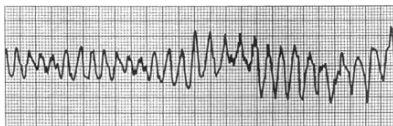
FIGURE 3.16 An episode of ventricular fibrillation.
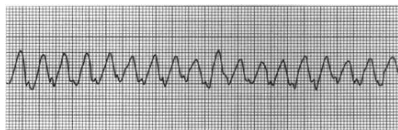
FIGURE 3.17 Ventricular tachycardia
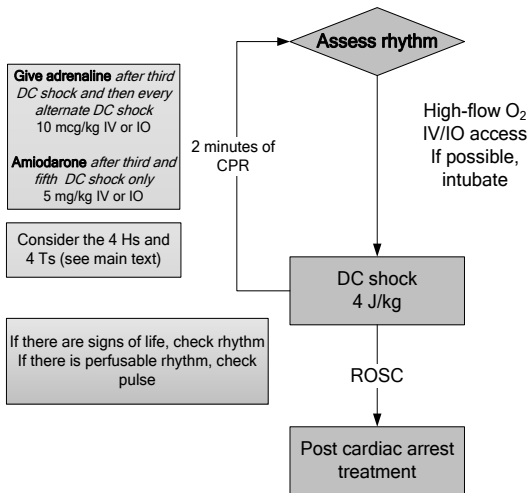
FIGURE 3.18 Algorithm for the treatment of shockable (VF and pVT) rhythms in infants. CPR, cardiopulmonary resuscitation; IV, intravenous; IO, intra-osseous; ROSC, return of spontaneous circulation.

If the patient is being monitored, the rhythm can be identified before significant deterioration occurs. With immediate identification of VF/pVT, asynchronous electrical defibrillation of 4 joules/kg in infancy should be undertaken immediately and the protocol continued as described below.

In unmonitored patients, basic life support will have been started in response to the collapse, and VF/pVT will be identified when the cardiac monitor is put in place.

Section 3 Advanced Life Support for the infant

An asynchronous shock of 4 joules/kg should be given immediately and CPR immediately resumed without reassessing the rhythm or feeling for a pulse. Immediate resumption of CPR is vital because there is a pause between successful defibrillation and the appearance of a rhythm on the monitor. Cessation of chest compressions will reduce the likelihood of a successful outcome if a further shock is needed. However, no harm accrues from 'unnecessary' compressions.

Paediatric paddles (4.5 cm) should be used for infants. One electrode is placed over the apex in the mid-axillary line, while the other is placed immediately below the clavicle just to the right of the sternum. If the paddles are too large, one should be placed on the upper back, below the left scapula, and the other should be placed on the front, to the left of the sternum.

For infants under 1 year of age, a manual defibrillator which can be adjusted to give the correct shock is recommended. However, if an AED is the only defibrillator available, its use should be considered, preferably with paediatric attenuation pads.

If the shock fails to defibrillate, attention must revert to supporting coronary and cerebral perfusion as in asystole. Although the procedures for stabilising the airway and obtaining circulatory access are now described sequentially, they should be undertaken simultaneously under the direction of a resuscitation team leader.

The airway should be secured, the patient ventilated with high-flow oxygen, and effective chest compressions continued at a rate of 100–120 per minute, with a compression depth of at least one-third of the antero-posterior diameter of the chest, and a ratio of 15 compressions to 2 ventilations. As soon as is feasible, a skilled and experienced operator should intubate the infant's airway. This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Once the patient has been intubated and compressions are uninterrupted, the ventilation rate should be 20 breaths per minute. It is important for the team leader to check that the ventilations remain adequate when chest compressions are continuous.

Obtain circulatory access. Whenever venous access is not readily obtainable, intra-osseous access should be considered early on in infants, as it is rapid and effective. In each case any drug is followed by a crystalloid flush (2–5 mL).

Two minutes after the first shock, pause the chest compressions briefly to check the monitor. If VF/VT is still present, give a second shock of 4 joules/kg and immediately resume CPR, commencing with chest compressions. Consider and correct reversible causes (the 4Hs and 4Ts) while continuing CPR for a further 2 minutes.

Section 3 Advanced Life Support for the infant

Pause briefly to check the monitor. If the rhythm is still VF/VT, give a third shock of 4 joules/kg.

Once chest compressions have resumed, give adrenaline 10 micrograms/kg in infants IV and amiodarone 5 mg/kg intravenously or intraosseously, flushing after each drug.

After completion of the 2 minutes of CPR, pause briefly to check the monitor, and if the rhythm is still VF/VT give an immediate fourth shock of 4 joules/kg and resume CPR.

After a further 2 minutes of CPR, pause briefly to check the monitor and if the rhythm is still shockable, give an immediate fifth shock of 4 joules/kg.

Once chest compressions have resumed, give a second dose of adrenaline 10 micrograms/kg and a second dose of amiodarone 5 mg/kg intravenously or intra-osseously. An amiodarone infusion can be continued if there is refractory VF/pVT of 15 mg/kg over 24 hours in infancy.

After completion of the 2 minutes of CPR, pause briefly before the next shock to check the monitor. Continue giving shocks every 2 minutes, minimising the pauses in CPR as much as possible. Give adrenaline after every alternate shock (i.e. every 4 minutes) and continue to seek and treat reversible causes.

Note: After each 2 minutes of uninterrupted CPR, pause briefly to assess the rhythm on the monitor.

In addition, if at any stage there are signs of life, such as regular respiratory effort, coughing or eye opening, stop CPR and check the monitor.
- If the rhythm is still VF/VT, continue with the sequence as described above.
- If the rhythm is asystole, change to the asystole/PEA sequence.
- If organised electrical activity is seen, check for signs of life and a pulse. If there is ROSC, continue post-resuscitation care.
- If there is no pulse (or a pulse of < 60 beats/minute) and no other signs of life, continue the asystole/PEA sequence.

In VT or VF that does not respond to the above sequence consider giving magnesium sulphate 25–50 mg/kg.

Sodium bicarbonate

Section 3 Advanced Life Support for the infant

If VF/VT is due to tricyclic antidepressant overdose or hyperkalaemia, sodium bicarbonate may be helpful. Give 1 mmol/kg (1 mL/kg of an 8.4% solution or 2 mL/kg of a 4.2% solution) in infants.

Amiodarone

Amiodarone is the treatment of choice in shock-resistant ventricular fibrillation and pulseless ventricular tachycardia. The dose of amiodarone for VF/pulseless VT is 5 mg/kg via rapid IV/IO bolus in infants.

Lidocaine is an alternative to amiodarone if the latter is unavailable. The dose is 1 mg/kg IV or IO as a bolus in infants.

It is DC shock that converts the heart back to a perfusing rhythm, not the drug. The purpose of the anti-arrhythmic drug is to stabilise the converted rhythm, and the purpose of adrenaline is to improve myocardial oxygenation by increasing coronary perfusion pressure. Adrenaline also increases the vigour and intensity of ventricular fibrillation, which increases the success rate of defibrillation.

Drugs used in non-shockable and shockable cardiac arrest
Oxygen

Although 100% oxygen must be used during the resuscitation process, once there is return of spontaneous circulation (ROSC) this can be detrimental to tissues that are recovering. Pulse oximetry should be used to monitor and adjust for oxygen requirement after a successful resuscitation. SaO_2 should be maintained in the range 94–98%. **Always ensure that oxygen delivery is discontinued during defibrillation shocks, to avoid the risks of explosions and fire.**

Adrenaline

Adrenaline is the first-line drug for treatment of cardiac arrest. Its effect is to increase blood flow to the brain and myocardium by constricting alternative arterioles. It renders the myocardium more susceptible to defibrillation.

The initial IV or IO dose is 10 micrograms/kg (0.1 mL/ kg of 1 in 10 000 solution) in infants. In infants with no existing IV access, the intra-osseous route is recommended as the route of choice, as it is rapid and effective. In each case, adrenaline is followed by a 0.9% saline flush (2mL).

Sodium bicarbonate

Good basic life support is more effective than alkalizing agents, which may be considered if spontaneous circulation has not returned after the first or second dose of adrenaline. Sodium bicarbonate is recommended in the treatment of patients with VT/VF due to hyperkalaemia and tricyclic antidepressant overdose (see above).

Section 3 Advanced Life Support for the infant

The dose is 1 mmol/kg in infancy (1 mL/kg of an 8.4% solution or 2 mL/kg of 4.2% solution).

- Sodium bicarbonate must not be given in the same intravenous line as calcium, otherwise precipitation will occur.
- Sodium bicarbonate inactivates adrenaline and dopamine, so the line must be flushed with Ringer-lactate or Hartmann's solution if these drugs are subsequently given.
- **Sodium bicarbonate must not be given via the intra- tracheal route.**

Glucose

Hypoglycaemia is defined as a glucose concentration of less than 2.5 mmol/litre (45 mg/dL).(see Section 22)

All infants can become hypoglycaemic when seriously ill. Blood glucose levels should therefore be checked frequently, and hypoglycaemia must be corrected. If it is suspected and blood glucose levels cannot be measured, always give 2.5 mL/kg of 10% glucose preferably IV or IO or alternatively enterally (via a gastric tube).

Section 4 Pain control in the infant

Special issues with regard to pain in the newborn infant
- Most studies (some of them controlled) have shown that neonates (both premature and full term) react to pain.
- Infants can easily be forced to put up with suffering.
- Small doses should be measured and given with an oral syringe.
- Adequate general anaesthesia, using morphine when needed, should be given for all surgical procedures on neonates.
- Local anaesthetics must be used when they would be used in an older child undergoing the same procedure.

Pain control during procedures in neonates
- A sugar-dipped dummy, coated with 2 mL of 25–50% sucrose 2 minutes before the procedure, can be helpful.
- Breastfeeding during procedures may be equally helpful.
- In all cases, comfort and containment (swaddling) should be provided by a parent or nurse.

Local anaesthetic drugs
Infiltration (the most widely used method)
Lidocaine 0.5–2%
- Used for rapid and intense sensory nerve block.
- Onset of action is within 2 minutes; the procedure must not be started until an anaesthetic effect is evident.
- Effective for up to 2 hours.

Doses:
infants maximum dose given locally 3 mg/kg – 0.3 mL/kg of 1% solution or 0.6 mL/kg of 0.5% solution (Preparation of lidocaine 0.5% solution.
Combine:
− lidocaine 1%, 1 part
Ringer-lactate or Hartmann's solution or sterile distilled water, 1 part.

Do not use local anaesthetic containing adrenaline in areas served by an end artery or with a poor blood supply (e.g. finger, toe, penis), as tissue necrosis will occur.

Local infiltration into an abscess is not recommended, because local anaesthetics are ineffective in inflamed tissues.

Complications of local anaesthesia;
Prevention of complications
Use the lowest effective dose.
- Inject slowly.
- Avoid accidental injection into a vessel. There are three ways of doing this:

- − the moving needle technique (preferred for tissue infiltration): the needle is constantly in motion while injecting, which makes it impossible for a substantial amount of solution to enter a vessel
- − the plunger withdrawal technique (preferred when considerable amounts are injected into one site): the syringe plunger is withdrawn before injecting, and if blood appears the needle is repositioned and another attempt is made
- − the syringe withdrawal technique: the needle is inserted and the anaesthetic is injected as the syringe is being withdrawn.

Symptoms and signs of lidocaine allergy and toxicity Lidocaine can be absorbed through mucous membranes in a large enough dose to be toxic.
Symptoms of allergy: shock, redness of skin, skin rash/hives, bronchospasm, vomiting, serum sickness.

Table 4.1 Lidocaine toxicity

Mild toxicity	Severe toxicity	Life-threatening toxicity (very rare)
Numbness of lips and tongue	Sleepiness	Tonic–clonic convulsions
Metallic taste in mouth	Disorientation	Respiratory depression or arrest
Dizziness/lightheadedness	Muscle twitching and shivering	Cardiac depression or arrest
Ringing in ears	Slurred speech	
Difficulty in focusing eyes		

- • Direct intra-arterial or IV injection of even a small amount may result in cardiac arrhythmias and convulsions (see above).
- • Resuscitative facilities and healthcare professionals with resuscitative skills should be present.

Local anaesthetics given through the surface of the skin or mucous membranes

1. Lidocaine: apply on gauze to painful mouth ulcers before feeds (apply with gloves, unless both the family member and the patient are HIV-positive, in which case the family member does not need protection from infection). It acts within 2–5 minutes.

2. TAC (tetracaine–adrenaline–cocaine): apply to a gauze pad and place over open wounds; it is particularly useful when suturing. Care needs to be taken close to mucous membranes to avoid toxicity from absorption of cocaine. If available, other

Section 4 Pain control for the infant

topical anaesthetic agents such as lidocaine–adrenaline–tetracaine seem to be equally effective and avoid the potential toxicity associated with cocaine.

Systemic drug treatment for pain

Paracetamol ±
Aspirin ±
Non-steroidal anti-inflammatory drugs (NSAIDs)

STEP 1

Morphine for moderate to severe pain
± Paracetamol or NSAIDs or both
± Adjuvants *

STEP 2
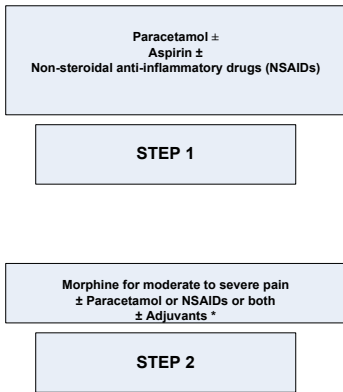
*An adjuvant is another drug (e.g. steroid or anxiolytic) or type of treatment that can prevent and relieve pain.

Non-opiate analgesics

Paracetamol

1. This is the most widely used analgesic and anti-pyretic.
2. It does not cause respiratory depression.
3. It is dangerous in overdose but a very safe and effective drug if used in recommended doses.
4. It is given by mouth, rectally or intravenously.
5. The maximum daily dose should not be given for more than 3 days.
6. Caution is needed in patients with liver impairment.
7. There are no anti-inflammatory effects.
8. *Paracetamol can be combined with NSAIDs and both have a morphine-sparing effect, lowering the dose, and therefore severity of side effects of morphine.*

Non-steroidal anti-inflammatory drugs (NSAIDs) (e.g. ibuprofen, diclofenac)

Opiate analgesics: Morphine

Morphine is the most important drug in the world for pain control, and the WHO recommends that it should be universally available.

In resource-limited countries it is mostly administered orally, which is useful for chronic or anticipated pain but less effective for acute pain. The latter requires IV administration of morphine.

Section 4 Pain control for the infant

At an appropriate dose, analgesia occurs without impaired consciousness. Nausea and vomiting are rare with oral treatment, but when morphine is given intravenously for the first time it may produce this side effect.

Intravenous use of morphine

- In single doses it has minimal haemodynamic effects in a supine patient with normal circulating blood volume.
- In hypovolaemic patients it can contribute to hypotension. Therefore: monitor the patient's cardiovascular status and have an IV fluid bolus of Ringer-lactate or Hartmann's solution ready (10 mL/kg for a neonate).
- In excessive dosage it can produce a dose-dependent depression of ventilation and decreased respiratory rate, leading to apnoea.
- Patients who are receiving morphine in hospital (where it is often intravenously administered) need observation and/or monitoring of respiratory rate and sedation.
- Morphine is better controlled by the IV than the IM route. If using the IV route, give a small dose initially and repeat every 3–5 minutes until the patient is comfortable. Individuals vary widely with regard to the dose needed to provide pain relief.
- It is rarely appropriate to give morphine intramuscularly, and **for patients who are in shock, giving morphine IM is dangerous, as it can be initially poorly absorbed, and then quickly absorbed when perfusion improves, potentially leading to too high a blood level of the drug.**
- Intravenous morphine can be dangerous in situations of raised intracranial pressure without the means to provide respiratory support.

Naloxone
Naloxone is an opiate antagonist that reverses the sedative, respiratory-depressive and analgesic effects of morphine, and so should be given to treat morphine overdose.

Table 4.2 Orally administered drugs for mild or moderate pain

Medicine	*Neonate 0–29 days*	*Infant 30 days to 3 months*	*3 months to 1 year*
Paracetamol	10 mg/kg every 6–8 hours. Maximum 4 doses in 24 hours 5mg/kg if jaundiced	10 mg/kg every 4–6 hours	15mg/kg every 6 hours
Ibuprofen	***Not recommended***	***Not recommended***	50 mg 3 times daily after food
Diclofenac	***Not recommended***	***Not recommended***	Over 6 months 0.3 to 1mg/kg 3 times daily after food

Section 4 Pain control for the infant

Preparations:
Paracetamol: oral suspension, 120 mg/5 mL,

Table 4.3 Intravenous paracetamol for mild or moderate pain

Age/weight	Dose	Maximum dose in 24 hours
Preterm over 32 weeks	7.5 mg/kg every 8 hours	25 mg/kg
Term neonate	10 mg/kg every 4–6 hours	30 mg/kg
Infant > 1 month	15mg/kg every 4-6 hours	60mg/kg

Intravenous paracetamol
1. Paracetamol IV is formulated as a 10 mg/mL aqueous solution (in ready-to-use 50-mL and 100-mL vials for infusion over 15 minutes).
2. It is useful, effective and safe.
3. The peak analgesic effect of IV paracetamol occurs within 1 hour, with a duration of approximately 4–6 hours.
4. Ensure that the correct dose is given, as serious liver toxicity can occur in overdose.
5. Side effects are rare. They include rashes, blood disorders and hypotension on infusion.
6. Caution is needed in patients with severe renal impairment, severe malnutrition (and thus low reserves of hepatic glutathione) or dehydration.
7. Paracetamol helps to reduce the amount of narcotics required when used in combination with them.

Table 4.4 recommended doses for oral and rectal morphine

Age	Initial dose (adjust according to response)	Interval
1–3 months	50–100 micrograms/kg	Every 4 hours
3-12 month	80-200 micrograms/kg	Every 4 hours

Parenteral morphine
Intravenous morphine is only needed if oral or rectal preparations are not going to be absorbed (e.g. in shock) or where rapid emergency onset is needed. IV morphine is potentially less safe, especially if staff shortages mean that the correctly calculated dose is not given.

Table 4.5 Intermittent IV (bolus) morphine dosage. We suggest that the total dose recommended is drawn up in 10mls 0.9% saline and that 2ml boluses of this solution are given every 3--5 minutes until the patient is comfortable. Also, if pain returns despite regular paracetamol/nonsteroidal analgesia, further dose of oral/IV morphine can be given within 6 hours if the respiratory rate is normal and the patient is not sedated.

Age	Dose	Interval	Maximum dose
Neonate	25–100 micrograms/kg	Every 6 hours	
1–6 months	100--200 micrograms/kg	Every 6 hours	2.5 mg/dose
6-12 months	100 micrograms/kg	Every 4 hours	2.5 mg/dose

Monitoring during morphine administration:
Side effects occur only in overdose and should not be seen at the doses stated here. They include the following:

1. Respiratory depression. If the respiratory rate is:
•   < 20 breaths/minute in patients aged less than 12 months

Alert medical staff and ensure that bag/valve/mask and naloxone are available.

Monitor SaO_2 as appropriate (it should be higher than 94% in air).
2. Constipation. Use prophylactic laxatives.
3. Monitor for urinary retention.
4. Patients with liver and renal impairment may need lower doses and longer time interval between doses
5. .Caution in patients with head injuries

Naloxone doses to reverse opioid induced respiratory depression

Always ventilate with bag/valve/mask first if patient is unresponsive before giving naloxone. This is because arrhythmias and pulmonary oedema can be caused if naloxone is given to a patient with high blood carbon dioxide concentrations.

Dose: Neonate to1 month of age: 5-10 microgram/kg repeated every 2--3 minutes until adequate response
One to 12 months 5-10 micrograms/kg and then if need a repeat give 100 micrograms/kg

Preparations: Ampoule 20 microgram/mL
Give IV or IM if IV is not possible. Repeat after 2–3 minutes if there is no response; the second dose may need to be much higher (up to 100 micrograms/kg). An IV infusion

may be needed if protracted or recurrent depression of respiration occurs because naloxone is short acting compared with most opioids.

(For the newborn, to treat respiratory depression due to maternal opioid administration during labour or delivery 200 microgram as a single IM dose is recommended or 60 microgram/kg)

Section 5: Immediate care of the newly born infant

The baby at risk of developing problems at birth
Preterm birth: Preventative strategies
Minimising the risk of surfactant deficiency: this can be halved if the mother is given a short course of high-dose steroid treatment before delivery:
- dexamethasone, 12 mg IM, two doses 12 hours apart
- or dexamethasone, 6 mg IM, four doses 12 hours apart.

Stopping premature uterine contractions (see Maternity section of Textbook)
- Give 20 mg nifedipine orally. Up to three further doses can be given at 30-minute intervals if uterine contractions persist.
- If this stops labour, give 20 mg nifedipine orally three times a day for the next 3 days.

Other problems associated with preterm birth include:
- Increased risk of infection and hypothermia
- Nutritional difficulty. Breast milk is ideal, and everything possible should be done to help the mother sustain her lactation until the baby is ready to feed reliably from the breast. A limited ability to suck and swallow usually appears from 32 weeks' gestation, but it remains unpredictable, unreliable and uncoordinated until 36 weeks' gestation. In the event that breastfeeding cannot be initiated immediately after birth, mothers should be encouraged to start expressing breast milk, to be given by nasogastric tube or cup and spoon.
- Partial breastfeeding can also help the mother to sustain her lactation, but in any event the mother should regularly express milk. Some mothers might find expressing breast milk difficult and may require help with this.

Infection prevention in the newborn
It is important to identify babies at risk of infection prior to delivery. If identified, the mother should be given antibiotics. Many of the babies who become infected during delivery develop respiratory signs very soon after birth, but in a few, the features are those of neonatal sepsis. In addition, there are a proportion of babies who are initially asymptomatic, and therefore prophylactic antibiotics should be commenced in the infant if there are risk factors for infection.

When to consider antibiotics for the mother and new-born infant
1. Symptomatic ascending infection in utero needs urgent treatment. If this is overlooked, both the mother's and the baby's life will be in danger.
2. Asymptomatic infection is, however, a much commoner problem. This occasionally progresses so rapidly once labour starts that, unless treatment is started at once, the baby will die even if the most appropriate antibiotic is given immediately after birth. Because such infection by definition is silent, it is important that treatment be considered in any mother going into active spontaneous labour before 35 weeks' gestation.
3. Membrane rupture can be both a sign of and a risk factor for, ascending bacterial infection. What most people mean by premature rupture of membranes (PROM) is really preterm pre-labour rupture of membranes (PPROM), where the membranes rupture before there is an overt sign of uterine activity or any detectable uterine

contractions. When this happens in the preterm baby, it is often a sign of the start of some sort of ascending infectious process. This process has already weakened the amniotic membranes and may stimulate the onset of preterm labour. Antibiotics must be given to the mother.

4.   Treatment of the mother with antibiotics should also be considered **at any gestation** if the mother's membranes rupture more than 18 hours before delivery. If premature rupture of membranes occurs before the onset of premature labour contractions then infection is more likely.

Maternal fever (> 38°C) in labour is a strong indication for initiating antibiotics for the mother. Similarly, foul-smelling or purulent liquor requires IV antibiotic treatment of the newborn from birth without waiting for any signs of infection.

In mothers with PPROM who show signs of being clinically infected give IV antibiotics.

In PPROM where there is no evidence of infection and no evidence of labour you can delay delivery by 1 week or more (on average) by giving the mother amoxicillin or, better still, erythromycin. In mothers who are in active labour 5 or more weeks before term and who give a clear history that the membranes had ruptured before they were able to detect any uterine contractions, the risk of the baby becoming infected during delivery can be reduced substantially by giving antibiotics IV (ideally probably both penicillin and gentamicin) during labour.

Antibiotic management of perinatal infection

Where facilities allow, a blood count, C-reactive proteins and blood cultures should be taken before starting antibiotics. Because a range of bacteria can be involved, **treatment of the baby** needs to protect against group B streptococcal, coliform and Listeria infection, making a combination of ampicillin and gentamicin the best strategy:

●   Give ampicillin 50–100 mg/kg IV 12-hourly **plus** gentamicin 5 mg/kg every 24 hours IV if more than 32 weeks' gestation, and 3 mg/kg if less than 32 weeks.

The WHO recommends that a neonate with risk factors for infection (i.e. membranes ruptured > 18 hours before delivery, maternal fever > 38°C before delivery or during labour, or foul-smelling or purulent amniotic discharge) should be treated with prophylactic antibiotics (IM or IV ampicillin and gentamicin) for at least 2 days. After this the neonate should be reassessed and treatment continued only if there are signs of sepsis (or a positive blood culture).

Hypothermia (see below)

Hypothermia seriously increases the risk of surfactant deficiency and hypoglycaemia, and must be avoided.

Management at delivery of a baby not needing resuscitation

1.   Deliver the baby on to the mother's abdomen or a warm surface, dry and cover.
2.   Clamp cord when pulsation stopped, usually between 1 and 3 minutes after birth
3.   Prevent hypothermia by nursing skin to skin.
4.   Initiate early breastfeeding.
5.   Minimise infection by hand washing, cord care and using clean materials.
6.   Give an injection of vitamin K.

Section 5 Immediate care of the newborn

Most babies do not need any resuscitation at birth but only require basic care to prevent infection and hypothermia. Extensive mouth suction, facemask oxygen, and vigorous stimulation in order to provoke a first gasp or cry are unnecessary rituals without clinical justification. As long as the baby becomes pink, and starts to breathe without distress, most babies should stay with their mothers and have a first feed at the breast within minutes of birth.

Colostrum, the initial milk with a clear, yellowish and thick appearance, is an extremely nutritious and concentrated feed rich in immunoglobulins (it is only present during the first 3 to 4 days). Mothers should be informed of its benefits and that it is ideal for their baby to feed on this as soon after birth as possible and as frequently as possible.

Preventing heat loss after birth
● Once any necessary resuscitation process has finished and as soon as the baby becomes pink, and starts to breathe without distress, give to the mother for skin-to-skin contact and the first feed at the breast. This not only prevents hypothermia but also helps better uterine contraction following delivery.
● The practice of using water or oil to clean the skin within a few hours of birth before the body temperature has stabilised can make the baby dangerously hypothermic. A simple drying of the skin with a warm towel or sheet is all that is required.
● The mother's own body is the most effective source of warmth, so long as the baby is first well dried to minimise evaporative heat loss. A larger sheet or blanket can then be used to protect both mother and baby from the convective heat loss caused by draughts.
● Babies have relatively large heads. Covering the head with a shawl, blanket or woollen cap can reduce heat loss.
● Heat and water loss through the skin can be a particular problem in babies born before 32 weeks' gestation. This can be limited initially by wrapping all but the face in a clean plastic wrapping such as cling film or a food- grade plastic bag with a hole cut in the end of the bag for the baby's head to protrude, for a few hours after birth. Remember that plastic over the face can cause death from suffocation. If plastic bags or cling film are not available, the preterm baby must be wrapped well in a clean towel or blanket. However, plastic bags are very good for preventing heat loss, but only in conjunction with an overhead heat source or heated mattress. If the fluid in the bag gets cold it will cool the baby quicker than drying and wrapping.
● Heat supplementation can be provided by locally built and maintained incubators, overhead heating systems, but most effectively in low resource settings by skin-to-skin (kangaroo) care.
● The first bath must be delayed for at least 24 hours.

Managing the placenta, cord and umbilical stump
Babies often become relatively anaemic 4 to 6 months after birth because red cell production does not keep pace with body growth. This problem can be minimised by ensuring that blood intended for the baby is not left in the placenta at birth.

If the baby is held higher than the placenta (i.e. on the mother's abdomen) while the cord is still pulsating, blood will drain out of the baby and into the placenta, so hold the (covered) baby just below the placenta for 1-2 minutes if the cord is still pulsating. If the

Section 5 Immediate care of the newborn

cord is clamped before it stops pulsating, this will also reduce the normal 'placental transfusion' at birth, especially if the uterus has not yet contracted.

Do not artificially 'milk' blood from the placenta into the baby, it is possible to leave the baby with so many red cells that the blood becomes thick and polycythaemic. Neonatal polycythaemia (see Section 13) has many complications, including putting the circulation under strain, making the capillary circulation very sluggish, and increasing the risk of jaundice.

The cord must be cut cleanly, and the cut stump secured in a manner that minimises the risk of late haemorrhage.

The umbilical stump will shrink as it dries out. Plastic clamps that shut down further as the cord starts to shrink are very effective. They are relatively inexpensive, and they do make it possible to cut the stump about 2–4 cm from the skin. An elastic band, if carefully applied, is a cheap and well-tested alternative. A stump that is left too long provides a reservoir where bacteria can breed and multiply with great speed, and therefore should not be permitted. A length of 2–4 cm is ideal.

Recent studies in resource-limited countries have shown that for home deliveries the application of 4% chlorhexidine solution immediately after birth can prevent omphalitis. Other possible antiseptics include surgical spirit or iodine.

Often the cord manifests a little 'stickiness', which may be of no concern. However, a local antiseptic should be applied if a red skin flare suggests early spreading staphylococcal cellulitis. Such babies **must also be given** an oral anti-staphylococcal antibiotic (cloxacillin or flucloxacillin). If the skin around the stump becomes oedematous with increasing redness, IV cloxacillin or oral flucloxacillin (25 mg/ kg three times a day for 7 days) must be given. Babies who are systemically unwell always need urgent broad-spectrum antibiotic treatment, IV or IM, for septicaemia.

Ensuring that all mothers receive at least two injections of tetanus toxoid 1 month apart during pregnancy can eliminate the risk of neonatal tetanus.

The risk of cross-infection during or after birth
The WHO estimates that infection is responsible for one-third of all neonatal deaths (over 3000 deaths a day). Kangaroo (skin to skin) mother care has significantly reduced the number of neonatal deaths from infection by colonising babies with the mother's bacteria rather than those of the hospital.

Perform neonatal examination before discharge of all babies

If not already given, ensure that vitamin K 1 mg IM is administered

Resuscitation of the newborn Infant

Most infants breathe well and do not need active 'resuscitation' at birth. Simply drying the infant with a warm dry sheet/towel will in most cases stimulate a cry from the infant thus expanding the lungs. Attempts to clear the airway, to stimulate breathing, or to give facial oxygen are unnecessary. Therefore, **routine airway suctioning is not needed.**

Around 5% of infants do not breathe spontaneously after delivery. However, breathing can be started in almost all these infants by correctly applying bag-and-mask ventilation. With lung inflation there is an immediate and easily detectable rise in heart rate. It may be difficult to identify the infant's pulse rate by palpation at any site, so the best way to determine the heart rate is to listen over the chest with **a stethoscope**.

Far less commonly, infants are born cyanosed, shocked, limp and hypotonic. Around 1% do not respond to bag- and-mask ventilation, and need further help with advanced resuscitation.

Guidelines from the 2010 International Liaison Committee on Resuscitation (ILCOR) The main changes that have been made to the Neonatal Life Support (NLS) guidelines relevant to resource-limited countries are as follows:
● The use of food-grade plastic wrapping (cling film) is recommended to maintain body temperature in very small preterm infants.
● Ventilatory resuscitation may be started with air. However, where possible, additional oxygen should be available if there is no rapid improvement in the infant's condition.
● Adrenaline should be given by the IV route, as standard doses are likely to be ineffective if given via a tracheal tube.
● If there are no signs of life after 20 minutes of continuous and adequate resuscitation efforts, discontinuation of resuscitation may be justified.

Guidelines from 2015 ILCOR
The temperature of newly born infants should be actively maintained between 36.5°C and 37.5°C after birth. Even the mild hypothermia that was once felt to be inevitable and therefore clinically acceptable carries a risk. The admission temperature should be recorded as a predictor of outcomes as well as a quality indicator.

Early use of nasal CPAP should also be considered in those spontaneously breathing preterm infants who are at risk of developing respiratory distress syndrome (RDS).

First call for help
Start the clock or note the time. Keep the infant dry and warm and assess their breathing and heart rate.
● Infants are born small and wet. They get cold very easily, especially if they remain wet and in a draught. Whatever the problem, **dry the infant well, including the head.** Remove the wet towel, and wrap the infant in a dry towel. It is helpful if the towels are warm.
● There is good evidence that for very preterm infants (30 weeks' gestation or earlier), placing the infant under a radiant heater after drying, and immediately covering

the head and body, apart from the face, with clean plastic wrapping, is the most effective way of keeping these very small infants warm during resuscitation.

●　　　　Drying the infant immediately after delivery will provide significant stimulation during which colour, tone, breathing and heart rate can continue to be assessed.

●　　　　Observing the breathing, skin colour, heart rate and tone helps to document the infant's condition and assess their response to resuscitation.

●　　　　Reassess these observations regularly (particularly the heart rate), every 30 seconds or so, throughout the resuscitation process. The first sign of any improvement in the bradycardic infant will be an increase in heart rate.

●　　　　A healthy infant may be born blue but will have good tone, will cry within a few seconds of delivery, will have a good heart rate (the heart rate of a healthy new-born infant is about 120–150 beats/minute) and will rapidly become pink during the first 90 seconds or so. An ill infant will be born pale and floppy, not breathing, and with a slow (<100) or very slow (<60) heart rate.

●　　　　The heart rate of an infant is best judged by listening to the chest with a stethoscope. It can also sometimes be felt by palpating the base of the umbilical cord, but a slow rate at the cord is not always indicative of a truly slow heart rate, and, if the infant is not breathing, must not delay the immediate application of lung inflations. In addition, if the infant is not breathing, feeling for peripheral pulses is potentially harmful as it delays the onset of life-saving lung inflations. If a stethoscope is not available, you can listen to the heart by placing your ear on the infant's chest or using a Pinard's stethoscope.

Airway: open the airway and keep it open

●　　　　Before the infant can breathe effectively the airway must be open.

●　　　　The best way to achieve this in an infant who is not breathing well is to place the infant on their back with the head in the neutral position (i.e. with the neck neither flexed nor extended). Most newborn infants will have a relatively prominent occiput, which will tend to flex the neck if the infant is placed on their back on a flat surface. This can be avoided by placing some support using a folded nappy or cloth under the shoulders of the infant, but be careful not to overextend the neck.

●　　　　If the infant is floppy it may also be necessary to apply chin lift or jaw thrust. The best way to stabilise an infant's condition at birth is to ensure that the upper airway remains unobstructed. The infant will then have little difficulty in drawing air into the lungs when it takes its first spontaneous gasp or cry. Unfortunately, books often talk of the need to keep the airway 'clear', giving the false impression that the infant is going to find it difficult to breathe unless all the fluid and mucus is first sucked out of the way. There is no evidence that this is ever necessary unless the infant is meconium stained or does not breathe well. Moreover, blind deep suction of the nose or mouth can stimulate the vagus nerve, leading to bradycardia, apnoea and laryngospasm. However, the upper airway of any infant who is born limp and hypotonic certainly needs to be opened and maintained in just the same way as the airway of any other unconscious patient. In an unconscious patient, pharyngeal tone decreases even more than it does during sleep, causing the upper airway to narrow or close. When such a patient is laid on their back the tongue also falls back, further obstructing the airway. There are three key ways to counter this:

1　　　　Hold the head in the neutral position and
2　　　　Support the chin or
3　　　　Push the jaw forward

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Figure 6..1 Neutral position of the head and neck in a new-born infant

Neutral position
Face parallel to surface
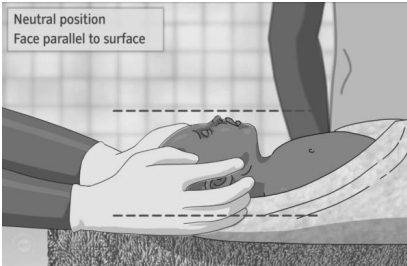
Figure 6.2 Chin lift in a new-born infant

If tone is poor it may also be necessary to support the chin. It is important to support the bony part of the chin. Pressure anywhere else may merely push the base of the tongue backwards, making matters worse.

If tone is very poor it may be necessary to use one or two fingers under each side of the lower jaw, at its angle, in order to push the jaw forwards and outwards (Jaw thrust), but this will require a second person to give inflation and ventilation breaths with the bag-valve-mask.

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Figure 6.3 Jaw thrust in a new-born infant. Note that the operator's thumbs are in a position to hold a mask in place.

Tracheal obstruction
Although it is rare for debris to completely block the trachea, this should be suspected if an infant tries to breathe but remains cyanosed and bradycardic, with laboured breathing and marked inter-costal and/or sternal recession. This is one of the few situations where tracheal intubation can be life-saving.

How to manage meconium
Around 15% of infants have meconium- stained liquor at birth. Meconium aspiration syndrome (MAS) can occur in about 1 in 10 such infants. The development of MAS is not entirely dependent on suctioning at birth. It is possible for infants to aspirate meconium into the large airways in utero if there is hypoxia and gasping. However, some infants may aspirate meconium during delivery, and these are the ones in whom the risk of MAS can be reduced by suctioning when the infant's head is on the perineum.

Studies based on experience from Africa and India have shown that suctioning the mouth of infants with meconium-stained liquor during birth when the head is at the perineum has dramatically reduced the incidence of meconium aspiration syndrome (MAS) and death. There is subsequently no need for further suctioning after birth if the infant breathes well.

What to do if the trachea appears to be blocked
If the infant is born through meconium and is unresponsive at birth, the oropharynx should be inspected and cleared of meconium. If intubation skills are available, the larynx and trachea should also be cleared under direct vision.
If meconium has entered the trachea, resuscitation here is only possible if the accumulated debris can be immediately removed. The easiest way to do this is to pass an endotracheal tube and then remove the debris by direct suction to the endotracheal tube. Sometimes the meconium debris is so large that it cannot be sucked through the tube. The tube can then be removed and replaced with a clean tube to clear the

remaining obstructive material. Suction may also make it easier to see the larynx during intubation.

Giving mask ventilation for the infant who is not breathing before the meconium has been cleared (as above) may force the meconium deeper into the lungs.

Breathing
● If the infant is not breathing adequately give **five inflation breaths** as soon as possible. Until now the infant's lungs will have been filled with fluid. Aeration of the lungs in these circumstances is best with slow inflations at pressures of about 30 cmH$_2$O with the bag and mask; these are called 'inflation breaths'. These initial ventilation breaths should last 2–3 seconds each. The aim is to mimic the initial breaths taken by a normal infant to open the airways, remove lung fluid and achieve its functional residual capacity.

If the baby is very preterm, such inflation breaths may injure immature lungs: give lower pressure ventilation breaths (see below) in this situation.
● If the heart rate was below 100 beats/minute initially then it should rapidly increase as oxygenated blood reaches the heart. If the heart rate does increase then you can assume that you have successfully aerated the lungs and there is adequate tissue oxygenation. If the heart rate increases but the infant does not start breathing, then continue to provide regular ventilation breaths at a rate of about 30–40 breaths/minute until the infant starts to breathe.
● The chest may not move during the first one or two breaths as fluid is displaced. Adequate ventilation is usually indicated by either a rapidly increasing heart rate or a heart rate that is maintained at more than 100 beats/minute. Therefore reassess the heart rate after delivery of the first five breaths. It is safe to assume that the chest has been inflated successfully if the heart rate improves. Once the chest has been seen to expand and the heart rate has increased, ventilation should be continued at a rate of 30–40 breaths/minute. Continue ventilatory support until regular breathing is established.
● If the heart rate does not increase following inflation breaths, then either you have not aerated the lungs or the infant needs more than lung aeration alone. By far the most likely possibility is that you have failed to aerate the lungs effectively. If the heart rate does not increase, and the chest does not passively move with each inflation breath, then you have not aerated the lungs.
Under these circumstances consider the following:
o Are the infant's head and neck in the neutral position?
o Do you need jaw thrust?
o Do you need a second person's help with the airway or to squeeze the bag? A relative or ward orderly can be shown immediately how to effectively squeeze the self-inflating bag while you ensure that the mask is held firmly and in the best position on the face over the mouth and nose with the airway open.
o Is there an obstruction in the oropharynx (laryngoscope and suction under direct vision)?

Bag-and-mask inflation of the lung
Having positioned the infant correctly it is usually easy to inflate the lungs using a self-inflating bag and mask.

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Remember that the infant cannot breathe through the bag-valve-mask system, so do not leave the mask sealed to the face and expect the infant to breathe from the bag. The valve between the bag and the mask prevents this. When the infant is breathing, remove the mask and watch closely to ensure that adequate breathing continues.

Most infants will respond to bag-and-mask ventilation by gasping and then starting to breathe on their own without further support. If this does not happen, it is still easy to confirm that lung aeration has been achieved, because the heart rate will rise reliably and consistently above 100 beats/minute. If lung aeration has been achieved and the infant still has a slow heart rate, proceed to support the circulation (C).

 If oxygen is available, applying this through the bag and mask may also help.

Correct bag-and-mask ventilation is the single most important skill needed to provide active resuscitation.
There is good evidence that most infants can be resuscitated using mask resuscitation without any need for tracheal intubation. However, some infants require early intubation, so the equipment and the skill to intubate should be available.

Evidence suggests that air is safer for initial resuscitation. However, where possible additional oxygen should be available for use if there is not a rapid improvement in the infant's condition. Equally, hyperoxia should be avoided, especially in the preterm infant. If a pulse oximeter is available this can be done. Try to keep the SaO2 between 88% and 95%.

When to cut and clamp the cord in an infant who needs resuscitation at birth
There are advantages to delaying clamping of the cord for 2 minutes after birth to allow placental transfer of blood to the infant (see above). However, it is important to ensure that by doing this there is no harm to the mother (e.g. if she needs resuscitation) or to the infant (e.g. if they require resuscitation). Usually **the umbilical cord is clamped and cut immediately if infant needs active resuscitation.**

Mouth-to-mouth and nose resuscitation (see Section 2)
Most current guidelines on neonatal care steer clear of discussing the role of mouth-to-mouth resuscitation. The risk of HIV infection or hepatitis has further fuelled that reluctance. However, there is no doubt that this can be an effective way of reviving an apparently lifeless infant in the absence of equipment. Remember the following:
●   Keep the upper airway open by optimising the position of the head and jaw as described above.
●   Cover the infant's nose and mouth with your mouth (or cover the mouth of a big infant and just pinch the nose).
●   Use the pressure you can generate with your cheeks, and try to aerate the lung by slow inflations for 2–3 seconds.
●   Only use as much air for each breath as you can keep in your cheeks (i.e. do not 'blow' air into the infant, but just small puffs).
●   Watch for chest movement, and allow time for lung recoil.
●   Once the chest starts to move, sustain what has been achieved with 20–25 artificial breaths/minute.

Checking progress before moving on

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●   If the heart rate has not risen to over 100 beats/minute after the five initial breaths or within 30 seconds of adequate ventilation, something is wrong. The most likely problem is that you have not successfully ventilated the infant. Never move on to deal with the issues covered under letter C of the resuscitation alphabet until you are quite sure you have achieved objectives A and B. To do so is quite futile. Chest compressions will never restore the circulation until the blood being massaged from the lung to the heart contains oxygen.

●   Look to see whether the chest moves each time you apply mask pressure. Movement should not be difficult to see once the first few breaths have aerated the lungs. It is usually easier to judge success with your eyes than with a stethoscope. In a newborn, breath sounds can be heard when only the airway is being aerated, so are not a good way to judge ventilatory success.

●   Check that the infant's head is well positioned. Check chin support and jaw thrust, and that the mask is correctly applied with no air leaks. Ask a second person to help you position the infant optimally and provide inflations by squeezing the bag while you hold the airway open and the mask in place.

●   Few infants need support with their breathing once their lungs have been aerated. Most will gasp, cry or breathe just as soon as an attempt is made to get air into the lungs, and then continue breathing adequately.

—   However, a few may benefit from further support if they do not start to breathe regularly, or only gasp occasionally. Some may have suffered severe hypoxia in utero, and a few may be drowsy because of drugs given to the mother during labour. Check that the heart rate remains normal (above 100 beats/minute) and that there is no central cyanosis (best judged by looking at the colour of the tongue).

—   Try to assess whether there is hypoxemia (cyanosis or SaO_2 less than 90% with a pulse oximeter), if the infant's breathing remains laboured and irregular or if the infant's colour remains blue. Give oxygen then if it is available, preferably with SaO_2 monitoring. Hyaline membrane disease, meconium aspiration syndrome, pneumonia or transient tachypnoea of the newborn are most likely.

Other possibilities include:
o   intra-partum pneumonia (common)
o   diaphragmatic hernia
o   pneumothorax
o   pulmonary hypoplasia (possibly associated with a skeletal or renal abnormality)
o   cyanotic congenital heart disease (although this usually takes a little time to appear)
o   persistent fetal circulation.

●   If breathing requires continuous support it is important to try and reduce mask inflation pressures to little more than half of what was needed to aerate the lung in the first place. It is easy to over-ventilate an infant with healthy lungs and to wash out so much of the carbon dioxide that normally provides the main stimulus to breathing that all such activity stops for a while. There is evidence that sustained over-ventilation can reduce cerebral blood flow.

Endotracheal intubation
As discussed earlier, most infants who need resuscitation can be managed with bag-valve-mask intubation. However, occasionally endotracheal intubation is required, but this must be done by someone skilled and practised in the technique. It is most likely to be required for prolonged resuscitation, in meconium aspiration, and in preterm infants with surfactant deficiency. A straight-bladed laryngoscope is preferred, and tube sizes

are around 3.5 mm for a term infant and 2.5 mm for a preterm infant. Sizes larger and smaller than these should be available.

Resuscitation of preterm infants
●   Infants with surfactant deficiency may have difficulty in expanding their lungs, and in developing a normal functional residual capacity at birth.
●   However, the preterm lung is quite a delicate structure with relatively little elastic support, and any use of undue pressure or excessive ventilation during resuscitation can damage the lungs.

While an inspiratory pressure of 30 cmH2O may well be necessary to begin aerating the lungs at birth, the pressure should be reduced as rapidly as possible to a level that ensures that the chest is moving adequately. The key aim must be to conserve such surfactant as already exists by sustaining the lung's functional residual capacity (an objective best achieved by providing at least 5 cmH$_2$O of positive end-expiratory pressure (PEEP). Aim to achieve this consistently throughout transfer to the nursery. This can be achieved using nasal prongs (nasal PEEP), thus avoiding tracheal intubation altogether (see Textbook).

Circulation: chest compressions
●   Most infants needing help at birth will respond to successful lung inflation with an increase in heart rate followed quickly by normal breathing. Chest compression should be started only when you are sure that the lungs are being aerated successfully.
●   If the heart rate remains very slow (less than 60 beats/minute) or absent following 60 seconds of ventilation with good chest movements, start chest compressions.
●   In infants, the most efficient method of delivering chest compressions is to grip the chest in both hands in such a way that the two thumbs can press on the lower third of the sternum, just below an imaginary line joining the nipples, with the fingers over the spine at the back. This can only be done if there is a second operator ventilating the lungs (see Figure 6.4).
●   If you are alone, the two-thumb method is not possible, as ventilations also need to be provided. In this situation, use the first two fingers of one hand to depress the lower sternum, while the other hand holds the mask in place (Figure 45.5). Then move the hand from the sternum to squeeze the bag.
●   Compress the chest quickly and firmly, reducing the antero-posterior diameter of the chest by about one-third.
●   Because oxygenation is such an important part of neonatal resuscitation, the recommended ratio of compressions to inflations in newborn resuscitation is 3:1.
●   Chest compressions move oxygenated blood from the lungs back to the heart and out into the ascending aorta. From there the two coronary arteries will then quickly deliver oxygen to the failing anoxic heart muscle. It is important to allow enough time during the relaxation phase of each compression cycle for the heart to refill with blood, at the same time ensuring that the chest is inflating with each breath.

Figure 6.4 Two-thumb compression of the chest, with a second operator ventilating the lungs, here using a T-piece as an alternative to bag and mask.

Figure 6.5 Two-finger chest compressions

● The rate of chest compressions is around 120/minute. However, with pauses for ventilation, the actual total number of compressions is less than 120/minute.

Drugs
Rarely inflation of the lungs and effective chest compression will not be sufficient to produce adequate circulation and perfusion in infants. In these circumstances, drugs may be helpful. However, drugs are needed only if there is no significant cardiac output despite effective lung inflation and chest compression.

Very few drugs have proved to be of benefit. The drugs used are adrenaline (1:10 000) and dextrose (10%). Drugs are best delivered via an umbilical venous catheter. In those where IV access is not possible, the intra-osseous route may be used. Each injection of a drug should be followed with a bolus of 2–3 mL of Ringer- lactate or Hartmann's solution. Unfortunately, most of the infants in whom cardiac output only returns after drug treatment require specialist neonatal care (often with mechanical ventilation) and do not survive to discharge. Most of those who do survive later develop profound disabling spastic quadriplegia.

Where the cause of the infant's terminal apnoea is a sudden and much more abrupt hypoxic event (such as shoulder dystocia or an occasional case of late cord prolapse)

these reservations may be less valid. Here there is at least anecdotal evidence that the outlook is much less bleak if the circulation can be restarted.

Acidosis not serious enough to precipitate circulatory standstill (asystole) will nearly always correct itself spontaneously within 90 minutes once the circulation has been restored and the infant starts to breathe for him- or herself. It does not therefore call for sodium bicarbonate, the use of which is controversial. Indeed, giving bicarbonate may increase carbon dioxide levels, worsening intracellular acidosis, and increases the amount of sodium that the potentially compromised kidney will need to excrete over the next few days.

- *Adrenaline*: The recommended dose of adrenaline is 10 micrograms/kg body weight (0.1 mL/kg body weight of 1:10 000 solution). If this is not effective, a dose of up to 30 micrograms/kg (0.3 mL/kg body weight of 1:10 000 solution) may be tried. Ideally, have ready-made and well-labelled 1:10 000 adrenaline solutions available on all emergency trolleys. In situations where this is not available in a ready-made state it could be prepared by adding 1 mL of 1:1000 solution to 9 mL of normal saline or Ringer-lactate or Hartmann's solution. It is potentially dangerous to leave inadequately labelled and made up doses of adrenaline around, as giving the same volume of 1:10000 as 1:1000 solution could cause cardiac arrest. Do not use a higher dose by these routes (IV) as it is harmful.
- *Glucose*: The recommended dose of glucose is 200 mg/ kg (2 mL/kg of 10% dextrose). Higher concentrations or larger doses can induce hyperglycaemia, which is associated with cerebral oedema and cerebral haemorrhage, and may lead to rebound hypoglycaemia. It is known that severe hypoglycaemia is rare immediately after birth, but tends to present after 1–2 hours. However, hypoglycaemia (less than 2.5 mmol/litre (45 mg/dL)) is a potential problem for stressed or hypoxic neonates, so 10% dextrose should be considered in cardiac arrest, as the heart will not recover in the presence of hypoglycaemia. This should be followed by an infusion of 5 mL/kg/hour of 10% glucose if there is confirmation of hypoglycaemia by a blood test. This should be continued until feeding is well established.

Never give any drug into the umbilical artery.

- Naloxone (nalorphine) can be used to reverse profound opiate-induced respiratory depression, but has no real role in neonatal resuscitation. If it does prove necessary, it is best to give it intramuscularly and give a full 200-microgram 'depot' dose irrespective of body weight. If naloxone is given as a single dose IV it will be eliminated from the body faster than the opioid drug, causing a return of the respiratory depression, and therefore the infant may stop breathing again without a naloxone infusion. Naloxone does not reverse the respiratory depressing effects of non-opiate drugs.

Acute blood loss as a cause of circulatory arrest (circulatory volume support)
- Sudden acute blood loss is a rare, but often unrecognised, cause of acute circulatory collapse. Bleeding from an aberrant placental blood vessel (vasa praevia) or snapped umbilical cord can rapidly lead to hypovolaemic death. The response to a rapid generous infusion of any IV fluid can be equally dramatic. Speed is of the essence. Circulatory collapse probably does not occur until the infant has lost 30–40 mL/kg of blood, but 20 mL/kg of Ringer-lactate or Hartmann's solution will usually

reverse the immediate critical hypovolaemia rapidly. The initial intravenous fluid bolus should be 10 mL/kg of Ringer-lactate or Hartmann's solution or blood group O Rh-negative blood (if immediately available). This can be repeated once if there is no or only minimal response. A packed red cell transfusion using group-specific or group O Rh-negative duly cross-matched blood can be given later to correct the associated anaemia.

- Other less well-recognised causes of hypovolaemic collapse include acute feto–maternal blood loss, sudden twin-to-twin transfusion, and accidental incision of the placenta during Caesarean delivery and cord ligature that has come off and not been detected.

Apart from these specific indications, fluid should not be used during neonatal resuscitation. There is no evidence to suggest benefit from routine use, which only compounds the problem of fluid balance that can develop over the next 2 to 3 days if severe intra-partum stress causes secondary renal failure.

Poor response to resuscitation
If the infant either fails to respond or shows a poor response to resuscitation, the most likely problem is inadequate oxygenation. The following steps should be considered:
- Check the airway and ventilation.
- Check for technical faults if using equipment.
  o Is the oxygen attached?
  o Is the airway blocked?
  o Is the endotracheal tube in the correct place?
- Re-examine the chest to see if a pneumothorax has developed. This is not common, but may cause a problem. Drain a tension pneumothorax with a small cannula over needle (21 gauge) in the second intercostal space in the mid-clavicular line. This should be followed by the insertion of a chest drain (see Section 34).
- Consider the possibility of a congenital heart lesion (see Section 25) if the infant remains cyanosed despite breathing and having a good heart rate.
- Consider the possibility of maternal opiates or sedation, such as diazepam or phenobarbitone, if the infant is pink, well perfused, but requires assisted ventilation.
- Shock, caused by acute blood loss, should respond to a rapid bolus of 10–20 mL/kg of O-negative blood (see above).
- Consider the possibility of hypoglycaemia.

Stopping resuscitation
Even with the most effective resuscitation, not all infants will survive. If the infant has been without a cardiac output after 20 minutes of resuscitation and does not respond despite effective ventilations and chest compressions, the outcome is unlikely to be altered by the use of drugs, although these should be considered. The decision to stop resuscitation should be taken by the most senior healthcare worker present, and the reason for the decision should be clearly documented. Explain sensitively to the parents that the infant has died. The infant should then be handled in accordance with cultural preference and practice.

Vitamin K prophylaxis against haemorrhagic disease of the newborn
Following resuscitation/stabilisation, all new-born infants should receive vitamin K 1 mg IM. Vitamin K is given to prevent haemorrhagic disease of the new-born (HDN), which may cause significant bleeding and even death. The IM route is preferred as it provides

a depot over many weeks. Similarly, neonates requiring surgery, those with birth trauma, preterm infants and those exposed in utero to maternal medication that is known to interfere with vitamin K are at especially high risk of bleeding and must be given vitamin K 1 mg IM. This is often forgotten in the rush to get the infant to the nursery.

Figure 6.6 Algorithm for resuscitation of the baby at birth

Section 7 Care of the newborn: preterm and low birth weight
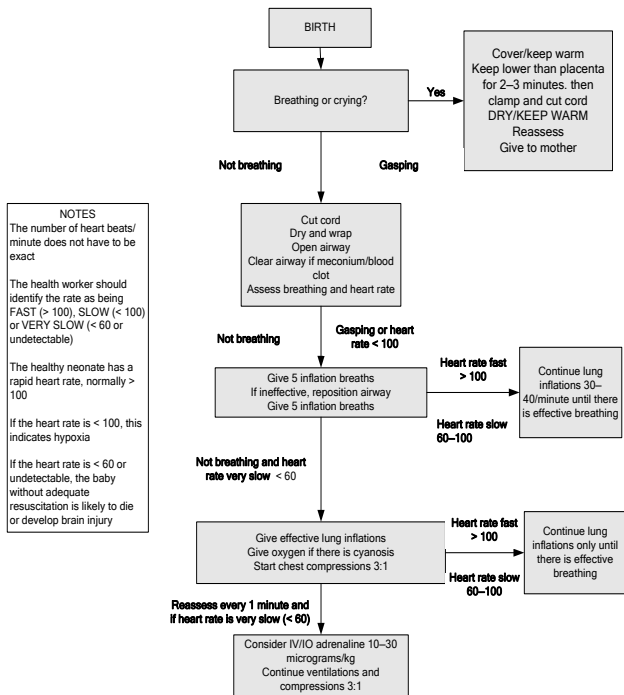
Clinical care of the neonate

Prematurity and low birth weight
A low-birth-weight infant is one weighing less than 2.5 kg at birth. Low birth weight may be attributable to preterm delivery or intrauterine growth restriction.

A preterm infant is one born before 37 completed weeks have elapsed since the first day of the last menstrual period (259 days). Most preterm infants are born after 32 weeks' gestation.

A small-for-gestational age (SGA) infant is one whose birth weight falls below the 10th percentile on a birth weight centile chart.
Probably at least 25% of SGA infants are just constitutionally small by virtue of maternal weight, and not secondary to poor placental perfusion. The mean birth weight of infants born to mothers 4 feet 10 inches (147 cm) tall is about 500 grams less than that of infants born to mothers 6 foot 0 inches (183 cm) tall. This discrepancy increases to about 1 kg if extremes of mid-pregnancy weight are also taken into account.

Intrauterine growth restriction (IUGR) refers to a slowing of fetal growth velocity. Most but not all IUGR infants are SGA at birth. Some IUGR infants are wasted.

A large-for-gestational age (LGA) infant is one whose birth weight is greater than the 90th percentile on a birth weight centile chart.

For most clinical purposes it is sufficient to classify infants as 'low birth weight', 'preterm' or 'small-for- gestational age'.

Assessing gestational age
Sometimes a mother cannot recall the date of her last menstrual period. The infant's gestational age can then be assessed to within ± 2 weeks based on a combined physical and neurological score (*see* Table 3.3.1 in Textbook). Wasted infants underscore on physical criteria.

Low birth weight and/or preterm infants

Infants with birth weight in the range 2.25–2.5 kg
These infants are normally strong enough to start feeding themselves. They need to be kept warm and closely observed for infection, but otherwise no special care is required.

Infants with birth weight in the range 1.75–2.25 kg
These infants sometimes need extra care, but can normally stay with their mothers to receive feeding and warmth, especially if skin-to skin contact can be maintained. Close monitoring by a healthcare worker is required.

Feeds can be started within 1 hour of delivery. Many of these infants will be able to suck and can be breast- fed. Those who cannot breastfeed should be given expressed breast milk with a cup. When the infant is sucking well from the breast and gaining weight on a daily basis, they can be weaned off cup feeds.

Section 7 Care of the newborn: preterm and low birth weight

These infants should be reviewed at least twice a day to assess their feeding ability, fluid intake and the presence of any danger signs including signs of serious bacterial infection. Such problems will necessitate close monitoring in a neonatal nursery (if available) in a similar way to the very low birth weight infant. The risk of keeping the infant in hospital (including hospital- acquired infections) should be considered.

Infants with birth weight below 1.75 kg

These infants are at risk of hypothermia, apnoea, hypoxaemia, sepsis, feed intolerance and necrotising enterocolitis. The smaller the infant, the greater these risks. All infants with a birth weight below 1.75 kg should be admitted to a special care or neonatal intensive care unit (if available).

Other treatments for low-birth-weight and/ or preterm infants

Oxygen

Oxygen should be administered via nasal cannulae, nasal prongs or a head box if there are signs of respiratory distress, such as moderate to severe recession (preterm infants may show mild recession with normal breathing), and definitely in the presence of cyanosis.

Pulse oximetry to measure oxygen saturation is a vital part of oxygen usage in the preterm infant. Retinopathy of prematurity (ROP), previously known as retrolental fibroplasia, which leads to lifelong blindness in many cases, is caused by high blood levels of oxygen saturation in the preterm infant. There is no good evidence for the optimum oxygen saturation for preterm infants. On the one hand it is important to avoid hypoxia, which would lead to brain damage, as the infant is likely to have respiratory problems from surfactant deficiency, and on the other hand unrestricted oxygen may cause ROP, which would lead to blindness.

Current advice is that the infant born at or before 32 weeks' gestation or weighing less than 1500 grams at birth should have a target oxygen saturation of 86–92%, which is higher than the oxygen saturation to which the fetus is exposed in utero.

Prevention of hypothermia

To prevent hypothermia, nurse the infant in skin-to-skin contact between the mother's breasts ('kangaroo care') or clothed in a warm room, or in an incubator. A hot water bottle wrapped in a towel can be useful for keeping the infant warm if no power for heating is available, but take care not to burn the infant.

Aim for an axillary temperature of 36.5–37.5°C, with the feet warm and pink. When the mother is asleep or if she is ill, a clean incubator can be used. Incubators should be washed with disinfectant between infants, and should be of a basic design that can be used appropriately by the staff available.

Fluids

It is best to give fluids enterally. However, if the infant is not well enough (e.g. due to severe respiratory distress), give IV fluids (see Section 14). Initially, consider giving approximately 2–4 mL of expressed breast milk every 1 to 2 hours through a nasogastric tube. This can be adjusted depending on the weight and the amount of IV fluids that the infant is receiving. With increasing age and weight gradually increase the volume and timing

of each feed (the maximum time interval between feeds should not exceed 4 hours). The total fluid intake of enteral feeds plus IV fluids per 24 hours should adhere to the following fluid management guidelines:

- 60 mL/kg on day 1
- 80–90 mL/kg on day 2
- 100–120 mL/kg on day 3
- 120–150 mL/kg on day 4
- 150–180 mL/kg thereafter.

Some infants can be fed with a cup. Use only expressed breast milk if possible. If 2–4 mL per feed is tolerated (i.e. there is no vomiting, abdominal distension, or gastric aspirates of more than half the feed) the volume can be increased by 1–2 mL per feed each day. Ideally, aim to have feeding established in the first 5 to 7 days so that the IV fluids can be tapered off. Reduce or withhold feeds if signs of poor tolerance occur. As the infant grows, recalculate the feed volume based on the higher weight. Feeds may be increased over the first 2 weeks of life to 150–180 mL/ kg/day based on a 3- to 4-hourly feeding pattern.

How to give gastric feeds (see also Section 44 on gastric tube placement)
Place the baby's lips on the breast even though he or she is unable to suck or attach before each feed. Place expressed breast milk (EBM) in the syringe. Only use fresh milk or milk that has been stored in a refrigerator, and that has not been out of the fridge for more than 1 hour in a hot climate.
Check that the tube is in the stomach before every feed or administration of enterally given drugs. Also check that there is not more than 10% of the previous feed in the stomach by gentle aspiration using a 2- or 5-mL syringe. Connect the syringe containing EBM and remove the plunger, giving the milk by gravity over 10–15 minutes per feed. Only if the feed does not flow in should you gently push with the plunger for a few seconds only to get it started. Never push the whole feed in. Observe the infant closely during the feed for signs of respiratory distress that might be due to lung aspiration. Replace the tube every 7 days, or sooner if it is blocked.

Give enteral feeds only if there is no abdominal distension or tenderness, bowel sounds are present, meconium has been passed, and there is no apnoea, low aspirates, no vomiting and adequate stool output.

TABLE 7.1 Guide to volumes of each feed given every 3–4 hours at different infant weights

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8
Total (mL/kg/day)	60	80	100	120	140	150	160	170
1.25–1.4 kg	10	15	18	20	22	24	25	≥26
1.5–1.9 kg	15	17	19	21	23	25	27	≥27
2.0–2.4 kg	20	22	25	27	30	32	35	≥35
≥2.5 kg	25	28	30	35	35	≥40	≥45	≥50

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Danger signs associated with infection in the neonate

Common danger signs

- Infant feeding less well than before.
- Infant lying quietly and making few spontaneous movements.
- Hypothermia or fever > 38ºC.
- Capillary refill time > 3 seconds.
- Respiratory rate ≥ 60 breaths/minute.
- In-drawing of the lower chest wall when breathing, *or* grunting.
- Cyanosis.
- History of a convulsion.

Less common but important signs

- Low respiratory rate (< 20 breaths/minute) or apnoea.
- Jaundice.
- Abdominal distension.

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Blood glucose levels
Check blood glucose levels every 6 hours until enteral feeds are established, and immediately if there are any danger signs of infection.

Infection
Observe carefully and constantly for infection (see Box above)

Apnoea
Monitor for apnoea, ideally with a pulse oximeter (which are now affordable and available in many resource-limited countries), supplemented by close visual monitoring of the infant by the mother or a close relative.

Feed intolerance
Observe closely for this and regularly check with the mother

Discharge and follow-up of low-birth-weight infants
Discharge when:

- there are no danger signs (see Box above) of serious infection
- the infant is gaining weight (at least 20 grams per day for 3 consecutive days) on breastfeeding alone
- the infant is able to maintain temperature in the normal range (36–36.5°C axillary) in an open cot or with skin- to-skin care
- the mother is confident and able to take care of the infant.

Section 7   Care of the newborn: preterm and low birth weight

Low-birth-weight infants should be given all scheduled immunisations by the time of discharge from the health facility or soon after.

Counsel the parents before discharge on:
- exclusive breastfeeding
- keeping the infant warm
- the danger signs that necessitate seeking care (see Table 7.2), plus advice on when to return for healthcare
- basic life-saving actions to use in the event of an emergency, particularly mouth-to-mouth and nose ventilation if prolonged apnoea occurs.

Low-birth-weight infants should be followed up at regular intervals following discharge for weighing, assessment of feeding, and assessment of general health until they have reached 2.5 kg in weight.

TABLE 7.2 Danger signs for parents of discharged newborns

Seek advice immediately if any of the following occur:	Seek advice very quickly if any of the following occur:
Convulsion(s)	Infant refuses feeds
ANY bleeding	Minor diarrhoea or vomiting
Severe diarrhoea or vomiting	Minor breathing problems
Infant appears unresponsive	Infant is less active/interested
Severe breathing problems	Infant feels abnormally hot
Infant feels cold	Jaundice

Enteral feeding for the newborn infant in hospital

Type of milk

Breast milk will provide the nutrients required by almost all infants. However, for preterm infants the following supplements are needed:

Vitamin D supplements

Term breastfed infants generally do not need extra vitamin D. However, this is only true if the mother has an adequate vitamin D status. Maternal vitamin D deficiency during pregnancy and lactation is common in resource-limited countries, contributing to the low vitamin D content of breast milk. Newborn infants of mothers who have dark skin or wear concealing clothes are also at greater risk of vitamin D deficiency at birth.

Large amounts of calcium and phosphorus are transferred from the mother to the infant during the last 3 months of pregnancy, helping the infant's bones to grow. Therefore, a preterm

infant may not receive sufficient amounts of calcium and phosphorus for this purpose. Vitamin D helps the body to absorb calcium from the intestines and kidneys. Very preterm infants require adequate vitamin D supplements. Liver problems such as cholestasis and prolonged use of diuretics or steroids may also cause problems with blood calcium levels.

Therefore, without further supplementation, preterm and also some full-term breastfed infants may be at risk of vitamin D deficiency. This risk may be minimised either by supplementing the mother with large amounts of vitamin D (4000 IU/day) during pregnancy and lactation, or by supplementing the infant directly (400 IU/day) during the period of lactation.

Phosphorus supplements

These may be needed in the case of very small infants, who can become hypophosphataemic (i.e. with plasma phosphorus levels of < 1.5 mmol/litre). If untreated, this may result in metabolic bone disease. The addition of a concentrated phosphorus salt (50 mg/kg/day of phosphorus) to feeds will prevent this. Adding 0.05 mL/kg of a 4 mmol/ mL phosphorus solution to each of eight feeds per day will give 50 mg/kg/day of supplemental dietary phosphorus.

Vitamin A supplements

In resource-limited countries, vitamin A supplementation of the newborn infant reduces mortality. Preterm infants are at high risk of vitamin A deficiency. This important subgroup of our infant population is not only born with inadequate body stores of vitamin A, but is also often unable to tolerate routine oral supplementation. Vitamin A supplementation programmes significantly reduce infant mortality as well as the incidence of xerophthalmia, respiratory infection, and morbidity from gastrointestinal disease. Oral supplementation of 4000 IU/kg/day has been recommended for very-low-birth-weight (VLBW) (<1500 grams at birth) infants from establishment of full enteral feeding until discharge from the neonatal unit. Supplementing term newborn infants with vitamin A (100 000 IU as a single dose) within 48 hours of birth reduces infant mortality by almost 25%, with those of low birth weight deriving the greatest benefit. Alternatively, a single dose of 200000 units can be given to all postpartum mothers within 6 weeks of delivery, when the likelihood of pregnancy is very low, and when infants benefit most from the presence of vitamin A in breast milk.

Vitamin K supplements

All neonates should be given vitamin K 1 mg IM within 1 hour of birth. Those who require surgery, those with birth trauma, those who are preterm and those exposed before birth to maternal medication (that can interfere with vitamin K) are at high risk of bleeding and must be given vitamin K. If the need for surgery only becomes

apparent some time after birth, we suggest that a repeat dose should be given before surgery.

Multivitamin preparations

A multivitamin preparation (preferably containing adequate vitamins A and D) for preterm infants may be commenced from 3 weeks of age. A supply of vitamin D (400 IU/day) is particularly important for bone mineralisation.

Iron supplements

Iron supplements for preterm infants are usually commenced from about 6 weeks of age. Preterm infants have reduced iron stores compared with term infants, especially if the umbilical cord is clamped early. The daily dietary iron supplementation is 2–4 mg/kg of elemental iron, up to a maximum of 16 mg/day.

Breast milk banking

The WHO recommends that low-birth-weight babies who cannot be fed their own mother's breast milk should receive donor milk. The high maternal mortality and morbidity in low-income countries mean that there are many infants who cannot be put to the breast within a few hours of birth, and donor milk is suitable for them. In addition, there is evidence that human donor milk reduces the incidence of severe infection and necrotising enterocolitis (NEC) in low-birth-weight babies, compared with formula milk.

It is possible to establish safe donor milk banks in resource-limited settings, provided that there is a micro- biology laboratory able to process donor samples, and a power supply to keep the banked milk frozen. A nurse or other staff member must be trained in good hand hygiene, and the importance of labelling and storage. Only simple equipment is needed.

Milk can be collected from lactating mothers who are known to have tested negative to HIV and syphilis, and are non-smokers. Ideally they have also been screened for hepatitis infections. Milk is collected by hand expression under supervision (as 'drip milk' is lower in calories than expressed milk). A 1-mL aliquot from each donor's sample is sent to the microbiology lab. If colony-forming organisms are grown, the whole sample is discarded. Milk can be stored in a refrigerator while awaiting the microbiology results.

Milk is pasteurised by heating for 30 minutes at 62.5°C, and then cooled and frozen. Pasteurised frozen milk can be stored for up to 6 months from the date of collection. Supplementary formula feeding for the infants of mothers who cannot provide breast milk, or whose mothers have died, may be needed after discussion with the infant's carers.

Section 7   Care of the newborn: preterm and low birth weight

Fluid and electrolyte management for the neonate in hospital
When giving fluid or blood intravenously, best practice is to use an in-line infusion chamber/burette to avoid fluid overload.

Fluid requirements
Body water content is high at birth and urine output is low for the first few days. Therefore, giving large volumes of fluid in the first few days may make an infant oedematous and worsen any respiratory disease. A simple general rule is to start an ill newborn infant who cannot take enteral fluids (breast milk) on 60 mL/kg/day IV as 10% dextrose solution, increasing in daily steps of 20–30 mL/kg/day to a maximum of 140–180 mL/kg/day. However, in a small-for-gestational- age infant it may be necessary to begin with 70–90 mL/kg/ day in order to meet the glucose requirements.

Ideally, use a 100-mL paediatric intravenous burette where 60 drops = 1 mL and therefore 1 drop/minute = 1 mL/hour.

So for an infant weighing 1.8 kg on day 1: 1.8 × 60 = 108 mL
In each hour the fluid will be 108 ÷ 24 = 4.5 mL, which corresponds to 9 drops every 2 minutes.

The rate of insensible water loss (mainly through the skin) is high in some circumstances, particularly in infants under 29 weeks' gestation or when an overhead heater (radiant warmer) rather than an incubator is used. Helpful measures for reducing insensible water loss in such cases include the following:
• Place the infant from below the neck in a clean plastic bag to maintain humidity. Maintaining humidity helps to keep very premature infants warm by reducing evaporative heat loss.
• Clothe the infant, or wrap the body below the head with bubble wrap or aluminium kitchen foil (with the shiny side facing inward towards the infant).
• When an overhead heater is used, the infant should not be covered, and the heater output must be adjusted in direct response to the infant's skin temperature (typically achieved by a continuous temperature probe servo system). Alternatively, a plastic bag over the infant's body from the neck down can help to preserve heat.
• In the first week of life, high rates of insensible water loss will be reflected by high rates of weight loss (more than 10% of birth weight), and often an increase in the plasma sodium concentration to 150 mmol/litre or higher. If either occurs, the infant is dehydrated and fluid intake should be increased by 30 mL/kg/day. When nursing a low-birth-weight infant under an overhead heater, it is advisable to add an extra allowance of 30 mL/kg/day right from the start (i.e. start on day 1 at 90 mL/kg/day rather than 60 mL/kg/day).

Note, however, that even 30 mL/kg/day might not be enough to meet the insensible losses of a very preterm infant (29 weeks or less) under a radiant heater. Such infants are much better nursed in closed incubators.

In very-low-birth-weight infants, enteral feeds should be advanced slowly in 20–30 mL/kg/day increments. Infants who are being enterally fed but who are unable to breastfeed

can be given expressed breast milk by orogastric tube or cup. A general plan for fluid enhancement is as follows:

- day 1: 60 mL/kg/day
- day 2: 85 (range 80–90) mL/kg/day
- day 3: 110 (range 100–120) mL/kg/day
- day 4: 135 (range 120–150) mL/kg/day
- day 5 and thereafter: 165 (range 150–180) mL/kg/day.

Monitor the fluid intake by weighing the infant daily and recording the frequency of urine output.

To weigh the baby, first place a blanket in the scales, set them to zero and then place the baby naked in the scales and cover the infant with the blanket to keep them warm. Fluid intake may need to be adjusted frequently to maintain fluid balance. Urine output can be monitored by measuring the difference between wet nappies (diapers) and a dry one using accurate scales. Generally, expect at least eight wet nappies in a 24-hour period. Look out for signs of fluid overload (oedema) or dehydration. If possible measure the plasma electrolytes, but remember that these cannot be interpreted without information on body weight and urine output.

Electrolyte requirements when giving IV fluids

Sodium requirements
Infants over 48 hours of age need some sodium supplementation in a dose of 2–3 mmol/kg/day. This can most easily be given by adding 20 mL/kg of normal saline (0.9%) to the daily requirement of 10% glucose to make up the total daily fluid volume needed. This gives approximately 3 mmol of sodium per kg.

Adding sodium is open to many errors. Ready-made neonatal fluids are available in some countries, and may be used to avoid this problem in some situations. The sodium requirements of very preterm infants may be much higher as urinary sodium losses may approximate 10 mmol/kg/ day in those of 29 weeks' gestation or less.

Sodium can be commenced on the third day of life (after 48 hours) in infants receiving intravenous fluids, but if there is respiratory distress it is wise to wait until the diuresis associated with recovery begins (this is often delayed until the third or fourth day of life).

Potassium requirements
Potassium supplementation in a dose of 1–2 mmol/kg/ day will meet requirements and can be provided by adding mathematically correct and small amounts of potassium chloride to a 10% glucose fluid. If IV potassium is given, the plasma potassium concentration must be monitored daily. Potassium can be added to fluids but this should be done very carefully. Remember that too much IV potassium can be fatal. The concentration of KCl in

peripheral IV solutions should never exceed 40 mmol/litre. Do not add KCl until the urine output is well established.

Remember that it is best to give potassium and calcium supplements orally, unless very low serum values are identified.

Glucose requirements
Infusing glucose at the following rates will match the normal hepatic glucose output and therefore maintain the blood glucose concentration at an acceptable level:

- term infant: 3–5 mg/kg/minute
- preterm, appropriate weight for gestation: 4–6 mg/kg/ minute
- small for gestational age: 6–8 mg/kg/minute.

A solution of 10% glucose at 60 mL/kg/day will give 4 mg (0.22 mmol) glucose/kg/minute.

These infusion rates provide minimal glucose requirements to maintain a normal blood glucose level, but higher rates will be required for growth. Consider hyperinsulinism as a cause of the problem if an infant requires higher rates of infusion to maintain normoglycaemia. Always use 5% or ideally 10% glucose/dextrose for peripheral IV infusions; an umbilical venous catheter will be needed if high glucose requirements or limits on fluid volume necessitate a more concentrated solution which will be damaging to thin peripheral veins.

Composite maintenance fluid
An alternative way to make a simple composite maintenance fluid is by adding the following to give a total volume of 100 mL:
- 1/5 dextrose saline (0.18% normal saline with 5% dextrose) = 71 mL
- 7.4% KCl = 2 mL
- 10% calcium gluconate = 2 mL
- 25% dextrose = 25 mL
  Total volume = 100 mL

Each 100 mL of the above solution would contain dextrose 10%, KCl 2 mEq, Ca 2 mEq and sodium 2.5–3 mmol.
Any such mixture must be prepared under sterile conditions.
KCl must not be added until urine output is well established.

Drug use in the newborn infant
Relatively few drugs are needed to deal with most common neonatal emergencies.

The IV route should be used if the infant is already being given IV fluids, as this will reduce the amount of pain to which the infant is subjected. There are dangers associated with rapid administration or breaking into an existing IV line, leading to an

increased risk of sepsis. Erecting an IV line merely to administer drugs also risks exposing the infant to dangerous fluid overload, unless a syringe pump can be used to control the rate at which fluid is infused.

Section 8 Hospital problems: feeding difficulties

Common problems requiring hospital care

Many emergencies can be prevented by attention to good feeding practices, providing adequate warmth and preventing infection. The more preterm or low birth weight the infant, the more likely it is that the following complications will occur:

- feeding difficulties
- poor temperature control, especially hypothermia
- infection – prevention and early recognition and safe management are essential
- polycythaemia
- respiratory distress and apnoeic attacks
- bleeding
- jaundice and neonatal anaemia
- reduced conscious level and seizures, including hypoglycaemia
- surgical problems.

Feeding difficulties

Infants born after 34 weeks are generally mature enough to suck and swallow well, but may be less demanding of feeds than term infants. Attention to the following can help all newborn infants, especially those born preterm, to establish breastfeeding:

- Encourage early and prolonged skin contact.
- Encourage small frequent feeds by waking the infant every 2 to 3 hours and putting them to the breast.
- If the infant will not latch on and suck, the mother can be encouraged to express breast milk and offer it to the infant by cup and/or spoon or if not accepted by orogastric or nasogastric tube.
- If an otherwise well infant on breast milk feeds is experiencing inadequate growth, an inadequate milk supply may be the problem. There are several possible causes for this, which can usually be identified by listening to the mother and then watching the infant feed. A relaxed mother will have a good 'let-down' reflex which gives the infant the more calorie-rich hind milk as well as the fore milk. The mother can tell when she has 'let down' by a tingling feeling in her breasts, and the infant starts to swallow rapidly. The infant must latch on properly for feeding to be successful, and this may need some assistance from the midwife. The best way to increase the milk supply for a hungry infant who is not thriving is to increase the feed frequency. Breast milk works on a demand-and-supply system, so the more the infant demands, the more the breast supplies. If the infant is not feeding vigorously enough to increase the milk supply, the mother should express milk after feeding and give it to the infant as described above.
- Avoid giving formula or breast milk by bottle. A small feeding cup (about the size of a medicine measuring cup, with a smooth rim) or a spoon can be used to feed the infant.

Section 8 Hospital problems: feeding difficulties

- Give expressed breast milk via orogastric or nasogastric tube if the infant is too unwell to suck or drink from a cup.
- As the infant becomes stronger, encourage a transition to demand breastfeeding.

Specific feeding problems

1. Ingested meconium/blood. Infants who have swallowed a lot of meconium or blood before birth may retch and appear distressed after birth. Such problems almost always settle within a few hours without any intervention.

2. Uncoordinated feeding. Infants born before 32 weeks' gestation often have difficulty sucking and swallowing in a coordinated way. Most will initially need some tube feeds. They are not likely to start gaining weight until they are taking at least 120 mL/kg of milk a day. Infants need to be fed regularly at least once every 4 hours, day and night. Breast milk can be supplemented with formula milk at this time if donor milk is not available. However, every effort needs to be made to sustain the mother's lactation by expression and by keeping the mother in hospital to be near her infant.

3. Regurgitation. Hurried frequent feeding may cause regurgitation. A poorly developed cough reflex can cause the infant to inhale milk into the lung, resulting in possible pneumonitis and even pneumonia. Newborn infants benefit from frequent small feeds every 2 to 3 hours. Feeds should be increased gradually over the first 3 to 5 days of life. Patience is required. Dehydration (and the risk of hypoglycaemia) need to be monitored, and can be prevented during this period by giving supplemental gastric or IV fluids so that total fluid intake (i.e. taking the gastric/IV and the oral intake together) does not fall below 120 mL/kg per day.

4. Feeding tubes. Tube feeding is the best option for infants who have not yet developed a coordinated suck and swallow reflex. Nasogastric tubes are easier to secure and less easily pushed out by the infant's tongue, but they can almost completely block one nostril, significantly increasing the work of breathing. Therefore, orogastric tubes are preferred if respiratory distress is present. Alternatively, a fine-bore nasogastric tube can be left in place and changed as required (up to a maximum of 7 days).

    Small frail infants should be handled as little and as gently as possible, and can be left lying undisturbed in their cots during a tube feed so long as the head end of the cot is elevated 25 cm.

Section 9 Hospital problems: temperature control

Temperature control and hypothermia prevention and treatment

1. Hypothermia can be due to a cold environment, but remember that starvation or serious infection can present as hypothermia.

2. Normal temperatures for newborn infants are 36.5– 37.5°C (axillary) if measured over 3 minutes, and lower (around 36.0–36.5°C) if measured over at least 1 minute. Rectal thermometers are difficult to use and can be dangerous. If the trunk is cold, the infant is almost certainly hypothermic

3. Use a low-reading digital thermometer, not a mercury thermometer. If the axillary temperature is less than 32°C, hypothermia is severe; if it is in the range 32–35.9°C the infant has moderate hypothermia. If the infant's temperature does not register on the normal thermometer, assume that they have hypothermia.

4. *Hypothermia can be prevented by the following measures:*

- Dry the infant well immediately after birth and place them in skin-to-skin contact with the mother. This is especially important for low-birth-weight infants who do not have other complications. For those with medical problems, warm the infant by skin-to-skin care. If there are adequate resources and staff, an overhead radiant heater or an air-heated incubator (set at 35–36°C) can be used.

- 'Kangaroo care' (skin-to-skin contact with the mother between her breasts and covered with a blanket) is the most effective method for all infants, especially for those of low-birth-weight. Randomised trials in both well-resourced and resource-limited countries have shown significant advantages to this technique for the infant and the mother, including an increased prevalence of breastfeeding, a reduced incidence of apnoea and a reduced risk of infection. Take care when examining the infant not to allow the temperature to fall (ideally room temperature in the hospital ward should be higher than 25°C).

- A cot heated with a hot-water bottle with the top screwed in tightly and wrapped in a clean towel can be just as effective if the above are not available. Ordinary domestic radiant heaters or electrical blower type heaters can also be effective.

- Cover the infant's head with a warm woollen hat and dress them in warm, dry clothes.

- Keeping the nappy dry is also very helpful.

- Avoid washing the infant before they are 24 hours of age.

- Do not leave the infant where there are any draughts.

- The infant should sleep either with or next to the mother during the night.

5. Avoid overheating by monitoring the axillary temperature 4- to 6-hourly.

6. Feed the infant 2- to 3-hourly, and continue with 4-hourly feeds during the night.

7. The development of incubators earlier in the twentieth century significantly reduced the mortality of preterm infants, but they are expensive, and require regular maintenance, thorough cleaning and sufficient numbers of trained staff. The nursing of infants in incubators is covered by standard texts, but Table 9.1 gives the settings from which to start, adjusting the incubator temperature up or down to maintain the infant's axillary temperature at 36.0–36.5°C.

TABLE 9.1 Incubator temperature guidelines

Weight of baby (grams)	Day 1	Day 2	Day 3	Day 4 and subsequent
<1200	35.0°C	34.0°C	34.0°C	33.5°C
1200–1500	34.0°C	34.0°C	33.5°C	33.5°C
1500–2500	33.5°C	33.0°C	32.0°C	32.0°C
>2500	33.0°C	32.5°C	31.0°C	30.5°C

8. Do not use antipyretic drugs to control fever in a newborn infant. Instead control the environment (e.g. remove some clothes, adjust incubator temperature) and always consider the possibility of serious infection

Prevention of neonatal infection

A newborn infant with risk factors for infection (membranes ruptured more than 18 hours before delivery, mother with fever > 38°C before delivery or during labour, or foul-smelling/purulent amniotic fluid) should be treated with prophylactic antibiotics (ampicillin and gentamicin IM or IV) for at least 2 days. After 2 days the infant should be reassessed and treatment continued if there are signs of sepsis (or a positive blood culture).

Simple measures that can prevent infection in the new- born include the following:
- Ensure a clean delivery environment for the mother and infant, including disinfectant cream for all maternal vaginal examinations (e.g. Hibitane cream).
- Good cord care: the WHO recommends that the cord be kept clean and dry. It should not be covered. Local applications of creams, ointments, etc. are generally not required except in high-risk settings, where application of an antiseptic is recommended. An antiseptic solution or cream such as 4% chlorhexidine has recently been shown to reduce omphalitis and resulting neonatal mortality. It should be applied immediately after birth and for several days thereafter if possible, preferably after every nappy change. Similarly, there is extensive successful experience with the application of surgical spirit or iodine solution to the cord.
- Exclusive breast feeding.
- Strict procedures for hand washing or the use of hand sprays or hand rubs for all staff and for families before and after handling infants.
- Not using water for humidification in incubators (where Pseudomonas can easily colonise).
- Cleaning incubators with an antiseptic before use (if skin-to-skin mother care is not possible).
- Strict sterility for all invasive procedures.
- Sterile injection practices.
- Remove intravenous drips when they are no longer necessary.
- Keep invasive procedures (e.g. blood sampling, unnecessary IV cannulation) to a minimum, only undertaking them when they are essential.

Early-onset sepsis (first 72 hours)

Early-onset sepsis usually occurs as a result of bacteria acquired by vertical transmission from mother to infant during labour and delivery. The most frequently observed organisms vary from one part of the world to another. Gram-negative enteric bacteria (especially *Escherichia coli and Klebsiella* species) predominate in many regions. Gram-positive cocci are also common, and include group B beta-haemolytic streptococcus, other streptococcal species, *Staphylococcus* and *Enterococcus*. Rarely *Listeria monocytogenes* is isolated from newborn infants with sepsis, especially when there are foodborne epidemics.

Section 10 Hospital problems: prevention neonatal infection

Maternal risk factors for early-onset sepsis
These include the following:
- maternal fever (especially 38°C or higher) before delivery or during labour
- pre-labour rupture of membranes
- prolonged rupture of membranes (18 hours or longer)
- preterm labour
- maternal bacteriuria during pregnancy (including E. coli and group B beta-haemolytic streptococcus)
- prior infected infant (group B beta-haemolytic streptococcus).

Early-onset sepsis in the newborn usually results from bacteria acquired from the mother at or shortly before delivery. These infants mostly present with respiratory distress, and have bacteraemia or pneumonia. However, vaginal cultures cannot be used to determine the choice of antibiotics when treating the symptomatic newborn.

Late-onset sepsis
Organisms are less likely to reflect those of the maternal genital tract, although the same pathogens may be identified in infants presenting from home. The most common infections are focal ones such as conjunctivitis, omphalitis, skin infections and meningitis. A circumcision wound can also be the site of serious infection.

In the hospital setting, infection is more commonly due to nosocomial pathogens, including coagulase-negative staphylococci, Gram-negative enteric bacteria (e.g. *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Enterobacter cloacae*), *Staphylococcus aureus*, *Pseudomonas* spe- cies, streptococcal species and *Enterococcus*. Fungal sepsis must also be considered. Investigate as for early- onset sepsis, with the inclusion of a lumbar puncture and suprapubic urine for analysis and culture if indicated, and treat empirically with parenteral broad-spectrum antibiotic therapy directed towards the most commonly encountered pathogens for the particular nursery. Once cultures are positive, therapy can be directed accordingly. (For details of treatment of sepsis, *see* Section 11.)
In seriously sick infants with suspected sepsis, priority should be given to the structured ABC approach, while simultaneously obtaining a blood culture followed by prompt administration of antibiotics. Other tests, such as a lumbar puncture if needed (see above), can be performed once the infant is stable and antibiotics have been started. (For details of treatment of sepsis, see Section 11.)

Infants who are vulnerable to maternal factors
The infant of a diabetic mother
If a diabetic mother is poorly controlled, her infant may be large for gestational age, putting him or her at risk of slow progress in labour and perhaps shoulder dystocia. At birth, the infant, although large, behaves in a similar manner to a preterm infant. There is a major risk of hypoglycaemia, caused by the intrauterine over-stimulation of the infant pancreas to produce abnormally high levels of insulin. The infant must be monitored at least

hourly for hypoglycaemia in the first 6 hours, and should then be monitored 4-hourly for hypoglycaemia, which should be treated as described above with an infusion of 10% dextrose. The infant of a diabetic mother has immature lung maturation and is liable to surfactant deficiency (*see* Section 14), poor feeding and jaundice. Polycythaemia is also more likely.

The infant of a mother who is dependent on alcohol or drugs of addiction
These infants have been exposed to significant levels of narcotic drugs or alcohol *in utero*, causing an increased risk of congenital abnormalities and of abnormal neurological development and behavior during childhood. Soon after birth they may show hyper-irritability and convulsions, requiring treatment and gradually reducing sedation as they are 'weaned off' the addictive drugs to which they have been exposed. These infants are also at risk of having been exposed to blood-borne viruses such as HIV and hepatitis B and C.

Neonatal illnesses   Neonatal sepsis

Recognising and treating neonatal infection

Bacterial sepsis (septicaemia) in the newborn infant may present with any number of subtle non-specific changes in activity or physical findings. A change in feeding pattern, vomiting, irritability, pallor, diminished tone and/ or decreased skin perfusion is suggestive of neonatal infection. Other presenting physical findings may include lethargy, apnoea, tachypnoea, cyanosis, petechiae or early jaundice. There may be fever, but this is not common, especially with bacterial infections occurring in the first week. However, temperature instability with hypothermia may be seen. Abnormal glucose homeostasis (hypoglycaemia or hyperglycaemia) and/or metabolic acidosis are commonly associated findings. Infants, especially preterm infants, are very prone to infection and can become ill very rapidly once infection takes hold. Antibiotic treatment is only likely to work if started early, but the recognition of early infection is not easy.

A WHO study showed that more than a third of all deaths in the first month of life in most resource-limited countries were caused by infection. It also found that more than 80% of these infants, when first seen, had **one or more of the following eight danger signs associated with infection in the neonate**:

- infant feeding less than well than before
- infant lying quiet and making few spontaneous movements
- hypothermia or fever > 38ºC
- capillary refill time > 3 seconds
- respiratory rate ≥ 60 breaths/minute
- indrawing of the lower chest wall when breathing, *or* grunting
- cyanosis
- history of a convulsion.

Less common but important signs include the following:

- low respiratory rate (< 20 breaths/minute) or apnoea
- jaundice
- abdominal distension
- skin infections.

All neonates with signs of sepsis need immediate hospital admission if they are not already there, and must be treated   with IV antibiotics for at least 10 days after blood and other appropriate cultures have been taken.

Ampicillin (or penicillin) plus gentamicin are the first-line drugs to be used. Consider adding cloxacillin or flucloxacillin if there are signs suggesting that *Staphylococcus aureus* is a cause (e.g. skin pustules, abscess, omphalitis). Blood cultures are ideal although not always possible before starting antibiotics. If the infant does not respond within 48 hours, consider changing the antibiotic. If there is a possibility of meningitis, risk of resistance or Gram-negative organisms, a third-generation cephalosporin such as cefotaxime or ceftriaxone should also be added.

Section 11 Neonatal illnesses: neonatal sepsis

Causes of early-onset sepsis (first 72 hours) Early-onset sepsis usually occurs as a result of bacteria acquired by vertical transmission from mother to infant dur- ing late pregnancy, labour and delivery. The most frequently observed organisms vary from one part of the world to another. Gram-negative enterics (especially *Escherichia coli and Klebsiella* species) predominate in many regions. Gram-positive cocci are also common, and include group B beta-haemolytic streptococcus, other streptococcal species, *Staphylococcus* and *Enterococcus*. Less commonly, *Listeria monocytogenes* is isolated from newborn infants with sepsis, especially when there are foodborne epidemics.

These infants mostly present with respiratory distress. However, vaginal cultures cannot be used to determine the choice of antibiotics when treating the symptomatic newborn.

Late-onset sepsis
Organisms are less likely to reflect those of the maternal genital tract, although the same pathogens may be identified in infants presenting from home. The most common infections are focal infections such as conjunctivitis, omphalitis, skin infections and meningitis. A circumcision wound can also be the site of serious infection.

In the hospital setting, infection is more commonly caused by nosocomial pathogens, including coagulase- negative staphylococci, Gram-negative enteric bacteria (e.g. *Klebsiella oxytoca, Klebsiella pneumoniae, Enterobacter cloacae), Staphylococcus aureus, Pseudomonas* species, streptococcal species and *Enterococcus*. Fungal sepsis must also be considered. Investigate as for early-onset sepsis (see below), with the inclusion of a lumbar puncture and suprapubic urine for analysis and culture if indicated, and treat empirically with parenteral broad-spectrum antibiotic therapy directed towards the most commonly encountered pathogens for the particular nursery. Once cultures are positive, therapy must be directed accordingly.

Laboratory evaluation of the unwell infant
In an infant who is generally unwell with no clinically obvious infective focus, the following investigations should be performed:

- Blood culture (about 1 mL of venous blood): This should be obtained from a peripheral vein after preparing skin with an antibacterial wash such as povidone-iodine and/or 70% ethanol or isopropyl alcohol. Blood culture is the gold standard for neonatal sepsis, but it is not 100% sensitive. The sensitivity may be further reduced if intrapartum antibiotics were administered to the mother antenatally. The results can be assessed at 48 hours.
- White blood cell and differential cell count are not helpful in most situations.
- Chest X-ray: This may be helpful if there are any respiratory signs, but not if it means taking the infant to another department in the hospital. A portable

chest X-ray is ideal.
- Lumbar puncture if indicated: cytology, chemistry, Gram stain and culture. Not routinely done on all infants with suspected infection unless there are neurological signs.
- C-reactive protein (CRP): This is an inexpensive and useful test which may take 12 hours to become positive after the onset of an infection if this is present.
- Blood glucose concentration.
- Serum bilirubin concentration: if the infant appears jaundiced.
- Surface cultures (ear canal, umbilical stump) and gastric aspirate cultures: these do not correlate with either the likelihood of sepsis or the causative agent in septic infants. **These cultures should not be obtained.**
- Midstream or suprapubic aspirate of urine for culture: This procedure is of little value in the infant with suspected sepsis shortly after birth, but may be positive in infants with new-onset symptoms later in the first week (≥ 3 days). A urinary tract infection should always be considered in neonates with late-onset sepsis.

In seriously ill infants with suspected sepsis, priority should be given to the structured ABC approach, while simultaneously obtaining a blood culture followed by prompt administration of antibiotics. Other tests, such as a lumbar puncture, can be performed once the infant is stable and antibiotics have been started.

Specific neonatal infections

Meningitis and/or septicaemia

Meningitis may occur at any time in the neonatal period, and is frequently fatal, with some survivors experiencing long-term sequelae. Survival and later prognosis depend on early diagnosis and rapid treatment. Confirmatory diagnosis from a lumbar puncture may take several hours. Therefore, it is urgent and appropriate to start antibiotic treatment empirically as soon as the diagnosis is suspected.

Presenting features of meningitis: These include lethargy, reduced or complete lack of willingness to take feeds, irritability, a high-pitched cry, apnoeic episodes, low- ered conscious level or even coma, hypotonia, convulsions, generalised signs of accompanying sepsis, and a bulging or tense anterior fontanelle.

Always measure and record the head circumference.

However, once signs such as the above are present, treatment may be unsuccessful and survivors may be handicapped. **Therefore any infant with the following danger signs should be started on antibiotics IV and the relevant investigations undertaken:**
- infant feeding less well than before
- infant lying quiet and making few spontaneous movements
- hypothermia or fever > 38°C

- capillary refill time > 3 seconds
- respiratory rate ≥ 60 breaths/minute
- indrawing of the lower chest wall when breathing, *or* grunting
- cyanosis
- history of a convulsion.

Less common but important signs include the following:

- low respiratory rate (< 20 breaths/minute) or apnoea
- jaundice
- abdominal distension
- skin infections.

Treatment of suspected bacterial septicaemia with or without early meningitis
1. Ensure that the airway is open and keep it open.
2. Ensure that the infant is breathing adequately, and if they are apnoeic, gasping or have a very low respiratory rate, consider ventilation using a bag and mask until they are breathing adequately.
3. If the infant is cyanosed, give them oxygen until they are pink or show normal oxygen saturation in air (> 92-93%).
4. Insert an IV cannula, using full sterile precautions. Umbilical vein catheterisation may be the most effective way to gain vascular access quickly in a shocked infant less than 1 week old (*see* Section 41). Otherwise it might be necessary to site an intra-osseous line or cannulate a scalp vein.
5. Take samples for full blood count, CRP, blood culture, lumbar puncture, blood glucose and other tests (urine microscopy and culture, chest X-ray, biochemical tests) if needed (and available). Failure to sterilise the skin rigorously can render blood culture results uninterpretable. Chlorhexidine, 0.5% aqueous solution, is a very effective antiseptic. Use two different swabs, applying each for 10 seconds, and then leave the skin to dry for 30 seconds. A keyhole drape and no-touch technique will reduce the risk of recontamination, especially when performing lumbar puncture or suprapubic aspiration.
6. If possible, check blood glucose levels, but if facilities do not allow this, give 2 mL/kg of 10% glucose IV over 2–3 minutes as an initial bolus, followed by 5 mL/kg of 10% glucose per hour for the next few days while enteral feeds are established. An infant who becomes alert and active immediately following the initial bolus is suggestive of hypoglycaemia (i.e. a blood glucose concentration of < 2.5 mmol/litre, or < 36 mg/dL), and this may be part of the problem. If an IV line cannot be inserted and hypoglycaemia is suspected, give expressed breast milk or 10% glucose by nasogastric tube or sublingual sucrose. Further intermittent monitoring of the blood glucose level should be undertaken and the infusion continued until it is clear that the infant is well enough to be fed orally.
7. Give the first dose of ampicillin plus gentamicin (or cefotaxime *or* ceftriaxone) intravenously using the dose regimen outlined at the end of this section. Remember to use the high meningitic dose if meningitis is suspected, and continue it

for the duration of therapy if meningitis is confirmed. If IV access is not immediately possible, give the initial antibiotic dose IM. Never wait for the results of cultures before starting antibiotics. Any delay can reduce the infant's chances of survival as well as leading to permanent damage if meningitis is present.

8. Start an IV infusion of 60 mL/kg/24 hours of 10% dextrose (or 1/5 normal saline with 5% dextrose) if at all possible.

9. If the infant is shocked, give an IV bolus of 10 mL/kg of Ringer-lactate or Hartmann's solution. This can be repeated twice (giving a total of 30 mL/kg) if the infant remains shocked. The use of inotropes (dopamine and dobutamine) (if available) can be considered in such situations, although the outlook is bleak if they are needed.

10. If the infant has any respiratory symptoms, take a portable chest X-ray (if facilities are available). Do not take a sick infant to an X-ray department for this, as the resulting information is not worth the risks of moving them. Look regularly to see whether cyanosis is developing, or use a pulse oximeter (if available) and give supplemental oxygen, preferably using nasal cannulae rather than a head box. Infants who become infected during delivery develop respiratory symptoms with progressive signs of septic shock within a few hours of birth. Do not give any-thing by mouth to an infant who is breathless, especially if there is additional evidence of oxygen dependency.

Points to consider

1. Undertake the ABC approach. Oxygen may be needed. If the conscious level is impaired, the airway may be at risk.

2. Be alert for the presence of seizures, and treat them as appropriate. Always consider meningitis as a possible cause. If there are any features suggestive of meningitis, perform a lumbar puncture at the same time as blood cultures or within 2 hours of starting antibiotic treatment, because the blood culture is sterile in 15% of infants with early meningitis. Do not delay antibiotic therapy pending the undertaking of a lumbar puncture. Treat seizures with phenobarbitone 20 mg/ kg IM or by slow IV injection. If needed, continue with phenobarbitone at a maintenance dose of 3–5 mg/kg/ day. Diazepam or midazolam can also sometimes be used to control seizures. However, always have a bag and mask available if diazepam or midazolam are given to stop fitting, as these drugs cause temporary apnoea in some patients, which can easily be managed with bag-and-mask ventilation until the infant is breathing adequately.

3. Microscopic examination of the CSF (in meningitis the white blood cell count is $\geq$ 25 cells/mm^3, low glucose levels and high protein levels with or without Gram stain can provide early confirmation of meningitis. Remember that a differential white blood cell count or a differential count in the CSF do not help with the decision to initiate or continue antibiotic treatment.

4. Surface swabs and gastric aspirate cultures have no diagnostic significance. However, urinary tract infection can occasionally be the primary focus of a Gram-negative septicaemic illness. Simple microscopy on a clean catch or

suprapubic urine specimen may be used to rule out a urinary tract infection. Identification of a urinary tract infection may suggest the need for ultrasound imaging of the renal tract and long-term prophylactic antibiotics.

5. Watch for, prevent and correct any sign of hypothermia (skin-to-skin mother care).

6. Antibiotics can be stopped after 48 hours if the blood cultures are negative and the infant is clinically well. If available, a normal CRP at 48 hours can help to exclude sepsis. If blood cultures are not available, continue the antibiotics for the full course appropriate for the site of infection (meningitis 14–21 days).

7. Think also of herpes infection, congenital TORCH infection (newborn intrauterine-acquired infections, including toxoplasmosis, parvovirus B19, syphilis, HIV, varicella, coxsackie, rubella and cytomegalovirus) or neonatal malaria (rare) in a malaria-endemic region.

Antibiotic treatment

1. Beta-lactam antibiotics plus aminoglycosides act synergistically in treating some of the most frequently encountered neonatal pathogens. Commonly used agents are ampicillin and gentamicin, but alternative broad-spectrum coverage may be used. Penicillin may be used if ampicillin is not available, but it has a narrower spectrum, limited to Gram-positive bacteria. Ampicillin may also provide better coverage for certain Gram- positive pathogens, including *Listeria*.

2. Third-generation cephalosporins such as cefotaxime and ceftriaxone may be used, but some Gram-positive bacteria may not be covered (e.g. *Enterococcus*, *Listeria*) if a penicillin derivative is not included. Infants with suspected Gram-negative meningitis and accompanying early-onset sepsis may benefit from inclusion of a third-generation cephalosporin which offers a theoretically greater penetration and killing power for enteric bacteria in the cerebrospinal fluid. These antibiotics may be given intramuscularly if IV access cannot be obtained. Frequent use of these drugs may contribute to the development of multi-drug-resistant strains of bacteria in nurseries. Ceftriaxone has a longer half-life and can be dosed once daily.

3. Cloxacillin (IV or oral) is preferable if septic spots are present, as these are usually caused by coagulase- positive staphylococci.

4. Second-line antibiotics (e.g. ciprofloxacin, vancomycin, meropenem, piperacillin-tazobactam, linezolid) may be helpful for treating nosocomial infections and resistant organisms. However, their use should be limited to proven multi-drug-resistant organisms. Advice can always be sought on these from nearby referral centres. Inappropriate use of these expensive antibiotics may lead to even more multi-drug-resistant organisms (the so-called 'superbugs'). It is recommended that these agents should only be used in specified clinical settings.

5. Empirical antibiotic therapy includes antibiotics used for neonatal sepsis (i.e. a beta-lactam antibiotic plus an aminoglycoside) and a third-generation cephalosporin (e.g. cefotaxime or ceftriaxone) with excellent CSF penetration and bactericidal effect on sensitive Gram-negative bacteria. Therapy can be adjusted once

the bacteria have been iden- tified and antibiotic sensitivities determined. The duration of treatment is at least 14 days for uncomplicated Gram- positive bacteria and 21 days for Gram-negative bacteria. The most frequently used initial combination is ampicillin and gentamicin (see the neonatal formulary at the end of this section). Benzyl penicillin may be preferable for known or suspected group B streptococcal infection. Cefotaxime or ceftriaxone is the drug of choice for most Gram-negative organisms, and ceftazidime is used for *Pseudomonas* infection.

Investigations for meningitis

- Lumbar puncture is potentially helpful if meningitis is suspected, and should be considered in all newborn infants with neurological signs. It is important to only attempt lumbar puncture once the infant has been stabilised, and ideally within 2 hours of initiating antibiotic treatment. Lumbar puncture is more likely than blood culture to identify the organism responsible, and within a shorter period of time.
- Cerebrospinal fluid (CSF) cell counts, chemistry and Gram stain would often point towards meningitis. An elevated CSF leucocyte count (≥ 25 white blood cells/ mm^3) with pleocytosis is characteristic of neonatal meningitis. The CSF protein level in meningitis may be high (> 2.0 g/litre in a term infant), and the CSF glucose level is typically low (< 30% of blood glucose value). Gram staining may reveal bacteria, but antibiotic therapy should not be directed on the basis of this result, as rapidly growing bacilli may be mistaken for cocci, or the state of the organism may result in variable staining.
- Sometimes the CSF picture in preterm infants who have sustained an intra-ventricular haemorrhage can show a mild reactive pleocytosis in the first few weeks of life, which can be quite misleading. If there is clinical suspicion this should be treated as bacterial meningitis until cultures are known to be negative.
- If a 'bloody tap' is obtained it is best to treat the infant as having meningitis, and repeat the lumbar puncture after 24–48 hours. The finding of many white cells or bacteria is significant even if the CSF is bloodstained.

Diarrhoea in the newborn
Special points to remember
1. Encourage frequent breastfeeding, as it helps in both preventing and treating diarrhoea in the newborn.
2. If the infant is dehydrated, give low-osmolarity oral rehydration solution (ORS) in addition to breast milk.
3. In the case of sick infants or those infants who are unable to feed orally, consider IV fluids.
4. If bloody diarrhoea occurs, it is best to assume that the infant has dysentery, and in initiate antibiotic therapy. Avoid the use of co-trimoxazole in the light of much better and more effective antibiotics with better side-effect profiles.
5. In the case of the septic and unwell infant, give IV antibiotics as outlined in Section 11.

Section 11 Neonatal illnesses: neonatal sepsis

Sometimes what is described as diarrhoea by the mother is in fact the normal loose breastfed stools of some infants in the first few days of life. Usually the number of stools passed per day declines quickly, and in some breastfed infants may be as infrequent as once daily.

Congenital syphilis

Congenital syphilis may be acquired from an infected mother via trans-placental transmission of *Treponema pallidum* at any time during pregnancy.

Clinical signs in infants may include any of the following:

- low birth weight with a heavy placenta
- palms and soles showing a red rash, grey patches, blisters or skin peeling
- abdominal distension due to large liver and spleen
- jaundice
- anaemia
- some low-birth-weight infants with syphilis show signs of severe sepsis, with lethargy, respiratory distress, skin petechiae or other signs of bleeding.

Investigation

No newborn infant should be discharged from hospital without determination of the mother's serologic status for syphilis at least once during pregnancy, and also at delivery in communities and populations in which the risk of infection with congenital syphilis is high.

If you suspect syphilis, perform a venereal disease research laboratory (VDRL), rapid plasmin reagent (RPR) or rapid syphilis test.

Treatment

All newborns of mothers with syphilis should be investigated and treated.

Infants should be treated for congenital syphilis if they have proven or probable disease demonstrated by one or more of the following:

1 physical, laboratory or radiographic evidence of active disease
2 a reactive result on maternal or infant VDRL testing where the mother has not had 3 weekly doses of benzathine penicillin.

Parenteral benzyl penicillin remains the preferred drug for treatment of an infant with any signs of congenital syphilis.

Asymptomatic neonates born to VDRL-positive or RPR-positive women should receive 37.5 mg/kg (50 000 units/kg) of benzathine benzyl penicillin as a single IM dose into the anterolateral thigh whether or not their mothers were treated during pregnancy. Routine CSF examination is not required. Ensure that the needle is not in a vein when this drug is given, by drawing back and ensuring that no blood is in the needle, as it can cause cardiac arrest and severe CNS damage if given IV.

Section 11 Neonatal illnesses: neonatal sepsis

Newborn infants with any signs of congenital syphilis should receive:

- aqueous benzylpenicillin, 100,000–150000 IU/kg/day administered as 30 mg/kg or 50000 IU/kg/dose IV every 12 hours, during the first 7 days of life, and every 8 hours thereafter for a total of 10 days

or

- procaine benzylpenicillin, 50 000 IU/kg by IM injection, as a single daily dose for 10 days (ensure that it is not injected into a vein).

Early congenital syphilis generally responds well to penicillin. Recovery may be slow in seriously ill infants with extensive skin, mucous membrane, bone or visceral involvement.

If the patient is allergic to penicillin (this is unusual), give ceftriaxone 80 mg/kg IM/IV once daily for 10 days or give erythromycin, 7.5–12.5 mg/kg orally, four times a day for 14 days but erythromicin is less effective.

Treat the mother and partner for syphilis, and check for other sexually transmitted infections.

The infant of a mother with tuberculosis (see Textbook)

- If the mother has active lung tuberculosis and was treated for less than 2 months before birth, or was diagnosed with tuberculosis soon after birth, the infant should be evaluated for congenital tuberculosis.
- Congenital tuberculosis is rare but should always be considered in sick neonates or infants, especially in areas where HIV/tuberculosis co-infection is common.
- Women with tuberculosis who have been treated appropriately for 2 or more weeks and who are not considered contagious can breastfeed. Reassure the mother that it is safe for her to breastfeed her infant.
- If a mother has completed tuberculosis chemotherapy during pregnancy or has inactive disease, her infant should be given BCG at birth.
- If she has active disease or is still requiring treatment, the infant should be given isoniazid 10 mg/kg once daily for 3–6 months.
- Once the mother and infant are both on appropriate treatment, the infant may breastfeed unless the mother has multi-drug-resistant TB. A tuberculin test and chest X-ray is then performed on the infant. If they are negative, BCG is given; if it is positive, full investigations for tuberculosis are undertaken. If no evidence of disease is detected, isoniazid is continued for another 3–4 months.
- If tuberculosis is suspected, full treatment with 4 drugs is given at standard doses (*see* textbook).
- At the age of 6 weeks, re-evaluate the infant, noting weight gain and taking an X-ray of the chest if possible. Congenital TB is most often intra-abdominal, so look for signs suggesting this. If there are any signs or findings suggestive of active disease,

start full anti-tuberculosis treatment according to national guidelines. If at the age of 6 weeks the infant is doing well and tests are negative, continue prophylactic isoniazid to complete 6 months of treatment. Delay BCG vaccination until 2 weeks after treatment is completed. If BCG vaccine has already been given, repeat it 2 weeks after the end of the isoniazid treatment.

- If the mother is suspected of having multi-drug-resistant tuberculosis, an expert in tuberculosis disease treatment should be consulted.

Skin, eye and mucous membrane infections
Conjunctivitis
Most conjunctivitis presents as 'sticky eyes', but this may not always be of bacterial origin, especially if it occurs in the first few days. However, a bacterial process must be considered in all cases. Infants with a serous discharge without significant conjunctival inflammation may simply have **blocked naso-lacrimal tear ducts**. This usually responds to gentle pressure/massage applied in a downward motion along the nose immediately adjacent to the eyes. The discharge may be cleaned from the eye with sterile 0.9% saline drops. Show the parent how to clean the infant's eyes with sterile normal saline or boiled and cooled clean water. The eyes should be wiped from the inside to the outside edge using a clean cotton wool swab for each eye. The hands should always be washed before and after the procedure.

If the condition worsens or if there is conjunctival inflammation or a purulent discharge, use of topical therapy should be considered. Erythromycin, tetracycline, neomycin or chloramphenicol ophthalmic ointments or drops may be considered. Sometimes this condition is due to chlamydia. Apply the ointment 2 to 4 times a day for 5 days after washing away any pus with sterile normal saline as described above. Treat this level of infection as an outpatient, but review every 48 hours.

Gonococcal conjunctivitis
A severe rapidly progressive purulent conjunctivitis occurring within the first few days must always be assumed to be due to *Neisseria gonorrhoeae*, which must be promptly identified and aggressively treated in hospital with parenteral antibiotics and irrigation. Most strains are now resistant to penicillin. Swab the eye for microscopy (Gram-negative intracellular diplococci) and culture (special medium is required, such as Thayer–Martin agar with incubation under increased carbon dioxide). Treatment should be initiated immediately before culture confirmation. Treatment with IV penicillin for 7 days has been used successfully, but because of increased world-wide resistance (penicillinase-producing gonococcus), a third-generation cephalosporin is often selected as the first-line therapy:

- ceftriaxone 125 mg IM, as a single dose
- *or* cefotaxime 25 mg/kg (maximum 125 mg) IM, as a single dose
- *or* cefixime 20 mg/kg orally, as a single dose.

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It is important to repeatedly clean the eye, or irrigate with saline until pus formation stops. It is vital to prevent corneal rupture and subsequent blindness.

In the case of a presumed or diagnosed gonococcal or chlamydial infection, the mother and partner should also be treated.

In countries with a low rate of sexually transmitted diseases, staphylococcal and Gram-negative organisms are more likely to be responsible. Staphylococcal infections can be treated with cloxacillin or flucloxacillin 30 mg/kg orally or IV every 6–8 hours for 5 days.

Chlamydial conjunctivitis

Chlamydia trachomatis is a common cause of infectious conjunctivitis in the newborn infant. It typically presents between 5 and 14 days. The presentation can vary from mild to moderate conjunctival erythema, and from scant mucoid discharge to copious purulent discharge. Eyelid oedema, chemosis or pseudo-membrane formation may also be present. Corneal involvement is unusual initially, although untreated chlamydia conjunctivitis can result in varying degrees of conjunctival scarring and corneal infiltrates.

Chlamydia can be confirmed by culture or rapid antigen detection, but these are highly specialised procedures that may not be readily available. Without a positive laboratory diagnosis, treatment is based on clinical severity. If the condition is mild, clean the eye only. If it is moderate, use a topical antibiotic and consider giving erythromycin 10 mg/kg orally, 6-hourly for 14 days. This effectively treats this infection and may also eradicate upper respiratory tract colonisation. Drug interactions with erythromycin include increased serum levels of digoxin, theophylline and potentially caffeine.

If the condition is severe, beware gonococcal infection, irrigate and use IV or IM cefotaxime or ceftriaxone.
Ensure that the mother is appropriately referred for treatment.

Skin pustules

Skin pustules are most commonly caused by *Staphylococcus aureus*. Most often these occur in small clusters in an otherwise healthy asymptomatic infant. Topical therapy with chlorhexidine 0.5% may be all that is needed in most of these cases. Oral therapy with a penicillinase-resistant penicillin (e.g. flucloxacillin 25 mg/kg 6-hourly) or first-generation cephalosporin (e.g. cephalexin 25 mg/kg 6–12-hourly for 7 days) may also be used if extensive pustules are found. If septicaemia is suspected, septic investigations and IV antibiotics after hospitalisation may be needed. Sometimes staphylococcal pustules can be difficult to distinguish from erythema toxicum (a benign, non-infectious newborn rash).

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Umbilical infection
A clinically relevant infection of the umbilical stump (omphalitis) presents as redness and oedema of the skin extending from the umbilicus. This should be distinguished from the ooze resulting from an umbilical granuloma, which may develop after a few weeks. If there is skin redness plus oedema extending from the umbilicus, appropriate antibiot- ics, usually anti-staphylococcal, should be used. Clean the area with soap and warm water and remove or drain pus and crusts. Dry and paint the area with antiseptic such as gentian violet, or use a simple alcohol swab to clean the area at the time of every nappy change. If there is only a 'sticky cord', manage it with local treatment only. Pus can be easily removed with a swab, whereas normal cord degeneration cannot be removed.

Cellulitis
This is most commonly caused by streptococci, but *Staphylococcus aureus*, Gram-negative enterococcus and anaerobes should also be considered when infection occurs at sites where there have been breaks in the skin. Treatment with parenteral antibiotics (e.g. Flucloxacillin, a penicillinase-resistant penicillin, and gentamicin, an amino-glycoside) should be directed against both Gram-negative and Gram-positive bacteria. Omphalitis may become rapidly progressive and spread to deeper tissues. Infection with *Clostridium* is common in the setting of poor maternal immunity or poor umbilical cord care, and can cause neonatal tetanus.

Scalded skin syndrome
This is a rare infection caused by toxin-producing staphylococcal organisms which leads to a toxic reaction producing the effect of both serious infection and burns. Treat it with IV cloxacillin or flucloxacillin.

Superficial candidiasis ('thrush' and 'monilial' rash)
Superficial candidiasis of the oral mucosa ('thrush') commonly manifests as white patches which are not easily scraped with a spatula. The nappy area may also be affected ('monilial' rash). Unlike irritant dermatitis, the erythema extends into skin folds and there may be small raised erythematous lesions. Treat with oral nystatin suspension, 1 mL after feeds (divide it between each cheek with a small syringe). Topical nystatin ointment may be used to treat the skin rash, but only in combination with oral nystatin. Keep the nappy area dry. Apply local treatment to the mother's nipples if they are also infected.

Warning: Excess and inappropriate antibiotic usage, besides being costly and generating a lot of nursing work, also leads to multi-drug resistance. Excess use can cause overt *Candida* infection (thrush), and also risks the eventual emergence of multi-drug-resistant organisms, especially in a hospital setting. The wide- spread use of ampicillin has caused many coliform organisms to become increasingly resistant to this antibiotic, while units that use cefotaxime extensively are starting to encounter serious *Enterobacter* and other multi-drug-resistant Gram-negative sepsis.

Drugs used to treat severe infection in the neonate

Ampicillin (or amoxicillin)

Give 100 mg/kg per dose IM or IV where meningitis is a possibility, and 50 mg/kg per dose in other situations. Give one dose every 12 hours in the first week of life, every 8 hours in an infant aged 1–3 weeks, and every 6 hours in an infant older than this. Oral dosing can sometimes be used to complete a course of treatment.

Benzyl penicillin

Give 60 mg/kg (100 000 units/kg) IV if meningitis or tetanus is a possibility. Give 30 mg/kg (50 000 units/kg) per dose in all other situations, including syphilis. Time the interval between each dose as for ampicillin. Oral dosing (with phenoxymethylpenicillin) can sometimes be used to complete a course of treatment.

Cefotaxime

Give 50 mg/kg per dose IV or IM. Time the interval between each dose as for ampicillin, except in meningitis, where doses are given 6-hourly.

Chloramphenicol

This remains a useful antibiotic, although there is a serious risk of death from liver failure if the dose suggested here is exceeded. **Warning: The problem is not the dose but incorrect mixing, as the bottle contains 1000 mg, so it is easy to overdose.** Give a 25 mg/kg loading dose IM followed by 12.5 mg/kg once every 12 hours to infants less than 1 week old. Give this dose every 8 hours to infants aged 1–4 weeks, unless there is evidence of liver damage or renal failure. Infants older than this can be given 12.5 mg/ kg once every 6 hours from the outset. Oral dosing can be used to complete any course of treatment. (The dose can be doubled in those over 1 month of age with severe infection.) Be very careful if the IV dose has to be diluted to obtain the correct dosage.

Cloxacillin (or flucloxacillin)

Give 100 mg/kg per dose IM or IV if serious infection is present, and 50 mg/kg per dose in other situations. Time the interval between each dose as for ampicillin. Oral treatment can often be given to complete a course of treatment (25 mg/kg standard, 50 mg/kg severe, 100 mg/kg in infec- tions such as osteomyelitis).

Erythromycin

Give 12.5 mg/kg per dose orally once every 6 hours. There is no satisfactory IM preparation.

Eye drops (and ointments)

Prophylactic chloramphenicol 0.5% eye drops or 1% eye ointment can be used to minimise the risk of gonococcal infection (IM/IV ceftriaxone is being used for overt infection). Tetracycline ointment 1% should be used (with oral erythromycin) to treat chlamydia conjunctivitis (this condi- tion is not prevented by silver nitrate use). *Pseudomonas* infection requires treatment with systemic antibiotics and topical gentamicin 0.3% eye drops.

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Gentamicin

Give 5mg/kg IM or IV once every 24 hours. If the infant weighs less than 2 kg, give 4 mg/kg per dose. Leave 36–48 hours between each dose if there is renal failure.

- If the infant is less than 32 weeks' gestation, give 4–5mg/kg 36-hourly.
- If the infant is more than 32 weeks' gestation, give 4–5mg/kg 24-hourly.

Hepatitis B vaccine

Give 0.5mL IM into the thigh as soon as possible after or within 12 hours of birth. Remind the mother that the infant will require booster injections at 6 weeks and 14 weeks after birth. Infants born to mothers infected during pregnancy or who are known high-risk carriers with a positive hepatitis B e-antigen should also be given 200 units of hepatitis B immunoglobulin (HBIG) IM into the other thigh within 24 hours of birth. Breastfeeding can safely continue.

Isoniazid

See Textbook for the latest advice on the treatment of infants with TB or suspected TB.

Metronidazole

Give a 15mg/kg loading dose and 7.5mg/kg per dose once every 12 hours in infants less than 4 weeks old, and every 8 hours in infants older than this. Treatment can be given IV or orally, but solubility makes IM use unsatisfactory. If the IV route is used, start the maintenance dose 24 hours after loading. If the oral route is used, give the first dose 12 hours after loading.

Miconazole

This controls infection with candida ('thrush') more effectively than topical nystatin. Use the oral gel at least four times a day and the skin cream twice a day for at least 7 days. Topical treatment with 0.5% aqueous gentian violet for not more than 4 days may be equally effective. Oral nystatin drops (1 mL four times a day) can be used to reduce heavy intestinal tract carriage.

Nevirapine

See Section 12, Textbook and national protocols for the latest   advice on the use of Nevirapine in the prevention of mother- to-child transmission of HIV infection.

Procaine and benzathine penicillin

Give asymptomatic infants born to mothers with evidence of untreated syphilis a single 37.5mg (50000 units/kg) dose of benzathine penicillin IM injection. Never give this drug IV.

Infants thought to be infected at birth are often given procaine penicillin 50 mg/kg (50 000 units/kg) IM once a day for 10 days, but repeated IM injections can cause a sterile abscess with subsequent muscle fibrosis and atrophy. IV benzylpenicillin for 10 days (as specified above) is just as effective. Infants born to mothers who have been fully treated for syphilis (1.8 grams, or 2.4 mega-units, of benzathine benzylpenicillin) at least 4 weeks before birth need no further treatment after birth.

TABLE 11.1 Antibiotics for use in the neonatal period

Drug	Route	Single dose	Frequency	Postnatal age	Gestation
Ampicillin	IV, IM	50–100 mg/kg	12 hourly	< 7 days	Any
		50–100 mg/kg	8 hourly	7–21 days	Any
		50–100 mg/kg	6 hourly	> 21 days	Any
	Reduce dose frequency in severe renal impairment and birth asphyxia				
	Use higher doses in case of suspected Group B strep infection or meningitis				
Benzyl Penicillin	IV, IM	25–50 mg/kg	12 hourly	< 7 days	Any
		25–50 mg/kg	8 hourly	7–21 days	Any
		25–50 mg/kg	6 hourly	> 21 days	Any
	Reduce dose frequency in severe renal impairment and birth asphyxia				
Cefotaxime	IV, IM	50 mg/kg	12 hourly	< 7 days	Any
		50 mg/kg	8 hourly	> 7 days	Any
	Reduce dose by 50% in severe renal impairment				
Ceftazidime	IV, IM	50 mg/kg	12 hourly	< 7 days	Any
		50 mg/kg	8 hourly	> 7 days	Any
	Reduce dose interval to 24 hours in severe renal impairment				
Ceftriaxone	IV, IM	50 mg/kg	24 hourly	Any	Any
	Avoid in infants < 36 weeks' gestation or if jaundiced. Follow special IM preparation instructions				

Drug	Route	Single dose	Frequency	Postnatal age	Gestation
Chloramphenicol	IV, IM	12.5mg/kg	12 hourly	<7 days	
		12.5mg/kg	8 hourly	>7 days	
	There is a serious risk of death from liver failure if the dose suggested is exceeded				
	Oral dosing can be used to complete any course of treatment				
Cloxacillin	IV, IM	50mg/kg	12 hourly	<7 days	Any
		50mg/kg	8 hourly	>7 days	Any
	Double the dose in severe infection and if CNS is involved Increase dose interval to 24				
	hours in severe renal impairment				
Erythromycin	PO	12.5mg/kg	6 hourly		
	There is no satisfactory IM preparation				
Gentamicin	IV	5mg/kg	48 hourly	<7 days	<29 weeks
		4mg/kg	36 hourly	>7 days	<29 weeks
		4mg/kg	36 hourly	<7 days	30–33 weeks
		4mg/kg	24 hourly	>7 days	30–33 weeks
		4mg/kg	24 hourly	<7 days	>34 weeks
		4mg/kg	24 hourly	>7 days	>34 weeks
	Trough and peak levels are not needed				
Isoniazid	*See* Textbook for details on its use.				
Metronidazole	IV, PO	5mg/kg	(*loading dose*)		
		7.5mg/kg	12 hourly	<28 days	
		7.5mg/kg	8 hourly	>28 days	
	Infuse over 30 minutes				
	Injection solutions can be given rectally				

Miconazole	Oral gel		6 hourly		
	Skin		6 hourly		
	This controls infection with Candida *('thrush')*				
	better than topical nystatin Use for at least				
	7–10 days				

0.5% Aqueous Gentian violet	Apply			Once daily for 4 days		
Oral nystatin drops	PO	1 mL		6 hourly		
	Can be used to reduce heavy intestinal tract carriage					
Nevirapine	*See* Textbook for details on its use.					
Procaine penicillin	IM	10 mg/kg		Single dose		
	Give to asymptomatic babies born to mothers with evidence of untreated syphilis					
		100 mg/kg		Once daily		
	Babies thought to be infected at birth are often give once daily for 10 days					
	Never give this drug IV					
	Rarely repeated IM injections can cause a sterile abscess with subsequent					
	muscle fibrosis and atrophy Alternatively					
	IM or IV benzylpenicillin for 10 day (as specified above) is just as effective					
	Babies born to mother fully treated for syphilis need not further treatment after birth					
	(Maternal treatment = 1.8 grams or 2.4 mega units of benzathine benzylpenicillin at least 4 weeks before birth)					

Varicella zoster (chickenpox) in pregnancy and the neonate
Pregnant women and newborn infants are at risk of severe disease from varicella, involving serious effects on organs such as the lungs. In chickenpox, patients are infectious for 48 hours prior to emergence of the rash and until all of the skin lesions are crusted over. The incubation period is 10–21 days.

Perinatal infection
If a neonate is exposed (mother has a rash) around the time of birth (from 5 days before to 2 days after delivery), there is a 17–30% risk of dangerous infection. This is characterised by skin lesions, disseminated intravascular coagulation, pneumonitis and hepatitis, and it has a mortality of up to 30%.

Prevention of neonatal chickenpox if the mother is infected from 7 days before to 7 days after birth
Give VZIG to the neonate as soon as possible after delivery. Isolate the mother and infant.
In addition, give IV aciclovir to the neonate if the onset of maternal symptoms was between 4 days before, and 2 days after the birth.

Doses of VZIG and aciclovir in the neonate
Aciclovir 10–20 mg/kg IV every 8 hours for at least 7 days. Side effects include nausea, vomiting, diarrhoea, headache and nephrotoxicity. Reduce the dose or dosage interval in patients with impaired renal function.
Varicella zoster immunoglobulin (VZIG): 250 mg by deep IM injection. Anaphylaxis is rare, but ensure that adrenaline is available.

Prevention of mother-to-child transmission (PMTCT) of HIV infection and ante retroviral treatment (ART) in pregnancy

Updated consolidated ART guidelines published by the WHO in 2013 now recommend that all pregnant and breastfeeding women should be commenced on ART (one simplified triple regimen), and that this should be maintained for at least the duration of MTCT risk (i.e. throughout breastfeeding). They suggest that, particularly in generalised epidemics, all pregnant and breastfeeding women with HIV should initiate ART as lifelong treatment. In pregnancy the focus is no longer on 'when or what to start' but on 'whether to stop' treatment after delivery. Ideally, a CD4 count should be obtained before deciding whether ART for PMTCT only (i.e. stopping after delivery) is an option.

As most women should continue ART following delivery, an effective link with HIV treatment programmes is essential.

National programmes are encouraged to move from the previous Option 'A' to Option 'B' or 'B+'.

Option B+: all pregnant and breastfeeding women infected with HIV should be started on ART as lifelong treatment. This is particularly important in generalised epidemics where high fertility, long duration of breastfeeding, limited access to CD4 to determine ART eligibility, and high partner sero-discordance rates all increase the risks of transmission to the woman's partner and babies.

Option B: in some countries (e.g. where CD4 counts are available), in the case of women who are not eligible for ART for their own health, consideration can be given to stopping the ARV regimen after the period of risk of mother-to-child transmission has ended.

Single-dose nevirapine (sdNVP) for women in labour is no longer recommended (unless it is combined with other ART) because it causes the virus to develop high levels of drug resistance.

The treatment focus is shifting to consider ART for the mother's health, to utilise more effective ART drugs, and to extend coverage throughout the MTCT risk period. **All women should be started on ART in pregnancy.**

Women diagnosed with HIV during labour or immediately postpartum

If a woman is diagnosed with HIV infection during labour or immediately postpartum, ART should be commenced immediately for PMTCT. This regimen can be modified later when the woman has been assessed (with CD4 count) with regard to whether she requires lifelong ART for herself.

Postpartum, women should be assessed and a CD4 count obtained. Ideally they should continue their triple-drug ART regimen lifelong unless their CD4 count is > 500 cells/mm^3. It will take weeks for the maternal viral load to be reduced, and

therefore it is important to give the mother ART in labour which will cross the placenta (and enter the baby) in addition to starting ART prophylaxis in the infant

If a woman is diagnosed with HIV infection postpartum and plans replacement (formula or bottle) feeding, refer her for HIV care and evaluation for treatment.

TABLE 12.1 WHO guidelines for ART in pregnancy for HIV-infected women who have not had previous ART

	For pregnant women for PMTCT only	For infants of mothers given a short course of ART for PMTCT
Option B	Preferred regimens: TDF + 3TC (or FTC) + EFV (as fixed-dose combination) Alternative regimens: AZT + 3TC + EFV or TDF + 3TC (or FTC) + EFV	*Baby being breastfed:* daily nevirapine (NVP) for 6 weeks *Baby being bottle-fed:* daily nevirapine (NVP) for 4–6 weeks or AZT twice a day for 4–6 weeks

	For pregnant women being given lifelong ART	For infants of mothers on lifelong ART
Option B+	Preferred regimens: TDF + 3TC (or FTC) + EFV (as fixed-dose combination) Alternative regimens: AZT + 3TC + EFV or AZT + 3TC + NVP or TDF + 3TC (or FTC) + NVP	*Baby being breastfed:* daily nevirapine (NVP) for 6 weeks *Baby being bottle-fed:* daily nevirapine (NVP) for 4–6 weeks or AZT twice a day for 4–6 weeks

TABLE 12.2 WHO guidance for ART for women diagnosed with HIV in labour/immediately postpartum

	For the mother	For the infant
Option B	Start (triple) ART immediately. Continue until 1 week after exposure to breast milk has	*Baby being breastfed:* Daily NVP from birth for 6 weeks, consider extending to 12 weeks
Option B+	Start (triple) ART immediately. Continue lifelong	*Baby being bottle-fed:* Daily NVP from birth for 6 weeks

Section 12 Neonatal illnesses: prevention HIV infection

**Co-trimoxazole prophylaxis to prevent *Pneumocystis jiroveci (formerly carinii)*
*pneumonia (PCP)***

HIV-exposed infants (born to women with HIV whether or not they are part of a
prevention of mother-to-child transmission (PMTCT) programme.) should be given
cotrimoxazole prophylaxis from 4–6 weeks of age, and this should be continued until
HIV infection has been excluded and the infant is no longer at risk of acquiring HIV
through breastfeeding.

Co-trimoxazole prophylaxis has been shown to be very effective in HIV-infected
infants and children in reducing mortality and the likelihood of PCP as a cause of
severe pneumonia. PCP is now unusual in countries where prophylaxis is routine.
Co-trimoxazole also protects against common bacterial infections, toxoplasmosis and
malaria.

Co-trimoxazole should be discontinued:

- If severe cutaneous reactions such as Stevens–Johnson syndrome occur or if there is
  renal and/or hepatic insufficiency or severe haematological toxicity (severe
  anaemia or pancytopenia). It is contraindicated in infants with glucose-6-
  phosphate dehydrogenase (G6PD) deficiency.
- In an HIV-exposed infant, only after HIV infection has confidently been excluded:
  - For a non-breastfeeding child under 18 months of age, this is by negative DNA or
    RNA virological HIV testing.
  - For a breastfed HIV-exposed child under 18 months of age, negative virological
    testing is only reliable if performed 6 weeks after cessation of breastfeeding.
  - For a breastfed HIV-exposed child over 18 months of age, negative HIV antibody
    testing 6 weeks after stopping breastfeeding.

Recommended dosages of 6–8 mg/kg TMP once daily should be used.
For infants under 6 months of age, give 2.5 mL of suspension (40 mg TMP//200 mg SMX in 5
mL) or 1 paediatric tablet (or ¼ adult tablet, 20 mg TMP/100 mg SMX: tablets can be
crushed).
Use weight-band dosages rather than body-surface- area doses.
If the infant is allergic to co-trimoxazole, dapsone is the best alternative. Give dapsone
after 4 weeks of age in an oral dose of 2 mg/kg/24 hours once daily.
If the patient is G6PD-positive, consider giving pentamidine or atovaquone.

Management of the delivery

Labour can be a worrying time for the HIV-positive woman, particularly because of possible
underlying fears about her own HIV infection and the risk of infecting her baby. She
will need reassurance and support, and it is important to ensure she knows that with
all of the interventions that are given her baby is more likely to be HIV-negative than
infected.

Get close to her, greet her and be seen to shake hands with her, to help to reduce the stigma around touching those infected with HIV. Support her relatives, and encourage her to tell her partner so that he can be tested for HIV. Promote safer sex and advise her to use condoms to prevent transmission of HIV.

Standard precautions should be used when caring for women in labour, whether or not they have HIV infection. Always wear gloves when touching body fluids, and dispose of single-use syringes and needles safely.

During delivery, to reduce MTCT:
- avoid artificial rupture of membranes
- avoid prolonged rupture of membranes
- avoid unnecessary episiotomy, but also avoid a tear.

Both blood and placenta will contain HIV, so wear gloves, an apron and eye protection. Avoid direct contact of blood on your skin. Blood on intact skin should be washed off immediately. HIV-positive blood on an open wound or splashed into the eye can transmit HIV and should be washed immediately (use soap and water for a wound, and water for an eye) and managed in the same way as a needle-stick injury (with post-exposure prophylaxis with ART).

Other considerations for managing HIV infection in pregnancy
HIV-2 infection
HIV-2 is much less transmissible than HIV-1 (the MTCT risk is 0−4%).
Non-nucleoside reverse transcriptase inhibitors (NNRTIs) such as NVP and EFV are not effective against HIV-2, and a triple nucleoside reverse transcriptase inhibitor (NRTI) combination is recommended.

Treatment of HIV-2 infection

Mother requires treatment: AZT + abacavir (ABC) + 3TC

PMTCT only: AZT from 14 weeks and continued until delivery

Infant of mother with HIV-2: AZT twice a day until 4–6 weeks

Essential postnatal care for HIV-exposed infants
1   Completion of ART prophylaxis regimen.
2   Routine newborn and infant care, including routine immunisation and growth monitoring.
3   Co-trimoxazole prophylaxis.

4   Early HIV diagnostic testing and diagnosis of HIV-related conditions.

5   Continued infant feeding counselling and support, especially after early HIV testing.

6   Nutritional support throughout the first year of life, including support for optimal infant feeding practices, and provision of nutritional supplements and replacement foods if indicated.

7   ART for HIV-infected infants when indicated.

8   Treatment monitoring for all infants receiving ART.

9   INH prophylaxis when indicated.

10   Adherence support counselling for caregivers.

11   Malaria prevention and treatment where indicated.

12   Diagnosis and management of common childhood infections and conditions, and integrated management of childhood illness (IMCI).

13   Diagnosis and management of TB and other opportunistic infections.

Polycythaemia

This potentially harmful condition occurs in up to 4% of births and risk factors are: being both small or large for gestational age or wasted, born to mothers with diabetes and being one of a multiple birth. Milking of the umbilical cord at birth, by increasing the amount of blood transferred into the baby, can also produce this condition.

Polycythaemia is defined as a venous haematocrit > 65% or Hb > 22 g/dL. Capillary samples can have higher haematocrit and if > 65% should be confirmed by venous sample.

Screen high risk babies at 2, 12 and 24 hours by capillary blood measurement of PCV and then if > 65% confirm by venous sample.

Above a PVC of 65%, the viscosity of blood increases exponentially and can produce dangerously reduced capillary perfusion in many organs which, when most severe, can produce cerebral, renal or mesenteric vein thrombosis.

Clinical presentation
The baby can be hypotonic, drowsy, have poor sucking, be irritable, jittery and, when severe, have convulsions. These are important danger signs.

There may also be jaundice, hypoglycaemia, and hypocalcaemia. With a high PCV the blood glucose reading may be falsely low with a normal serum glucose.

Management
First exclude dehydration and if present treat by supervised feeding and if necessary increased enteral or even IV fluids. The best way to check for dehydration is to weigh the baby and compare weight with that at birth. Normally babies lose 5–7% of their body weight in the first 3–4 days and regain their birth weight level by 10–14 days. If the baby's weight at 24 hours of age has dropped by 5% or more from the birth weight then dehydration is present.

Check blood glucose and bilirubin levels and treat appropriately (see below).

If venous PCV is 65–70% and no signs, bilirubin is below phototherapy levels, and no hypoglycaemia is present treat conservatively by regular examination for the signs described above, ensure adequate fluid intake by direct observation of feeding and regular accurate weighing before and after feeds.

If venous PCV is 70–75% and there are no danger signs, treat polycythaemia by giving additional fluid of 20 mL/kg per day enterally or IV. Also treat jaundice with phototherapy and hypoglycaemia with glucose, if necessary IV.

Section 13 Neonatal illnesses: polycythaemia

If PCV is > 75% or there are any danger signs as described above then partial exchange transfusion (PET) should be undertaken urgently.

PET involves the exchange of 20 mL/kg by repeatedly taking off aliquots of blood of up to 5 mL at a time and replacing IV with equal volumes of Ringer-Lactate or 0.9% saline. Ideally blood will be taken from a peripheral vein but, if this is not possible, either use an umbilical venous catheter placed aseptically or a peripheral arterial cannula. At the end of the procedure, re-check the PCV, Hb, bilirubin and blood glucose levels (ideally also blood calcium value). Continue to monitor the clinical state of the baby and the PCV until it is shown to fall below 60%. Re-check blood glucose and bilirubin levels as appropriate.

Respiratory disorders

Features of respiratory distress in the newborn

These include:

- tachypnoea (respiratory rate ≥ 60 breaths/minute)
- recession of the chest wall and sternum
- expiratory grunting
- nasal flaring
- prolonged apnoea (lasting for more than 20 seconds) or intermittent shorter apnoea with cyanosis or severe falls in oxygen concentration (< 90%)
- gasping
- tachycardia
- SaO_2 < 92% in air
- cyanosis is a relatively late presentation of a respiratory or cardiac cause.

These signs are relatively non-specific, arising from conditions that affect the respiratory system, as well as from cardiac, neurological and metabolic abnormalities.

Cardinal signs that characterise distress due to respiratory disorders

- Central cyanosis in room air.
- Tachypnoea: respiratory rate ≥ 60 breaths/minute (always measure over at least 1 minute, as the infant's breathing may be irregular).
- Retractions (recessions): tugging of the soft tissues between the ribs or at the edges of the rib cage.
- Grunting: a prolonged expiratory effort, usually with an audible noise.

Two of these signs are sufficient to make the diagnosis. Cyanosis may not be present, especially if the infant is receiving oxygen.

If pulse oximetry is available, the SaO_2 in infants with respiratory impairment will usually be less than 92% in air (often less than 90% in more severe cases).

Causes of early respiratory distress

'Early' respiratory distress (presenting in the first 12 hours of life) may result from a number of causes, including the following:

- 'transient tachypnoea of the newborn' associated with a delay in clearing of fetal lung fluid
- congenital pneumonia or sepsis (e.g. group B streptococcus sepsis)
- surfactant deficiency (hyaline membrane disease or respiratory distress syndrome)
- pneumothorax
- meconium aspiration
- congenital abnormalities of the lung or airways (including diaphragmatic hernia)
- hypothermia.

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Maternal fever during labour and prolonged rupture of the membranes (more than 18 hours) particularly point to pneumonia or sepsis. Pneumonia may also be due to congenital syphilis. Pneumothorax should be considered if the infant has been resuscitated using positive-pressure ventilation (although it has also been described as occur- ring spontaneously in about 1% of normal term infants). Transient tachypnoea is more common among infants delivered by elective Caesarean section (in the absence of spontaneous labour). Surfactant deficiency and infection are the most likely causes in preterm infants.

Congenital heart disease does not usually cause early respiratory distress. Cyanosis/severe hypoxaemia is the more likely presentation (*see* Section 25). Respiratory distress associated with heart failure normally occurs after the first week of life, in association with tachycardia, pallor, sweating, hepatomegaly and excessive weight gain.

Causes of respiratory distress in the newborn
Common causes
- Lack of surfactant causing respiratory distress syndrome in the preterm infant.
- Infection acquired before or during delivery.
- Transient tachypnoea of the newborn (wet lung).

Less common causes
- Meconium aspiration.
- Persistent pulmonary hypertension of the newborn.
- Pneumothorax.

Rare causes
- Pulmonary hypoplasia.
- Congenital abnormalities (e.g. diaphragmatic hernia, choanal atresia, tracheo-oesophageal fistula).
- Pulmonary haemorrhage.
- Metabolic causes (inborn error of metabolism).

Non-respiratory causes
- Congenital heart disease.
- Hypothermia.
- Severe anaemia.

Principles of treatment of respiratory diseases of the newborn
- Ensure that the airway is open and that it remains so. Thick secretions from the throat may be cleared by intermittent suction using direct observation
- Ensure that the infant is breathing. If the infant is apnoeic, gasping or has a very

slow respiratory rate, use chest inflations with a bag valve mask to re-establish breathing.

- The infant should be offered enough supplemental oxygen to treat any degree of central cyanosis and ideally to keep SaO_2 in the normal range (86–92% in preterm and 92–96% in term infants). It should never be in the hyperoxic range (above 96%), especially in a preterm infant who is receiving additional inspired oxygen.
  - Oxygen should be given either with an oxygen concentrator or from cylinders. An oxygen supply must be available at all times in areas where newborn infants are treated.
  - Pulse oximetry should be employed (if available) to assess initial disease severity, to monitor subsequent progress, and to ensure that such supplies of oxygen as are available are optimally used. Wind-up versions of pulse oximeters are available (www.PET.org.za).
  - Tents and incubators are not an efficient way of giving oxygen. Giving oxygen into a clear plastic hood (head box) placed over the head stops the oxygen supply from dropping every time a tent or incubator door is opened. Oxygen is an expensive resource and must not be wasted by giving it into incubators.
  - Nasal cannulae optimise the efficient use of the available oxygen supply. They prevent wasting of oxygen, and also make it very much easier to move and handle the infant without disrupting the supply. However, they make it rather more difficult to quantify how much oxygen is needed to control cyanosis.
- Infants should ideally have their actual oxygen needs monitored and adjusted at regular intervals.
  - Measuring the inspired oxygen concentration needed is one way of assessing the infant's changing condition. This can be done using a combination of a pulse oximeter with an inspired oxygen monitor placed in a head box next to the infant's face.
  - A simpler alternative for achieving this objective involves titrating the oxygen flow to maintain saturation on the pulse oximeter in the range 92–96% for term infants and lower, at 86–92%, for preterm infants, as described earlier.
- Keeping the infant fully clothed with a pulse oximeter attached makes it possible to dispense with any other monitoring of pulse and respiration, thus keeping the infant warm with minimal handling.
- If assisted ventilation is available and there is very severe respiratory failure, an arterial or capillary blood gas measurement can be helpful for determining the severity of respiratory acidosis and the need for ventilator sup- port such as nasal continuous positive airway pressure (nCPAP) or intubation and ventilation.
- Infants with serious respiratory distress should not be offered feeds until their condition has stabilised. Support expression of milk by the mother so that she is ready to provide breast milk when her infant has recovered. In such situations, IV infusion of 10% glucose (60 mL/ kg/day) is safest. If there are no facilities for IV infusion, breast milk or 10% glucose may be given in limited quantities (up

to 60 mL/kg/day) by orogastric tube. Nasogastric tubes may contribute to upper airway resistance, so an orogastric tube is preferred in infants with respiratory distress, although it is more difficult to keep in place, so compromise may some- times be necessary.

- Infants less than 2 days old should be started on an IV infusion of 10% dextrose at 60–90 mL/kg/24 hours. For infants more than 3 days old sodium chloride should be added to 10% dextrose to provide 2–3 mmol/kg/day and used at the age-appropriate giving rates (*see* fluid management section 8). It is recommended that in neonates it is best to use a paediatric burette (chamber) where 1 mL = 60 micro-drops (1 drop/minute = 1 mL/hour). Caution: A standard infusion set gives 20 drops/mL and can lead to dangerous fluid over-load if it is not carefully controlled.

- **Give antibiotics IV or IM (the IV route is preferable) at least for the first 48 hours in all infants with respiratory distress, as bacterial infection is a likely reason for the infant's respiratory problems. Take blood for culture first wherever possible. Antibiotics can be stopped if the blood culture results are negative and the infant is well after 72 hours.**

- In order to gain further insight into the probable cause of the problem, a portable chest X-ray machine (if available) can be useful.

- Take stringent steps to prevent nosocomial cross-infection within the unit. This can be a particular problem not only with some bacterial infections (e.g. *E. coli, Klebsiella*), but also with some troublesome viral infections (e.g. respiratory syncytial virus, RSV) that are more commonly seen later in the first month of life.

Management issues in specific respiratory conditions
Primary surfactant deficiency (respiratory distress syndrome (RDS), or hyaline membrane disease)
The principles of treating IRDS are as follows:

1 Minimal handling of the infant.
2 Supplementary oxygen.
3 IV fluids.
4 No oral feeding.
5 Continuous positive airways pressure (CPAP).
6 Avoidance of hypothermia.
7 Give antibiotics IV or IM (the IV route is preferable) at least for the first 48 hours in all infants with respiratory distress. Take blood for culture first wherever possible. Antibiotics can be stopped if the blood culture results are negative and the infant is well after 72 hours.

- Surfactant deficiency is by far the commonest cause of respiratory distress in a preterm infant in the first 3 days of life. It is a self-limiting condition, because birth always triggers a gradual increase in surfactant production. The challenge therefore is to support the infant for the first 2–3 days (72 hours) of life without

doing further damage to the lung, until such time as the deficiency resolves itself.

- The key features of RDS (cyanosis, an expiratory 'grunt', tachypnoea, and intercostal and/or subcostal recession) become clinically obvious within 4 hours of birth. Supplemental oxygen, minimal handling and IV fluid,   keeping the infant 'nil by mouth', have been the standard ingredients of care for the last 50 years. Elective surfactant administration (which is expensive) and ventilation (which is complex) have become the standard approach to management in the last 20 years. However, it is now becoming clear that the very small infant can pay a high price for chronic tracheal intubation, which by interrupting ciliary flow can interfere with the way that necrotic material is normally cleared from the lung.

- Most infants will manage well for themselves as long as they are offered help in preventing the lung from closing down and becoming airless for the 72-hour period it takes for the surfactant production to 'switch on'. The expiratory grunt that is a characteristic feature of this condition is the infant's own method of sustaining positive end-expiratory pressure (PEEP) and holding the alveoli open. Making the infant breathe against a constant positive airway pressure gradient achieves the same result. By applying this pressure at the nose (nasal CPAP), the complications associated with tracheal intubation can often be avoided.
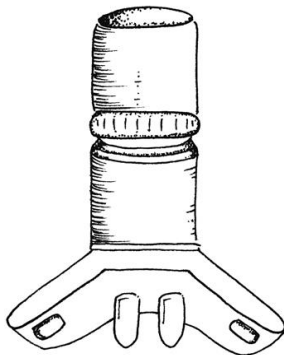

FIGURE 14.1 Nasal continuous positive airway pressure (CPAP) nasal prongs.

- To be maximally effective, we now know that CPAP should be applied as soon as there is any evidence of respiratory distress in a preterm infant. CPAP given

via   paired short cannulae or a specially made nasal mask is probably best, as it minimises airway resistance.
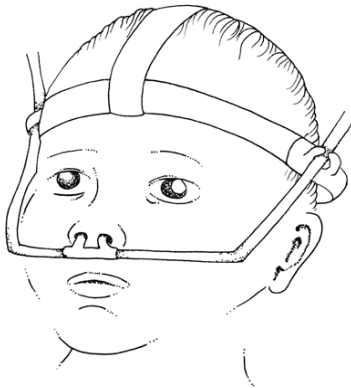

FIGURE 14.2 Nasal continuous positive airway pressure (CPAP) equipment in place.

Even though the 3-mm nasal cannulae that are normally used to provide supplemental oxygen can provide some CPAP, especially when higher flow rates are applied (6–8 litres/minute), there is a need for an air–oxygen blender to ensure that excessive and harmfully high concentrations of oxygen are not given. Humidification of the air–oxygen mixture is also required. Purpose-built CPAP systems with special nasal cannulae are better able to provide   pressures of 5–8 cm H$_2$O (a system specially designed for resource-limited settings based on an oxygen concentrator has recently become available: www.diamedica.co.uk). In general, all that is then required is a controlled flow of blended humidified air and oxygen with a simple device for producing controlled adjustable CPAP. Regular nursing attention is necessary to make sure that the nasal cannu- lae remain correctly positioned and do not cause necrotic pressure damage to the nose. This is a skill that does not take long to acquire.

Transient tachypnoea of the newborn

This is almost indistinguishable from RDS. However, unlike RDS, the signs do not progress with time in the hours after birth. Most of these infants are born at or near term. All are tachypnoeic, and a few are obviously cyanosed for 6–12 hours after birth. The condition seems to be caused by a delay in clearing lung fluid after birth. All of these infants will recover on their own so long as handling is kept to a minimum and they are

not fed until their respiratory signs have subsided. Some need supplemental oxygen, but few need it for more than 72 hours. The condition appears to be more common after Caesarean section.

Give antibiotics IV or IM (the IV route is preferable) at least for the first 48 hours in all infants with respiratory distress, as bacterial infection maybe a contributory cause of the infant's respiratory problems. Take blood for culture first wherever possible. Antibiotics can be stopped if the blood culture results are negative and the infant is well after 72 hours.

Aspiration pneumonia

Aspiration of particulate matter can occasionally almost completely block the trachea. More commonly it can also cause a chemical pneumonitis. Meconium can be particularly irritant in this regard, making the term infant very oxygen dependent for the best part of a week. Aspiration of particulate matter may also trigger persistent fetal circulation (see below).

Contrary to the findings of some studies originating from well-resourced centres in the developed world, suctioning meconium-stained infants during deliveries as soon as the head is on the perineum has made a dramatic difference to the risk of meconium aspiration syndrome in India and South Africa.

Nevertheless, with minimal handling, IV fluid and supplemental oxygen, most of these infants can be expected to make a complete recovery provided that there has been no associated hypoxic cerebral damage. Providing unnecessary respiratory support may actually make matters worse by increasing the risk of pneumothorax. Antibiotics should probably be given until it is clear that there is no associated bacterial infection.

Aspiration after birth can cause a similar picture. Milk can block the trachea, but it seldom causes much of an inflammatory reaction. However, gastric acid can be much more damaging. Recurrent minor unrecognised reflux and aspiration is probably more common than a single massive episode of aspiration, and it can certainly over time render the infant quite oxygen dependent. Infants who are hypotonic and have a poor cough reflex or repeated apnoea are probably at particular risk in this regard. Aspiration is common after an apnoeic event.

Give antibiotics IV or IM (the IV route is preferable) at least for the first 48 hours in all infants with respiratory distress, as bacterial infection maybe a contributory cause of the infant's respiratory problems. Antibiotics can be stopped if the blood culture results are negative and the infant is well after 72 hours.

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Bacterial pneumonia
This should be managed as outlined in the section on suspected infection, remembering that there may be septicaemia as well as pneumonia.

Persistent fetal circulation
This is relatively common in resource-limited countries, and is a potentially life-threatening condition leading to poor lung perfusion after birth. It may complicate fetal hypoxia, meconium aspiration, early bacterial pneumonia, diaphragmatic hernia, respiratory distress syndrome or (very occasionally) be a primary disorder.

After birth the pressure in the pulmonary vessels remains high, so that the normal fall in pressure in the right atrium, right ventricle and pulmonary arteries does not occur. As a result of this, the blood flows via the fetal circulation (i.e. the foramen ovale and ductus arteriosus) from the right side of the heart to the left. This blood has not been oxygenated, so the infant soon becomes cyanosed. It is difficult to differentiate this from a congenital cardiac malformation. Serious cyanosis in an infant with a well- aerated lung on chest X-ray and progressive acidosis can cause rapid self-perpetuating cyclical deterioration.

The treatment in the first instance is oxygenation, minimal handling, IV fluids and avoidance of oral feeds. Metabolic acidosis should be vigorously and rapidly corrected or even over-corrected. Drugs that cause pulmonary vasodilation, such as sildenafil or magnesium sulphate, have been used to some effect. However, they can lead to serious hypotension and should be used very selectively in a controlled environment in specialised centres.

Give antibiotics IV or IM (the IV route is preferable) at least for the first 48 hours in all infants with respiratory distress, as bacterial infection maybe a contributory cause of the infant's respiratory problems. Take blood for culture first wherever possible. Antibiotics can be stopped if the blood culture results are negative and the infant is well after 72 hours.

Survival is more likely in a unit that is capable of providing sustained respiratory support, and early transfer should be considered when possible.

Pneumothorax
This is present more frequently than expected, and may occur spontaneously in up to 1–2% of infants. It is often asymptomatic, and may be associated with meconium aspiration, too high inflation pressures used during mechanical ventilation or resuscitation, and respiratory distress syndrome. It does not automatically need to be treated, unless there is progressive respiratory distress. Confirmation by chest X-ray (if available) is often too time- consuming, especially in the case of a **rapidly developing tension pneumothorax**. It may be possible to diagnose a pneumothorax clinically by simple observations. The abdomen is often

distended by downward displacement of the liver and spleen. The breath sounds may be reduced on the affected side. A hyper-resonant chest with mediastinal shift (trachea deviated away from the side of the suspected pneumothorax) and rapidly deteriorating clinical condition with severe hypoxaemia and/or cardiovascular compromise (bradycardia, hypotension) strongly suggests a tension pneumothorax. This requires an immediate needle thoracocentesis followed (if this results in an immediate improvement in respiratory and cardiovascular function) by the insertion of a chest drain into the fourth or fifth intercostal space in the mid to anterior axillary line (*see* Section 34). In an emergency situation with a rapidly deteriorating cardiac and respiratory function, this must be done without prior X-ray confirmation. Transillumination can be useful if a 'cold light' (fibre-optic light source) is available (the affected side may glow brightly).

A pneumothorax that does not result in severe respiratory distress, and is not under tension, may spontaneously resolve without mechanical removal of the pleural air, but oxygen and careful monitoring are required.

Lung hypoplasia due to oligohydramnios Chronic loss of liquor for many days before birth can occasionally impede lung growth enough to threaten survival, but what looks like a serious problem at delivery can occasion- ally resolve quite rapidly after 1–2 days. However, where the oligohydramnios is due to bilateral renal agenesis or dysplasia, the prognosis for survival is very poor. The stiff- ness of the small malformed lungs in these cases causes marked intercostal and subcostal recession with unrelievable cyanosis. Chest X-ray will often reveal an untreatable pre-terminal pneumothorax. The infant's face may appear flattened and there may be limited extension of the elbows and knees due to oligohydramnios.

Congenital malformations

The most common congenital defect causing respiratory distress soon after birth is **diaphragmatic hernia.** This occurs in 1 in 4000 births, and more commonly affects the left side. Clinical examination reveals respiratory distress, and reduced air entry on the affected side with a displaced apex beat and scaphoid abdomen. The chest X-ray is diagnostic. It used to be thought that early surgery improved the likelihood of survival, but it is now known that this is not the case. Therefore immediate transfer does not have to be considered until the infant's initial respiratory problems have stabilised. During the interim period, an IV line and open nasogastric tube should be in place to keep the gut empty of gas, and feeding should be withheld. Restricted lung growth means that only about 50% of these infants have any chance of survival. Use a headbox rather than nasal cannula oxygen, and place an open nasogastric tube to prevent bowel distension, which makes the condition worse.

Management of diaphragmatic hernia

This includes the following:

- oxygen supplements

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- minimal handling
- IV fluids and withholding of oral feeds
- a nasogastric tube to keep the stomach empty
- stabilisation of respiration with mechanical ventilation fol- lowing intubation or continuous negative extra-thoracic pressure (CNEP) can be helpful if available
- transfer to surgical care if the infant responds to treatment.

A number of rare generalised skeletal abnormalities that affect rib growth also cause severe untreatable lung hypoplasia.

Congenital heart disease can occasionally cause overt cyanosis from birth, but there are seldom any associated signs of respiratory distress (*see* Section 25).

Apnoeic/hypoxaemic episodes

Apnoea is the cessation of respiration or a hypoxaemic event associated with signs of cardiorespiratory decompensation (bradycardia, cyanosis and pallor). Apnoeic episodes are common in preterm infants under 32 weeks' gestation ('apnoea of prematurity'). In term infants, apnoea usually signifies an underlying pathological condition.

Apnoea of prematurity

This is often characterised by a brief cessation of respiration that responds to gentle tactile stimulation, and may vary significantly in duration and severity, especially in very- low-birth-weight infants. Sometimes, isolated bradycardia with brief oxygen desaturation events is identified without a clinically apparent apnoea. The aetiology of apnoea of prematurity is often a mixture of impaired central nervous system respiratory control ('central apnoea'), intrapulmonary shunting and upper airway obstruction. Sometimes recurrent apnoea is associated with gastro-oesophageal reflux, particularly in neurologically compromised infants with poor airway-protective reflexes.

Oral theophylline or caffeine, by its effect on the respiratory centre, may reduce or even eliminate the severity and frequency of apnoeic events. Caffeine has become the preferred methylxanthine by some neonatologists, because it has a long half-life (allowing once daily dosing), fewer side effects and serum levels do not have to be monitored. Continuous positive airway pressure (CPAP) or rarely mechanical ventilation may become necessary to control recurrent apnoea.

The diagnosis of 'apnoea of prematurity' is one of exclusion, as various other processes may cause or exacerbate apnoea. In the case of a preterm infant, these include the following:

- respiratory distress (surfactant deficiency, pneumonia, pulmonary oedema due to a persistent ductus arteriosus)
- intraventricular haemorrhage
- hypoglycaemia
- over-heating or hypothermia
- sepsis
- severe anaemia may also contribute to apnoea.

Pulmonary parenchymal disease

Any condition that causes decreased lung compliance or impaired gas exchange can contribute to apnoea. Appropriate pulmonary support should be provided for adequate gas exchange, and the underlying pulmonary condition should be treated.

Airway obstruction

This may result from simple malpositioning of the head (e.g. hyper-flexion or hyper-extension of the neck), especially in preterm infants. Congenital airway anomalies

such as tracheo-oesophageal fistula or an aberrant thoracic blood vessel compressing the trachea (vascular sling) may also present as apnoea. Maintaining proper head positioning or surgical correction of the underlying anomaly should be provided.

Infection
Infection must always be excluded and antibiotics adminstered until infection has been ruled out by subsequent clinical findings and laboratory results (complete blood counts, chest X-ray, blood cultures, etc.).

Convulsions (see Section 21)
Convulsions may present primarily as apnoea. This possibility should be considered especially in term or near-term infants with no other identifiable cause of apnoea. In such cases there may be a poor response to positive pressure ventilation. Convulsions in the first 1 to 3 postnatal days are usually due to intra-partum hypoxia. If there is a history of an operative vaginal delivery (e.g. forceps) or other birth trauma, this may indicate the possibility of an intracranial haemorrhage.

Maternal medication
A common cause of apnoea in the newborn can be intra-partum morphine or pethidine administration for maternal pain or sedation during the last 4 hours before delivery. The effects can be reversed by administering naloxone hydro- chloride (100 micrograms/kg, usually given IM). Naloxone should not be given if there is a history of drug abuse with narcotics in pregnancy, as acute neonatal narcotic withdrawal may be precipitated (*see* Section 4).

Exposure to high levels of magnesium sulphate given to the mother for eclampsia has also been associated with apnoea in the immediate postnatal period. This is usually a self-limiting process that very rarely requires mechanical ventilation.

Continuous monitoring, preferably with a pulse oximeter, is needed especially if the infant becomes bradycardic or cyanosed with the apnoea.

Treatment
- Gentle stimulation is usually all that is required to start the infant breathing again.
- Bag-and-mask resuscitation may occasionally be called for, and there should always be equipment immediately available and ready to use (not locked away in a cup-board) should this be necessary.
- If available, oral caffeine may reduce the number of episodes in a preterm infant. Caffeine seldom causes the tachycardia and other side effects associated with theophylline. It is advisable to continue caffeine for 4–5 days after cessation of apnoea. Recurrent apnoea that does not respond to caffeine occasionally requires a period of nasal CPAP or mechanical ventilation.
- If an apnoea monitor is available it can be used, but a pulse oximeter with the alarm turned on for hypoxaemia is much safer, as apnoea (absent ventilation)

can occur despite continued breathing movements. This will also identify any baseline low oxygen saturation which, when treated, may help to prevent apnoea.

TABLE 15.1 Caffeine doses for apnoea of prematurity given intravenously or orally

Preparations	Each dose	Dose frequency	Notes on administration
Caffeine citrate	20 mg/kg	Loading dose	If oral dose is too large, divide into two and give 1 hour apart
	5–8 mg/kg maintenance	Once daily	
Caffeine base	10 mg/kg	Loading dose	Give IV loading dose over 30–60 minutes diluted as much as possible
	2.5–4 mg/kg maintenance	Once daily	

Bronchiolitis

This is a lower respiratory viral infection, typically most severe in young infants, occurs in annual epidemics, and is characterised by airways obstruction and wheezing. Respiratory syncytial virus is the most important cause. It is much more serious in preterm infants and in those with congenital heart disease. Secondary bacterial infection may occur and is common in some settings. Episodes of wheeze may occur for months after an attack of bronchiolitis, but will eventually stop.

Clinical features of bronchiolitis

- Infants are coryzal, have a cough and may feed poorly or even be unable to suck and feed. There may be vomiting.
- The nose is often obstructed by secretions.
- On examining the chest, there may be hyperinflation, chest wall in-drawing, nasal flaring, grunting, wheeze and fine crackles at the lung bases.
- Young infants may present with apnoeic/hypoxaemic episodes which may be recurrent and life-threatening.
- There may be hypoxaemia, with SaO_2 less than 94%, with or without cyanosis.
- Some infants will have such severe respiratory distress that there is gasping; this is pre-terminal.

Treatment

1. Only supportive treatment (e.g. oxygen, gentle suction of the nose, and fluids) is of benefit. Antibiotics and bronchodilators have no role. However, in the most severe cases and unless you are certain that pneumonia is not present, it is safer to give antibiotics and a trial of a bronchodilator (stop the bronchodilator if it is not helping).
2. Non-invasive respiratory support to help to overcome small airway obstruction (nasal CPAP and continuous negative extrathoracic pressure (CNEP)) may be valuable (see Textbook). CNEP may be more effective because of the nasal blockage that accompanies bronchiolitis.
3. Give oxygen by nasal cannulae to keep SaO_2 in the range 94–98%. Check that the nasal cannulae are in the correct place, and check frequently that they are not blocked by secretions.
4. Nasal clearance. Gentle nasal suction should be used to clear secretions in patients in whom nasal blockage is thought to be causing respiratory distress. This may be aided by saline nasal drops or spray.
5. Ensure that daily maintenance fluids are achieved. If this is not possible by mouth, use nasogastric feeding. This should be considered in any patient who is unable to maintain oral intake or hydration (use the mother's expressed breast milk if possible and if tolerated).
6. If the patient is vomiting despite nasogastric feeding, or severe respiratory distress is present, give fluids IV.
7. If there are signs of pneumonia, give antibiotics (*see* Section 17).

8. If fever (≥ 39°C or ≥ 102.2°F) is causing distress, give paracetamol. High fever is the exception rather than the rule in bronchiolitis, and should make you suspect bacterial infection.

Failure to improve

If the condition worsens suddenly, consider pneumothorax, although this is uncommon. Tension pneumothorax associated with major respiratory distress and shift of the heart requires immediate relief by needle thoracocentesis (i.e. placing a needle to allow the air that is under pressure to escape) (*see* Section 34). If needle thoracocentesis is helpful, insert a chest tube with an underwater seal until the air leak closes spontaneously and the lung expands (*see* Section 34). The signs of pneumothorax in severe bronchiolitis may be difficult to detect clinically. However, needle thoracocentesis in the absence of a pneumothorax may cause one, so if you are unsure, take a chest X-ray. Even on a chest X-ray, the diagnosis may be very difficult due to the areas of hyperlucency in bronchiolitis caused by air trapping.

If respiratory failure develops, nasal continuous positive airways pressure (CPAP) or continuous negative extrathoracic pressure (CNEP) may be of benefit (*see* Textbook for details).

If apnoeic episodes develop (this is most likely in premature infants), give bag-valve-mask resuscitation, then nasal CPAP or CNEP. Sometimes **intubation and ventilation may be needed in a high-dependency ward (if available)**; if so, contact an anaesthetist urgently.

Infection control

Bronchiolitis is infectious and easily transmitted to other infants in hospital. Babies in the neonatal unit are particularly at risk. The following strategies may reduce the risk of cross-infection (*see* Textbook):

- hand washing between patients
- the wearing of gloves and aprons
- ideally isolate the affected patient, but close observations are needed
- restrict visiting by anyone with symptoms of upper respiratory tract infection.

Pneumonia in infancy

Pneumonia is responsible for around two million deaths annually in children under 5 years of age. In resource- limited countries, most of these infections are bacterial, and the most common causative bacteria are *Streptococcus pneumoniae* and *Haemophilus influenzae*. In severely malnourished children, *Klebsiella pneumoniae* and *Staphylococcus aureus* are common causative organisms.

Immunisation
Pneumococcal conjugate vaccine has been introduced to the primary immunisation schedule in many well-resourced countries, and reduces the incidence of X-ray-proven pneumonia in infants by around one-third. The HiB vaccine (against encapsulated *Haemophilus influenzae* type B) will not protect against unencapsulated *H. influenzae*, which causes some cases of pneumonia in resource-limited countries. Nevertheless, the HIb vaccine is very effective against other very serious infections caused by *H. influenzae* (e.g. meningitis, epiglottitis), and should be given to all infants in every country.

Management of the infant with acute lower respiratory infection (ALRI)

Children at greatest risk of dying from an ALRI have the following risk factors:
- age under 1 year
- malnutrition
- pneumonia as a complication of infection with measles, pertussis, malaria or HIV.

Diagnosis of ALRI

In many hospitals in resource-limited countries, special tests (e.g. blood culture, microbiology of respiratory secretions, X-rays) may be limited or unavailable. However, because the prevalence of bacterial pneumonia is high, the diagnosis must usually be made clinically. This will not be 100% accurate, so a few children may receive antibiotics unnecessarily (i.e. clinical diagnosis has less than 100% specificity). However, it is more important not to miss children who do need antibiotics (i.e. clinical diagnosis should have a good sensitivity). Clinical diagnosis may be as accurate as an X-ray and more helpful in deciding whether treatments such as oxygen are indicated. The clinical features will also help to decide how severe the infant's infection is and what treatment is appropriate.

The following clinical features should be recorded:
- The presence of cyanosis, which is best seen in the lips or tongue. It may be missed if the lighting is poor or if the infant is anaemic (e.g. due to co-infection with malaria), and it can be difficult to detect in black infants. Cyanosis is a late sign of respiratory problems, and if possible oxygenation should be assessed with a pulse oximeter. Normal saturation at sea level (SaO_2) is greater than 94%.
- Inability of the infant to drink.

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- The presence of chest wall in-drawing (an inward motion of the lower chest wall when the infant breathes in).
- The presence of grunting (expiratory braking).
- The presence of hyperinflation (bronchiolitis).
- Elevated respiratory rate. Respiratory rate is measured over 1 minute, using a suitable timing device. The respiratory rate varies with age. Table 17.1 lists the abnormal values for respiratory rate for various age groups.

TABLE 17.1 WHO definition of abnormally fast breathing

Age	Abnormally fast breathing
<2 months	≥ 60 breaths/minute
2–12 months	≥ 50 breaths/minute

Remember that conditions such as severe anaemia, dehydration and high fever are themselves accompanied by a raised respiratory rate.

A high fever in an infant with breathing difficulties may be due to pneumonia, bacterial tracheitis or even epiglottitis. If the airway is clear, the most likely diagnosis is pneumonia. Although high fever and respiratory signs are the usual way for pneumonia to present, pneumonia should always be considered in the list of causes of abdominal pain and neck stiffness.

Clinical examination (or chest X-ray) cannot reliably differentiate between a viral pneumonia and a bacterial one, so all cases are treated with antibiotics.
Features of pneumonia include the following:
- fever, cough, breathlessness and lethargy
- pleuritic chest pain, abdominal pain and neck stiffness (these indicate pleural involvement).

- signs of consolidation:
  - dull percussion
  - reduced breath sounds
  - bronchial breathing may be absent in an infant
- chest X-ray may show pleural effusion or empyema as well as consolidation.

FIGURE 17.1 Right middle lobe pneumonia. Note the loss of the right heart border.

FIGURE 17.2 Left lower lobe pneumonia. Note that the silhouette of the diaphragm cannot be seen on the left. The right middle lobe is also affected.

FIGURE 17.3 Right upper lobe pneumonia. Note that the horizontal fissure is pulled up.

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Auscultation should always be undertaken, but only after first checking for cyanosis, observing the breathing pattern and the other signs as described above. Important clinical signs include evidence of the following:

- consolidation or effusion/empyema
- wheeze
- bronchiolitis (hyperinflation with crackles at the lung bases)
- alveolitis (e.g. in HIV-induced *Pneumocystis* pneumonia) with end-inspiratory crackles.
- pericardial involvement (rare)
- pneumothorax (rare).

A chest X-ray may be helpful if there is any doubt about the diagnosis or if the infant is seriously ill.

Figures 17.1, 17.2 and 17.3 show the appearance of lobar pneumonia affecting different lobes.

Additional features of ARLI usually include a fever and a cough.

Table 17.2 gives guidelines for the assessment and treatment of acute respiratory infection.

Diagnosis of severe pneumonia

This is diagnosed by the presence of cough or difficult breathing plus at least one of the following:

- central cyanosis
- inability to breastfeed or drink, or vomiting after every drink
- convulsions, lethargy or unconsciousness
- severe respiratory distress.

In addition, some or all of the other signs of pneumonia may be present, such as the following:

- fast breathing:
  - age < 2 months: ≥ 60 breaths/minute
  - age 2–11 months: ≥ 50 breaths/minute
- nasal flaring
- grunting (in young infants)
- lower chest wall in-drawing
- chest auscultation signs of pneumonia:
  - decreased breath sounds
  - bronchial breath sounds
  - crackles
  - abnormal vocal resonance (decreased over a pleural effusion, and increased over lobar consolidation)
  - pleural rub.

Obtain a chest X-ray and measure SaO_2 (if available).

For infants with no evidence of pneumonia but with signs suggesting a chest infection, look for ear and throat infections or infections in another system and treat accordingly.

TABLE 17.2 The management of children with different severities of acute lower respiratory tract infection (ALRI) (modified from the WHO *Pocket Book of Hospital Care for Children*, second edition 2014)

Sign or symptom	Classification	Treatment
Central cyanosis and/or SaO$_2$ <90% Severe respiratory distress (e.g. head nodding, gasping, chest wall indrawing, grunting) Fast breathing as below under 'pneumonia that is not severe' Decreased breath sounds and/or bronchial breathing Crackles in the lung fields Vocal resonance and percussion suggesting consolidation and/or effusion Pleural rub Inability to drink, vomiting, reduced consciousness	Severe pneumonia	Admit to hospital Give IV/IM appropriate antibiotics* Give oxygen Manage the airway Treat high fever if present If the child has HIV infection, refer to specific guidelines (Textbook and *see* Section 12)
Fast breathing but no chest wall in-drawing: ≥60 breaths/minute in a child aged < 2 months ≥50 breaths/minute in a child aged 2–11 months Definite crackles on auscultation	Pneumonia that is not severe	Home care (but depends on home circumstances and overall clinical state of the child) Give an appropriate antibiotic* Advise the mother when to return if treatment fails on amoxicillin and more appropriate second-line treatment is needed Follow up in 2 days
No signs of pneumonia or severe pneumonia	No pneumonia Cough or coryza	Home care Advise the mother to return for follow-up in 5 days if not improving If coughing for more than 14 days, consider investigations for TB, asthma, inhaled foreign body, pertussis, HIV, bronchiectasis and lung abscess (*see* Textbook for details).

* See details of antibiotics, routes of administration and durations for different categories of pneumonia in section 'Antibiotics' below

Section 17 Neonatal illnesses: pneumonia

Oxygen

Children with severe or very severe pneumonia are likely to be hypoxaemic. However, cyanosis is a late sign of hypoxaemia.

Oxygen must always be available in sufficient quantity to provide 24-hour treatment without depending on the availability of a reliable electricity supply.

Give oxygen if the child shows any of the following:

- restlessness (if oxygen makes the infant more comfortable)
- severe chest wall in-drawing
- a breathing rate of more than 70 breaths/minute
- grunting (in an infant under 2 months of age)
- gasping
- if a pulse oximeter is available, SaO_2 of less than 94% (at sea level; lower values will be normal at high altitude, and normal values of SaO_2 should be known for healthy local children in your area if it is at high altitude). Aim to maintain SaO_2 in the range 94–98%.

Give oxygen until the signs of hypoxia (e.g. severe lower chest wall in-drawing, high breathing rates and/or $SaO_2 < 94\%$ in air) are no longer present.

Oxygen delivery

A good source of oxygen is an oxygen concentrator. This is a durable piece of equipment, but it requires a continuous supply of mains electricity to provide oxygen. It works on the 'molecular-sieve' principle, removing nitrogen from room air. The alternative is cylinder oxygen, but cylinders must be replenished regularly and need to be available at all times, which is expensive and may give rise to transport difficulties. **A combination of the two supplies of oxygen is essential.** An oxygen generator which can provide oxygen and fill cylinders when the electrical power is available (e.g. Diamedica equipment). The concentrator or cylinder should be connected to a low-flow meter. The use of a flow splitter will allow up to four children to receive oxygen from one source. The oxygen should be delivered to the child using nasal cannulae. These should be only 2–3 mm long, to avoid nasal irritation.

Figure 17.4 shows the delivery of oxygen through nasal cannulae.

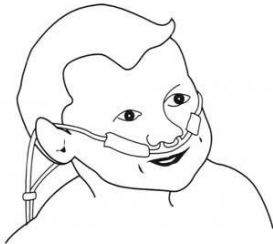**A mask should be used to give high-flow oxygen during resuscitation.**

FIGURE 17.4 Nasal cannulae for delivering oxygen. The cannula has been taped to the infant's cheeks, close to the nostrils. The tubing is run under the infant's shirt to stop them pulling it, and leads to the low-flow meter and oxygen concentrator or cylinder. A flow splitter may be used.

Nurses should check frequently that the nasal cannulae are not blocked with mucus and are in the correct position, and that all connections are secure.

Antibiotics

Infants who are vomiting or who require IV fluids should have their antibiotics given intravenously (preferably), or intramuscularly if vascular access is difficult to achieve or maintain, for the first 48 hours. Some antibiotics, such as gentamicin, are always given IV or IM. Certain antibiotics are reserved for specific circumstances, such as high-dose co-trimoxazole for suspected *Pneumocystis jirovecii* pneumonia, and flucloxacillin or cloxacillin for pulmonary abscess or bacterial tracheitis where *Staphylococcus aureus* is likely to be responsible. These are described at the end of the section on antibiotics.

For severe pneumonia:

- Give ampicillin 50 mg/kg IM/IV or benzyl penicillin 50000 units/kg that is 30 mg/kg IM or IV every 6 hours plus gentamicin 7.5 mg/kg IM or IV once a day for 5 days. Then, if the infant responds well, complete treatment with oral amoxicillin (25 mg/kg three times a day, maximum 500 mg, or 1 gram in severe cases) plus IM or IV gentamicin 7.5 mg/kg once daily for a further 5 days.
- Alternatively, if the above are not available, give chloramphenicol (25 mg/kg IM or IV every 8 hours) until the infant has improved. Then continue orally four times a day for a total course of 10 days.
- Or use ceftriaxone (80 mg/kg IM or IV once daily) or cefotaxime (50 mg/kg IV 6-hourly) for 10 days.
- If the infant does not improve within 48 hours, switch to gentamicin (7.5 mg/kg IM or IV once a day) and cloxacillin (50 mg/kg IM or IV every 6 hours), as described below for possible staphylococcal pneumonia.

For pneumonia that is not severe:

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- Treat the infant as an outpatient.
- Give amoxicillin 40mg/kg twice a day for 5 days.
- Give the first dose at the clinic, and teach the mother how to give the other doses at home.
- In infants aged 2–12 months who have some of the signs suggestive of non-severe pneumonia without a high fever but with wheeze, the most likely diagnosis is bronchiolitis. This is caused by a virus, and in the absence of signs suggesting the development of secondary bacterial infection (severe pneumonia), antibiotics are not necessary. The WHO recently published the following conclusion: Antibiotics are not routinely recommended for children aged 2 months to 5 years with non-severe pneumonia (that is, fast breathing with no chest indrawing or danger signs) with a wheeze but no fever (temperature below 38°C), as the cause is most likely to be viral. **In infants under 2 months of age antibiotics should always be given under these circumstances.**

Treatment for infant with any degree of pneumonia
1. Nurse in a thermo-neutral environment (lightly clothed in a warm room at around 25°C).
2. *Fever*. Remember that fever may not be simply due to the pneumonia. Consider other diagnoses, such as malaria.

If the infant has fever (≥ 39°C or ≥ 102.2°F) that appears to be causing distress, give paracetamol oral or rectally, 10–15 mg/kg 4- to 6-hourly. *NSAIDS are not appropriate.*
3. Remove by gentle suction under direct observation any thick secretions in the throat which the infant cannot clear.
4. Ensure daily maintenance fluids appropriate for the infant's age, but avoid over-hydration.
5. Encourage breastfeeding and oral fluid intake.
6. If the infant cannot drink, insert a nasogastric tube and give maintenance fluids in frequent small amounts. If the infant is taking fluids adequately by mouth, do not use a nasogastric tube, as it increases the risk of aspiration pneumonia. Encourage eating as soon as food can be taken. When the infant is recovering, nutritional rehabilitation may be necessary.

Failure to improve If the infant has not improved after 2 days, or if their condition has worsened, re-examine them thoroughly, looking for signs of pleural effusion/empyema and other causes of fever. If possible, obtain a chest X-ray. This may show a pleural effusion or empyema (*see* Textbook for details of management) into which antibiotics cannot penetrate, or it may show the characteristic pneumatocoeles (lung abscesses) of staphylococcal pneumonia.

Also consider *Mycoplasma pneumoniae* or *Bordetella pertussis* infections. Pertussis should be recognisable because of the characteristic nocturnal emetic cough and the whoop in the child over 2 years of age.

Prescribe erythromycin if either of these infections is suspected. It should be given orally as follows:

- 125 mg 6-hourly (infants)

Pneumonia that does not respond to standard antibiotics within 2 weeks
Tuberculosis
An infant with persistent fever for more than 2 weeks and signs of pneumonia should be evaluated for tuberculosis. If another cause of the fever cannot be found, tuberculosis should be considered and treatment for tuberculosis, following national guidelines, may be initiated and response to anti-tuberculous treatment evaluated.

Infants who are HIV-positive or in whom HIV is suspected
Some aspects of antibiotic treatment are different in infants who are HIV-positive or in whom HIV is suspected. Although the pneumonia in many of these children has the same aetiology as in children without HIV, pneumocystis pneumonia (PCP), often at the age of 4–6 months, is an important additional cause which must be treated when present (*see* Textbook). While confirming the diagnosis, give ampicillin plus gentamicin as described above for severe pneumonia.

Staphylococcal pneumonia
Staphylococcal pneumonia is suspected if there is rapid clinical deterioration despite treatment, a pneumatocoele or necrotising pneumonia with effusion on chest X-ray, numerous Gram-positive cocci in a smear of sputum, or heavy growth of *Staphylococcus aureus* in cultured sputum or empyema fluid.

- Treat with flu/cloxacillin (50 mg/kg IM or IV every 6 hours) and gentamicin (7.5 mg/kg IM or IV once a day) for at least 7 days.
- When the infant improves, continue cloxacillin/flucloxacillin orally four times a day for a total course of 3 weeks. Note that cloxacillin can be substituted by another anti- staphylococcal antibiotic, such as oxacillin, flucloxacillin or dicloxacillin.

Severe dehydration
This may be a problem in pneumonia, arising from high fever and poor fluid intake
Look for signs of dehydration or shock (tachycardia, weak pulse, poor peripheral circulation, and capillary refill time prolonged by more than 3 seconds).

- If the infant is shocked: Site an intravenous line and give a bolus of crystalloid – for example, Hartmann's solution, Ringer-lactate or colloid 10–20 mL/kg (10 mL/ kg in a neonate).
- If the infant is not shocked but is clinically dehydrated (*see* Section 28): Give oral rehydration solution (ORS), 15–20 mL/kg/hour for 2 hours orally or via nasogastric tube. Encourage breastfeeding.

Management of ALRI in special circumstances
Management of the infant under 6 months of age

Section 17 Neonatal illnesses: pneumonia

Young infants with severe ALRI/pneumonia may not cough, but rather they may present with apnoea, poor feeding or hypothermia. Remember that in infants under 2 months of age, the abnormal respiratory rate cut-off is higher (≥ 60 breaths/minute). For infants aged 2–12 months the cut-off is ≥ 50 breaths/minute.

Note that some chest wall in-drawing is normal during REM (dream) sleep in all infants.

All infants with severe ALRI/pneumonia should be admitted to hospital for treatment.

Bronchiolitis is a frequent diagnosis, and usually involves hypoxaemia due to ventilation to perfusion mismatch. Oxygen is usually required. Additional respiratory support (*see* Section 35) may also be necessary if available, especially if there is apnoea or severe respiratory distress leading to exhaustion.

Grunting (a short expiratory noise at the start of expiration) is common and usually an indication for oxygen.

Avoid using chloramphenicol in infants under 2 months of age (there is a risk of development of 'grey baby syndrome'). Use benzylpenicillin or ampicillin plus gentamicin instead.

Respiratory infection in neonates may rapidly develop into septicaemia, shock and death, so it is essential to act quickly.

Haemorrhage in the neonate

Causes of haemorrhage

An infant's blood volume approximates 80 mL/kg of body weight. Peripartum haemorrhage of relatively small amounts of blood can therefore result in hypovolaemic shock in the newborn. Common causes may include a slipped ligature on the umbilical cord, intrauterine feto–maternal haemorrhage (diagnosed by the Kleihauer–Betke test), or subgaleal haemorrhage. Vasa praevia or an accidental incision of the placenta during Caesarean section are other causes.

The Kleihauer–Betke test is a blood test used to measure the amount of fetal haemoglobin transferred from the fetus to the mother's bloodstream. It is usually performed on Rhesus-negative mothers to determine the dose of Rho(D) immune globulin needed to inhibit the formation of Rh antibodies in the mother and prevent Rh disease in future Rh-positive children. It is also the standard method of quantitating feto–maternal haemorrhage. The test exploits the differential resistance of fetal haemoglobin to acid. A standard blood smear prepared from the mother's blood is exposed to an acid bath. This removes adult haemoglobin, but not fetal haemoglobin, from the red blood cells. Subsequent staining makes fetal cells (containing fetal haemoglobin) appear rose-pink in colour, whereas adult red blood cells are only seen as 'ghosts'. The percentage of fetal to maternal cells is calculated under a microscope.

Bleeding in the first week of life is uncommon, but may signify haemorrhagic disease of the newborn or clotting factor deficiency.

Presenting features

The infant will appear pale with weak peripheral pulses, tachypnoea and a tachycardia that may exceed 200 beats/ minute. Blood pressure may be low or undetectable even in a term infant but is very difficult to measure in neonates. The haematocrit and haemoglobin concentration may be normal in an infant with acute hypovolaemic shock, and are an unreliable early indicator of the amount of blood lost in the first few hours after the bleed. Obvious blood loss rarely results in hypovolaemic shock.

Common sites of blood loss include the umbilical stump and the gastrointestinal tract. In the latter case, there may be doubt as to whether blood is of maternal origin (blood swallowed at delivery or from a bleeding nipple) or infant origin. In some cases this can be resolved by the Apt test.

Apt test

Mix 1 part of the blood-containing fluid (vomitus, gastric aspirate or liquid stool) with 5 parts of distilled water. Centrifuge it, and then mix 1 mL of the supernatant with 1.25 mL of 0.25% sodium hydroxide (NaOH). A yellow- brown colour signifies

maternal blood, whereas fetal haemoglobin remains pink. The solution must be pink to start with.

Treatment

- In an emergency in a shocked infant take a blood sample for blood grouping and cross-matching. Give O Rh-negative or cross-matched blood (20 mL/kg) at a rate depending on the degree of shock (usually the first 10 mL/kg can be safely given over 5 minutes), monitoring the response and reducing the rate of infusion as improvement occurs. Sometimes a further 10–20 mL/kg of cross-matched blood may be necessary.
- If O-negative or cross-matched blood is not available, use 10–20 mL/kg of 4.5% albumin or Ringer-lactate or Hartmann's solution.
- If there is overt bleeding, take a blood sample for blood grouping and cross-matching, haemoglobin, platelet count, film and clotting studies. Then give 1 mg of vitamin K (phytomenadione or phytonadione) IV. If bleed- ing continues, give 20 mL/kg of fresh-frozen plasma (if available). Administer platelets if the count is < 60 000/ mm^3. Bleeding due to haemorrhagic disease of the newborn usually stops within 30 minutes of vitamin K administration.

Jaundice

Many infants become jaundiced for a few days after birth. This is because bilirubin released from the breakdown of red blood cells has to be excreted by the infant after birth. *In utero*, bilirubin would cross the placenta to reach the maternal liver, from where it would be processed and eliminated. The neonatal liver takes time to develop normal functioning. The serum bilirubin level usually rises after the first 24 hours of life and peaks at 100–300 µmol/litre by 3 to 5 days after birth.

Causes of "physiological jaundice" in the neonatal period include the following:

- increased breakdown of red blood cells in the first few days of life
- reduced lifespan of red blood cells (70 days, compared with 120 days in the adult)
- less efficient metabolism of bilirubin by the immature liver entero-hepatic circulation of bilirubin.

'Physiological jaundice' is common, affecting at least one-third of normal term infants. Jaundice can be considered physiological and does not require treatment or investigation if the following criteria are met:

- Jaundice is not present in the first 24 hours of life.
- The infant is well, and free from signs of infection, without enlargement of the liver or spleen.
- The bilirubin concentration does not exceed 300 µmol/ litre (approximately 17 mg/dL) at any stage (term infants only). A much lower acceptable level is set for preterm infants.
- The bilirubin concentration reaches a peak on the fourth or fifth day of life.
- The jaundice has fully resolved by the end of the second week of life.

The risk of jaundice can be reduced by encouraging early unrestricted demand breastfeeding.

There is no evidence whatsoever to support the widely held belief that giving extra water for the infant to drink either reduces the risk of jaundice or is helpful in treatment. In fact the opposite has been shown. Giving water is likely to reduce the frequency of breastfeeds and increase the risk of jaundice. Dehydration should be avoided by encouraging frequent breast feeds.

Assessing the degree of jaundice

Various means of estimating the degree of jaundice make it easier to determine which infants really need any intervention. Healthcare professionals can sometimes make a rough estimate of the degree of jaundice by looking at the skin colour (this is best undertaken in natural daylight), but it can only be divided into simple categories such as 'slight', 'moderate' or 'severe'. The face is often the first part of the body to show signs of jaundice. The trunk usually only becomes yellow as jaundice deepens. Finally, the

palms of the hands and soles of the feet become jaundiced. These observations are just estimates, and sometimes have to be confirmed by other means. Jaundice in the newborn infant can be missed in infants with dark skin, but can be more easily judged once the skin is blanched free of blood by finger pressure.

Bilirubin encephalopathy (kernicterus) in the absence of overt haemolysis is excessively uncommon in the term infant, unless the serum bilirubin level exceeds 425 µmol/ litre.

Note:

- µmol/litre divided by 17.1 = mg/dL
- mg/dL multiplied by 17.1 = µmol/litre.

Several electronic devices have been developed for assessing skin colour, but none have yet been shown to work significantly better than the simple 'icterometer' devised in 1960 and still in use. Jaundice is assessed by pressing the   clear plastic of this simple device against the tip of the nose (or against the gums or tongue in a dark-skinned infant, where it has been shown to be accurate in a South African study), and then matching the colour of the skin against the icterometer's colour scale. Levels in excess of 350 µmol/litre are unlikely to be missed if a blood sample is taken once the icterometer reads 3.5 µmol/litre or more. This too little known device, which costs only US$39, is still made by Cascade Health Care Products of Salem, Oregon, in the USA. Measuring the degree of jaundice by this method is of no value once phototherapy has been started.

The bilirubin concentration can be most simply and accurately measured by simple spectrophotometry of serum obtained by centrifuging blood in a capillary tube. Several easily operated machines are available. Ward- based devices for assessing the bilirubin content of a spun micro-haematocrit tube optically are accurate until the level exceeds 350 µmol/litre, and are adequate for most clinical purposes. If these devices are used, staff should be trained in this technique, and the machine should be calibrated daily and checked with control specimens of known bilirubin content. Using dirty tubes (or cuvettes), haemolysed or lipaemic samples can produce significant errors. Use plastic tubes, not glass ones, to avoid HIV infection if the tubes break.

The accurate measurement of values in excess of 350 µmol/litre is only possible in a biochemistry laboratory. A laboratory spectrophotometer reading is needed before initiating an exchange transfusion.

Direct/conjugated bilirubin presents no threat to the brain. It only accounts for a small fraction of the total serum bilirubin level in the first week of life. Decisions about treatment should therefore be based on the total serum bilirubin level, remembering that even laboratory estimates have limited precision.

Collecting blood

Only a small amount of blood is needed to check the bilirubin level. **It is essential not to enter the underlying bone with a needle, and can lead to osteomyelitis, so must**

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be avoided.

Try to use a disposable ideally automatic lance designed for neonates, but never use the same lance for more than one infant, because of the risk of transmitting hepatitis or HIV infection. It is not necessary or appropriate to try to sterilise the skin first, so long as it is cleaned with warm clean water and then dried. **Never penetrate the heel by more than 2mm.** The infant will also show fewer signs of distress if held or given something to suck during the procedure.

Grip the foot firmly enough to make it go red but not white. Stab the back of the foot just once and then squeeze gently and intermittently to stimulate blood flow. The use of a standard lance should optimise blood collection because it helps to ensure that the skin is punctured to a standard depth (never more than 2mm). A shallower prick is unlikely to reduce the pain inflicted because it will almost certainly prolong the procedure. A double puncture may help if a lot of blood is needed. Slight finger pressure exerted through a cotton ball on the site for about a minute is usually enough to stop any further bleeding after the procedure is over. The healthcare worker should be careful not to prick their own finger.
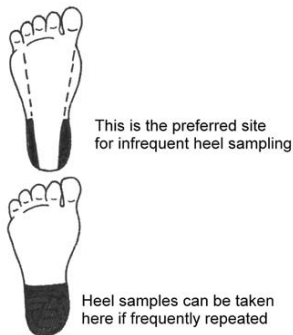

This is the preferred site for infrequent heel sampling

Heel samples can be taken here if frequently repeated

FIGURE 19.1 The safest positions in bold from which to obtain capillary blood from the foot of a neonate.

Biliary atresia

In prolonged jaundice (jaundice persisting beyond 14 days of age), it is important to determine not only the total bilirubin concentration but also the proportion of conjugated bilirubin. Conjugated bilirubin is not neurotoxic, but its presence signifies the presence of biliary obstruction attributable to potentially serious conditions such as neonatal hepatitis or biliary atresia.

Section 19 Neonatal illnesses: jaundice

The history can be informative if laboratory investigations are not available. The presence of pale un-pigmented stools or dark urine would be suggestive of biliary obstruction. Urine can also be tested with a reagent strip for bilirubin (if positive for bilirubin, the diagnosis of biliary obstruction is supported, provided that the infant is not receiving photo- therapy when unconjugated bilirubin appears in the urine). It is important to identify biliary atresia promptly, as operative intervention is more likely to be successful if undertaken within 8 weeks of birth. Even mild jaundice merits review if the stool becomes grey or putty coloured rather than yellow or green. Similarly, in the absence of a neonatal screening programme (a situation that is prevalent in the majority of resource-limited countries), it is important that congenital hypothyroidism and glucose-6-phosphate dehydrogenase (G6PD) deficiency are identified. This can be done by tests including T4, TSH, G6PD assay, bilirubin (total and direct), complete blood picture and reticulocyte count.

Breast milk jaundice

Around 10% of breastfed infants are still slightly jaundiced 1 month after birth. Laboratory investigations seldom reveal anything that needs treatment, and the infant is otherwise well. This scenario may be suggestive of breast milk jaundice. However, it is important that other common causes, including congenital biliary atresia, hypothyroidism and G6PD deficiency, are ruled out. Remember that breast milk jaundice is a diagnosis of exclusion.

Ill infants with continuing jaundice should be given a prophylactic 1 mg IM injection of vitamin K if it is not clear that they received such an injection at birth, to minimise the risk of potentially fatal late vitamin K deficiency bleeding.

Pathological jaundice

There is an increasing risk that high levels of unconjugated serum bilirubin will breach the blood–brain barrier, causing critical damage to many cells in the brain. This becomes more likely if, in the presence of haemolysis, the unconjugated serum bilirubin level is allowed to rise above 350 µmol/litre. Indeed, in a small preterm infant who is also ill, the safe limit may be nearer to 250 µmol/litre, or sometimes even less.

Once this happens there is nothing that can usefully be done to reverse the resultant brain damage. Infants may manifest this by seizures, or by becoming stiff with arching of the back and neck signifying a severe encephalopathy. Many of these infants will die after becoming severely ill. The survivors will almost all become severely deaf, and the majority may develop athetoid cerebral palsy.

Causes of abnormally raised bilirubin levels

These include the following:
- haemolytic disease
- neonatal sepsis
- polycythaemia
- severe malnutrition
- hypothyroidism

- congenital infection (usually obstructive jaundice):
    - syphilis
    - toxoplasmosis
    - cytomegalovirus
    - rubella
    - hepatitis.

In the first week of life, the following factors may lead to jaundice that is sufficiently severe to require treatment:

- Preterm delivery: Even moderate prematurity significantly increases the risk of early or severe jaundice and associated sequelae. Consequently, the bilirubin treatment charts give lower treatment thresholds for infants born at 31–34 weeks' gestation. At less than 31 weeks, treatment is started at even lower bilirubin levels.
- Haemolytic disease: This may be isoimmune (e.g. Rh or ABO incompatibility) or due to red blood cell disorders (e.g. hereditary spherocytosis, G6PD deficiency).
- Infection: Haemolysis and impaired elimination of bilirubin may be associated with septicaemia. Congenital infection (e.g. syphilis) may also be associated with jaundice, but other features such as rash, hepatosplenomegaly and thrombocytopenia will be present, and there is usually a significant conjugated bilirubin level.
- Polycythaemia.
- Rarer causes: These include inborn errors of metabolism (e.g. galactosaemia), congenital hypothyroidism, other intrauterine infections and neonatal malaria.
- Obstructive jaundice: This rarely presents in the first week of life, but is important in the differential diagnosis of prolonged jaundice.

Haemolysis

Clinically noticeable jaundice within 24 hours of birth, especially if the mother is blood group O and the infant is blood group A or group B, or the mother is Rhesus negative and the infant is Rhesus positive, should suggest the possibility of a haemolytic disease.

Term infants with physiological jaundice seldom need treatment with phototherapy unless there is an unusually high rate of red cell breakdown. However, phototherapy should be started as soon as jaundice becomes apparent if there is evidence of haemolytic disease. The trend in the bilirubin level should then be checked twice a day (the level cannot be assessed from skin colour once phototherapy has commenced).

Section 19 Neonatal illnesses: jaundice

Investigation

A good principle to remember is to measure bilirubin levels and investigate if:

- jaundice appears on day 1 in any infant
- jaundice appears on day 2 in any preterm infant
- the palms of the hands or soles of the feet are yellow in any sick neonate and in any infant of any age.

Jaundice should never last for more than 3 weeks.

In an infant who develops jaundice in the first 24 hours, the most likely causes are infections, haemolytic disease and polycythaemia. The history and examination may be helpful. It is important to determine whether the mother has previously had affected infants, or if she is known to have a hereditary haemolytic disorder, or if risk factors for infection or clinical signs of sepsis exist. Hepatosplenomegaly could be suggestive of congenital infection.

The following suggest a high risk for haemolysis:

- red cell antibodies in the mother's blood
- a positive Coombs' or direct antiglobulin test on blood samples from the umbilical cord
- a packed cell volume > 65%, Hb > 220 g/litre
- a family history of G6PD deficiency or congenital spherocytosis
- a history of previous children being seriously jaundiced in the first week of life
- otherwise unexplained neonatal anaemia at birth (haemoglobin level < 140 g/litre or haematocrit < 40%).

Useful laboratory tests include the following:

- the mother's and infant's ABO and Rhesus blood groups.
- Save serum to cross-match if exchange transfusion is needed
- direct Coombs' test (if positive this indicates an isoimmune haemolytic anaemia)
- complete blood count and reticulocyte count (anaemia and reticulocytosis indicate haemolysis, high PCV suggests polycythaemia and/or abnormal white blood cells indicate possible infection)
- peripheral blood smear (abnormal red cell morphology
- and/or fragmented red cell forms suggest a specific red cell disorder and/or haemolysis)
- G6PD screen
- syphilis serology
- thyroid function tests (T4, TSH)
- urine test for non-glucose-reducing substance (for possible galactosaemia)
- ultrasound scan of liver.

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Treatment

The bilirubin treatment charts (*see* Table 19.1) show intervention levels for the two principal treatments (i.e. phototherapy and exchange transfusion). In general, the smaller the infant and the sicker the infant, the more urgent the need to intervene. Bilirubin in plasma is normally bound to albumin, but in a sick acidotic infant less binding occurs, and more 'free' bilirubin will be available to enter the central nervous system. Therefore, consider intervening about 40 μmol/litre below the indicated line in such circumstances. The specific bilirubin levels for which phototherapy and exchange transfusions need to be considered in infants born before 31 weeks' gestation are less certain. A frequently used guideline is to initiate phototherapy when the bilirubin level approaches 85 μmol/litre/kg birth weight (which equals approximately 5 mg/dL/kg birth weight), and to consider an exchange transfusion for levels above 170 μmol/litre/kg birth weight (which equals approximately 10 mg/dL/kg birth weight).

TABLE 19.1 WHO recommendations (2012) for phototherapy and exchange transfusion levels of unconjugated bilirubin

	Phototherapy		Exchange transfusion	
Age	Healthy newborns ≥ 35 weeks' gestation	Newborns < 35 weeks' gestation or any risk factors	Healthy newborns ≥ 35 weeks' gestation	Newborns < 35 weeks' gestation or any risk factors
Day 1	Any visible jaundice		260 μmol /litre (15 mg/dL)	220 μmol /litre (10 mg/dL)
Day 2	260 μmol /litre (15 mg/dL)	170 μmol /litre (10 mg/dL)	425 μmol /litre (25 mg/dL)	260 μmol /litre (15 mg/dL)
Day 3+	310 μmol /litre (18 mg/dL)	250 μmol l/litre (15 mgd/L)	425 μmol /litre (25 mg/dL)	340 μmol //litre (20 mg/dL)

Phototherapy

This uses light in the blue-green region of the spectrum (not ultraviolet) to convert bilirubin to its water-soluble isomer lumirubin, which can be excreted in urine and stools.

In infants who are very yellow, it is best to use light from a bank of at least six 60-cm 20-watt fluorescent strip lights suspended not more than 30 cm above the unclothed infant (lights placed 60 cm from the infant are only about half as effective). Placing a white sheet under and round the infant will increase the effectiveness of any treatment. While under phototherapy, it is important to monitor body temperature and to protect the infant from draughts. It is also standard practice to mask the eyes to protect them from the bright light. The infant should be nursed naked in an incubator, under a radiant heater or in a cot, allowing maximum skin exposure. Feeding, especially breastfeeding, should continue

without interruption, as more frequent breastfeeding is helpful not only in eliminating meconium from the bowel but also in enhancing bilirubin clearance via the stools and urine. During phototherapy, the infant can be removed for breastfeeds as necessary (intermittent treatment has been shown to be as effective as continuous treatment). Fluid other than breast milk (e.g. breast milk substitute, water, sugar water) should **not** be given. However, the total daily fluid intake may need to be increased by about 10%, especially in preterm infants, in order to minimise additional water losses from evaporation and convection.

Side effects of phototherapy include rashes and profuse watery stools, but these are rare and do not require treatment. Phototherapy can be stopped when the serum bilirubin level is 50 µmol /litre (3 mg/dL) below the phototherapy threshold.

Exchange transfusion

Bilirubin levels that rise above certain threshold values place the infant at risk of developing bilirubin encephalopathy (kernicterus). In such cases, the bilirubin level needs to be immediately lowered with a double-volume exchange transfusion. A volume of the infant's blood equal to the body weight in kg × 2 × 80 mL is exchanged in small aliquots with O Rhesus-negative blood, or blood cross-matched against maternal antibodies.

Double-volume exchange transfusion is recommended in term infants if:

- they are haemolysing or are ill and have a bilirubin level higher than 300 µmol/litre
- they are well and not haemolysing but their bilirubin level is higher than 425 µmol/litre.

The functions of exchange transfusion include the following:

- removal of maternal antibodies
- removal of antibody-coated red blood cells before they haemolyse
- correction of anaemia
- lowering of total bilirubin levels, if there is sufficient time for equilibration between intravascular and extravascular levels.

Exchange transfusion is generally only undertaken if the rate of red blood cell breakdown is likely to exceed the ability of phototherapy to control bilirubin levels. This is very likely to occur in infants with a positive Coombs' test who are already anaemic (because of fetal haemolysis) at birth. A cord blood haemoglobin level of less than 140 grams/litre serves to identify most of these infants.

Human immunoglobulin 500 mg/kg, given as an IV infusion over 2 hours, reduces the number of infants who require an exchange (especially if due to Coombs' posi- tive Rhesus or ABO incompatibility). It also decreases the number who require a 'top-up' transfusion for late neonatal anaemia.

Section 19 Neonatal illnesses: jaundice

How to undertake an exchange transfusion

1   Calculate the infant's circulating volume (= 80 mL/kg). Twice this amount of blood will be required. Do not exceed this (usually 1 bag of whole blood = 450 mL). Do not use blood that is more than 4 days old.

2   Check that the blood has either the same ABO group as the infant or is blood group O Rhesus-negative and in addition is compatible with the mother's serum.

3   Ensure that the infant is closely monitored throughout the procedure.

4   This is a sterile procedure, so gloves and gowns must be worn and universal precautions applied.

5   Secure umbilical vein access. Pass the umbilical venous catheter (UVC) as described in Section 8.4.B, and check its position with an X-ray (if available). Ideally it should be positioned in the vena cava just outside the right atrium, but a position below the liver is also acceptable if the line will sample and flush easily. A line positioned in the liver should not be used.

6   Ideally, use a blood warmer (especially for low-birth- weight infants). Otherwise warm the blood bag by placing it under the mother's clothing next to the skin.

7   Set up a closed circuit with either a four-way tap, or two three-way taps. The four links are:

- the infant
- the syringe for removing and replacing blood
- the blood to be transfused
- the route for discarding the infant's blood.

8   Make sure that the total blood in and out is recorded. Plan to spend 1.5–2 hours on the procedure.

9   Decide on the size of aliquot you will be exchanging with each draw and infusion. This is roughly as follows:

- baby weighing < 1500 grams: 5 mL
- baby weighing 1500–2500 grams: 5–0mL
- baby weighing 2500 grams: 10–15 mL.

If you use small aliquots, remember to add an allowance for the 'dead space' in the tubing between the syringe and the baby. You should draw out each aliquot over 2–3 minutes to avoid abrupt changes in blood pres- sure, and replace over 3–4 minutes with the observer keeping a running total.

10   Send the first aliquot for measurement of bilirubin, electrolyte and calcium concentrations.

11   Halfway through the procedure check the blood glu- cose, calcium and potassium concentrations.

12   Measure them again, together with the bilirubin con- centration, at the end of the procedure.

13   Sometimes it is necessary to exchange more than once in quick succession. Symptomatic hypocalcaemia may occur as the citrate in donor blood binds calcium. This responds best to halting the procedure for 15 minutes. Giving calcium gluconate is of little benefit and may be hazardous, so is best avoided.

Although the potassium concentration of the blood is often 8–10 mmol/litre, this does not usually cause significant hyperkalaemia.

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Exchange transfusion should only be undertaken once all of the attendant risks have been considered. Even in experienced hands, 1% of infants may suffer a sudden cardiac arrest during or shortly after the procedure. This should respond to prompt intervention using the approach adopted when dealing with cardiac arrest at birth, but the infant needs to be monitored closely, and staff need to be prepared for such a possibility if this is not to prove fatal. Air embolism can kill within minutes, and faulty technique can cause sudden hypo- or hypervolaemia, or introduce later sepsis. The use of donor blood more than 5 days old can cause serious hyperkalaemia and an arrhythmia. Blood used straight from the fridge at 4°C can impose a major cold stress. Cytomegalovirus (CMV) infection may occur if the blood does not come from a CMV-negative donor. It is also critical to avoid causing HIV or hepatitis B or C infection. In addition, there is a definite but poorly understood risk that the procedure will trigger serious necrotising enterocolitis. If possible it is best to avoid the use of heparinised blood.

Because of all these risks, if at all possible exchange transfusion should ideally be undertaken in a neonatal unit where the staff are experienced in the use of this procedure.

The neonate with anaemia

Causes of anaemia

These include the following:

- haemorrhage
- twin-to-twin transfusion
- feto–maternal transfusion
- placental abruption
- haemolysis due to
- Rhesus incompatibility
- ABO incompatibility.

Treatment of anaemia

Haemolysis may continue for several weeks after birth even if it is not severe enough to require intervention in the first week of life. An attempt should therefore be made to check all infants with a positive Coombs' test for late anaemia when they are about 6 weeks old. Infants with a capillary haemoglobin level of less than 80 grams/litre or a haematocrit (PCV) of less than 25% should then receive a 'top-up' transfusion of 20 mL/kg of cross-matched or group O Rhesus-negative blood given over 2 hours. Red cell concentrate or packed cells are preferable. Daily folic acid (1 mg/day) for at least 1 week can help to reduce anaemia.

The neonate with seizures, spasms or reduced conscious level

Seizures (also called fits or convulsions) have been reported to affect about 0.1% of term infants and 10% of those weighing less than 1500 grams at birth.

Presenting features

Seizures may be subtle (apnoea, staring, lip smacking/ grimacing, deviation of the eyes, cycling movements of the limbs) or more obvious (tonic extensor posturing or clonic movements). Involvement of a limb or one side of the body does not necessarily imply a focal cause in the neonate. A bulging anterior fontanelle may suggest intracranial haemorrhage or infection. It is important to always measure and note the head circumference. Sometimes involuntary movements (e.g. extreme jitteriness) or benign myoclonic jerks can be difficult to distinguish from seizures. The presence of associated autonomic instability and/or lateral eye deviations may signal seizure activity, whereas the absence of these findings or elimination of these movements when the limbs are restrained indicate a non-seizure event.

TABLE 21.1 Differentiating between seizures and jitteriness

Well but jittery infant	Infant with clonic seizures
No abnormal eye movements	Abnormal eye movements
No apnoea	Apnoea
No colour changes	Pallor or cyanosis
No heart rate changes	Tachycardia
Easily triggered by handling and stopped by gentle passive flexion of the affected	Independent of handling
Rhythmical movements	Jerky with fast and slow components that are not equal

Causes of seizures

These include the following:

- hypoxia
- hypoglycaemia
- meningitis
- drug-related seizures
- sepsis
- polycythaemia
- tetanus

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- hypocalcaemia
- hyper- or hyponatraemia
- metabolic abnormalities

Hypoxic ischaemic encephalopathy is the most common cause of seizures in a term infant. Onset is usually within the first 24 hours, and it almost never starts after the third day

Intracranial haemorrhage, subarachnoid haemorrhage or cerebral infarctions: these are also common causes of neonatal seizures. With subarachnoid haemorrhage, seizures may or may not be focal. However, unilateral tonic–clonic seizures are often observed with cerebral infarction. Although intraventricular haemorrhage occurs most frequently in low-birth-weight infants or at gestational ages under 32 weeks, very rarely it may manifest in term or near-term infants with neonatal seizures. Always give 1 mg IV vitamin K

infection: although meningitis is not the commonest cause of neonatal convulsion, it must always be excluded by lumbar puncture and antibiotics commenced urgently pending the results of culture

Metabolic causes of seizures include:
- hypoglycaemia: always check blood glucose levels
- hypocalcaemia: check plasma calcium and magnesium levels
- hyponatraemia and hypernatraemia: seizures are uncommon unless the plasma sodium level is < 120 mmol/litre or > 160 mmol/litre. Seizures in infants with hypernatraemia may result from associated cavernous sinus thrombosis. A rapid fall or rise in serum sodium level, as may occur with too rapid therapeutic correction, may be more injurious than the absolute value of serum sodium level. A slow correction is desirable in such situations

Bilirubin encephalopathy (see above section on jaundice)

Maternal substance abuse, particularly opiate withdrawal

Tetanus remains a problem in many low-resource countries.

When managing neonatal seizures it is best to focus on the limited number of conditions where immediate treatment can have a major impact on long-term outcome. There are many situations where seizures are simply the outward sign of damage that cannot be reversed, even though it may be possible to stop continuing seizure activity from making matters worse.

Focal seizures can sometimes be the sign of what was otherwise a silent haemorrhagic infarction of part of the brain. While investigation would explain what was going on, it would not alter management.

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If the infant is alert and well between episodes of seizure activity, appears normal on examination, and is feeding normally, sometimes it may be perfectly appropriate to do nothing.

Investigations

These should include the following:

- lumbar puncture and blood culture
- full blood count, PCV, CRP
- blood glucose, calcium, urea and electrolytes, and blood ammonia (if available)
- arterial blood gas analysis to help further assess acid–base status
- cranial ultrasound (if available)
- intracranial imaging (head computed tomography if available)
- baseline and follow-up electroencephalograms (if available) may aid diagnosis and treatment
- save urine, plasma and CSF for metabolic studies (if available) if seizures are protracted.

Treatment

Management of a neonate with seizures is as follows:

- Airway management.
- Breathing management.
- Circulatory access.
- Give glucose, IV or intra-osseous (2 mL/kg of 10% glucose).
- Give antibiotics, IV or IM, as there is a strong possibility of meningitis or sepsis.
- Stop the seizures with an anticonvulsant:
  - phenobarbitone, 20 mg/kg over 5 minutes IV or IM
  - paraldehyde, 0.2 mL/kg IM or 0.4 mL/kg rectally
  - diazepam 300 microgram/kg IV slowly or 500 micro- gram/kg rectally.
- Treat hypoglycaemia if present.
- Monitor the heart rate and respiratory rate, and oxygenation (ideally with pulse oximetry). Treat low SaO_2 or cyanosis with oxygen.
- Consider anticonvulsant therapy: the earlier the fits appear, the more frequent they are (more than two or three per hour) and the longer they last (more than 3 minutes), the more likely it is that anticonvulsants will be needed. Fits that interfere with respiration need to be treated and may require respiratory support.

Emergency treatment of hypoglycaemia

Hypoglycaemia is a common cause of seizures in infants. Ideally, do a rapid bedside test for low blood glucose levels and act accordingly, but if this test is not available then a test dose of 2 mL/kg of 10% glucose should be given IV or, if venous access is not available, intra-osseously.

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Anticonvulsant treatment

Phenobarbital is the first-line drug for neonatal seizures. Give a 20 mg/kg IV loading dose slowly followed by 3–5 mg/ kg once every 24 hours. Seizure control may be achieved more quickly if the first dose is given IV, but this loading dose must be given slowly, over at least 5 minutes, to minimise the risk of shock, hypotension or laryngospasm. Some texts recommend the use of a higher dose if the standard dose fails, but this can cause respiratory depression. Always have a bag-valve-mask available to support ventilation. With hypoxic encephalopathy usually only a loading dose is needed. Seizures have been reported to respond to this dose 40% of the time. An additional 10mg/kg may be required if seizures persist or recur (70% response rate).

Phenytoin is the second-line drug for neonatal seizures. Initial seizure control with this drug requires the presence of a saline-filled IV line (because the drug crystallises out in dextrose solutions). The same problem also renders the IM route unavailable. Give a 20 mg/kg loading dose (diluted in 10–15mL of normal saline) IV slowly over 10–20 minutes (monitor for hypotension and cardiac arrhythmia, making sure that the drug does not leak into the tissues), and then 2mg/kg IV or by mouth once every 8 hours. Infants more than 2 to 3 weeks old may need a considerably larger maintenance dose. Oral absorption of phenytoin may be quite unpredictable, so this would need to be monitored. Phenytoin is dangerous in infants with hypoxic ischaemic encephalopathy who may also have ischaemic hypoxic heart injury.

Paraldehyde is the third-line drug for neonatal seizures. Give a single 0.4 mL/kg dose mixed with an equal volume of mineral oil by the rectal route. This route offers excellent bioavailability of the drug. The same dose can be repeated once if seizures persist. Give within 10 minutes after preparation when using a plastic syringe (because paraldehyde interacts with many plastics). Paraldehyde can also be given by the IM route. However, problems with muscle necrosis make this a less desirable route.

Diazepam is an alternative to phenobarbital as first line treatment for neonatal seizures. However, it is vital that hypoglycaemia has been excluded and treated before giving this drug. A working bag-valve-mask of suitable size must be ready next to the infant before this drug is given. It can be given IV at a dose of 300 microgram/kg over 5 minutes or rectally in a dose of 500 microgram/kg. The rectal dose can be repeated once after 10 minutes if the infant is still fitting.

Clonazepam: The loading dose is given as 100 micrograms/kg by slow IV infusion. It should not be administered for more than 3 days.

Midazolam: This has an immediate effect but a short duration of action. It can be given into the buccal cavity or IV. Like diazepam it can cause respiratory depression, so a

bag and mask must be available when it is used, and the infant must be monitored closely. **Anticonvulsants may precipitate a need for respiratory support. Therefore, always have a bag-valve-mask available.**

Once seizures are controlled, maintenance therapy (which is rarely needed) with a single agent (usually phenobarbitone) is often possible. Discontinuation of treatment depends on the underlying aetiology, but aim to withdraw anticonvulsants as soon as possible.

It is essential to consider the four main treatable causes of fitting, namely hypoglycaemia, hypocalcaemia, meningitis and tetanus, as any delay in diagnosis could have serious consequences.

Table 21.2 Anticonvulsant details for the neonate and infant

Drug	Route	Single dose	Frequency	Postnatal age	Gestation
Paraldehyde	Rectal	0.2 to 4 mL/kg	(***loading dose***)		
		Can repeat once 4–5 hours later			
	Use injection rectally or ready-diluted rectal solution Dilution with an equal volume of olive oil or any edible oil If using a plastic syringe administer immediately IM injections may cause sterile abscessed (maximum 1 mL at one site)				
Phenobarbitone	IV, IM, PO	20 mg/kg	(***loading dose***)	*followed by maintenance 12–24 hours later*	
	Slow IV over 5 minutes. Loading dose may be repeated by at 10 mg/kg if clinically indicated				
		3–5 mg/kg/24 hours	Once daily	*Once daily generally but with time may need to be give 12 hourly*	
	Monitor plasma levels				
	Therapeutic range: 15–30 mg/L although increasing up to 40 mg/L should be considered in resistant seizures				
Phenytoin	IV, PO	15–20 mg/kg	(***loading dose***)		
	Give IV infusion over 20–30 minutes diluted in 10 mL of normal saline				
		1.5–3 mg/kg	12 hourly		
	Slow IV over 20–30 minutes *Usual maximum dose 7.5 mg/kg 12 hourly Therapeutic range 5–17 mg/L Oral dose is poorly absorbed particularly in premature infants* *Wide variation in levels, so monitor and adjust dose and interval accordingly* *Measure trough level. May not reach steady state for up to 14 days*				

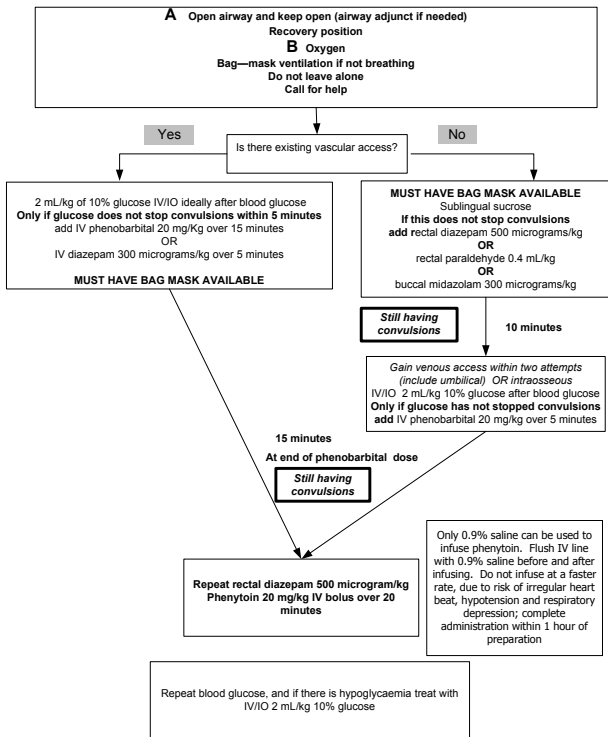# Section 21 Neonatal illnesses: fits, reduced consciousness

A **Open airway and keep open (airway adjunct if needed)**
Recovery position
B Oxygen
Bag—mask ventilation if not breathing
Do not leave alone
Call for help

Is there existing vascular access?

Yes   No

2 mL/kg of 10% glucose IV/IO ideally after blood glucose
Only if glucose does not stop convulsions within 5 minutes
add IV phenobarbital 20 mg/Kg over 15 minutes
OR
IV diazepam 300 micrograms/kg over 5 minutes

MUST HAVE BAG MASK AVAILABLE

MUST HAVE BAG MASK AVAILABLE
Sublingual sucrose
If this does not stop convulsions
add rectal diazepam 500 micrograms/kg
OR
rectal paraldehyde 0.4 mL/kg
OR
buccal midazolam 300 micrograms/kg

Still having convulsions   10 minutes

*Gain venous access within two attempts
(include umbilical) OR intraosseous*
IV/IO 2 mL/kg 10% glucose after blood glucose
Only if glucose has not stopped convulsions
add IV phenobarbital 20 mg/kg over 5 minutes

15 minutes

At end of phenobarbital dose

Still having convulsions

Only 0.9% saline can be used to infuse phenytoin. Flush IV line with 0.9% saline before and after infusing. Do not infuse at a faster rate, due to risk of irregular heart beat, hypotension and respiratory depression; complete administration within 1 hour of preparation

Repeat rectal diazepam 500 microgram/kg
Phenytoin 20 mg/kg IV bolus over 20 minutes

Repeat blood glucose, and if there is hypoglycaemia treat with IV/IO 2 mL/kg 10% glucose

Figure 21.1 Pathway of care for the neonate who is convulsing

Section 22 Hypoglycaemia

Hypoglycaemia (glucose < 2.5 mmol/litre or < 45 mg/dL)

Hypoglycaemia is a common problem in the nursery; it can occur in infants who appear well, as well as in those who are sick. It is important to identify any infant at risk and implement preventative and curative measures as early as possible. Untreated symptomatic hypoglycaemia can result in brain damage.

Infants at risk of developing hypoglycaemia include the following:
- infant of diabetic mother
- preterm infant
- small-for-gestational-age or wasted infant
- large-for-gestational-age infant
- post-term infant
- sick infant with infections and respiratory failure
- infant who is not receiving adequate breast milk.

The definition of hypoglycaemia is controversial, and no studies have determined an absolute value at which organ dysfunction will occur. However, it is known that a prolonged low level of symptomatic hypoglycaemia is associated with brain injury. At the time of writing, most neonatologists prefer to maintain blood glucose levels above 2.5 mmol/ litre (45 mg/dL).

Causes of neonatal hypoglycaemia

Increased utilisation of glucose (hyperinsulinism)
- Infants of diabetic mothers.
- Respiratory distress.
- Abrupt interruption of high glucose infusion.
- Polycythaemia.
- Hypothermia.

Rare causes of hyperinsulinism include:

- erythroblastosis fetalis
- islet-cell hyperplasia
- Beckwith–Wiedemann syndrome
- insulin-producing tumours
- maternal beta-agonist tocolytic therapy
- rarely malpositioned umbilical arterial catheter infusing a high concentration of glucose into coeliac and mes- enteric arteries (T11–T12), stimulating insulin release.

Decreased production/stores of carbohydrate
- Prematurity.

Section 22 Hypoglycaemia

- Small-for-gestational-age or wasted infant.
- Inadequate caloric intake.

Mixed increased utilisation and/or decreased production from other causes
- Perinatal stress (e.g. due to hypoxia, sepsis, shock, hypothermia).

Rare causes
- Defects in carbohydrate metabolism (galactosaemia, fructose intolerance, glycogen storage disease).
- Endocrine deficiency (adrenal insufficiency, hypotha- lamic insufficiency, glucagon deficiency).
- Defects in amino acid metabolism (maple syrup urine disease, propionic acidaemia, methylmalonic acidaemia, tyrosinaemia).

Diagnosis of hypoglycaemia

There are few data on normal blood glucose levels in the first week of life, particularly for healthy breastfed term infants. Moreover, there is little evidence that a transient low blood glucose concentration in term infants who show no physical signs is harmful. However, asymptomatic hypoglycaemia may rapidly progress to symptomatic hypoglycaemia. Fits due to hypoglycaemia typically start in a previously well infant on or after the second day of life.

Indications for measuring the blood glucose concentration of a term infant include lethargy, poor feeding, temperature instability, respiratory distress, new-onset apnoea/bradycardia, jitteriness, pronounced hypotonia, diminished consciousness and seizures. The association between such signs and low blood glucose levels is described as 'symptomatic hypoglycaemia'.

Beware of blaming 'hypoglycaemia' alone for these signs. Remember that an infant who seems drowsy may be infected, and low blood glucose concentration may merely be an associated finding. It is important to try to establish the underlying cause of the problem.

- Although laboratory estimates of blood glucose concentration are ideal for diagnosing and managing this condition, reagent strips can be helpful.
- The blood glucose concentration in the first 6 hours of life is very often low (1.5–2.0 mmol/litre). There is no evidence that this is harmful for otherwise healthy term infants, who adapt by mobilising other fuels. Consequently, early testing (under 6 hours of age) is pointless, unless neurological signs are pre- sent or there are other conditions that necessitate testing.
- In newborn infants the serum glucose concentration is about 0.5 mmol/litre lower than that of whole blood.

Section 22 Hypoglycaemia

When to test
- Symptomatic infants (lethargy, poor feeding, temperature instability, respiratory distress, new-onset apnoea/   bradycardia, jitteriness and seizures) should be tested immediately.
- Infants at risk should be tested within 1 hour after birth (as such infants rapidly become hypoglycaemic after delivery) and then 3-hourly until blood glucose levels are stable at 2.5 mmol/litre (45 mg/dL) or higher. Continue to monitor until feeds are well established.
- In infants with hypoglycaemia, check the blood glucose concentration every 20–30 minutes from the beginning of treatment, then hourly until it is stable at 2.5 mmol/ litre (45 mg/dL) or higher. Continue to monitor frequently (every 4–8 hours) during treatment, and while decreasing supplemental IV glucose infusions.

Laboratory diagnosis
- Reagent strips are useful and rapid, but in general are less reliable than laboratory plasma glucose measurements. Reagent strips may show a glucose level as much as 15% lower than plasma glucose levels. Whenever possible, it is preferable to use a calibrated glucometer.
- Laboratory plasma glucose determinations (if available) are useful for confirming hypoglycaemia detected by reagent strips, but blood samples must be processed promptly for accurate values, as glycolysis occurs in   standing whole blood samples. Do not wait for labora- tory confirmation before initiating therapy.

Management of hypoglycaemia
Infants at risk of hypoglycaemia but appearing to be well
1. Initiate early feeding within 1 hour after birth with breast milk or formula (only if breast milk is not available) and repeat every 2–3 hours.
2. Feeding with 5% glucose is not recommended in infants, because milk provides more energy.
3. Infants of diabetic mothers are unlikely to develop hypo- glycaemia on the second day of life if tests in the first 24 hours are satisfactory.

Infants with symptomatic hypoglycaemia who are unable to feed or who failed correction of glucose levels with enteral feeding
1. Establish an IV line using sterile precautions, and take a sample for blood culture and other biochemical tests (if available).
2. Give an IV glucose bolus 200 mg/kg over 5 minutes (2 mL/kg of 10% glucose in water). If the infant almost immediately becomes more alert and active 'on the end of your needle' you have made the diagnosis, even before the laboratory report comes back.
3. In such situations it is then important to keep the blood glucose level stable by starting a sustained   infusion of 10% dextrose at 5 mL/kg/hour (or 5–8 mg/

kg/minute) for the next 2 to 4 days while gradually building up oral feeds.

4. If further episodes of symptomatic hypoglycaemia occur, the bolus should be repeated and the infusion rate increased by 10–15%.

5. An infant who seems drowsy may be infected, and a low blood glucose concentration may be an associated finding rather than the main cause of the problem. It is important to exclude infection and initiate antibiotics if indicated.

6. When administering boluses, never use higher concentrations of glucose (> 10%) because of the risk of intra-ventricular haemorrhage and/or cerebral oedema.

7. The concentration of glucose in the maintenance fluids can be adjusted in accordance with the total daily fluid requirements.

8. If using concentrations higher than 12.5%, a central venous line or umbilical venous catheter needs to be inserted because of the risk of tissue damage in the event of fluid extravasation.

9. Most infants will correct hypoglycaemia with infusion of 5–8 mg/kg/minute; not infrequently, however, infants with severe intrauterine growth restriction or wasting and those with hyperinsulinism may require infusion rates of up to 12–15 mg/kg/minute.

10. When normal blood glucose levels have been stable for 12–24 hours and the infant is tolerating enteral feeding, decrease the IV glucose infusion by 10–20% each time levels are higher than 2.5 mmol/litre (45 mg/dL).

11. Always decrease the IV infusion gradually because of the risk of precipitating hypoglycaemia.

12. If you are unable to gain IV access, a feed of breast milk should be started if the infant is conscious. Hypostop Gel, an oral glucose mixture containing 500 micrograms of glucose/mL, can be helpful (if available). Apply 1–2mL to the oral mucosa.

13. If hypoglycaemia persists beyond the first 48 hours of life and requires large infusions of glucose (greater than 8 mg/kg/minute), evaluation for endocrine or metabolic disorders should be considered.

Tetanus

Do not forget tetanus. Neonatal tetanus has to be considered if a previously well and still conscious infant starts to develop increasingly frequent muscle spasms 3–14 days after birth. This becomes more relevant if there is any doubt about the way the umbilical cord was managed at birth, or if there is no proof that the mother was ever immunised with tetanus toxoid vaccine. Involuntary muscle contractions are typically triggered by quite light touch or sound, and the hands and jaw are often held firmly clenched.

Treatment of tetanus

1. Airway and breathing are frequently compromised. Secure and maintain the airway, and ensure adequacy of ventilation. Oxygen may help if the spasms are causing cyanosis, but in severe cases survival may be dependent on the availability of respiratory support, sometimes with tracheostomy to protect the airway. Intubation may trigger very dangerous spasm of the airway and must be undertaken by a skilled professional.

2. Insert an IV line for drug and antibiotic administration.

3. Give high-dose benzyl penicillin 60 mg/kg IV one dose every 12 hours in the first week of life, every 8 hours in an infant aged 1–3 weeks, and every 6 hours in an infant over 3 weeks of age. Oral dosing (with phenoxymethylpenicillin) can sometimes be used to complete a course of treatment.

4. Give a 150 unit/kg dose of IM human tetanus immunoglobulin. Other IM injections must be avoided, as they will provoke spasms.

5. If the infant is in acute spasm, this should be terminated by giving diazepam by bolus IV infusion over 15 minutes (dose 200 micrograms/kg) or rectally (400 micrograms/kg). Ensure that for IV infusion, diazepam is diluted to 100 micrograms/mL, and that extravasation (very irritant) does not occur. Slow and incomplete absorption means that IM diazepam is not effective. Always ensure that a bag and mask are immediately available when giving diazepam, in case apnoea occurs.

6. Also give an IV loading dose of 25–40 mg/kg of magnesium sulphate over 20–30 minutes.

7. Subsequently give 10–20 mg/kg of magnesium sulphate IV 2- to 4-hourly to control spasms. If this is not available or does not control spasms, give IV diazepam 200 micrograms/kg every 4–6 hours.

8. Stop diazepam if magnesium sulphate alone controls the spasms.

9. Reduce the dose of diazepam if apnoeic episodes occur.

10. Always have a bag and mask available in case the patient stops breathing as a result of the diazepam plus magnesium.

11. When stable, a nasogastric tube, ideally passed by an anaesthetist, will allow fluids, food and drugs to be given with minimal disturbance. Feeds need to be given frequently (ideally hourly) and in small amounts due to reduced gut motility.

Regular breast milk feeds via a nasogastric tube are essential.

12. Excision of the umbilical stump is not indicated.
13. The disease itself does not induce immunity, so after recovery tetanus vaccine must be given for future prevention.
14. Treat any obvious umbilical infection with an additional broad-spectrum antibiotic.
15. Minimise handling, provide care in a quiet dark room and give frequent small tube feeds of breast milk..
16. Immunising the mother (give two 0.5-mL doses 1 month apart) will prevent a similar tragedy in any future pregnancy.
17. Severe cases may need muscle paralysis and ventilation in a specialist unit (if available).

Section 24 Neonatal fits and hypoxic ischaemic encephalopathy

Other causes of fits and hypoxic ischaemic encephalopathy

Rule out any other cause, including biochemical causes other than hypoglycaemia.

Remember that biochemical disturbance may not be the main underlying problem. In many infants, the evidence of hypoglycaemia or any other biochemical disturbance is only a sign of another more serious underlying illness. Of these, by far the most important treatable condition is meningitis. Unless the infant is otherwise entirely well it is important not to miss this possibility.

Other important diagnostic possibilities include hypocalcaemia, hyponatraemia and hypernatraemia. Often a history and clinical features will aid the recognition of these biochemical abnormalities, and a serum level will clinch the diagnosis. Any existing problem will be made worse if hypernatraemia is corrected too rapidly.

Fits due to hypocalcaemia (a serum total calcium level of < 1.7 mmol/litre), with or without hypomagnesaemia, are generally benign and occur unexpectedly in an otherwise well but hyper-reflexic infant more than 2 to 3 days old. As with hypoglycaemia, signs may settle 'on the end of the needle' if the infant is given 1–2 mL/kg of 10% calcium gluconate in equal dilution as a slow IV infusion. Such seizures usually respond extremely well to oral supplementation. It is appropriate to investigate the mother for an unrecognised endocrine abnormality (if facilities allow this). Do not allow IV calcium to go outside the vein as it will cause severe tissue damage.

Toxic substances provided by a traditional healer create important causes of seizures and reduced conscious level in neonates in some countries.

Bilirubin encephalopathy
Infants with brain damage due to jaundice are stiff and semi-conscious, but seldom have fits. Signs usually appear quite abruptly 3 to 6 days after birth, but by the time they appear it is too late to initiate treatment.

Inborn errors of metabolism
Other more complex biochemical disturbances are usually associated with metabolic acidosis and progressively deepening coma in an infant who was initially well for 1 to 2 days after birth. They are generally too complex to treat without substantial biochemical support, but it may be appropriate to take specimens for later diagnostic evaluation, because many of these conditions are familial and genetically determined. Pyridoxine deficiency is one of the few rare treatable conditions.

Section 24 Neonatal fits and hypoxic ischaemic encephalopathy

Meningitis
>   *See* Section 11.

Hypoxic-ischaemic encephalopathy

This is an abnormal neurological state of infants who have suffered significant lack of oxygen and/or circulation to vital organs before, during or immediately after birth. It is characterised by the following:

- signs of fetal distress in labour, cord blood pH < 7.0, and low Apgar score (3 at 5 minutes) despite appropriate resuscitation measures
- neonatal neurological abnormalities soon after delivery
- evidence of multi-organ dysfunction such as oliguria (signifying acute tubular necrosis), increased transaminase levels (hepatic necrosis), necrotising enterocolitis or myocardial dysfunction.

Hypoxic-ischaemic encephalopathy-related problems in the days after birth

- Reduced consciousness and/or convulsions: treat with phenobarbital and check glucose levels to rule out hypoglycaemia.
- Apnoea: this is common after severe perinatal asphyxia, and is sometimes associated with convulsions. Manage with oxygen administered by nasal cannulae and resuscitation with bag and mask.
- Inability to suck: feed with expressed breast milk via a nasogastric tube. Beware of delayed emptying of the stomach, which may lead to regurgitation of feeds.
- Poor motor tone: the infant may be floppy or have limb stiffening (spasticity).

Sarnat's clinical grading system (*see* Table 24.1) may be used to help to guide treatment and give some indication of the prognosis.

Treatment of hypoxic ischaemic encephalopathy

1. Treatment is generally supportive, with close attention to monitoring of good respiratory function, glucose levels and fluid balance.
2. Avoid hyponatraemia, which may result from inappropriate antidiuretic hormone secretion and excessive IV hypotonic solutions.
3. Acute renal failure is often present; if so, restrict fluids to measured urine output and gut losses plus 15 mL/kg/24 hours for full term and 24 mL/kg/24 hours for preterm infants (to reflect insensible losses), and avoid giving potassium supplements.
4. Seizures are treated as described above. Note, however, that increasing doses of anticonvulsants may precipitate a need for mechanical ventilation and confound the clinical staging criteria below, which apply only to non- sedated infants.
5. Keep the axillary temperature at 35.5–36.0°C. Avoid overheating.

Section 24 Neonatal fits and hypoxic ischaemic encephalopathy

Cooling infants for 72 hours under carefully controlled conditions has shown improved neurological outcomes. This should only be undertaken by experienced teams.

Prognosis

The prognosis is good in stage 1, guarded in stage 2 and very poor in stage 3.
About 50% of stage 2 infants will recover without sequelae. Infants in stage 3 will either die or be left severely disabled. A decision must therefore be made with the family about the implementation or continuation of intensive care in such cases.

TABLE 24.1 Sarnat's grading of hypoxemic-ischaemic encephalopathy

	Mild (stage 1)	Moderate (stage 2)	Severe (stage 3)
Conscious level	Hyper-alert	Lethargic	Stuporose
Muscle tone	Normal	Hypotonic	Flaccid
Seizures	Rare	Common	Severe
Feeding	Sucks weakly	Needs tube feeds	Needs tube feeds or IV fluids
Respiration	Spontaneous	Spontaneous	Absent

Management

Once bacterial meningitis has been excluded, intrapartum hypoxia or birth trauma will turn out to be the underlying problem in most infants presenting with fits in the first 2 to 3 days of life. Most of these infants already look stressed and unwell within a few hours of birth. The onset may be a little more sudden and abrupt in the preterm infant who suffers a sudden intra-ventricular haemorrhage. These infants usually become progressively more stuporose and unresponsive over time, and there is relatively little that can be done to improve the long-term outlook. An attempt should be made to minimise hypoxia, and anticonvulsant treatment is sometimes initiated in the hope that it will reduce the number of apnoeic episodes. Many of these infants are too ill to accept even tube feeds, and, where this is the case, it may be appropriate to minimise the risk of hypoglycaemia by giving IV glucose. Unfortunately, an infusion of more than 3 mL/kg of 10% dextrose per hour may result in water retention if there is accompanying renal failure. The outlook is fairly bleak for infants who have not recovered and started to feed normally within 1 week of birth.

Other less common causes of neonatal seizures/fits
Drug-related seizures:

Accidental infiltration of the fetal scalp during the injection of lidocaine into the maternal perineum prior to episiotomy can cause fits simulating intrapartum hypoxia. With supportive treatment there is every prospect of complete recovery.

Section 24 Neonatal fits and hypoxic ischaemic encephalopathy

Some infants born to drug-dependent mothers show signs of drug withdrawal, starting 1 to 2 days after delivery. A small minority may have seizures. Minimal handling in a quiet dark room with small frequent feeds and a more gradual withdrawal from the drug to which they have been exposed is the only treatment usually necessary.

Congenital brain abnormalities: It is said that up to 10% of otherwise unexplained neonatal seizures are associated with the existence of some underlying cerebral problem (often cortical dysgenesis). Some of these infants will benefit from continuing anticonvulsant treatment.

Congenital heart disorders

Congenital heart disease occurs in 5–8 in 1000 live births.

Cardiac defects may present as any of the following:

- cyanosis in the newborn period
- cyanosis in the older infant
- cardiovascular collapse in the newborn period
- cardiac failure in infancy
- an asymptomatic murmur.

This section explains how to recognise the presence of congenital heart disease in each of these clinical scenarios, and provides enough information to make a working diagnosis. Management decisions can then be made when modern imaging techniques are not immediately available.

The cyanotic newborn infant

Is there a cardiac problem?

When an infant is referred as a 'blue baby', first check to see whether there is genuine central cyanosis. Examine the lips and tongue for blue discoloration, and confirm the clinical impression by measuring the oxygen saturation (less than 94% is abnormal). If there is central cyanosis this may have a cardiac or respiratory cause.

TABLE 25.1 Features that distinguish cardiac from respiratory cyanosis in the neonate

Cardiac cyanosis	Respiratory cyanosis*
Mild tachypnoea but no respiratory distress	Respiratory distress
May have cardiac signs on examination	Chest X-ray: abnormal lung fields
Arterial blood gas: paO_2 decreases, $paCO_2$ decreases or normal	Arterial blood gas: paO_2 decreases, $paCO_2$ increases or normal
Fails hyperoxia test	Passes hyperoxia test

*A respiratory cause for cyanosis is more likely in infants born preterm.

The *hyperoxia test* is performed as follows:

1 Ensure that there is good IV access.
2 Monitor oxygen saturations continuously.
3 Give 100% oxygen for 10 minutes.
4 Take an arterial blood gas sample in the right arm (preductal).
   - If pO_2 is lower than 20 kPa (150 mmHg), a cardiac cause of cyanosis is likely (the test is 'failed').
   - If pO_2 is higher than 20 kPa, a respiratory cause of cyanosis is likely (the test

is 'passed').

- Although pulse oximetry cannot reliably be used in place of an arterial blood gas, a resting saturation of less than 80% and a saturation of less than 90% after 10 minutes in 100% oxygen suggests cyanotic heart disease requiring early intervention.
- Note: Oxygen administration could cause closure of the arterial duct, precipitating profound hypoxaemia in some types of cyanotic congenital heart disease.
- Prostaglandin E (which opens the duct) should there- fore be available at the time of the test and should be given if oxygenation deteriorates.

Persistent pulmonary hypertension of the newborn (PPHN) may often mimic cyanotic heart disease using these clinical criteria. However, PPHN is usually distinguished by a history of fetal distress, resuscitation is often needed at birth, and there may be neurological signs. Improvements in oxygenation may be possible after intubation and ventilation, and saturations in the right arm may be significantly higher than those in the feet, suggesting right-to-left shunting across the arterial duct.

What type of cardiac defect is present?

Cyanotic cardiac defects can be divided into three broad categories, as described below.

Once cyanotic congenital heart disease is suspected, attempt to place the defect in one of the three categories. This may be done using Table 25.2, which describes the typical findings in each physiological category. These guidelines assist the clinician but are not infallible, and the nature of the defect is sometimes only clear after echocardiography.

TABLE 25.2 Features that help to distinguish the three types of cyanotic congenital heart defect

	Low pulmonary blood flow	Complete TGA	Common mixing lesion
PaO$_2$ at rest	Often ≤ 35 mmHg	Often ≤ 35 mmHg	Often ≥ 45 mmHg
SaO$_2$ at rest	< 80%	< 80%	80–90%
Pa O$_2$ hyperoxia test	Often ≤ 50 mmHg	Often ≤ 50 mmHg	75–200 mmHg
SaO$_2$ hyperoxia	< 90%	< 90%	90–100%
Chest X-ray	Reduced pulmonary vascular markings	Normal or increased pulmonary vascular markings with or without narrow mediastinum	Normal or increased pulmonary vascular markings

Cyanotic congenital heart diseases

Low pulmonary blood flow

In defects where there is low pulmonary blood flow the physiology is the same regardless of the precise anatomy of the defect. Deoxygenated blood returning from the systemic veins cannot flow through the right side of the heart to the lungs. The pulmonary blood supply is therefore via the arterial duct. The deoxygenated blood from the right side of the heart shunts to the left side of the heart (via either an atrial or a ventricular septal defect), and the left ventricle receives both deoxygenated blood from the right heart and oxygenated blood from the pulmonary venous return. Blood entering the aorta is therefore not fully oxygenated, and the infant appears cyanosed. If the duct closes, the infant becomes profoundly cyanosed and is unlikely to survive unless pulmonary blood flow is rapidly restored. This is duct-dependent pulmonary circulation, an example of which is shown in Figure 25.1.
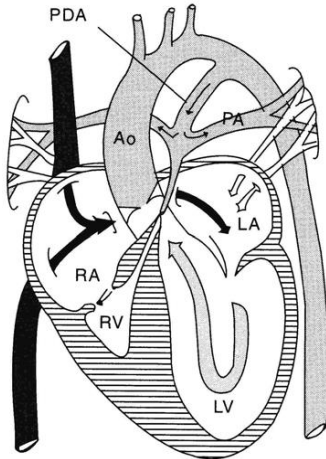
FIGURE 25.1 The circulation in pulmonary atresia with intact ventricular septum. PDA, patent ductus arteriosus; Ao, aorta; PA, pulmonary artery; LA, left atrium; LV, left ventricle; RV, right ventricle; RA, right atrium.

Section 25 Congenital heart disorders

Complete transposition of the great arteries (TGA)

Here the aorta arises from the right ventricle and the pulmonary artery arises from the left ventricle (*see* Figure 25.2). Systemic venous return enters the right side of the heart and is recirculated to the systemic arteries. Pulmonary venous return enters the left side of the heart and is recirculated to the lungs. Oxygenated blood and deoxygenated blood are therefore separated in two parallel circuits. Oxygenated blood enters the systemic circulation only when there is mixing between the two circuits. Mixing occurs at atrial level (across the foramen ovale) and at ductal level (while the duct remains open). Systemic oxygen saturation reflects the amount of mixing (which in turn depends on the size of these communications). If the atrial communication is small, oxygenation may therefore be duct dependent.
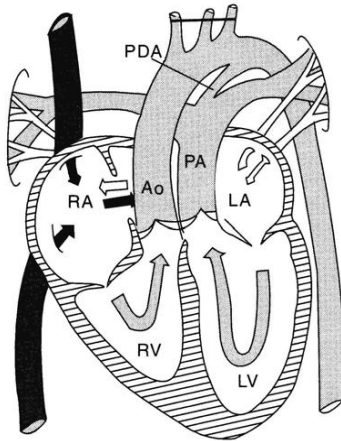
FIGURE 25..2. Transposition of the great arteries. For explanation of abbreviations, see legend to Figure 25.1.

Common mixing lesions

In common mixing lesions, oxygenated pulmonary venous blood and deoxygenated systemic venous blood mix in one of the cardiac chambers. An example is shown in Figure 25.3 The systemic output is therefore only partly oxygenated. The relative amounts of pulmonary and systemic blood in the mixture determine the oxygen saturation

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and the mode of presentation. If pulmonary blood flow is high, cyanosis is minimal, and the infant usually presents at about 2 months of age in heart failure. If pulmonary blood flow is low (the complex lesion may coexist with pulmonary stenosis), cyanosis is severe and is often detected early.
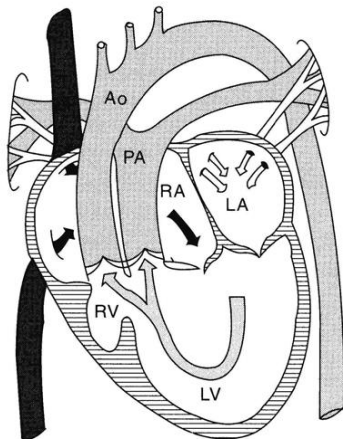
FIGURE 25.3 The circulation in double-outlet left ventricle. For explanation of abbreviations, see legend to Figure 25.1.

Once the defect has been placed in one of these categories, immediate management decisions can be made. Although it is not imperative to reach a more specific diagnosis, an anatomical diagnosis can sometimes be made using clinical information and simple investigations.

TABLE 25.3 Conditions with low pulmonary blood flow

Critical pulmonary stenosis
Pulmonary atresia with intact ventricular septum
Tetralogy of Fallot (with severe right ventricular outflow tract obstruction)
Pulmonary atresia with ventricular septal defect
Absent right atrioventricular connection

Section 25 Congenital heart disorders

Pulmonary atresia with intact ventricular septum and critical pulmonary stenosis

These two conditions are similar pathologies with either complete or almost complete closure of the pulmonary valve. Both are often associated with hypoplasia of the right ventricle. There is either no murmur or a soft murmur at the lower left sternal border (tricuspid regurgitation). The chest X-ray usually shows a normal heart size. The precordial leads on the ECG usually show decreased right ventricular voltages (small R waves in leads V1 and V2) and dominant left ventricular voltages (prominent S waves in leads V1 and V2 and prominent R waves in leads V5 and V6).

Tetralogy of Fallot and pulmonary atresia with ventricular septal defect

These two pathologies are also similar, but in Fallot's tetralogy the right ventricular outflow tract is patent, albeit narrow, generating a high-pitched ejection systolic murmur at the upper left sternal border. In both defects, the cardiac silhouette on the chest X-ray has a concavity on the left heart border where there is usually a convexity produced by the right ventricular outflow tract and pulmonary artery. The ECG shows dominant right ventricular voltages (normal neonatal RS progression).

Absent right atrioventricular connection (also known as tricuspid atresia)

There is often a long harsh systolic murmur (this may arise from a restrictive ventricular septal defect or pulmonary stenosis). The precordial leads on the ECG show decreased

right ventricular voltages and dominant left ventricular volt- ages. The QRS axis is characteristically directed to the left and superiorly between 0 and –90 degrees.

Complete transposition of the great arteries(TGA)

There is usually no murmur. The ECG shows dominant right ventricular voltages (normal neonatal RS progression). Therefore, if a newborn is severely cyanosed and otherwise appears clinically normal, actively look for a narrow mediastinum on the chest X-ray to help to make the diagnosis.

Management of defects with low pulmonary blood flow or complete TGA

1. Do not give oxygen after the hyperoxia test, as it may precipitate ductal closure.
2. Start IV prostaglandin E (PGE) to maintain ductal patency. There are two formulations, namely prostaglandin E1 (PGE1) and prostaglandin E2 (PGE2). Commence PGE1 at 25 nanograms/kg/minute or PGE2 at 5 nanograms/kg/minute.
3. PGE infusion is made up by adding 30 micrograms/kg of prostaglandin to 50 mL of 5% dextrose (if the pump runs at 1 mL/hour this is equivalent to 10 nanograms/kg/minute).
4. If oxygen saturations are initially very low and fail to improve 10 minutes after starting PGE, intubate and ventilate the baby in air and increase the PGE dose to 50 nanograms/kg/minute. The dose can be increased further to a maximum of 100

nanograms/kg/minute if there is still no response. Prostaglandin sometimes causes vasodilation, so fluid boluses may be required at high doses of PGE in order to maintain blood pressure.

5. PGE often causes hypoventilation and apnoea (particularly at doses of PGE2 above 10 nanograms/kg/minute). If oxygen saturations initially improve with PGE and then start to fall, assess respiratory effort. If respiration is shallow or slow, intubate and ventilate in air.

6. If oxygen saturations start to fall after PGE is started, and respiratory effort appears adequate, increase the PGE dose stepwise until a response is seen. At doses over 10 nanograms/kg/minute watch very carefully for hypoventilation.

7. Arrange for an urgent paediatric cardiology review and transfer the infant to a cardiac centre.

8. Defects with poor pulmonary blood flow usually require a systemic to pulmonary artery shunt (modified Blalock–Taussig shunt) to provide a stable pulmonary blood supply. Where special interventional expertise is available it may be possible to implant a coronary stent in the duct to maintain patency and avoid surgery.

9. TGA often requires enlargement of the inter-atrial communication by balloon atrial septostomy, followed by an arterial switch operation if surgical expertise is available.

10. For critical pulmonary valve stenosis or pulmonary atresia, early intervention to open the pulmonary valve is required. This can be done by the trans-catheter route if interventional expertise is available.

TABLE 25.4 Cyanotic defects with high pulmonary blood flow

Truncus arteriosus
Total anomalous pulmonary venous connection
Double-outlet left ventricle
Absent right atrioventricular connection with large ventricular septal defect (tricuspid atresia)
Pulmonary atresia with large or multiple aorto-pulmonary collateral arteries
TGA with large ventricular septal defect

Management of common mixing lesions

1. Monitor the infant on the neonatal unit if available

2. Arrange for an echocardiogram as soon as possible to define the anatomy.

3. If oxygen saturations fall progressively to less than 70%, commence PGE and arrange for an urgent paediatric cardiology review.

4. Once the anatomy is defined it may be possible to discharge the baby without further treatment (after paediatric cardiology advice has been obtained).

Section 25 Congenital heart disorders

The older infant with cyanosis

Is there a cardiac problem?

When an older infant presents with cyanosis, cardiac pathology is likely if:

- Respiratory distress is not severe.
- There is no carbon dioxide retention.
- Respiratory pathology is not evident on the chest X-ray.
- The cardiovascular examination is abnormal (see below).

What type of cardiac defect is present?

The cyanotic defects that commonly present after the neonatal period are tetralogy of Fallot and cyanotic defects with high pulmonary blood flow. They may escape detection at birth because cyanosis is initially only mild. In tetralogy of Fallot, there is right ventricular outflow tract obstruction and a large ventricular septal defect (VSD) (right ventricular hypertrophy and aortic override are the other components of the tetralogy). The right ventricular outflow tract obstruction limits blood flow to the pulmonary arteries, causing deoxygenated blood to shunt right to left across the VSD, resulting in cyanosis. With time, the right ventricular outflow tract obstruction usually becomes more severe, causing further reductions in pulmonary blood flow, more right to left shunting, and increasing cyanosis.

Cyanotic defects with high pulmonary blood flow

In cyanotic defects with high pulmonary blood flow (mostly common mixing defects), pulmonary flow increases as pulmonary vascular resistance decreases over the first few weeks of life, resulting in progressively worsening cardiac failure.

Findings in defects with high pulmonary blood flow

- May present with cardiac failure at 2–6 weeks of age.
- Active praecordium.
- Murmur usually present (may be systolic, diastolic or continuous).
- Increased pulmonary vascular markings on chest X-ray.

Management of cyanotic defects with high pulmonary blood flow

Define the anatomy by echocardiography. Manage cardiac failure medically (*see* Section 26). Surgical correction or pulmonary artery banding will be necessary in most cases.

Findings in Tetralogy of Fallot

- May present with increasing cyanosis.
- May present with an ejection systolic murmur at the upper left sternal border.
- Reduced pulmonary vascular markings on chest X-ray, and concavity on the left heart border where there is usually a convexity produced by the right ventricular outflow tract and pulmonary artery.

- Infants are often asymptomatic, but there may be sudden periods of increased cyanosis known as hyper- cyanotic spells.

Characteristics of hypercyanotic spells

- Spells often occur on waking from sleep or after feeding.
- The infant becomes restless and agitated.
- There is increased cyanosis and pallor.
- Respiration is often rapid and shallow.
- In severe spells, crying is followed by limpness or loss of consciousness.
- Spells usually last 1–5 minutes, but may last longer when severe.
- The ejection systolic murmur shortens or becomes inaudible.

Management of tetralogy of Fallot

1. The anatomy should be confirmed by echocardiography, preferably within a few weeks of presentation, and surgical correction should be carried out between 6 and 12 months of age (although it can be carried out later).
2. **Hypercyanotic spells may be life-threatening. If an i n f a n t starts to have such spells, discuss this with a paediatric cardiologist immediately, as it is an indication for urgent surgery.**
3. If hypercyanotic spells are more than a few minutes in duration, treat them urgently as follows:
   - Knee–chest position.
   - Give oxygen by face mask.
   - Give an IV bolus of Ringer-lactate or Hartmann's solution 10–20 mL/kg, as during spells infants are often relatively hypovolaemic.
   - Give IV or IM morphine, 100 microgram/kg (or IV ketamine 1 mg/kg).
   - Give IV propranolol at an initial dose of 20 micrograms/kg with a maximum of 100 micrograms/kg (have isoprenaline ready in case of excessive–blockade).
   - Adrenaline may make spells worse
   - .General anaesthesia and artificial ventilation are needed in intractable cases.
4. If cyanosis persists, consider an emergency aorto-pulmonary shunt.

Neonatal cardiovascular collapse

Is there a cardiac problem?

When an i n f a n t presents in shock in the first month of life, the working diagnosis is often dehydration or sepsis. The following features help to distinguish cardiac causes of poor systemic output from non-cardiac causes:

- collapse in the first 2 weeks of life
- poor feeding, lethargy and tachypnoea prior to collapse
- hepatomegaly
- pulmonary oedema and cardiomegaly on chest X-ray
- lack of response to intravascular volume expansion.

Section 25 Congenital heart disorders

What type of cardiac defect is present?

Left heart obstruction is the most likely cardiac cause of cardiovascular collapse with low systemic output in the first 2 weeks of life:

TABLE 25.5 Left heart obstruction

Critical aortic stenosis
Hypoplastic left heart syndrome (HLHS)
Coarctation of the aorta
Interrupted aortic arch
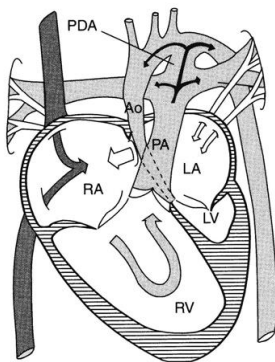
FIGURE 25.4 Hypoplastic left heart. For explanation of abbreviations, see legend to Figure 25.1.

Hypoplastic left heart syndrome (HLHS)

In hypoplastic left heart syndrome all of the left heart structures are small (*see* Figure 25.4). There is insufficient forward flow through the left ventricle and the aortic valve to support the systemic circulation. Pulmonary venous return cannot pass through the left heart, so it crosses the atrial septum and enters the right atrium, mixing with systemic venous return. Mixed pulmonary and systemic venous blood enters the right ventricle and is pumped to the pulmonary arteries and also across the arterial duct to supply the systemic circulation. Ductal flow passes to the descending aorta and retrogradely around the aortic arch to supply the head and neck vessels and the coronary arteries. Ductal flow is not fully oxygenated, so there is a

degree of central cyanosis. When the duct closes, the cardiac out- put falls precipitously, the infant becomes shocked, and cardiac failure develops. This is duct-dependent systemic circulation. The haemodynamics are the same in critical aortic stenosis.

Coarctation of the aorta

Coarctation of the aorta consists of a narrowing in the descending aorta close to the aortic end of the arterial duct. Contractile tissue may extend from the duct into the aorta so that when the duct closes it draws in the adjacent section of aorta, causing obstruction. Flow to the head and neck vessels is maintained, but flow to the lower body distal to the coarctation site is dramatically reduced. The infant becomes shocked and acidotic. Cardiac failure develops secondary to high systemic afterload. This is also an example of the systemic circulation depending on ductal patency (although systemic blood flow may not directly depend on a right-to-left shunt through the duct). In interrupted aortic arch, perfusion to the lower part of the body depends on right-to-left ductal flow and presentation is similar to that with coarctation.

The following features help to distinguish between the lesions causing left heart obstruction:

- If all of the pulses are weak or absent, consider HLHS or critical aortic stenosis.
- If the right arm pulses are palpable and the femoral pulses are weak or absent, consider coarctation or interrupted aortic arch (note, however, that all pulses may initially be impalpable if the cardiac output is poor).
- If four limb blood pressures demonstrate significantly lower blood pressures in the legs than in the right arm (a gradient of more than 20 mmHg), consider coarctation or interrupted aortic arch.
- Coarctation often presents towards the beginning of the second week of life.
- HLHS often presents in the first 2 days of life.
- In HLHS, the ECG shows reduced left ventricular volt- ages (small R waves in leads V5 and V6).

Other cardiac causes of cardiovascular collapse in the first few weeks of life are supraventricular tachycardia (SVT) and cyanotic congenital heart disease with duct-dependent pulmonary blood flow (when the duct closes, the ensuing profound hypoxaemia causes acidosis and cardiovascular collapse). SVT should be evident on the ECG and cyanotic heart disease should be suspected when the oxygen saturation remains low after instituting the management described below for left heart obstruction.

Emergency management of low systemic output secondary to left heart obstruction

1. Check the ECG (to exclude SVT as a cause of collapse).

2. Obtain peripheral IV access if not already established (if IV access is difficult, intra-osseous access should be obtained).

3. Give a fluid bolus of 10 mL/kg Ringer-lactate or Hartmann's solution if not already given.

4. Intubate and ventilate if there is significant respiratory distress (high PEEP, 8–10 cmH2O).

5. Once ventilated, commence prostaglandin E1 or E2 100 nanograms/kg/minute (give for 30 minutes, then reduce to 2 nanograms/kg/minute, reducing again to 10 nanograms/kg/minute when stabilised). If the initial clinical condition is not poor, commence PGE2 at a lower dose of 10 nanograms/kg/minute, which should avoid PGE-related apnoea and the need for ventilation.

6. Admit the infant to a paediatric ICU (if available)

7. Check blood sugar levels, full blood count, urea and electrolytes, coagulation, calcium levels and magnesium levels, and correct abnormalities.

8. Take blood cultures and treat with IV antibiotics, as sepsis cannot be excluded.

9. Check arterial blood gas (using the right arm if possible).

10. Give IV furosemide 1 mg/kg if the chest X-ray shows pulmonary oedema.

11. Insert central venous access and an arterial line.

12. Reassess whether further intravascular volume is needed (give if the central venous pressure is low).

13. Give dopamine 5–10 micrograms/kg/minute if perfusion remains poor or the blood pressure remains low.

14. Give adrenaline 0.1–0.2 micrograms/kg/minute if perfusion remains poor or the blood pressure remains low (by central venous access only).

15. If acidosis is profound and not improving with other measures, give IV sodium bicarbonate 4.2% (2ml/Kg).

16. Ask for an urgent paediatric cardiology review and advice.

Asymptomatic murmurs

When a newborn infant presents with an asymptomatic murmur, first examine them for cyanosis and measure the oxygen saturation. If there is desaturation, refer the infant for an echocardiogram, as cyanotic congenital heart disease requires a detailed anatomical assessment. Tetralogy of Fallot is the most likely diagnosis. If cyanosis is excluded, the nfant may have an innocent cardiac murmur or one of the following defects.

TABLE 25.6 Initially asymptomatic heart lesions

Left-to-right shunts	Left or right heart obstruction
Small to moderate-sized VSD	Pulmonary stenosis
Small to moderate-sized PDA	Aortic stenosis
Atrial septal defect (ASD)	Coarctation of the aorta
Partial AVSD	

Innocent murmurs are characterised as follows:
- The Still's murmur is a vibratory short systolic murmur heard at the lower left sternal border or apex.
- The venous hum is a soft continuous murmur heard best below the clavicles, and is abolished by pressure over the jugular vein or lying down with the neck flexed.
- The pulmonary flow murmur is a soft ejection systolic murmur at the upper left sternal border, and may be confused with an ASD or mild pulmonary stenosis.
- The neck bruit is an ejection systolic murmur that is maximal above the clavicle and may be confused with aortic stenosis.

The cardiac defects are characterised as follows:

1. In coarctation, the right arm blood pressure is often elevated, the femoral pulses are weak or impalpable, and there is brachiofemoral delay.
2. The patent ductus arteriosus (PDA) has a continuous murmur that is loudest in the left infraclavicular region.
3. The ventricular septal defect (VSD) has a harsh pan- systolic murmur that is loudest at the lower left sternal border radiating to the lower right sternal border.
4. Aortic stenosis, pulmonary stenosis, atrial septal defect (ASD) and partial atrioventricular septal defect (AVSD) all have an ejection systolic murmur at the upper left sternal border.
    - In aortic stenosis the ejection systolic murmur is harsh and may be heard at the upper right and left sternal border. The murmur radiates to the carotid arteries and there is often a carotid thrill. There may be an ejection click at the apex if the stenosis is at valvar level.
    - In pulmonary stenosis, the ejection systolic murmur is harsh and radiates to the back. There may be an ejection click along the left sternal border if the stenosis is at valvar level.
    - In an atrial septal defect (ASD) there is a soft ejection systolic murmur at the upper left sternal border from increased flow across the pulmonary valve. There is sometimes a fixed widely split second heart sound, and there may be a mid-diastolic murmur at the lower left sternal border (from increased flow across the

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tricuspid valve) when the left-to-right shunt is large.

— In partial atrioventricular septal defect (AVSD) there is an abnormal atrioventricular valve and a defect in the atrial septum. There may be a blowing pansystolic murmur at the lower left sternal border or apex from atrioventricular valve regurgitation. The ejection systolic murmur may mimic an ASD, but the defect is distinguished by a superior QRS axis on the ECG.

Unless the murmur is clearly innocent, perform an ECG and chest X-ray.

Right ventricular hypertrophy (RVH) is indicated by an R wave in lead V1 > 98th centile for age ($\geq$ 20 mm is always abnormal), a neonatal RS progression beyond the neonatal period (dominant R waves in lead V1 and dominant S waves in lead V6) or an upright T wave in lead V1 before the teenage years.

Left ventricular hypertrophy (LVH) is indicated by T inversion in leads V5 and V6, loss of the Q wave in lead V6 or the amplitude of the R wave in lead V6 plus S wave in lead V1 > 98th centile for age (> 50 mm is always abnormal). RVH may indicate significant right heart obstruction or high pulmonary artery pressure (secondary to a large left-to-right shunt or pulmonary vascular disease). LVH may indicate significant left heart obstruction. Cardiomegaly and increased pulmonary vascular markings on the chest X-ray may indicate a large left-to-right shunt.

Any infant who is thought to have an anatomical defect on the basis of the clinical examination, or any infant with an abnormal ECG or chest X-ray, should if possible be referred to a paediatric cardiologist for an echocardiogram and opinion. If there is evidence of a significant left-to-right shunt in a VSD or PDA, the refferal should be as soon as possible, as there is still a risk of pulmonary vascular disease even when the infant does not present in heart failure.

Heart failure in infancy

Introduction

Heart failure occurs when the heart is unable to pump enough blood to meet the metabolic needs of the body. The term is often used to indicate the clinical changes that occur when the cardiac pump cannot meet the workload it is presented with. This may occur either because the pump is weak (due to a primary abnormality of the cardiac muscle) or because the workload imposed on the heart is higher than normal. The latter is the case in congenital heart disease, where heart failure occurs because the heart is pumping against a high resistance (in the case of obstructive lesions) or because it is volume loaded (commonly in left-to-right shunting cardiac lesions).

Left-to-right shunting cardiac defects are the commonest cause of heart failure in infancy identified in well-resourced countries.

In resource-poor countries most heart failure is related either to severe anaemia or to fluid overload when treating infections or severe malnutrition, particularly with IV fluids or during blood transfusion (see below).

The physiology of left-to-right shunts

A large defect between the ventricles or great arteries allows free communication between the left and right sides of the heart. Left and right heart pressures therefore equalise, and pulmonary artery pressure is maintained at systemic level. The pulmonary vascular resistance then determines the pulmonary blood flow. In the newborn period the pulmonary vascular resistance is high, which limits the pulmonary blood flow and therefore the left-to-right shunt across the defect. Over the first 6 weeks of life, the pulmonary vascular resistance gradually falls, allowing pulmonary blood flow and the left-to-right shunt to increase. This gives rise to heart failure, which usually appears after 4 weeks of age. If the pulmonary arteries are exposed to high pressure and flow for a prolonged period, pulmonary vascular disease develops. This normally becomes significant between 12 and 18 months of age. High pulmonary vascular resistance secondary to pulmonary vascular disease reduces the left-to-right shunt, and symptoms of heart failure gradually resolve. Eventually, pulmonary resistance becomes so high that flow across the defect becomes right to left, and cyanosis develops (Eisenmenger's syndrome). The pulmonary artery pressure remains high throughout, and it is only the amount of flow through the lungs that changes.

Section 26 Heart failure in infancy

Is heart failure present?

TABLE 26.1 Diagnosis of heart failure secondary to **congenital** heart disease **in infancy**

Symptoms	Signs
Easily tired	Failure to thrive
Poor feeding	Tachypnoea
Breathlessness (particularly during feeds)	Increased respiratory effort
	Tachycardia > 160 bpm
Sweaty (particularly during feeds)	Sweating
	Pallor
	Hepatomegaly
	Gallop rhythm

What type of cardiac defect is present?
Heart failure in the first few weeks of life is a medical emergency.
The following causes should be considered:

- supraventricular tachycardia
- complete atrioventricular block
- high-output cardiac failure
- left heart obstruction.

Perform an ECG to detect supraventricular tachycardia and heart block. Check the haemoglobin level, as severe anaemia may cause high-output cardiac failure. Also examine the baby for cranial and hepatic bruits, as cranial and hepatic arteriovenous malformations are a potential (although very rare) cause of high-output cardiac failure.

If these tests are negative, refer the infant to a paediatric cardiologist (if available), as a left heart obstructive lesion is likely and there may be duct-dependent systemic circulation. Consider the use of prostaglandin to keep the ductus arteriosus open until the referral can be achieved.

Heart failure in infancy presenting after the first few weeks of life may be caused by any of the following:
- the left-to-right shunting lesions listed in Table 25.6
- cyanotic congenital heart defects with high pulmonary blood flow
- the same causes that present in the first few weeks of life
- myocarditis or cardiomyopathy.

Section 26 Heart failure in infancy

TABLE 26.2 Common left-to-right shunting lesions that cause heart failure

Large ventricular septal defect (VSD)
Atrioventricular septal defect with large ventricular component (AVSD)
Large persistent ductus arteriosus (PDA)

Examine the child for cyanosis and measure the oxygen saturation. It should be possible to detect those children with cyanotic defects immediately (note, however, that children with AVSD are sometimes mildly desaturated).

Next, attempt to detect the children with left-to-right shunts, looking for the following features which are present in significant shunts:

- hyperdynamic precordial impulse
- apical impulse displaced laterally and inferiorly
- apical mid-diastolic murmur (from increased flow across the mitral valve)
- loud second heart sound (from increased pulmonary artery diastolic pressure)
- cardiomegaly and increased pulmonary vascular markings on the chest X-ray
- signs of heart failure and pulmonary oedema on the chest X-ray in severe cases.

If these examination findings are not present and there is no evidence of SVT or a hyperdynamic circulation (see above), a left heart obstructive lesion should be considered. Some of these are eminently treatable conditions and if they are suspected the infant should be referred for paediatric cardiology review without delay.

If there is evidence of a large left-to-right shunt, refer the infant to a paediatric cardiologist within a few weeks. These signs must not be missed, as a remediable cardiac defect is rendered inoperable by delay.

Although it is not imperative to make a more specific diagnosis, the following clinical features discriminate between the three most common left-to-right shunts:

- The persistent arterial duct has a continuous murmur that is maximal in the left infra-clavicular area.
- A large ventricular septal defect has a quiet pansystolic murmur that is maximal at the lower left sternal border radiating to the lower right sternal border. There may also be a soft ejection systolic murmur at the upper left sternal border from increased flow across the pulmonary valve.
- An atrioventricular septal defect with a large ventricular component may have a blowing pansystolic murmur at the lower left sternal border or apex from atrioventricular valve regurgitation. The ECG shows a characteristic superior QRS axis (between −30 and −180 degrees).

Heart failure in later infancy

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In addition to the symptoms seen in early infancy (easily tired, poor feeding, breathlessness particularly during feeds, excess sweating particularly during feeds), older infants may have shortness of breath when lying flat.

The signs of heart failure are cyanosis or SaO_2 < 94%, basal lung crepitations, failure to thrive, tachypnoea > 50 breaths/minute for infants aged 2–12 months

There is usually increased respiratory effort, sweating, pallor and hepatomegaly.

In addition to the congenital heart defects described in Section 25 the following causes of heart failure should be considered:

- Severe anaemia
- Severe malnutrition
- Excessive intravenous fluids
- Rheumatic fever
- Myocarditis
- Cardiomyopathy
- Infective endocarditis
- Constrictive pericarditis (rare and most often caused by tuberculosis).

Anaemia is a common and often severe problem in poorly resourced settings. When the haemoglobin falls below 7 g/dl cardiac output must increase to maintain oxygen delivery and heart failure frequently develops with a haemoglobin < 5 g/dl). The treatment is careful blood transfusion, but the increase in intravascular volume may precipitate worsening heart failure. Blood must therefore be infused slowly in small boluses and an exchange transfusion may be needed if there is clinical deterioration. Furosemide 1 mg/kg IV may be given during transfusion.

Protein-calorie malnutrition is also an important cause of cardiac failure in disadvantaged countries (*see* Textbook) with specific contributions from certain vitamin deficiencies (*see* Textbook). Although cardiac failure is unusual at presentation, it may occur after several days of re-feeding. Rapid re-feeding can cause a hyper- metabolic state, demanding an increase in cardiac output which cannot be met by the malnourished heart which has a decreased cardiac reserve. The problem is exacerbated by coexisting anaemia, blood transfusion, inappropriate intravenous fluid administration and high sodium diets.

Management of heart failure
Monitor heart and respiratory rates, respiratory distress and oxygenation regularly during treatment of acute heart failure. It is necessary to both control the symptoms of failure and to determine and treat the underlying cause.

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1. Treat severe anaemia if present, be careful with IV fluids and ensure adequate nutrition.
2. Nasogastric feeding if there is inadequate oral intake.
3. For older infants nurse sitting up with legs dependent
4. Treat hypoxaemia with oxygen to keep SaO_2 > 94%.
5. In an emergency where there is pulmonary oedema, give furosemide 1 mg/kg IV which should produce a diuresis within 2 hours. If the initial dose is ineffective, give 2 mg/kg IV and repeat after 12 hours if necessary.
6. For chronic heart failure give oral furosemide 1 mg/kg once a day, twice a day or three times a day.
7. Spironolactone 1 mg/kg once a day or twice a day in combination with furosemide, matching the dose frequency, to enhance diuresis and prevent furosemide-related hypokalaemia.
8. If furosemide is used without spironolactone, oral potassium 3–5 mmol/kg/day, should be given (supplemental potassium is not required if furosemide is given for less than 4 days).

If more than twice daily diuretics are required, consider using captopril. Captopril should be commenced in hospital with a 100 microgram/kg test dose. The dose should then be increased gradually over a number of days 100–300 microgram/kg 2–3 times a day to a maximum total dose of 4 mg/kg daily. After the test dose and each increment monitor the blood pressure carefully, as hypotension is common. Reduce the dose if significant hypotension occurs. Monitor urea and electrolytes daily while building up the dose, as renal failure is a well-recognised side effect. Stop spironolactone when the captopril dose is greater than 500 micrograms/kg per day as both drugs cause potassium retention. Do not give captopril if there is left heart obstruction.

Shock in infancy

In the early stages of shock the body has mechanisms to try to combat this process. The sympathetic nervous system attempts to protect the vital organs by diverting blood away from muscle, skin and the digestive system and directing it to the heart, brain and kidneys. This gives rise to some of the earlier signs of shock, such as cold peripheries, increased capillary refill time, cerebral anxiety or agitation, tachycardia to increase cardiac output, and reduced urine output as the kidneys conserve fluid.

Later signs such as depressed consciousness, weak pulses, falling blood pressure and acidotic breathing show that the body's compensation mechanisms are failing. It can be seen that it is vital to recognise and treat shock in the patient as soon as possible, as this will give the best chance of patient recovery.

Clinical diagnosis of shock

The signs of shock are listed below, although not all of them are present in all types of shock.

- **Tachycardia** (best measured with a stethoscope).
- **Weak pulse** (ideally central – brachial, femoral or carotid, but difficult in infancy).
- **Low blood pressure** (this is a late sign and very difficult to measure in infants).
- Extreme central pallor (severe anaemia).
- Raised respiratory rate (due to acidosis).
- **Cold skin with poor circulation**.
- **Prolonged capillary refill time (CRT) > 3 seconds.**
- Increased skin sweating in some cases.
- Agitation and anxiety (this is an early sign).
- **Reduced conscious level.**
- Reduced urine output (this is an early sign).

The WHO diagnosis of shock includes all of the above signs that are highlighted in bold type.

The problem is that shock is quite difficult to diagnose in the early stages, as some signs also occur as a result of medical causes other than shock. The diagnosis in the early stages depends on the following:

- tachycardia, which is a very useful sign of shock, but also occurs with fever and with anxiety or fear
- anxiety and/or agitation and persistent crying
- prolonged capillary refill time, which also occurs in dehydration and is influenced by environmental temperatures and by how hard the nail bed or sternum is pressed
- cold skin, which is also dependent on environmental temperature
- reduced urine output, which is also dependent on fluid intake.

Section 27 Shock in infancy

It is vital that if any of these early signs are noted in a patient that they are not dismissed as some unrelated cause, but are seriously considered as likely to be indicating the development of shock.

This is why it is so useful to have regular vital signs (pulse, respiration, conscious level, temperature and blood pressure) observations on patients, so that abnormal trends can be detected early.

It is also important to note that shock is not diagnosed on the basis of one physical sign alone, but on the basis of several signs occurring together. For example, a tachycardia alone does not diagnose shock, but if you note a tachycardia, you should look for cold limbs, prolonged capillary refill time, or a history suggestive of a cause of shock, such as a fever, severe diarrhoea or bleeding.

P*athological mechanisms that can cause shock*

The main mechanisms of shock:

- loss of fluid or blood: hypovolaemic shock (e.g. diarrhoea, blood loss)
- failure of the heart pump: cardiogenic shock (e.g. dysrhythmias, cardiomyopathy, myocarditis, malnutrition)
- abnormal function of vessels supplying nutrients and oxygen to tissues: distributive shock (e.g. sepsis, anaphylaxis)
- inadequate capacity of the blood to release oxygen that is dissociative shock (e.g. severe anaemia, carbon monoxide poisoning)
- restriction of circulation to the tissues: obstructive shock (e.g. some congenital heart diseases, tension pneumothorax).

In an individual with shock, often several of these mechanisms may coexist. Therefore, the clinician must consider which emergency treatments will be effective and which will be harmful for any particular patient. One of the most difficult situations is in the anaemic malnourished infant with sepsis, where fluid is required to expand the circulating volume, but the heart is already failing and cannot cope with a rapid fluid infusion.

Basic management of shock

Shock is managed according to the following principles:

- High concentrations of oxygen are safe and must be given regardless of the cause of shock.
- Airway and breathing stability or support must be established promptly first (the only exception is to control exsanguinating external bleeding).
- Frequent reassessment, at least after every therapeutic manoeuvre, is vital to avoid both under-infusing and over-infusing fluids.
- The underlying pathology must be treated to arrest the pathological process.

The clinical diagnosis of the cause of shock is not easy or definitive. Shock is a spectrum of conditions and mechanisms, and it is a clinical challenge.

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Immediate resuscitation is needed to maintain oxygenation and perfusion of vital organs. Once this is under way, the cause of shock needs to be found and treated.

Diagnosis depends on history, clinical examination, and response to treatment given. It is often possible to identify the cause of shock with a good history and a careful examination.

Investigations

- Haemoglobin measurement is essential.
- Blood glucose measurement is essential, as some signs of shock are the same as signs of hypoglycaemia.
- Plasma electrolyte measurements are helpful, especially sodium and bicarbonate.
- Lactate measurement is helpful (if available).

Choice of intravenous fluid

Fluid infused into the circulation should approximate to plasma in its electrolyte content, osmolality and pH.

Dextrose-only fluids

It is clear that although glucose or dextrose is necessary to prevent or manage hypoglycaemia, fluids containing only dextrose should never be used for IV fluid replacement or maintenance, or for the emergency management of shock.

TABLE 27.1 Diagnostic pointers to the clinical causes of shock in infancy

Diarrhoea and/or vomiting with signs of severe dehydration	Gastroenteritis (*see* Section 28 and volvulus, intussusception (*see* Section 29)
Fever, non-blanching (purpuric) rash	Meningococcal septicaemia
Urticaria, wheeze, oedema, exposure to allergen	Anaphylaxis
Trauma	Blood loss, tension pneumothorax, internal bleeding, spinal cord transection
Burns	Fluid loss from burns
Pallor, tachycardia, severe malaria, severe acute malnutrition	Severe anaemia, often with malnutrition and malaria
Fever, signs of shock and a very sick child	Septicaemia and malaria
Baby < 4 weeks old: cyanosis, with no response to oxygen, very weak pulses	Congenital heart disease
Very fast pulse, heart failure	Arrhythmia and cardiomyopathy (*see* Textbook)

The reason for this is that the dextrose is rapidly metabolised, so the effect of a dextrose-only IV fluid on the child's body is as if pure water had been given. The outcome of this treatment would be severe hyponatraemia, which could quickly lead to brain damage or death.

In addition, this pure water is rapidly moved out of the circulation and into the cells, and the state of shock is then worse than before the infusion.

Sodium-containing fluids

The fluid traditionally infused into the circulation for the management of shock has been 'normal saline' (0.9% sodium chloride, NaCl). This fluid has increasingly been shown to be potentially dangerous, especially in the sick patient. An infusion of normal saline causes a hyperchloraemic acidosis (a high chloride concentration leading to an acidosis), which in the shocked patient, who is already acidotic, causes a deterioration in the health of cells in vital organs, even though perfusion of the cells has been improved by the increased circulating volume.

There are sodium-containing alternatives to normal saline which are safer as they approximate more closely to human serum in content (*see* Table 27.2), although they are a little more expensive. We recommend the use of either Ringer-lactate or Hartmann's solution for all fluid replacement in shock. Recognising that not all hospitals will have access to these solutions immediately, there may sometimes be no alternative but to start fluid replacement with normal saline. However, if more than 20 mL/kg needs to be given, one of the safer alternatives should be used in these very sick infants if at all possible.

TABLE 27.2 Comparison of electrolytes, osmolality and pH levels in IV fluids with those in human serum

Fluid	Na^+ (mmol/litre)	K^+ (mmol/litre)	Cl^- (mmol/litre)	Ca^{2+} (mmol/litre)	Lactate or bicarbo	Osmolarity (mOsm/l	pH
Human serum/plasma	135–145	3.5–5.5	98–108	2.2–2.6	22–30	276 to 295	7.35–7.45
Ringer-lactate or	131	5.0	111	2.0	29	279	6.0
0.9% 'normal' saline	154	0	154	0	0	310	5.4

Initial management of shock

Even though it may be clear on initial inspection that the infant is in shock, the first priority must still be to call for help, manage the airway, manage breathing and then manage the circulation.

Section 27 Shock in infancy

Call for help.

Airway

At this stage also stop any obvious exsanguinating bleeding.

Assess the airway by the simple technique of gently shaking the baby and looking for a response. Any vocalisation such as a reply or crying indicates an open airway and some ventilation. In the absence of a response, formally open the airway with a head tilt/chin lift or a jaw thrust manoeuvre (*see* Section 2), and assess breathing by looking, listening and feeling for its presence.

Stop any obvious exsanguinating bleeding by applying external pressure.

Breathing

All infants with suspected shock must receive high-flow oxygen.
In the absence of spontaneous breathing, give assisted ventilation with a bag-mask.

Circulation

Intravenous access with a short wide-bore venous cannula, or placement of an intra-osseous line (see Section 43), is vital. More than one line is preferable, as rapid fluid resuscitation may be needed, although always start treatment as soon as the first access has been achieved and insert the second line when possible. Take blood samples for the following investigations: full blood count, glucose levels, electrolytes, blood culture (and, if relevant, cross-matching)

Nutritional status

While starting to give fluid, assess the infant's nutritional status.   Look for visible severe wasting or marasmus. The WHO recommended criteria are as follows: 'Look at the arms, legs and chest. The marasmic child does not look just thin, but appears to be all skin and bone. The skin looks too large for the body, there is no fat on the child and you will see the outlines of the ribs. There is also severe muscle wasting of the arms, legs and buttocks. The head may appear relatively large because of wasting of the body.' Use the mid upper arm circumference (MUAC) to assess marasmus, as the urgency of the child's need for treatment precludes a weight and height measurement.

Look also for Kwashiorkor. Check for oedema of both feet. Look at the bare feet. Press the top of the foot gently with your thumb for a few seconds. Oedema is present if a definite dent is left in the tissues. Look and feel to determine whether the infant has oedema of both feet.

Some assessment of weight will be necessary to calculate the amounts of fluid and antibiotics to be given.

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Severe anaemia

In very anaemic infants (with either obviously pale palms or haemoglobin levels of less than 3–4 grams/dL), crystalloid alone will worsen oxygen delivery to the tissues. These infants need blood, either packed cells or a partial exchange transfusion, in addition to initial slow fluid resuscitation.

The next step is to **give fluid intravenously**. In most cases this should be a crystalloid such as Hartmann's or Ringer-lactate solution, but give normal saline (0.9%) if this is all that is available. In infants, the initial volume of fluid to be given is usually 10 mL/kg, especially in severe anaemia or malnutrition which is 12.5% of the circulating volume. Shock is not usually clinically evident until 25% of the circulation has been lost, so any infant with signs of shock must have lost at least this amount of fluid from the circulation.

In infants with shock start with IV boluses of 10 mL/kg of crystalloid, or ideally blood if the cause of shock is haemorrhage or severe anaemia, and reassess after each bolus.

The next important step is to **reassess the patient's vital signs** to see whether the fluid has helped, and to ensure that circulatory overload has not given rise to a situation where more IV fluids may produce very dangerous heart failure (see Section 26 for clinical signs of this).

During this reassessment, give IV antibiotics, as shock without obvious fluid loss is probably sepsis.

At this point, some infants will need more crystalloid fluid, while others will not, or they will need other fluids (e.g. blood). Many will need additional treatments.

FIGURE 27.1 Pathway of care for the infant with shock that is not cardiac in origin.

Airway → Not open or at risk → Head tilt – chin lift / Jaw thrust / Oropharyngeal airway / Intubation

Open → Breathing → Inadequate → Assist ventilation

Yes

100% oxygen[1]–face mask and reservoir

[1] Oxygen is vital in all cases, especially severe anaemia and severe malnutrition

Circulation → Baseline assessment / Repeat observations regularly to assess response

IV or IO access → Take blood for: haemoglobin, glucose, cross-match, blood culture

If hypoglycaemic on blood test or suspected → 5 mL/kg 10% glucose IV or IO

Shock due to severe dehydration or sepsis if no severe anaemia or severe malnutrition

IV bolus 20 mL/kg Ringer-lactate or Hartmann's

IV antibiotics [2]

Assess response to fluid; if no better repeat IV bolus of 20 mL/kg Ringer-lactate or Hartmann's up to maximum of 40 mL/kg [3]

Shock due to bleeding

Stop bleeding IV/IO bolus 10 mL/kg Ringer-lactate or Hartmann's and urgently start successive 10 mL/kg boluses of whole fresh blood until shock resolves [3]

[3] When total of 40 mL/kg IV or IO given, request anaesthetist ideally present before more fluids or blood are given
Consider inotrope (dopamine infusion or adrenaline boluses) and IPPV + PEEP to support further IV fluid boluses if available

Severe malnutrition and/or severe anaemia on clinical examination and/or haemoglobin < 5 g/dL

Give 10 mL/kg Ringer-lactate or Hartmann's with 5% dextrose over 30–60 minutes while awaiting blood. Then give 10 mL/kg bolus of blood over 15 minutes, then reassess and repeat 10 mL/kg every hour until total of 40 mL/kg has been given[3], observing closely for circulatory overload

IV antibiotics [2]

[2] IV antibiotics for septicaemia – ceftriaxone or [ampicillin + gentamicin].
Add metronidazole if anaerobes may be present, especially if there is an intra-abdominal cause.
Correct any biochemical abnormality if possible (including bicarbonate if there is severe acidosis, especially when there is severe anaemia)
Re-evaluate after each step to check that the treatment is working, and for development of pulmonary oedema

The infant with severe dehydration

Dehydration

- Dehydration is loss of water, sodium and other essential electrolytes.
- The most common cause in resource-limited countries is gastroenteritis).
- Most cases can be treated with low-osmolarity oral rehydration solution (ORS) administered by mouth or nasogastric tube.
- In infants with severe malnutrition, use a solution with lower sodium content, such as ReSoMal.
- It is important to also consider surgical causes of dehydration, such as intussusception and volvulus (see Section 29).

Dehydration classification

Dehydration is classified by estimating the percentage of body water lost according to clinical criteria (except in malnutrition, where clinical signs are more difficult to interpret; see below).

'No dehydration'

If there is less than 3% weight loss there are no clinical signs.

'Some dehydration'

If there is 3–9% weight loss, the following signs are seen:

- increased thirst
- drinks eagerly
- dry mucous membranes
- loss of skin turgor, tenting when pinched
- sunken eyes
- sunken fontanelle in infants
- restless or irritable behaviour
- decreased capillary refill time (> 3 seconds)
- decreased urine output.

'Severe dehydration'

If there is 10% weight loss or more, the following signs are seen:

- more pronounced signs than those seen in moderate dehydration
- lack of urine output
- lack of tears when crying
- inability to drink or drinking poorly (because of reduced conscious level)
- lethargy

plus

- hypovolaemic shock, including:
  - rapid and weak low-volume pulse (radial pulse may be undetectable) (use a

stethoscope and measure the heart rate which will usually be increased)
— altered consciousness or coma
— low or undetectable blood pressure
— cool and poorly perfused extremities
— severe nail bed or sternum decreased capillary refill time
— peripheral cyanosis
— rapid deep breathing (from acidosis).

It is important to realise that the above classification is made only to guide the start of treatment. Levels of dehydration are a continuous spectrum, not three separate and distinct categories. The only way to be absolutely certain about the percentage dehydration of a infant is to compare an accurate weight measured just before the onset of the diarrhoeal illness with an accurate current weight. It is very unlikely in most cases that the former weight will be available. In the case of the shocked patient, immediate treatment takes precedence over weighing the infant.

Emergency treatment of severe dehydration: principles of treatment

1. Recognise and treat shock.
2. Give a fluid bolus, 20 mL/kg IV of Hartmann's or Ringer-lactate solution (0.9% saline, or 'normal' saline, can be used if there is no alternative).
3. A second bolus may be needed if the child does not respond (*see* the 'shock' pathway in Figure 27.1).
4. It is unusual to need more than two boluses in cases of dehydration due to gastroenteritis alone, unless they are due to cholera (*see* Textbook). Consider other causes, such as septicaemia, diabetic ketoacidosis (check blood sugar levels), volvulus or intussusception (check whether vomit is bile- stained, and whether there is fresh blood in stools; *see* Section 29).
5. If septicaemia is suspected, treat with IV antibiotics.
6. Think of the most likely cause of the dehydration.
7. Estimate the level of dehydration (see above) to calculate the fluid deficit, maintenance needs and ongoing losses (see below).

Shock in dehydration: recognition and treatment

Infants with shock associated with dehydration will have a high and increasing heart rate, weak pulse, poor skin circulation time with prolonged capillary refill time (> 3 seconds), depressed conscious level, and low or even unmeasurable blood pressure.

These infants require immediate resuscitation (ABC) and emergency treatment.

Call for help (summon an anaesthetist if possible).

Section 28 Severe dehydration and gastroenteritis in infancy

Airway (in cases of reduced conscious level) Use an opening manoeuvre if the airway is not open or if it is partially obstructed. Keep the airway open. If there is immediate improvement but the airway closes without active opening support, consider airway adjuncts to sup- port the airway.

Suction if necessary under direct vision, but not routinely. If the child is deeply unconscious (P or less on the AVPU scale), the airway may need to be secured by intubation using experienced senior help (if available).

Breathing

Give 100% oxygen (using a mask with reservoir and a flow rate of at least 6 litres/minute) regardless of SaO2 (this increases oxygen delivery as well as improving tissue oxygenation).

For inadequate ventilation or depressed conscious level (check with the AVPU scale) with hypoventilation, respiration should be supported with oxygen via a bag and mask, and experienced senior help should be summoned (if available).

Circulation

Obtain vascular access to give IV boluses quickly. Insert an IV cannula and send blood for a full blood count, urea and electrolytes, cross-matching (if the patient is anaemic) and clotting. If peripheral veins are difficult to access, the external jugular vein or long saphenous vein cut-down are good alternatives, as is an intra-osseous infusion (*see* Section 8.4.B). If a skilled operator is available, an internal jugular or femoral vein central line is ideal, as it can also allow central venous pressure (CVP) measurements (if available).

Infants with normal nutrition

If the infant is not malnourished, give an initial rapid bolus of 10–20 mL/kg of Ringer-lactate or Hartmann's solution, but give normal saline (0.9%) if this is all that is available. It is essential that the bolus is given as rapidly as possible. Do not use 5% glucose or 0.18% saline/4% glucose solutions for resuscitation, which can be dangerous (risk of hyponatraemia and cerebral oedema). Boluses should be manually pushed in using a 20-to 50-mL syringe (with a three-way tap and linked to an IV giving set).

When this bolus of fluid has been given, review the infant's condition, looking to see whether there has been any improvement in pulse rate, conscious level, respiratory rate, capillary return and limb warmth, and blood pressure.

A further 10–20 mL/kg bolus will be required if signs of shock remain. Once a total of 40 mL/kg of boluses has been given IV, complications such as pulmonary oedema are more likely to occur. In an infant with shock from severe dehydration caused by diarrhoea, it would be unusual to need more than 40 mL/kg to improve the infant's circulation, unless cholera was the cause. In severe cases, where more

than a total of 40 mL/ kg is considered essential, intubation, ventilation, CVP monitoring and inotrope support might be indicated (if available), but **the diagnosis should be reviewed as this need is unusual in straightforward gastroenteritis.** Reconsider the diagnosis. For examples:

Surgical abdominal pathology, such as intussusception, peritonitis or volvulus (bile-stained vomiting, abdominal distension or tenderness) (*see* Section 29)

Additional pathology, severe anaemia, septicaemia or a cardiac problem.

Infants with severe malnutrition or severe anaemia
Fluids must be given much more carefully. Give 15 mL/kg IV over 1 hour. The recommended solution is Ringer-lactate or Hartmann's solution, each with 5% glucose (insert 50 mL of 50% dextrose into a 500-mL bag of the bolus fluid, ideally after first removing 50 mL from the bag: not essential), but give normal saline (0.9%) if this is all that is available, also with 5% dextrose. At the same time, insert a nasogastric tube and give ReSoMal, 10 mL/kg/hour. Monitor carefully for signs of over-hydration: reassess the respiratory and heart rates every 15 minutes. It is also wise to give IV antibiotics in this situation, as it can be very difficult to distinguish septic shock from dehydration shock in infants with malnutrition.

Nutritional status
- While starting to give fluid, assess the infant's nutritional status. Look for visible severe wasting or marasmus. Follow the WHO criteria: 'Look at the arms, legs and chest. The marasmic infant does not look just thin, but appears to be all skin and bone. The skin looks too large for the body, there is no fat on the infant and you will see the outlines of the ribs. There is also severe muscle wasting of the arms, legs and buttocks. The head may appear relatively large because of wasting of the body.'
- Use the mid upper arm circumference (MUAC) to assess marasmus, as the urgency of the infant's need for treatment precludes a weight and height measurement.
- Look also for Kwashiorkor. Check for oedema of both feet. Look at the bare feet. Press the top of the foot gently with your thumb for a few seconds. Oedema is present if a definite dent is left in the tissues. Look and feel to determine whether the infant's has oedema of both feet.

Severe anaemia
In very anaemic infants (with either obviously pale palms or haemoglobin levels less than 3–4 grams/dL), crystalloid alone may worsen oxygen delivery to the tissues. These infants need blood, either packed cells or a partial exchange transfusion, in addition to initial slow fluid resuscitation.
If after 1 hour the infant is improving but still severely dehydrated, stop the IV fluids, but

continue nasogastric ReSoMal 10 mL/kg/hour for up to 5 hours (*see* Textbook for further details).

An infant with a haemoglobin level of less than 5 grams/dL will also need a transfusion of 10 mL/kg of packed cells over 4 hours, watching continuously for evidence of pulmonary oedema. If pulmonary oedema develops, furosemide 1 mg/kg IV may be required, but if possible pulmonary oedema of severity requiring diuretics should be avoided by a slow and vigilant approach to therapy in these very sick infants.

Keep the patient warm, but do not overheat them, as this will cause peripheral vasodilatation and reduce the blood supply to vital centres. Hypothermia will exacerbate poor peripheral perfusion, acidosis and coagulation abnormalities.

Elevate the patient's legs (raise the foot of the cot).

Importance of hypoglycaemia in shock

If the infant has a reduced level of consciousness or has a convulsion, hypoglycaemia may be present. Always measure the blood glucose concentration in this situation. However, if blood glucose measurement is not possible, always treat as for presumed hypoglycaemia and, in addition to the IV fluids given above, give 2 mL/ kg of 10% glucose IV or, if there is no IV access, by intra-osseous needle.

While treating shock, reassess the infant, ideally continuously, until signs of shock have resolved.

When signs of shock have resolved

When shock has resolved and the patient's level of consciousness has returned to normal, the remaining estimated fluid deficit MUST be taken by mouth or by gastric tube, especially if there is malnutrition and/or anaemia (due to the danger of a large IV fluid volume). Use WHO Plan B (*see* Textbook).

Electrolyte disturbances in dehydration from diarrhoeal illnesses

Knowledge of the levels of serum electrolytes rarely changes the management of infants with diarrhoea. Indeed, these values are often misinterpreted, leading to inappropriate treatment. The disorders described below are usually adequately treated by oral rehydration therapy (ORT).

Hypernatraemia

Some infants with diarrhoea develop hypernatraemic dehydration, especially when given drinks that are hypertonic due to their sugar content (e.g. soft drinks, commercial fruit drinks) or salt. These draw water from the infant's tissues and blood into the bowel, causing the concentration of sodium in extracellular fluid to rise. If the solute in the drink is not fully absorbed, the water remains in the bowel, causing osmotic diarrhoea.

Infants with hypernatraemic dehydration (serum Na^+ > 150 mmol/litre) have thirst that is out of proportion to other signs of dehydration. Their most serious problem is

convulsions, which usually occur when the serum sodium concentration exceeds 165 mmol/litre, and especially when intravenous therapy is given. Seizures are much less likely to occur when hypernatraemia is treated with ORS, which usually causes the serum Na^+ concentration to become normal within 24 hours.

It is absolutely essential that intravenous rehydration does not lower the serum Na+ too rapidly. Intravenous glucose solutions (5% glucose or 0.18% saline/4% glucose) are particularly dangerous and can result in cerebral oedema, which is usually fatal or permanently disabling. Check the serum sodium concentration, and if it is higher than 155 mmol/litre, reduce it slowly with oral rehydration solution over 48 hours. Too rapid a reduction in sodium levels leads to cerebral oedema.

Hyponatraemia
Infants with diarrhoea who drink mostly water, or watery drinks that contain little salt, may develop hyponatraemia (serum Na^+ < 130 mmol/litre). Hyponatraemia is especially common in infants with shigellosis and in severely malnourished infants with oedema. It is occasionally associated with lethargy and (less often) with seizures. ORS is safe and effective therapy for nearly all infants with hyponatraemia. An exception is infants with oedema, for whom ORS may provide too much sodium. ReSoMal (*see* Textbook may be helpful here.

Check the plasma potassium concentration
Hypokalaemia
Inadequate replacement of potassium losses during diarrhoea can lead to potassium depletion and hypokalaemia (serum K^+ < 3 mmol/litre), especially in infants with malnutrition. This can cause muscle weakness, paralytic ileus, impaired kidney function and cardiac arrhythmias. Hypokalaemia is worsened when base (bicarbonate or lactate) is given to treat acidosis without simultaneously providing potassium. Hypokalaemia can be prevented, and the potassium deficit corrected, by using ORS for rehydration therapy and by giving foods rich in potassium during diarrhoea and after it has stopped (e.g. bananas, coconut water, dark green leafy vegetables).

It is also essential to check blood potassium levels, especially if the infant has not passed urine, prior to replacing potassium IV, in order to avoid complications of hyperkalaemia secondary to pre-renal failure.

If it is necessary to give potassium intravenously (e.g. if serum K^+ is < 2.0 mmol/litre or there are ECG signs of hypokalaemia, namely ST depression, T-wave reduction and prominent U waves), great care must be taken. In acute depletion, an infusion at the rate of 0.2 mmol/kg/hour can be used and the serum K^+ level checked after 3 hours. The potassium for injection must be diluted before use and thoroughly mixed before being given. The maximum concentration of potassium that can be given through a peripheral vein is 40 mmol/litre. The maximum infusion rate of potassium is 0.5 mmol/kg/hour. The recommended concentration is 20 mmol/litre.

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Note: The injectable form of KCl usually contains 1.5 grams (i.e. 20 mmol of potassium in 10 mL), and can be given orally. The daily potassium requirement is 2.5–3.5 mmol/kg.

Zinc supplements
If dehydration is due to acute gastroenteritis give zinc supplements. Zinc supplements given during an episode of diarrhoea reduce the duration and severity of the episode, and lower the incidence of diarrhoea in the following 2–3 months. For these reasons, all patients with diarrhoea should be given zinc supplements as soon as possible after the diarrhoea has started. Give 10 mg/kg for infants less than 6 months old and 20 mg/kg for older infants for 14 days.

Further tests might include abdominal X-ray or ultra- sound scanning, if there is concern about a distended abdomen.

A surgical opinion is needed if there is bile-stained vomiting or abdominal signs.

Fluid requirements
WHO Plans A, B and C for gastroenteritis in children (*see* Textbook) include estimates of total fluid requirements, and assume that most infants will be drinking by 4 hours into treatment and thus able to 'self-regulate'. For patients for whom this is not the case, the following guidelines can be used.

Estimating fluid requirements
The amount of fluid needed in a 24-hour period must be calculated. It is the sum of:

Estimated fluid deficit + Maintenance requirements + Ongoing losses.

Deficit
If an accurate recent pre-illness weight is available, subtract the current weight to estimate lost fluid (1 kg = 1 litre of fluid). For example, an infant who weighed 9.2 kg is seen with diarrhoea and a weight of 8.3 kg. The estimated fluid loss in this case is (9.2 – 8.3) kg = 0.9 kg = 900 mL deficit, i.e. 10% dehydrated.
If no recent reliable weight is available:
1 Estimate the degree of dehydration.
2 Use the formula: percentage dehydration × weight (kg) × 10 = deficit (in mL).

For example, an infant whose weight is estimated to be 10 kg is 10% dehydrated. The estimated fluid loss in this case is 10 × 10 × 10 = 1000 mL (i.e. 40 mL/hour if replaced over 24 hours).

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Maintenanc*e*

The estimated maintenance fluid requirements based on body weight for an infant are shown in Table 28.1.

TABLE 28.1 Fluid requirements per day

Body weight	Volume of fluid needed per day	Volume of fluid needed per hour
First 10 kg of body weight	100 mL/kg	4 mL/kg

Ongoing losses

Estimated ongoing fluid losses are shown in Table 28.2

TABLE 28.2 Estimates of ongoing fluid losses in gastroenteritis

For each diarrhoea stool	give 50–100 mL	
For each vomit	2 mL/kg oral rehydration solution: give small frequent volumes via a spoon, syringe or cup	
For nasogastric tube aspirates	Replace volume for volume with either oral rehydration solution or Hartmann's or Ringer- lactate solution with added 5% or 10% glucose or normal saline with 5% or 10% glucose and 5 mmol/litre of potassium chloride	

Over-hydration

Signs of over-hydration, especially if there is cardiac failure (e.g. in severe malnutrition) are as follows:
tachycardia, increased respiratory rate, oedematous (puffy) eyelids, crepitations at lung bases, enlarged liver and raised jugular venous pressure (JVP) and pulmonary oedema on chest X-ray.

Management
- Stop giving IV fluids or oral rehydration solution, but give breast milk or plain water and food.
- Do not give a diuretic unless there is pulmonary oedema (crepitations in lungs), in which case give furosemide 1 mg/kg IV.

Reassess:

- ABC.

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- The state of intravascular rehydration.
- Plasma electrolytes (if possible).
- Urine output and urine electrolytes.
- Glucose levels.

Reduce fluid intake and continue to monitor, responding to changes in the infant's condition as described above.

Gastrointestinal disorders in the neonate and infant

Oesophageal atresia should always be considered in the infant with a history of polyhydramnios or excessive frothy salivation following delivery. Surgery is much more likely to be successful if it can be performed before aspiration pneumonia develops. Pass a large-bore catheter as far down the oesophagus as possible and aspirate frequently. If an X-ray shows that the tube has stopped at the level of the heart and has not entered the stomach, the diagnosis is made. Such an infant needs urgent referral for surgery, with steps taken to suck the blind upper oesophageal pouch clear of all accumulating secretions at least every 15 minutes before and during transfer. Site an IV line and ensure that the infant does not become hypoglycaemic.

Bowel obstruction

Severe vomiting, often associated with abdominal distension, in the first few days of life suggests the existence of a problem that requires referral for surgical review. This is particularly true if the vomit is green (bile stained), as this is suggestive of **duodenal atresia or bowel obstruction** requiring urgent surgical intervention.

If severe vomiting develops in a infant who has passed changing stool, the diagnosis of **volvulus, pyloric stenosis or intussusception** must be considered. Duodenal atresia is more common in infants with Down's syndrome. A paediatric surgical opinion should be sought.

Necrotising enterocolitis (NEC)

This is a serious condition with a mortality of approximately 20–40%. Preterm or small-for-dates infants are at increased risk of developing this condition. Prevalence is inversely related to birth weight and gestation. NEC is more common in ill infants. Although it is more common in infants who have received feeds, about 15–20% of affected infants may never have been fed. It is much less common in infants fed exclusively on human milk. NEC may occur in epidemics due to cross-infection in the nursery.

Presenting features

The condition should be suspected in an infant who had started to accept oral feeds and then develops an ileus or becomes lethargic and starts passing a bloody stool. The problem is caused by the sudden focal invasion of bacteria into an area of ischaemic gut, and an abdominal X-ray will often show gas accumulating within the gut wall.

Common signs of NEC include the following:
- abdominal distension or tenderness
- intolerance of feeding

- bile-stained vomit or bile-stained fluid up the nasogas- tric tube
- blood in the stools.

Features of multisystem failure, such as coagulopathy, petechial haemorrhage, oliguria and haematuria, may be associated with NEC.

Investigations
A plain abdominal X-ray may show an abnormal gas pat- tern in the form of:

- free intra-peritoneal air, best seen with a left side down (lateral decubitus) X-ray, where free air may be easily seen overlying the dense hepatic tissue
- intramural gas (pneumatosis intestinalis) or gas in the portal tracts of the liver.

A complete blood count with differential cell count, blood culture and serum electrolytes should be obtained. Regular weighing, frequent blood pressure measurements, and continuous heart and respiratory rate monitoring are required.

Treatment

- Stop all enteral feeds for at least 5 days and provide IV fluids, typically 120 mL/kg/day of 10% glucose with added electrolytes. Adjust fluids as indicated based on weight change, urine output and serum electrolyte values.
- If available, place an orogastric tube on low-pressure continuous suction or leave the tube open with inter- mittent gastric aspiration (every 4 hours). The goal here is to keep the intestines decompressed. The volume of gastric fluid aspirated is usually relatively small, so replacement fluid is seldom required.
- Start parenteral broad-spectrum antibiotics (usually ampicillin and gentamicin). Because of the probable   association of Gram-negative anaerobes, also give met- ronidazole, especially if there is pneumatosis, perforation or evidence of peritonitis. Broader-spectrum antibiotics may be considered in the presence of extensive disease or poor response, or based on culture results.
- Treat any accompanying shock with Ringer-lactate or Hartmann's solution or colloid, such as 4.5% albumin, 10mL/kg over 15 minutes. Repeat if necessary.
- Measure the haemoglobin concentration daily and transfuse if it falls below 10 g/dL. If the infant is bleeding, give 1 mg vitamin K IV and fresh-frozen plasma 10mL/ kg (if available).
- The principal goal of therapy is to rest the bowel and treat any contributing or evolving bacterial infection with antibiotics. The duration of this therapy is usually

10–21 days, depending on the severity of the process. Serial abdominal X-ray studies (if available) are indicated early in this disease to monitor for pneumatosis intestinalis or perforation. Ideally, parenteral nutrition should be given at this time in place of simple 10% glucose and electrolytes. Enteral feeds (breast milk) are reintroduced slowly at

the end of antibiotic therapy (initially 20–30 mL/ kg/day), with careful monitoring for abdominal distension or other signs of obstruction.

In seriously ill infants or infants who do not improve after 48 hours a surgical opinion should be sought.

Even in hospitals with good surgical support, perforation of the bowel is not necessarily an indication for a laparotomy. The conventional surgical approach has been laparotomy with resection of the perforated and adjacent necrotic bowel. A stoma and mucus fistula may be created with later anastomosis. An alternative surgical approach is to place a peritoneal drain, with laparotomy reserved for later complications, if they develop (e.g. bowel obstruction from adhesions or bowel wall strictures). Although there is some controversy about which approach is best, studies suggest that the overall mortality may be similar with either approach.

Immediate mortality is quite high, but many cases resolve without surgical intervention (although a stricture may occasionally develop about a month later in the affected area of gut), where it is usually possible to re-intro- duce feeds after about 5 days. An infant who is sucking and showing interest in feeding is usually ready for feeding. Intestinal perforation is generally the main indication for surgical intervention, but the prognosis really depends on whether there is generalised peritonitis and whether some part of the gut has become totally dead and gangrenous.

Pyloric stenosis

This is a classical cause of gastric outlet obstruction in infants. It has a prevalence rate of about 1.5 to 4 in 1000 live births among white populations, but is less common in Africans and Asians. It is more common in males than in females, with a ratio of between 2:1 and 5:1. There appears to be an increased risk to firstborn infants with a positive family history.

Presentation

Pyloric stenosis typically presents at 2–8 weeks of age, with a peak occurrence at 3–5 weeks. The vomiting is projectile and non-bilious. Occasionally there is coffee-ground vomiting due to gastritis or oesophagitis. The infant remains hungry after vomiting, and is otherwise not ill looking or febrile. Around 2–5% of infants have jaundice associated with indirect hyperbilirubinaemia. Non-bilious projectile vomiting, visible gastric peristalsis in the left upper abdomen, and in those presenting late a hypochloraemic hypokalaemic metabolic alkalosis are the cardinal features of pyloric stenosis.

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Diagnosis

A definite diagnosis can be made in 75% of infants with pyloric stenosis by careful physical examination of the upper abdomen. **An absolute prerequisite for this is a calm and cooperative infant, a warm environment, good light and patience.** With the patient in the supine position, in the mother's left arm and sucking on the left breast, and the surgeon sitting on the left side of the patient, the left hand is used to feel the classically described 'olive' to the right of the rectus muscle, often palpated against the spinal column. Visible gastric peristalsis is often noticed.

Investigations

- Ultrasonography is the most commonly used imaging technique for diagnosis. A positive finding is a pyloric canal length of 16 mm or more and a pyloric muscle thickness of 4 mm or more. A diameter of more than 14 mm is also considered abnormal.
- Blood investigations in an advanced situation may show the typical hypochloraemic hypokalaemic metabolic alkalosis.

Management

1. It is most important to prepare the patient appropriately and adequately for anaesthesia and surgery.
2. Intravenous fluid resuscitation with 5% glucose in 0.9% saline with 20–40 mEq/litre of potassium chloride is the optimal fluid.
3. Urine output and serum electrolytes should be monitored.
4. The stomach should be aspirated before the operation.
5. Ramstedt's pyloromyotomy performed through a right upper quadrant or supraumbilical incision is curative, and is associated with a low morbidity.
6. The majority of these patients can be started on feeds about 6 hours after surgery.
7. Those who present with haematemesis from gastritis may benefit from delay of feeding for an additional 6–12 hours after surgery.
8. Vomiting in the early post-operative period is thought to be secondary to discordant gastric peristalsis or atony.

Intussusception

This is the telescoping of a portion of the intestine into the lumen of an immediately adjoining part. Typically it occurs in a well-nourished infant aged 4–12 months. The male:female ratio is 3:2, and it is more common in Caucasians.

The pathogenesis of intussusception is unclear. It usually originates in the ileum close to the ileocaecal junction and proceeds into the ascending colon. In 2–8% of cases there is a specific lead point such as a Meckel's diverticulum, polyp or duplication cyst. Adenoviral infection resulting in lymphoid hyperplasia may act as a lead point.

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Clinical presentation
The infant is suddenly disturbed by what appears to be violent abdominal pain. The pain is colicky, intermit- tent and severe. With spasms the infant draws up the knees to the abdomen, screams, becomes pale and may sweat, and vomiting occurs soon afterwards. The infant may pass a normal stool, appears to recover immediately, and may resume normal eating habits, until stricken by another bout of colicky abdominal pain. The vomiting is initially reflex, but with a delayed diagnosis becomes secondary to intestinal obstruction and is often bile-stained.

Classically, the infant passes stool that resembles redcurrant jelly. Many parents describe this as the presenting symptom, and consequently it is often treated as bacillary dysentery initially.

The triad of pain, vomiting and blood per rectum is present in only one-third of patients. One in 10 infants with intussusception will have diarrhoea before signs and symptoms attributable to intussusception become obvious. This is often a cause for delay in diagnosis.

Pallor, persistent apathy and dehydration are common signs.

Abdominal examination reveals emptiness in the right lower quadrant and a sausage-shaped mass in the right hypochondrium, extending along the line of the transverse colon. The mass is not always easy to palpate, and its absence does not rule out an intussusception.

Fever and leukocytosis are common, and tachycardia results from episodes of colic and hypovolaemia from dehydration.

Investigations
Abdominal X-ray may show a soft tissue mass across the central abdomen with dilated loops of bowel.
Ultrasonography has become the standard non- invasive diagnostic test, and is very reliable. Doughnut (target or concentric ring) and pseudo- kidney sign suggest a diagnosis of intussusception.

Management
The most important aspect of treatment is adequate resuscitation prior to intervention. This involves establishing reliable IV access, collecting blood for baseline investigations and for cross-matching, passing a nasogastric tube for decom- pression, and giving IV fluids

and analgesia. Some patients may require one or more boluses of 10–20 mL/kg of albumin or Ringer-lactate solution when first seen.

Broad-spectrum IV antibiotics such as a combination of cefuroxime (25–50 mg/kg 8-hourly, depending on the degree of infection) and metronidazole (7.5 mg/kg 8-hourly IV over 20 minutes) are started, and the urine output is monitored.

Management is initially non-surgical (i.e. with the use of air or barium enema). Sedation should be used for the procedure.

A surgeon and theatre should be ready when the radiologist attempts reduction. If perforation occurs, surgery should be performed immediately.

An absolute contraindication to rectal reduction is evidence of peritonitis, indicating the presence of a gangrenous intestine.

If hydrostatic reduction fails and if the patient is stable, a repeat reduction may be attempted. Once the intussusception reduces, the infant should be observed overnight with careful monitoring of fluid and electrolytes.

If reduction fails, the infant is taken for surgery, where by gentle manipulation (pushing and not pulling) the intussusception can be reduced. The appendix may be removed, recorded and the parents informed. If a pathological lead point is found, a resection anastomosis is performed. If the bowel is not viable, it is resected and a primary anastomosis is performed. Feeds are started the day after the operation and increased gradually.

Intravenous antibiotics should be given for at least 48 hours, and longer (for 7 days) if peritonitis is present.

The interval between the onset of symptoms and institution of treatment is of paramount importance, and mortality can be reduced if the condition is recognised and treated early.

Intestinal obstruction

This is the most common condition requiring emergency surgery in infants. Most causes result from complications of congenital anomalies or from inflammatory conditions that affect the bowel.

Causes

- Extrinsic causes: incarcerated hernia and vascular bands, intussusception, anomalies of rotation (volvulus and Ladd's bands, paraduodenal and paracaecal

her- nias), post-operative adhesions.
- Intrinsic causes: inspissation of bowel contents (meco-nium ileus, distal intestinal obstruction syndrome in patients with cystic fibrosis), roundworm obstruction.
- Peristaltic dysfunction: Hirschsprung's disease.
- Inflammatory lesions: tuberculosis, Crohn's disease.

Symptoms and signs

Patients present with cramping abdominal pain with anorexia, nausea and vomiting, which progresses to become bile-stained. Abdominal distension occurs, with the degree being directly related to the site of obstruction in the gastrointestinal tract, such that the distension is greater the more distal the obstruction.

On examination, the patient may have tachycardia and signs of dehydration. Tenderness and hyperactive bowel sounds are present on abdominal examination.

Chest and abdominal films are taken to confirm the diagnosis of obstruction and rule out the presence of free air.

Treatment

The goal of treatment is to relieve obstruction before ischaemic bowel injury occurs. Intravenous access is established and blood collected for baseline investigations, including a full blood count, urea, creatinine and electrolytes, and cross-matching.

Intravenous fluids (Ringer-lactate or Hartmann's solu- tion with 10% glucose) are started according to the guidelines of 4 mL/kg/hour for the first 10 kg

Some patients may need one or more IV boluses (10– 20 mL/kg) with Ringer-lactate or Hartmann's solution or albumin at the start of resuscitation.

A nasogastric tube is passed for decompression.

Give broad-spectrum IV antibiotics such as:
- cefuroxime 50 mg/kg 8-hourly or 12-hourly in the neonate, and metronidazole 7.5 mg/kg 8-hourly IV or
- benzylpenicillin 50 mg/kg 6-hourly plus gentamicin 7 mg/kg once daily plus metronidazole 7.5 mg/kg 8-hourly.

Once the patient is adequately resuscitated and fluid and electrolyte imbalances have been corrected, laparotomy is performed and the cause treated. Transfer to a facil- ity where paediatric surgical and anaesthetic skills are available should be undertaken if the patient's condition will tolerate this. Otherwise, or in the absence of such a facility in the country, surgery should be performed.

At all times adequate analgesia should be given (*see* Section 4).

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Hirschsprung's disease

This is characterised by an absence of ganglion cells in the affected intestine. The incidence is about 1 in 4400–7000 live births; the male:female ratio is about 4:1, and in long segment disease it approaches 1:1. The longer the segment of aganglionosis, the higher is the familial incidence.

Associated conditions

These include Down's syndrome (4–16%), Waardenburg syndrome, multiple endocrine neoplasia 2A and Von Recklinghausen's disease. A higher incidence of enterocolitis has been noted in patients with Hirschsprung's disease and Down's syndrome.

Presentation

The usual presentation is with delay of passage of meconium beyond 48 hours after birth. (Around 95% of full-term infants pass meconium within 24 hours after birth, and the remainder pass it within 48 hours.) The infant then has episodes of constipation, abdominal distension, vomiting and poor feeding, and fails to thrive. They may also present with a history of constipation with explosive diarrhoea, the latter indicating the development of enterocolitis.

Differential diagnosis

Hirschsprung's disease should be considered in the differential diagnosis of any infant who has constipation dating back to the newborn period. However, constipation related to dietary and habitual problems needs to be carefully ruled out in order to avoid unnecessary X-rays and biopsies.

Examination

On examination the infant has a distended abdomen, and after a rectal examination there is often explosive passage of flatus and faeces.

A plain X-ray of the abdomen may show dilated bowel loops with paucity of air in the location of the rectum. Barium enema may show the characteristic coning, although a simple colonic dilatation can occur in any chronic constipation.

Rectal biopsy remains the gold standard for diagnosis. It should be performed at least 2 cm above the anal valves, as the normal anus has a paucity or absence of ganglion cells at the level of the internal sphincter. Although suction rectal biopsy with acetylcholinesterase staining has become the accepted standard for diagno- sis in most centres, a full-thickness rectal biopsy under general anaesthesia is equally useful if such facilities are not available.

Treatment

Enterocolitis remains the major cause of morbidity, and has a mortality rate of around 6–30%. It manifests clinically as explosive diarrhoea, abdominal distension and fever. The pathophysiology is not fully understood. The diagnosis is made on clinical grounds, and treatment is conservative, consisting of IV fluids and rectal washouts to decompress the colon.

Surgery

The surgical treatment of Hirschsprung's disease has evolved from a three-stage procedure (initial colostomy with multiple sero-muscular biopsies, pull-through of the ganglionic colon as the second stage, and closure of colos- tomy as the third stage) through a two-stage procedure (colostomy at the transition zone initially, and pull-through as a second stage) to a one-stage procedure without a colostomy. The essential prerequisite for a primary pull- through is adequate preparation with colonic washouts.

Perforative peritonitis

The causes of perforation include amoebiasis, typhoid, tuberculosis, roundworm perforation and Hirschsprung's disease.

Management starts with an adequate history and clinical examination, followed by chest and abdominal X-rays. Adequate resuscitation should be carried out as outlined in the section on intestinal obstruction. After this a laparotomy is performed and the cause treated. Treatment includes fluid resuscitation if necessary, and antibiotics (either a third-generation cephalosporin or an aminoglycoside plus metronidazole).

Practical procedures in the infant

Practical procedures should first be explained to the parents, any risks discussed with them and their consent obtained. Procedures on infants should avoid hypothermia. Good light is essential. Analgesia should be given where necessary, and invasive procedures only performed when essential.

Preventing hypothermia in infants especially the newborn and especially small and preterm infants
Skin to Skin (Kangaroo) Mother Care (KMC)
This technique has been shown to be the most effective way of achieving the following in poorly resourced environments

- Preventing hypothermia
- Promoting breast feeding and growth
- Reducing infection risk
- Monitoring and reducing apnea and stress in baby
- Empowering the mother

Consider the needs of mother and baby in order to establish successful KMC
Hypothermia: what is a normal body temperature for a baby?
Normal
Axillary temperature measured for 3 minutes = 36.5-37.4°C
Axillary temperature measured for 1 minute = 36.0-37.0°C
Hypothermia
Mild = 36-36.4°C
Moderate = 32.0 -35.9°C
Severe = below 32.0°C
What harm does hypothermia cause?
- Reduces the amount of oxygen available to the baby's tissues
- Causes tissue acidosis
- Stops the baby's lungs producing surfactant to help breathing
- Increases the likelihood of severe infection
- Slows weight gain
All these problems are worse in the pre-term and SGA baby
Why is the newborn baby so prone to hypothermia?
- High surface area to weight
- Absence of subcutaneous fat causes skin to lose heat easily
- Not very active
- Can not shiver
- Have limited special brown fat
All these issues are more severe in the pre-term and SGA baby

What can we do to prevent hypothermia?
- Keep the delivery room and post natal ward warm (over 25°C) and draft free
- Dry the baby well and wrap in a dry cloth

Section 30 Practical procedures: KMC

- Place baby skin to skin on mother's breasts, cover them both
- Hat on baby
- Do not bathe for at least 24 hours
- Keep baby in skin to skin care for as long as practicable

Keeping the baby in skin-to-skin contact with the mother prevents hypothermia
If the baby is too warm, the breasts cool the baby down
If the baby is too cold, the breasts warm the baby up
Preventing hypothermia improves growth, reduces infection risk and promotes lung maturation

Early, demand led breast feeding
KMC encourages early breast feeding (best within an hour of birth)
KMC facilitates successful and prolonged breast feeding
Therefore, growth is optimised and infant infection lessened

Reducing infection in the small baby
Being in skin-to skin contact with the mother as long as possible means that the baby can be colonised by the mother's bacteria (to which the baby has placentally transferred antibodies) instead of the dangerous hospital bacteria
Infant infection is lessened significantly

Apnea and stress in small babies
Low birth weight babies are prone to apneic episodes. KMC can reduce these as the mother's breathing and heart beat gives gentle stimulation.
Mother can be taught to recognise apnea and other abnormal breathing, stimulate the baby and call for help if needed
Studies show that babies in KMC are less stressed than babies in cots and incubators

KMC: benefits for mothers
Mothers report feeling empowered, more confident with their baby and more fulfilled with KMC
Fathers performing KMC felt more confident and content too!

It is important to note that KMC is beneficial to all babies, especially preterm and low birth weight infants but also including full term healthy babies,

What is needed for Kangaroo Mother Care?
- Mother who understands importance and is willing to learn
- Staff with skills and knowledge
- Supportive environment in hospital and at home

Mothers' needs
- Chair where mother can sit comfortably
- Beds arranged so mother is propped up when sleeping (chest 15 degrees up)
- Personal and clothing washing facilities
- Meal facilities
- Education on care of baby

- Recreational/income generating activities

Care of baby
- Position so that airway is open
- Show mother how to monitor breathing
- Show mother how to stimulate if apneic
- Monitor temperature: if low/high, consider infection

Teach mother about danger signs

What happens if the baby needs treatments?
- Naso or orogastric feeds
- Intravenous antibiotics
- Oxygen by nasal prongs
- Nasal CPAP

All can be given during KMC
Only phototherapy needs baby's skin exposed

When mother needs a break or she is ill
- Clothe the baby and put in a warmed cot with blankets
- Better still, other family members can do KMC too

KMC at home
Vital to continue KMC after discharge at home for several months to:
- Prevent hypothermia
- Improve weight gain
- Reduce infections

All family members can help, including older children

Technique of KMC
i)   *Moby wrap*
ii)   *Plain cotton binders*
iii)   *Kalafong wrap (The Thari) (see Figure 30.1 for how to do this)*
a.   This is a one-piece wrap, 3 metres long, with a central panel and 4 long straps
b.   With the lower edge of the central panel in the middle of the mother's lower chest, wrap the bottom straps around the back.
c.   Cross the bottom straps at the back and bring the straps to the front just below the breasts.
d.   Tie a square knot in front, being sure that the knot is on top of the centerpiece of fabric, which will provide a secure basis for the infant's legs and bottom. Make sure that the straps are tied just below the breasts and not around the waist because the waist level may position a small infant too low on the mother's chest, which may compromise the infant's airway.
e.   Wrap the top straps around the mother's back
f.   Cross the top straps at the back and bring them over the shoulders toward the front. Let the top straps dangle in front, leaving the centrepiece top edge loose so

that it creates a pouch for the infant.

g.   Position the infant prone inside the pouch created by the centerpiece. The infant's legs and arms should be flexed with the hands beside the infant's head.

h.   Gently turn the infant's head to the side if necessary. Be sure that the top edge of the centerpiece is at the midline level of the infant's ear.

i.   Pull the dangling top straps further forward. This action tightens the centre piece around the infant, securing the infant up against the chest. Tie the top straps to the bottom straps. Each strap is attached to the bottom strap with its own square knot.

j.   The wrap should be tied firmly and securely to enable the mother to release her hold on the infant so her arms are free while the infant remains safely contained.

k.   It is important that the upper edge of the centre piece pouch secures the infant's head at the level of the infant's ear, ensuring that a safe airway is maintained.

l.   The lower edge of the centrepiece can now be tucked into a skirt and blouses and jackets are easily worn over the wrap.

Choices of material from which to make the Thari

Polyester Cotton: It works well and is cheap. The material tears easily and it is not necessary to cut the material with a pair of scissors. Unfortunately, poly cottons are usually 110 cm wide and only 2 wraps can be made from 3 metres of material. If one can get cheap cotton material that is 150 cm wide, one is able to get 3 wraps from 3 metres of material.

Cotton knit (t-shirt material): T-shirt material stretches a lot. It would be better to use a material that contains lycra, but lycra is very expensive.

A thin Denim material also works well, it lasts very long especially if you use it over and over in the ward, but unfortunately denim is quite expensive.

Wraps can be made from old sheets, if funds are not available to buy new material.
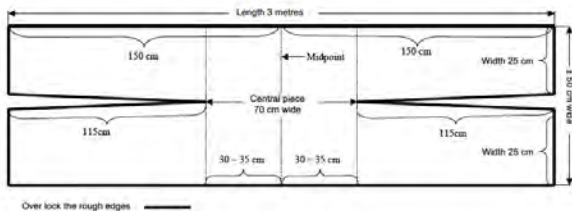
Figure 4. The KMC wrap.

Figure 5. Wrap bottom straps around back.

Figure 6. Cross bottom straps and bring to front.

Figure 7. Secure with square knot just below the breasts.

Figure 8. Wrap top straps around back.

Figure 9. Cross straps and bring over shoulders.

Figure 10. Straps crossed in back.

Figure 11. Position infant.

Figure 12. Tie top strap to bottom strap using square knot.

Figure 13. Tie other top strap to bottom strap.

Figure 14. Infant secure in KMC wrap.

Figure 15. Center piece properly positioned beneath infant's ear.

Figure 30.1 Applying the Kelafong KMC wrap

Teaching the mother how to assess the baby's breathing

i. The mother should be constantly aware of the baby's breathing by *looking* at the baby for respiratory or nasal movement, *listening* for breath sounds and *feeling* for exhaled air. The mother should be helped to recognise rapid breathing and chest wall recession indicating increased breathing efforts.

ii. If she cannot detect breathing or is not sure, she should call for help (either from a nurse or from another mother or relative to get a nurse) and stimulate the baby usually by gently pinching the ear.

Helping the mother to breast feed the infant while still in the wrap

Section 30 Practical procedures: KMC

Small infants should have a combination of demand feeding and scheduled feeding. While demand feeding is preferable as it is infant led, some infants may be too drowsy to demand and will therefore take insufficient milk for growth. If a baby is feeding less often than 2 hourly, he/she should be woken for a feed. There will be further discussion on this topic in the nutrition and growth lecture and breast feeding workshop.

The advantage of the baby remaining in the wrap while breast feeding is that he/she is still close to mother with all the sensory input to keep him calm and to encourage breast feeding. Also there is no period of exposure risking hypothermia.

i.   The wrap must be loosened and the baby's position moved so that he is able to comfortably access the breast in the usual nursing position.
ii.   An additional blanket may be needed if there are any exposed parts of the naked baby or the mother needs privacy.
iii.   Both breasts may be used, or if the baby is feeding frequently then only one breast per feed may be used.
iv.   Once the baby has finished feeding, he should be replaced in the upright open airway position and firmly secured again.

Enabling KMC to continue when mother is asleep
It is relatively easy to continue KMC when the mother is ambulant, seated or lying down but awake. She may be concerned about continuing KMC while asleep. The position for sleeping KMC is with the mother's upper body elevated at up to 30^0 to the horizontal by means of pillows.

Section 31 Practical procedures: restraint and analgesia

Restraining infants for procedures

Restraint is important both for the infant and for the clinician who is undertaking the procedure. Clearly, the procedure will be undertaken more quickly, safely and accurately if the infant is kept still. If facilities do not allow or if the procedure is unlikely to require repetition, physical restraint can be used. Ideally a parent or relative can actually hold the infant.

Analgesia and sedation for procedures

Some procedures have to be undertaken immediately, to save life. Clearly, there is no time to use analgesia in these circumstances, nor indeed much need to do so, as infants who are in such severe collapse will have significantly depressed conscious levels. Where there is consciousness, analgesia and/or sedation is a top priority.(For details on pain assessment and analgesia, see Section 4
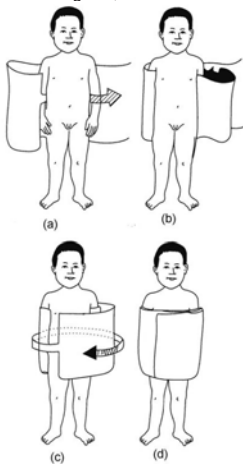
FIGURE 31.1 Wrapping an infant so that they can be held securely for a procedure. (a) and (b). One end of a folded sheet should be pulled through under the arms on both sides. (c) and (d). The other end is then brought across the front and wrapped round the infant.

For some procedures (e.g. chest tube insertion, dressing of burns), analgesia with a powerful drug such as ketamine should be considered, with a skilled healthcare worker (usually an anaesthetist) present and able to treat any adverse reactions immediately.

Section 31 Practical procedures: restraint and analgesia

For planned intubation, anaesthesia is induced first. For some rarely used procedures such as defibrillation for cardiac arrest caused by a shockable rhythm (see Section 3) there is neither time nor need for sedation, as the patient is unconscious.

If ketamine is being used, give 2–4 mg/kg IM. This takes 5–10 minutes to act and the effects last for about 20 minutes. Ketamine can also be given slowly IV in this situation, 250–500 microgram/kg IV, and repeated as required to control pain. **An anaesthetist or other expert in airway control must be present when ketamine is used.**

When giving any analgesia, manage the infant's airway, beware of respiratory depression and monitor oxygen saturation with a pulse oximeter (if available). Ensure that you have a resuscitation bag and mask available (and oxygen).
Always use local analgesia if appropriate (see Section 4).

Tracheal intubation

Aims
These are as follows:
- to secure the airway
- to protect the airway
- to facilitate prolonged and intra-operative ventilation
- for tracheo-bronchial toilet
- in the application of high airway pressures and positive end-expiratory pressure (PEEP) during cardiopulmonary resuscitation to improve ventilation and allow uninterrupted chest compressions.

The correctly sized tube is one that passes easily through the glottis and subglottic area with a small air leak detectable at 20 cmH$_2$O (sustained gentle positive pressure).

Size of tube
The correct size of tube is:
- one that can just fit into the nostril or
- in preterm neonates, 2.5–3.5 mm internal diameter or
- in full-term neonates, 3.0–4.0 mm internal diameter or
- in infants after the neonatal period, 3.5–4.5 mm internal diameter

Aids to intubation
Laryngoscope: blade (straight for neonates and infants because of long, floppy epiglottis).
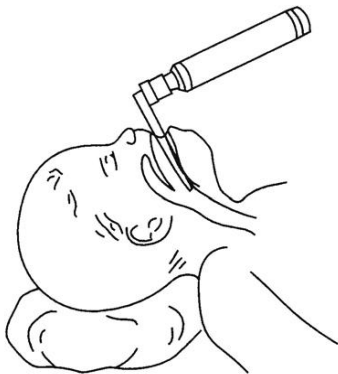
FIGURE 32.1 Straight-blade laryngoscope, suitable for infants.

Section 32 Practical procedures: tracheal intubation

Magill's forceps.
Introducer (not further than the end of the tube itself).
Syringe (cuffed tube).
Gum elastic bougie (over which the tube can pass).
Cricoid pressure (can aid visualisation of larynx).
Suction apparatus (this must be available).

Predicting difficulty
- Difficulty in opening mouth.
- Reduced neck mobility.
- Laryngeal/pharyngeal lesions.
- Congenital: Pierre-Robin syndrome, mucopolysaccharidoses.
- Acquired: burns, trauma.

If on viewing the infant's face from the side, the chin is unusually small (micrognathia), the intubation will be difficult, and senior help is required (but see below).

Complications
- Displacement: oesophageal, endo-bronchial, out of larynx!
- Obstruction: kinking, secretions.
- Trauma: lips to larynx.
- Hypertensive response.
- Spasm: laryngeal, pharyngeal.
- Aspiration: gastric contents.

Procedure
1. Prepare and check the equipment.
2. Choose an appropriate tube size, with one size above and one size below it available.
3. Get the tape ready to fix the tube.
4. Suction must be available.
5. Induce anaesthesia and give a muscle relaxant unless the patient is completely obtunded. Do not attempt the procedure in a semi-conscious infant.
6. Position the infant.
7. Neonates and infants: a neutral position (large occiput).
8. Oxygenate using a face mask and reservoir (if patient is breathing) or bag and mask ventilation to provide high flow oxygen.
9. Introduce the laryngoscope into the right side of the mouth.
10. Sweep the tongue to the left.
11. Advance the blade until the epiglottis is seen.
12. Straight blade: advance the blade beneath the epi- glottis, into the oesophagus. Pull back, and the glottis will 'flop' into view.
13. Recognise the glottis.
14. Insert the endotracheal tube gently through the vocal cords.
15. Stop at a predetermined length.
16. Confirm the correct placement.
17. The chest moves up and down with ventilation.

18. Listen to breath sounds in the axillae and anterior chest wall.
19. Confirm that there are no breath sounds in the stomach.
20. Oxygen saturations do not go down.
21. Carbon dioxide is measured from expired gases (ideal if available).

Secure the tube.
Secure with tape around the tracheal tube and on to the patient's face (see below).

Nasal intubation.
Although oral intubation is quicker and more reliable in an emergency, for prolonged ventilation nasal intubation is preferable, if a skilled operator is available, as the tracheal tube is more securely fixed. The technique is similar, but with the additional use of the Magill's forceps to grasp and guide the tracheal tube as it emerges into the posterior pharynx downward into the trachea through the vocal cords.

Fixation of endotracheal tubes
1. Two people should be available to do this, one of whom should hold the tube at all times.
2. Cut two strips of sticky zinc oxide tape (see below); they should reach from just in front of the ear across the cheek and above the upper lip to the opposite ear.

FIGURE 32.2 Tape for tracheal tube fixation.

3. If available, apply some benzoin tincture to the cheeks, above the upper lip and under the chin, which will make the tape stick well.
4. Make sure that the endotracheal tube is clean and that no old tape is left on it.
5. Start with the broad end of the tape, and stick this on to the cheek. Then wrap one of the thinner ends carefully around the tube. It is useful if it is still possible to see the endotracheal tube marking at the lips.
6. Tape the other half across the philtrum to the cheek.
7. The second tape starts on the other cheek, and the thinner half is stuck across the chin, while the other half is also wrapped around the tube (see below).
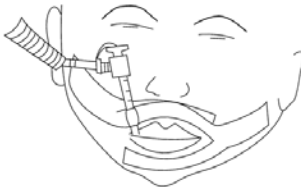
FIGURE 32.3 Taped tracheal tube

Cricothyroidotomy

Cricothyroidotomy is indicated if a patent airway cannot be achieved by other means. It must be performed promptly and decisively when necessary.
Call a surgeon and an anaesthetist (if available).
The relevant anatomy is shown in Figure 33.1.

FIGURE 33.1 Anatomy of the neck.

Surgical cricothyroidotomy
1. Place the patient in a supine position.
2. If there is no risk of neck injury, consider extending the neck to improve access. Otherwise, maintain a neutral alignment.
3. Identify the cricothyroid membrane in the following man- ner. Place your finger over the most prominent part of the thyroid cartilage (the Adam's apple). Move the finger downwards (i.e. towards the chest), keeping strictly in the midline. The first dip felt is the area of cricothyroid membrane.
4. Prepare the skin and, if the patient is conscious, infiltrate with local anaesthetic.
5. Place the index and middle fingers of your left hand on each side of the midline of the neck to stabilise the cri- cothyroid membrane, and to protect the lateral vascular structures from injury.
6. Make a small vertical incision in the skin, and press the lateral edges of the incision outwards, to minimise bleeding.
7. Make a transverse incision through the cricothyroid membrane, being careful not to damage the cricoid cartilage.
8. Insert a tracheal spreader, or use the handle of the scalpel by inserting it through the incision and twisting it through 90 degrees to open the airway.
9. Insert an appropriately sized endotracheal or tracheostomy tube. It is advisable to use a slightly smaller size than would have been used for an oral or nasal tube (e.g. size 6.0 mm internal diameter for age 12–16 years).
10. Ventilate the patient and check that this is effective. 11 Secure the tube to prevent dislodgement.

Complications of cricothyroidotomy
These include the following:
- asphyxia

Section 33 Practical procedures: cricothyroidotomy

- aspiration of blood or secretions
- haemorrhage or haematoma
- creation of a false passage into the tissues
- surgical emphysema (subcutaneous or mediastinal)
- pulmonary barotrauma
- subglottic oedema or stenosis
- oesophageal perforation
- cellulitis.

Section 34 Needle thoracocentesis, chest drain and chest tap

Emergency needle thoracocentesis

This procedure is used for the rapidly deteriorating patient who has a life-threatening tension pneumothorax. If this technique is used in a patient who does not have a tension pneumothorax, there is a 10–20% risk of producing a pneumothorax or causing damage to the lung, or both. In such cases, immediate insertion of a chest drain is mandatory. Patients who have undergone this procedure should ideally have a chest radiograph, and may require chest drainage if they subsequently need assisted ventilation.

Minimum equipment
Swabs for disinfecting the skin.
Large over-the-needle IV cannula (16-gauge, but 20- to 22-gauge in preterm infants).
20-mL syringe.

Procedure (see Figure 34.1)
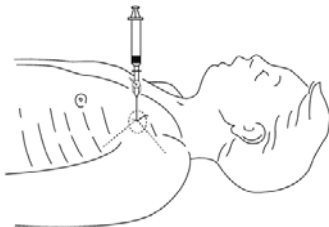
FIGURE 34.1 Position for inserting over-the-needle cannula for thoracocentesis.

1. Identify the second intercostal space in the mid-clavicular line on the side of the pneumothorax (the opposite side to the direction of tracheal deviation and the same side as the hyper-resonance).
2. Swab the chest wall with surgical preparation solution or an alcohol swab.
3. Attach the syringe to the over-the-needle IV cannula, ideally via a three-way tap.
4. Insert the cannula vertically into the chest wall, just above the rib below to avoid blood vessels, aspirating all the time.
5. If air is aspirated, remove the needle, leaving the plastic cannula in place.
6. Tape the cannula in place and proceed to chest drain insertion as soon as possible.

Complications
These include the following:
- local cellulitis
- local haematoma

- pleural infection
- empyema
- pneumothorax.

Insertion of a chest drainage tube

In a trauma emergency that requires a chest drainage tube, fluid resuscitation through at least one large calibre IV cannula, and monitoring of vital signs should be ongoing. Usually the patient will be receiving oxygen through a face mask with a reservoir. Chest drain placement should be performed using the open technique described here, as this minimises lung damage. In general, the largest size of drain that will pass between the ribs should be used.

Minimum equipment
- Skin disinfectant and surgical drapes.
- Scalpel with fine straight blade.
- Blunt forceps.
- Artery forceps.
- Large clamps × 2.
- Suture.
- Local anaesthetic if the infant is conscious.
- Scissors.
- Chest drain tube.
- Underwater seal or Heimlich flutter valve.
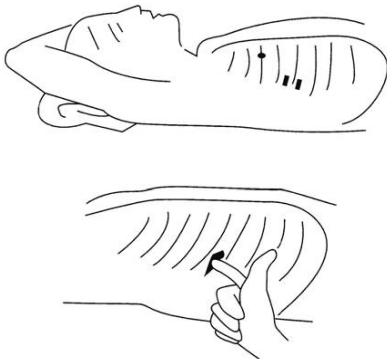
FIGURE 34.2 Sites for chest drain: 4th or 5th intercostal space in the anterior or mid-axillary line.

Section 34 Needle thoracocentesis, chest drain and chest tap

Procedure
1. Consider using analgesia.
2. Wash your hands and arms to the elbows, and wear a mask, surgical hat (bonnet), sterile gown and sterile surgical gloves.
3. Prepare the underwater seal with an assistant and take the sterile end of the tube, ready to connect to the chest tube once inserted. The 'seal' end should be covered by no more than 1–2 cmH$_2$O.
4. Decide on the insertion site (usually the fourth or fifth intercostal space in the anterior or mid-axillary line) on the side with the pneumothorax (see Figure 34.2).
5. Swab the chest wall with surgical preparation or an alcohol swab.
6. Use local anaesthetic if the infant is conscious. Morphine (50 micrograms/kg IV over 10 minutes) should also be given if the infant is conscious, but in the preterm infant who is not ventilated this may precipitate apnoea. Facilities to provide bag-and-mask ventilation and/or intubation should be immediately available, together with staff trained in their use.
7. Make a 1- to 2-cm skin incision along the line of the intercostal space, immediately above the rib below to avoid damage to the neurovascular bundle which lies under the inferior edge of each rib.
8. Using artery forceps, bluntly dissect through the subcutaneous tissues just over the top of the rib below, and puncture the parietal pleura with the tip of the forceps.
9. Clear the path into the pleura with artery forceps.
10. Advance the chest drain tube (use the largest size that can comfortably pass between the ribs) into the pleural space without the trocar in place, but using the artery forceps to help to guide it into the pleural cavity if necessary. Pass about 2-3 cm and then connect to the underwater seal. Ideally advance the chest drain tube into the pleural space during expiration.
11. Ensure that the tube is in the pleural space by looking for fogging of the tube during expiration.
12. Ensure that all of the drainage holes of the chest drain tube are inside the chest.
13. Connect the chest drain tube to an underwater seal. Check that the tube is in the right place by observing intermittent bubbling of the water in the drainage bottle.
14. Secure the tube using a suture passed through the skin at the incision site (after ensuring that adequate local anaesthetic has been administered) and tied around the tube.
15. Cover the puncture site in the chest wall with a sterile dressing, and tape the chest tube to the chest wall.
16. Obtain a chest radiograph if at all possible.

If the chest drainage tube is satisfactorily positioned and working, occasional bubbles will pass through the underwater seal. The water level in the tube may also rise and fall slightly with the respiratory cycle.

Complications of chest drainage tube insertion
- Dislodgement of the chest drain tube from the chest wall or disconnection from the drainage bag.
- Drainage bag elevated above the level of the chest, and fluid flowing into the chest cavity, unless there is a one-way valve system.

- Chest drain tube kinking or blocking with blood clot.
- Damage to the intercostal nerve, artery or vein. This might convert a pneumothorax to a haemopneumothorax, or result in intercostal neuritis or neuralgia.
- Damage to the internal thoracic artery if the puncture is too medial, resulting in haemopneumothorax.
- Incorrect tube position, inside or outside the chest cavity.
- Introduction of pleural infection (e.g. thoracic empyema).
- Laceration or puncture of intra-thoracic or abdominal organs. This can be prevented by using the finger technique before inserting the chest tube.
- Leaking drainage bag.
- Local cellulitis.
- Local haematoma.
- Mediastinal emphysema.
- Persistent pneumothorax from a large primary defect; a second chest tube may be required.
- Subcutaneous emphysema (usually at the tube insertion site).

Tapping the chest for diagnostic tests in pleural effusions or empyema

Diagnostic procedure
1. Consider giving the infant analgesia or light anaesthesia with ketamine.
2. Wash your hands and put on sterile gloves.
3. Clean the skin over the chest with an antiseptic solution (e.g. 70% alcohol).
4. Select a point in the mid-axillary line (at the side of the chest) just below the level of the nipple (fifth intercostal space; see Figure 34.2).
5. Inject about 1 mL of 1% lignocaine into the skin and subcutaneous tissue at this point.
6. Insert a needle or needle-over-catheter through the skin and pleura, and aspirate to confirm the presence of pleural fluid. Withdraw a sample for microscopy and other tests and place it in a container.
7. If the fluid is clear (straw-coloured or brownish), pull out
8. the needle or catheter after withdrawing enough fluid to relieve distress, and put a dressing over the puncture site. Consider a differential diagnosis of tuberculosis
9. If the fluid is thin pus or cloudy (like milk), leave the catheter in place so that you can draw out more pus several times a day. Make sure that you seal the end of the catheter so that no air can get in.
10. If the fluid is thick pus which cannot pass easily through
11. the needle or catheter, insert a chest tube as described above.

Continuous positive airway pressure (CPAP)

CPAP has several effects on the airway and lungs of the preterm and full-term infant. These include prevention of alveolar collapse, increased functional residual capacity (FRC), and splinting of the airway. It is therefore of most value when used early in the course of respiratory disease (i.e. before too much alveolar collapse has taken place). *Several units around the world use it successfully as first-line ventilatory support in even the smallest infants (< 750 grams birth weight).*

Indications for CPAP
These include the following:
- signs of significant respiratory distress (tachypnoea, recession, grunting, nasal flare)
- diseases with low FRC (respiratory distress syndrome, transient tachypnoea of the newborn, pulmonary oedema)
- meconium aspiration syndrome
- apnoea and bradycardia of prematurity
- tracheomalacia.

Requirements
- Low-resistance delivery system.
- Large-bore tubing.
- Short wide connection to patient.
- Consistent and reliable pressure generation.
- Appropriate snug-fitting nasal cannulae.
- Well-positioned and secured nasal cannulae.
- Prevention of leaks, mainly via the mouth, with a chinstrap.
- Optimally maintained airway.
- Ideally warmed humidified gas.
- Prevention of neck flexion or over-extension with a neck roll.
- Regular suction to remove secretions.
- Meticulous and consistent technique.

Monitoring
- Continuous heart and respiratory rate monitoring.
- Continuous pulse oximetry, ideally pre-ductal.
- Blood gas measurements in a well resourced unit. These need not be done regularly in the stable baby with low oxygen needs unless they are required in order to assess the degree of metabolic acidosis, but in those with high oxygen requirements (FiO2 > 40%) or in the unstable baby they should be checked regularly via an arterial line.

Complications
- Nasal septum erosion/necrosis: this is a result of ill-fitting nasal cannulae, and can be avoided by the fitting of snug, but not tight, cannulae (blanching of the overlying skin suggests that the cannulae are too large) which are held firmly in place to prevent rubbing as the infant moves.

- Pneumothorax: all methods of artificial ventilation are associated with this problem. However, the more effective the CPAP is the less the work of breathing and therefore the lower the risk of pneumothoraces should be. Any pneumothorax that does occur should be drained appropriately. It is inappropriate to discontinue the CPAP.
- Gastric distension from swallowed air: this is important and is easily overcome by the venting of any such air via an open orogastric tube.

Insertion and securing of nasal cannulae and administration of CPAP
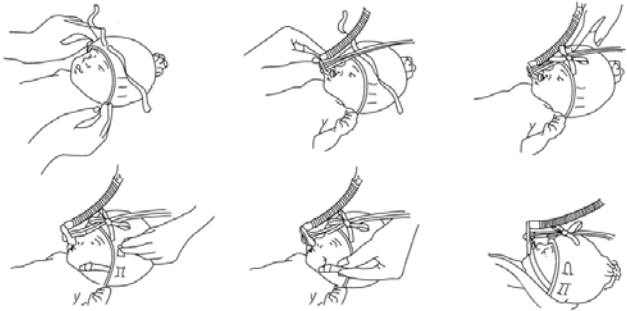
FIGURE 35.1 Securing nasal cannulae for giving continuous positive airway pressure (CPAP) in a baby. A special bonnet is used from which tapes hold the pipe carrying the air/oxygen mixture to the nasal cannulae to the forehead and a separate tape above the mouth to ensure the cannulae do not come out of the nasal passages..
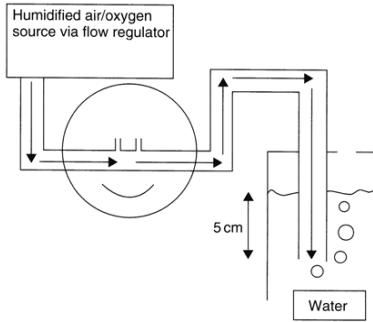
FIGURE 35.2 Simplified diagram of Hudson continuous positive airway pressure (CPAP). The gas flow is adjusted until a continuous trail of bubbles starts to appear in the water bottle, which is at the same height as the baby. This generates a CPAP of +5 cmH2O.

Circulatory procedures

Access to and support for the circulation is vital in emergency care, to draw blood samples for diagnosis and monitoring, to infuse fluid to restore circulating volume and improve perfusion, to transfuse blood and to give treatment drugs.

Also included are circulatory support procedures such as defibrillation and pericardiocentesis, and techniques for other non-parenteral routes of drug administration, includ ing intramuscular (IM), subcutaneous (SC) and intradermal (ID) injection

Minimising error in drug and fluid administration: giving injections

General points on safety
1. Drug vials once reconstituted do not contain preservatives or antiseptic. Therefore, multiple sampling from them is potentially hazardous.
2. For infants, dilute drugs to ensure that volumes can be accurately measured. For example, do not use doses of less than 0.1 mL for a 1-mL syringe.
3. Serious errors can occur if the dead space in the hub of the syringe is overlooked during dilution. For example, if the active drug is drawn into a 1-mL syringe up to the 0.1-mL mark, the syringe will contain between 0.19 and 0.23 mL. If the syringe is then filled with diluent to 1 mL, the syringe will contain approximately twice as much drug as was intended. Dilution must involve first half filling the syringe with diluent and then adding active drug by using the distance between two graduations on the syringe. Mix the two by moving the plunger, and then finally add further diluent to the total planned volume of active drug and diluent. For dilutions of more than 10-fold, use a small syringe to inject the active drug, connected by a sterile three-way tap to a larger syringe. Then add diluent to the large syringe to obtain the desired volume.
4. Many drugs are equally effective whether given orally or parenterally. Oral administration is safer and less expensive. The following antibiotics are as effective given orally as given IV in a baby who is taking feeds: amoxicillin, ampicillin, chloramphenicol, ciprofloxacin, co-trimoxazole, erythromycin, flucloxacillin, fluconazole, isoniazid, metronidazole, pyrimethamine, rifampicin, sodium fusidate, and trimethoprim.
5. If a drug is given down an orogastric or nasogastric tube, a proportion of the drug will remain in the tube unless it is flushed through.
6. Rectally administered drugs are less reliably absorbed than drugs given orally. Liquid formulations are better than suppositories in infants.
7. When giving IV drugs, do so slowly in all cases. After it has been injected into the line (ideally through a three- way tap), the normal IV infusion rate of the fluid going into the cannula can be used to drive the drug slowly into the patient.
8. If there is no background infusion, give sufficient follow-up (flush) of fluid (0.9% saline, sterile water or 5% glucose) to ensure that the drug does not remain in the cannula or T-piece. Give the flush over 2 minutes to avoid a sudden surge of drug (remember the hub).

9. If the IV drug needs to be given rapidly do this by administering a 2-mL bolus of 0.9% saline via a second syringe, not by temporarily increasing the infusion rate (sometimes the temporary increase becomes prolonged and dangerous).
10. Do not mix incompatible fluids IV.
11. For IV drug infusions (using a syringe/infusion pump: if available) given in addition to background IV infusions:
12. Adjust the total 24-hour IV fluid intake.
13. Never allow a surge of a vasoactive drug such as dopamine or epinephrine.
14. Never put more drug or background IV into the syringe or burette than is needed over a defined period of time.
15. Check and chart the rate of infusion, and confirm this by examining the amount left every hour.
16. Intramuscular injections need special precautions:

- IM injections are unsafe in shock, as they will be poorly absorbed from poorly perfused muscle tissue initially, and then (especially, for example, with opiates) a high dose may be released once recovery of the circulation occurs.
- To avoid nerve damage, only the anterior aspect of the quadriceps muscle in the thigh is safe in a small wasted infant under 1 year of age.
- Alternate between the legs if multiple injections are required.
- Do not give IM injections if a severe bleeding tendency is present.
- It is essential to draw back the plunger to ensure that the needle is not in a vein before injecting potentially dangerous drugs IM (e.g. adrenaline or lidocaine).

Care of intravascular lines
- Placement of central venous lines: check with a lateral X-ray that the line is placed well into a major vein, and if near the heart with the catheter tip ideally in the superior vena cava at the entrance to the right atrium.
- Placement of an umbilical arterial line should either be above the diaphragm in the thoracic aorta, or below the two renal arteries (at L4) to minimise the risk of renal or mesenteric artery thrombosis.
- All arterial lines can result in life-threatening haemorrhage or occlusion leading to ischaemia. Procedures to ensure that these complications do not occur should be in place.
- Never give a drug into an IV cannula that has started to tissue. Some drugs (e.g. those containing calcium) can cause severe scarring. Inspect the cannula tip site before and while injecting any drug IV.
- Local infection can become systemic, especially in neonates or in the immunosuppressed.
- Always remove the cannula if there is erythema in tissue around it and if lymphangitis is seen. If lymphangitis is present always take a blood culture from a separate vein and start IV or IM antibiotics.
- Always place cannulae aseptically and keep the site clean.
- There is no evidence that frequent changes of cannula site or infusion kit are of benefit. However, it is a good idea to change the giving set after blood transfusion or if a line of blood has entered the infusion tubing from the vein and clotted there, as this can act as a site for bacterial colonisation. Otherwise change the lines every 3 or 4 days.

Section 36 Circulatory procedures: safety of drugs and infusions

- Air embolism: if air reaches the heart, unlike blood it will stay there, especially if the patient is lying flat. Unless it is immediately aspirated, air in the heart can block the circulation.
- Umbilical venous and other central venous lines are particularly dangerous. There must be a tap or syringe on the catheter at all times, especially dur- ing insertion.
- An alternative source of air embolus is through the giving set, especially when pumps are being used.
- Blood loss.
    - In neonates this can occur from the umbilical stump.
    - From central venous or arterial lines, it can rapidly be fatal, and therefore all connections must be Luer locked and the connections to the cannula and its entry must be observable at all times.
    - Ideally, arterial lines should be connected to a pressure transducer and alarm.

Use of intravenous/intra-arterial (IV/IA) access
- When sampling from an IV/IA line, clear the dead space first (by three times its volume).
- Blood glucose levels cannot be accurately meaured from any line through which a glucose solution is infused, even if many times the dead space has been cleared.
- For blood culture, always use a separate fresh venous 'needle stab' sample.
- Never add anything to a line carrying total parenteral nutrition (TPN).
- Certain infusions, such as glucose > 10%, adrenaline and dopamine, are better given through a central vein. In an emergency, dopamine and adrenaline infusions can be given through a peripheral vein.
- If a continuous infusion is not required, a peripheral cannula can be stopped off with a sterile bung after flushing the drug in with 0.9% saline, sterile water or 5% glucose to clear the dead space (there is no evidence that a heparin lock is needed for a cannula in peripheral veins).
- Central venous catheters must be firmly anchored to the skin so they do not migrate into or out of position.
- After individual drug injections and without continuous infusion, a heparin lock is appropriate to prevent clotting of the line (10 units of heparin per 1 mL of 0.9% saline), particularly in double-, triple- or quadruple-lumen catheters (always use Luer lock connections to minimise extravasation).
- Peripheral artery lines should never be used for giving drugs.
- To maintain patency, a continuous low-rate (0.5–1.0 mL/hour) infusion of heparinised 0.9% or 0.18% saline is useful (heparin at 1 unit/mL). Clear the 1-mL dead space of the catheter before and after sampling, which must be done aseptically.
- In neonates and infants, frequent flushing with saline 0.9% can result in sodium overload. Therefore, consider using 0.18% saline or sterile water to achieve flushing.
- Do not add drugs to any line containing blood or blood products.
- Most IV drugs can be given into an infusion containing 0.9% saline or up to 10% glucose (the exceptions include amphotericin B, phenytoin and erythromycin).

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- If only one line is being used for an infusion and more than one drug needs to be given, try to wait 10 minutes between them. If this is not possible, separate by 1 mL of 0.18% saline/4% glucose, 0.9% saline or sterile water for injections. This is very important with an alkaline drug such as sodium bicarbonate.
- Always give the flush slowly over at least 2 minutes to ensure that the drug already in the line/vein does not move forward in the patient in a sudden rapid surge (especially if the catheter/vein contains an inotrope or vasoactive drug such as aminophylline, cimetidine, phenytoin or ranitidine, which can cause an arrhythmia).
- When two IV drugs need to be given together and there is only one IV catheter, terminal co-infusion using a T- or Y-connector next to the catheter can be used. It is important to know whether this is safe for the drugs in question.

Minimising IV infusion and IV drugs errors
Errors of both commission and omission occur. For example, excess IV fluids can be dangerous by causing circulatory overload, and inadequate IV fluids can be dangerous by causing hypoglycaemia (especially in the neonate, and commonly when a blood transfusion is being given and the infant is relying on IV glucose).

Extravasation can also result in the absence of a vital drug (e.g. morphine infusion for pain). Errors will always occur where human actions are involved, and it is essential to have systems in place to minimise these.

Steps to reduce errors and their impact
1. Prescribe or change infusion rates as infrequently as possible, ideally no more than once or twice daily.
2. Never have more than one IV infusion line running at the same time unless this is absolutely necessary (e.g. in major trauma or shock, where two lines are needed for volume replacement and also in case one line is lost at a critical time).
3. Use a burette in which no more than the prescribed volume is present (especially in infants).
4. Record hourly the amount given (from the burette, syringe or infusion bag) and the amount left.
5. Check the infusion site hourly to ensure that extravasation has not occurred.
6. Ensure that flushes are only used when essential, and are given slowly over at least 2 minutes.
7. Ensure that flushes do not overload the patient with sodium.
8. Be particularly careful with potassium solutions given IV (use the enteral route whenever possible).
9. Check and double check the following:
   - Is it the right drug? Check the ampoule as well as the box.
   - Is it the right concentration?
   - Is the shelf life of the drug within the expiry date?
   - Has the drug been constituted and diluted correctly?
   - Is the dose right? (two people can check the prescription chart.)
   - Is it the correct syringe? (Deal with one patient at a time.)
   - Is the IV line patent?
   - Is a separate flush needed? If so, has the flush been checked?

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- Are sharps (including glass ampoules) disposed of?
- Has it been signed off as completed (and ideally countersigned)?

Writing a prescription
1. Use block capitals.
2. Use approved names.
3. The dosage should be written in grams (g), milligrams (mg) or micrograms. Always write micrograms in full.
4. Volumes should be written in millilitres (mL).
5. Avoid using decimal places whenever possible. If this is not possible, they should be prefaced by a zero. For example, write 500 mg, not 0.5 g, and if a decimal place is used, write 0.5 mL not .5 mL.
6. Write times using the 24-hour clock.
7. Routes of administration can be abbreviated as follows: IV (intravenous), IM (intramuscular), PO (orally), SC (sub- cutaneous), NEB (nebuliser), RECT (rectally).
8. 'As required' prescriptions must be specific about how much, how often and for what purpose (indicate the maximum 24-hour dose).
9. Each drug should be signed for individually by a registered/licensed doctor or other qualified professional.
10. Stop-dates for short-course treatments should be recorded when first prescribed.

IV drug infusions in severely ill or injured infants in high-dependency care

Adrenaline: in 5% dextrose or 0.9% saline. Do not mix with bicarbonate
Dose: 0.05–2 micrograms/kg/minute: this is equivalent to 0.6 mL/kg of 1 in 1000 (600 micrograms/kg) in 100 mL run at 0.5–20 mL/hour.

As a short-term measure, place 1 mg (1 mL of 1 in 1000 adrenaline) in 50 mL of 0.9% saline. Give 1 mL (20 micrograms) to an infant under 1 year of age. Give IV slowly. Repeat as required (with ECG monitoring).

Aminophylline: in 5% dextrose or 0.9% saline
Loading dose (do not give aminophylline if theophylline has been received in the last 24 hours).
IV infusion over 20–30 minutes, 5 mg/kg for children under 12 years of age
Then give 1 mg/kg/hour if under 12 years: this is equivalent to 50 mg/ kg in 50 mL run at 1 mL/hour for those under 12 years, and 0.5 mL/hour for those over 12 years.

Dopamine: in 5% dextrose or 0.9% saline or undiluted (ideally via a central line). Do not mix with bicarbonate This can be mixed with dobutamine.
Give 2–20 micrograms/kg/minute (renal = up to 5 micro- grams/kg/minute): this is equivalent to 30 mg/kg in 50 mL run at 0.2–2 mL/hour.

Ketamine: in 5% dextrose or 0.9% saline

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Give 10–45 micrograms/kg/minute: this is equivalent to 50 mg/kg in 50 mL run at 0.6–2.7 mL/hour (maximum concentration 50 mg/mL).

Midazolam: in 5% dextrose or 0.9% saline or undiluted Give 1–6 micrograms/kg/minute (60–360 micrograms/ kg/hour): this is equivalent to 6 mg/kg in 50 mL run at 0.5–3 mL/hour.
Or give undiluted (5 mg/mL), run at 0.012–0.072 mL/ kg/hour.

Morphine: in 5% dextrose or 0.9% saline
Give 10–60 micrograms/kg/hour: this is equivalent to 1 mg/ kg in 50 mL run at 0.5–3 mL/hour.

Giving injections

Wash your hands thoroughly. **MUST use disposable needles and syringes**. Clean the chosen site with an antiseptic solution. Carefully check the dose of the drug to be given and draw the correct amount into the syringe. Expel the air from the syringe before injecting. Always record the name and amount of the drug given. Discard disposable syringes in a safe container.

Intramuscular route
In infants, use the outer side of the thigh midway between the hip and the knee, or over the deltoid muscle in the upper arm. Hold the muscle at the injection site between the thumb and first finger and push the needle (23- to 25-gauge) into the muscle at a 90-degree angle (45 degrees in the thigh). Draw back the plunger to make sure that there is no blood (if there is, withdraw slightly and try again). Give the drug by pushing the plunger slowly until the end. Remove the needle and press firmly over the injection site with a small swab or cotton wool for at least two minutes.

FIGURE 37.1 Holding a child for an intramuscular injection in the thigh.

Subcutaneous route
Select the site as described above for intramuscular injection. Pinch up skin and subcutaneous tissue between your finger and thumb. Push the needle (23- to 25-gauge) under the skin at an angle of 30–45 degrees into the subcutaneous fatty tissue. Do not go deep to enter the underlying muscle. Draw back the plunger to make sure that there is no blood (if there is, withdraw slightly and try again). Give the drug by pushing the plunger slowly until the end. Remove the needle and press firmly over the injection site with cotton wool for at least two minutes.
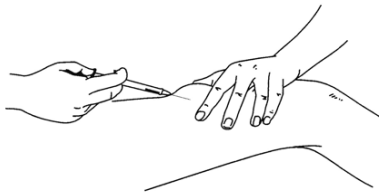
FIGURE 37.2 Giving a subcutaneous injection.

Intra-dermal route

Select an area of skin which has no infection or damage for the injection (e.g. over the deltoid in the upper arm). Stretch the skin between the thumb and forefinger of one hand. With the other hand, slowly insert the needle (25-gauge), bevel upwards, for about 2 mm just under and almost parallel to the surface of the skin. Considerable resistance is felt when injecting intra-dermally. A raised blanched bleb showing the surface of the hair follicles is a sign that the injection has been given correctly.
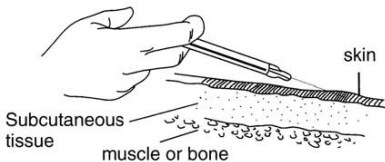
FIGURE 37.3 Giving an intradermal injection.

Gaining circulatory access

Peripheral venous cannulation

Preparation of kit
The following equipment is needed:

- 18- to 25-gauge IV cannula or butterfly needles
- 2-mL syringe and T-piece containing Ringer-lactate or Hartmann's solution or 0.9% saline for flushing
- tape or plaster of Paris for scalp veins
- a small splint (this can be made from a wooden spatula covered with gauze)
- alcohol swabs for skin cleaning
- local anaesthetic cream if available
- tourniquet (or assistant)
- cannula size: neonates: 24–25G
- infants: 22–24G

Procedure
1. Apply the tourniquet to distend the vein (do not forget to remove it after cannulation).
2. Choose a vein:
   - forearm
   - long saphenous vein (anterior to the medial malleolus)
   - back of the hand or front of the wrist
   - scalp.
3. Useful sites to cannulate include the dorsum of the feet and hands. The saphenous and antecubital veins are larger, but can be useful for percutaneously inserted 'long lines'. The antecubital veins are also useful for venepuncture for laboratory studies.
4. If possible, place the cannula close to the bone where it is more fixed.
5. Decide the direction of blood flow.
6. Clean the skin with antiseptic.
7. Fix and slightly stretch the skin with your other hand.
8. Pass the cannula through the skin at a slight angle (10–20 degrees). Be decisive.
9. Stop once you are through the skin.
10. Flatten the cannula to the skin and advance with the long axis of the cannula in the same direction as the vein. Be decisive.
11. Aim to pass it into the vein at the first attempt with steady advancement.
12. Always watch for blood appearing in the hub of the cannula.
13. As soon as blood is seen, stop.
14. Hold the needle still, and advance the cannula over the needle until the hub is at the skin.
15. Hold the cannula still.
16. Withdraw the needle.
17. Connect the connector, flush and fix. No subcutaneous swelling should be seen and there should be no resistance to injection.

18. If no blood is seen on advancing the cannula, but it is felt to be beyond the vein, stop.
19. Gently pull the cannula back in the same direction as advancement; if blood appears, stop once again. Follow the same procedure as if blood was seen on first advancement (transfixion technique).
20. Connect the T-piece and flush the cannula gently with Ringer-lactate or Hartmann's solution or 0.9% saline to confirm that it is in the vein.
21. If the cannula is satisfactorily inserted, tape it in place by looping a thin piece of the tape under the hub and round to form a 'V' shape fixing it to the skin.
22. When splinting, try to 'double back' the tape (i.e. put a short piece and a long piece back to back, leaving just the ends of the longer piece sticky). This helps to prevent excessive amounts of tape sticking to the baby, which is particularly important in the case of more immature babies whose skin is easily damaged.

FIGURE 38.1 Inserting an intravenous cannula into a vein on the back of the hand. The hand is flexed to obstruct venous return and thus make the veins visible.
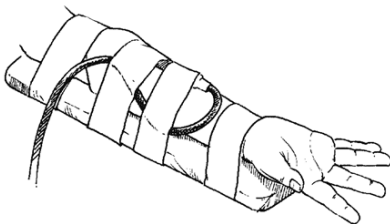
FIGURE 38.2 Arm splinted to prevent bending of the wrist.

Note on flushing lines
The smaller the syringe used, the greater the pressure exerted on fluid in the line. Therefore avoid using 1-mL syringes to flush a blocked line, as the line may rupture or tissue may be damaged by infiltration.

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Section 38 peripheral vein, scalp vein and external jugular cannulation

Care of the IV cannula
Secure the cannula when it has been introduced. This may require the splinting of neighbouring joints to limit the movement of the catheter. Keep the overlying skin clean and dry. Fill the cannula with Ringer-lactate or Hartmann's solution or 0.9% saline immediately after the initial insertion and after each injection.

Blood sampling from the IV cannula
If the patient needs blood samples at the time of cannulation it is often possible to take these as the cannula is inserted. Blood can be dripped from the end of the cannula into the appropriate bottles, or a syringe can be used to gently aspirate blood from the cannula. If the cannula has been flushed prior to insertion, the first 0.5–1 mL of blood should be discarded.

Common complications
Superficial infection of the skin at the cannula site is the commonest complication. The infection may lead to thrombophlebitis, which will occlude the vein and result in fever, and may progress to septicaemia. The surrounding skin is red and tender. Remove the cannula immediately to reduce the risk of further spread of the infection. Antibiotic treatment (effective against Staphylococcus aureus) should be given.

IV drug administration through an indwelling cannula
Attach the syringe containing the IV drug to the injection port of the cannula and introduce the drug. Once all of the drug has been given, inject 0.5 mL of Ringer-lactate or Hartmann's solution or 0.9% saline into the cannula until all of the drug has entered the circulation and the catheter is filled with the infusion fluid.

Safe IV infusions where no burettes are available
Mark the infusion bottle with tape for each hour to be given, and label each hour, or Empty until only the necessary amount to be given is left in the bottle.

Special sites for IV cannulae

Scalp veins
Procedure
1. Restrain the infant.
2. Shave the appropriate area of the scalp with a sterile razor.
3. Clean the skin.
4. Have an assistant distend the vein by holding a taut piece of tubing or bandaging perpendicular to it, proximal to (nearest to the infant's body) the site of puncture.
5. Fill the syringe with Ringer-lactate or Hartmann's solution or 0.9% saline and flush the butterfly set.
6. Disconnect the syringe and leave the end of the tubing open.
7. Puncture the skin and enter the vein. Blood will flow back through the tubing.
8. Infuse a small quantity of fluid to see that the cannula is properly placed and then tape it into position.

9. Care should be taken not to cannulate an artery, which is recognised by pulsation on palpation. If there is a pulsatile spurting of blood, withdraw the needle and apply pressure until the bleeding stops. Then look for a vein.
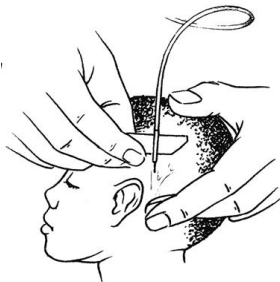
FIGURE 38.3 Inserting a scalp vein needle.

Scalp drips are generally more precarious than ones in the limbs, and need to be carefully observed. Infiltration into the soft tissues of the scalp can spread quickly and cause extensive necrosis if irritant. Shave the hair from an area about 2–3 cm around the site selected in order to allow for fixation by tape. Always ensure that the tip of the needle is not covered by dressings, so that infiltration is quickly seen.

External jugular vein cannulation
Procedure
1. Place infant in a 15–30-degree head-down position (or with padding under the shoulders so that the head hangs lower than the shoulders). Wrapping may be necessary to restrain the infant (see above).
2. Turn the head away from the site of puncture. Restrain the infant as necessary in this position.
3. Clean the skin over the appropriate side of the neck.
4. Identify the external jugular vein, which can be seen passing over the sternocleidomastoid muscle at the junction of its middle and lower thirds.
5. Have an assistant place their finger at the lower end of the visible part of the vein just above the clavicle. This stabilises it and compresses it so that it remains distended.
6. Puncture the skin and enter the vein pointing in the direction of the clavicle.
7. When free flow of blood is obtained, ensure that no air bubbles are present in the tubing, and then attach a giving set.
8. Tape the cannula securely in position. One of the most important points is to ensure that the cannula is properly secured in the vein by high-quality fixation. It is easily removed by the infant, so use plenty of tape!

Section 38 peripheral vein, scalp vein and external jugular cannulation
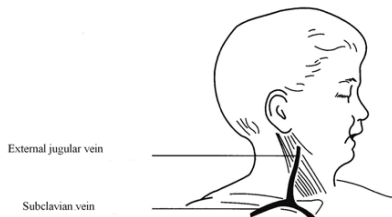
FIGURE 38.4 Position of the external jugular vein.

Be aware that there is a higher risk of air embolism than with peripheral venous cannulation.
If infusion through a peripheral vein or scalp vein is not possible, and it is essential to give IV fluids to keep the infant alive:

- set up an intra-osseous infusion
- or use a central vein
- or perform a venous cut-down.

All of these procedures are described below.

Section 39 Central venous cannulation

Central venous cannulation

This should not be used routinely. It should only be performed when IV access is urgent and, in the case of central veins, only by those who have been trained in the technique (it is best done by an anaesthetist). Remove the cannula from a central vein as soon as possible (i.e. when IV fluids or drugs are no longer essential, or when a peripheral vein can be cannulated successfully).

The aims of central venous cannulation are as follows:

- to obtain venous access when peripheral cannulation is not possible (however, in an emergency, intra- osseous cannulation is faster and easier).
- to monitor central venous pressure
- to obtain prolonged vascular access
- to obtain large-bore vascular access
- to administer certain drugs
- during resuscitation.

Procedure
Several routes are possible, but the most widely used are the femoral and internal jugular approaches. The femoral approach is easiest in the emergency situation.

Preparation of kit
The following equipment is needed:

- sterile pack
- sterile Seldinger wires
- cannula: single 16- to 22G cannula
- single, double or triple lumen if available (5 FG 5–8 cm length for an infant)
- syringe and Ringer-lactate or Hartmann's solution or saline
- suture and tape for fixing
- local anaesthetic with fine 25G needles.

Preparation of the infant
1. Explain what is going to happen to the parent .
2. Position the infant.
3. Sterilise the skin and maintain sterile technique.
4. Apply local analgesia to the skin (if the infant is conscious).

Two insertion techniques are available, namely:

- the same as in peripheral cannulation
- the Seldinger technique (wire)

ideally an ultrasound probe can help identify the vein and ensure the cannula when inserted is in the correct position in the lumen of the vein.

Seldinger method
1. Identify the vein with cannula on syringe (same approach as for peripheral cannulation); there must be good flow.
2. Stop, and pass the cannula over the needle.

3. Disconnect the syringe.
4. Pass the wire through the cannula to three-quarters the length of wire (if there is any resistance, stop, withdraw the wire with needle, and start again).
5. Holding the wire firmly, withdraw the needle over the wire.
6. Pass the dilator over the wire (it is sometimes necessary to make a small cut at the skin) and, holding the wire firmly, withdraw the dilator.
7. Pass the cannula/catheter filled with Ringer-lactate or Hartmann's solution or 0.9% saline over the wire (passage of the cannula should be smooth, meeting no resistance).
8. Hold the cannula, and withdraw the wire (gently if it sticks, do not force it).
9. Confirm correct placement by aspiration of blood.
10. Suture and fix with antiseptic ointment over the entry site.
11. Confirm the position with an X-ray.

Femoral vein cannulation
This is adequate for almost all needs, is technically much easier and has lower complication rates, particularly in neonates and infants. However, if it is not a sterile procedure, there is a risk of causing septic arthritis in the hip joint.

1. Position the patient supine with the leg slightly abducted. Place a towel under the buttocks to raise the pelvis.
2. Clean the skin and drape with sterile towels. Locate the vein by finding the femoral arterial pulsation 2 cm below the midpoint of the inguinal ligament. The vein lies immediately medial to the artery. If the infant is conscious, infiltrate the skin and subcutaneous area with 1% lignocaine.
3. With a finger on the femoral artery introduce the needle with syringe attached at an angle of 30–45 degrees to the skin along the line of the vein pointing towards the umbilicus. Advance the needle while aspirating.
4. When blood 'flashes back' into the syringe, stop advancing and remove the syringe from the needle. Feed the Seldinger guide wire through the needle, keeping hold of one end of the wire at all times.
5. Withdraw the needle over the wire, then feed the catheter over the wire into the vein.
6. Withdraw the wire and aspirate for blood to confirm the position. Then flush the catheter with Ringer-lactate or Hartmann's solution or 0.9% saline.
7. Suture the catheter in place.
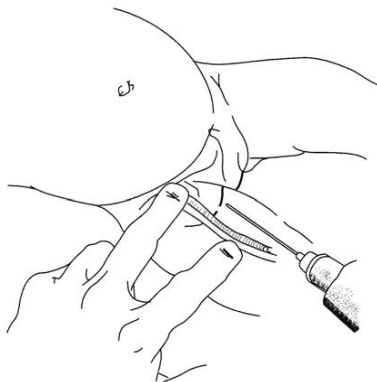
FIGURE 39.1 Femoral vein cannulation.

If you are unsure whether you are in a vein or an artery, consider transducing the pressure waveform.

Internal jugular vein
Use a head-down position for the internal jugular approach, as this increases vein distension and reduces the risk of air embolism.

Procedure
1. Place the infant in a 30-degree head-down position and turn their head to the left-hand side for the right-sided approach, which avoids the lymphatic duct.
2. Place a towel or roll under the shoulders to extend the neck.
3. Clean the skin and drape with towels, exposing the neck to the clavicle.
4. Identify the apex of the triangle formed by the two heads of the sternocleidomastoid and clavicle, and infiltrate local anaesthetic (if the infant is conscious). Alternatively, identify carotid pulsation medial to the sternomastoid at the level of the lower border of the thyroid cartilage, and the vein (usually) just lateral to this. Aim the needle at 30 degrees to the skin and towards the ipsilateral nipple (note that the neck is very short and the vein is superficial in the very young). Estimate the length of catheter from the point of skin entry to the nipple.
5. Direct the needle at 30 degrees to the skin, pointing towards the right nipple, and puncture the skin at the apex of the triangle.
6. Holding this position, advance the needle, aspirating all the time. If blood 'flashes back', stop advancing and remove the syringe from the needle. (If you do not cannulate the vein, withdraw the needle, but not out of the skin, and advance again slightly more laterally.)

7. Feed the Seldinger guide wire through the needle, always having control of one end of the wire.
8. Withdraw the needle over the guide wire and then feed the catheter over the wire into the superior vena cava.
9. Withdraw the wire, aspirate for blood and attach the infusion set. Do not leave the catheter open, as this may lead to an air embolism.
10. Suture the catheter in place and obtain a chest X-ray (if possible) to check for a pneumothorax and the position of the catheter tip, which should be in the superior vena cava (SVC), ideally at the junction of the SVC and the right atrium, but not in the right atrium.
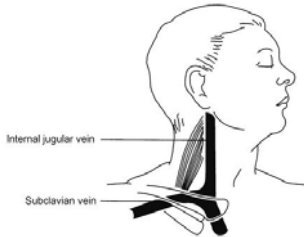
FIGURE 39.2 Position of the internal jugular and subclavian veins.

Cut-down venous cannulation

Indication
Continuous IV access is needed where percutaneous attempts have failed. In the emergency situation, intra- osseous access is faster and easier. Cut-down is less appropriate if speed is essential.

Preparation of kit
The following equipment is needed:
- skin prep (iodine, alcohol)
- scalpel
- suture
- IV cannula
- local anaesthetic
- curved artery forceps
- syringe and hypodermic needle
- sterile drapes.

Procedure
Identify landmarks. The long saphenous vein at the ankle is superior and medial to the medial malleolus of the ankle.

Long saphenous vein:
1. Immobilise the lower leg and clean the skin, as described above. Identify the long saphenous vein, which lies half a finger breadth (in the infant) superior and anterior to the medial malleolus.
2. Clean the skin and drape with sterile towels.
3. Infiltrate the skin with 1% lignocaine using a fine 24- to 25G needle, and make an incision through the skin perpendicular to the long axis of the vein. Bluntly dissect the subcutaneous tissue with haemostat forceps.
4. Identify and free a 1–2 cm section of vein. Pass a proximal and distal ligature.
5. Tie off* the distal end of the vein, keeping the ties as long as possible for traction.
6. Make a small hole in the upper part of the exposed vein, gently dilate the opening with the tip of a closed haemostat, and insert the cannula (without the needle/trocar in it) into this, while holding the distal tie to stabilise the position of the vein.
7. Secure the cannula in place with the upper ligature.
8. Attach a syringe filled with Ringer-lactate or Hartmann's solution or saline and ensure that the fluid flows freely up the vein. If it does not, check that the cannula is in the vein or try withdrawing it slightly to improve the flow.
9. Tie the distal ligature* around the catheter, and then close the skin incision with interrupted sutures.
10. Place antiseptic ointment (e.g. iodine) over the wound, and suture or tape the catheter to the skin (ensure that local anaesthetic is used at the suture site if the infant is conscious). Cover with sterile dressing.

* It is also possible to dispense with the proximal and distal ligatures and simply penetrate the vein directly with a plastic over-the-needle cannula as you would if

penetrating the skin externally. Once in the vein, remove the inner needle and secure in position.
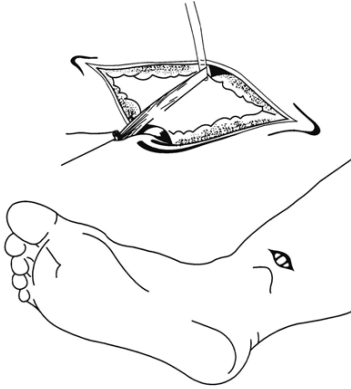
FIGURE 40.1 Cut-down incision showing vein: position of cut- down on long saphenous vein at ankle.

Complications
These include the following:
- haemorrhage or haematoma
- perforation of the posterior wall of the vein
- nerve transection
- phlebitis
- venous thrombosis.

Umbilical vein catheterisation

Indications
Where there is urgency during resuscitation of the newborn to give IV fluids and drugs. Temporarily for exchange transfusion. The catheter should not be left in position between exchanges.

Time of insertion
Catheterisation is usually easy in the first 4 days of life, and possible from 5 to 7 days. Passing an umbilical vein catheter is the quickest and easiest way to access the circulation in the newborn.

Preparation of kit
The following equipment is needed:
- gown and gloves
- sterile instruments including:
- fine scissors
- forceps
- scalpel
- silk suture for retaining
- French gauge umbilical catheter
- a sterile feeding tube may be satisfactory if an umbilical catheter is not available, but measure the length first so that you will know how much you have passed by measuring the length from the hub to the umbilical insertion. Cannulae designed for use as umbilical vein cannulae are usually marked in 5-cm increments
- a three-way tap
- 0.5% chlorhexidine or 10% povidone-iodine for clean- ing the skin
- sterile cotton wool balls
- sterile towels or drapes to cover the baby's abdomen
- sterile 2-mL syringe and connector filled with Ringer- lactate or Hartmann's solution or 0.9% saline.

FIGURE 41.1 Insertion and securing of a catheter in the umbilical vein. (a) Preparation of the umbilical cord. (b) Inserting the catheter into the umbilical vein, which is the larger thin-walled structure towards the head. Note the two umbilical arteries, which are thick-walled and towards the legs of the baby. (c) Securing the inserted catheter to prevent kinking.

Procedure

1 Assemble the syringe, three-way tap and catheter. Flush and fill the catheter with sterile 0.9% saline. Then close the tap to prevent air entry (which may cause air embolus).
2 Clean the umbilical cord and surrounding skin with 0.5% chlorhexidine or 10% povidone-iodine, and then loosely tie a suture around the base of the cord.
3 Cut back the cord to about 2 cm from the base.
4 Cover the skin with towels to form a sterile working surface.
5 Hold the cord at an edge with forceps.
6 Identify the vein. It is usually gaping, larger, and well separated from the two small thicker-walled arteries.
7 Hold the catheter approximately 2 cm from the end with non-toothed forceps, and insert the tip into the vein. Gently advance the catheter, which should pass easily.
8 Insert the catheter for a distance of 4–6 cm.
9 Check that the catheter is not kinked and that blood draws back easily. If there is a block, pull gently on the cord, pull back the catheter partly and reinsert.
10 The catheter can be secured by winding a suture round it several times and then passing a stitch through the cord base. An additional safeguard is to form two wings of tape which can then be taped to the abdominal wall, always remembering that it is preferable to use as little tape as possible in smaller babies. However, it is essential that the catheter does not fall out.

Section 41 Umbilical vein cannulation

Occasionally the umbilical vein is kinked and advance of the catheter is blocked at 1–2 cm beyond the abdominal wall. Gentle traction on the cord usually relieves this.

If obstruction occurs at more than 2 cm, and only partly gives way with pressure, the catheter is probably either wedged in the portal system or coiled up in the portal sinus. It is advisable to withdraw the catheter part way and reinsert it.

Care of indwelling umbilcal vein catheters
Leave the cord exposed to air. Remove blocked catheters.

Removal of the catheter
1 Use sterile technique.
2 Remove a specimen of blood for culture.
3 If possible, place a purse-string suture around the vessel at the base of the umbilicus and withdraw the catheter slowly.
4 Tighten the purse-string suture.
5 Apply pressure to the umbilical stump for 5–10 minutes.

Time of removal of catheter
Remove the catheter as soon as possible as dictated by the clinical state of the baby. The infection rate rises after 24 hours. Complications are more common with venous catheters than with arterial ones, so venous catheters should rarely be left in.

Complications
These include the following:
- thrombosis : survivors may develop portal vein thrombosis
- embolism from clots in the catheter, or from injected air
- vascular perforation
- vascular damage from hypertonic solutions (more common when the tip is in the portal system)
- haemorrhage from a disconnected catheter
- necrotising enterocolitis or bowel perforation may occur as a complication of exchange transfusion
- infection: there is no evidence that prophylactic antibiotics are of any value.

Intra-osseous needle insertion

Intra-osseous infusion is a safe, simple and reliable method of giving fluid and drugs in an emergency when venous access is not possible (e.g. in shock).

Site for needle
The first choice for the puncture is the proximal tibia. The site for needle insertion is in the middle of the antero-medial surface of the tibia, at the junction of the upper and middle third, to avoid damaging the epiphyseal plate (which is higher in the tibia), 2–3 cm below the tibial tuberosity. An alternative site for needle insertion is the distal femur, 2 cm above the lateral condyle.

Intra-osseous needles (15- to 18-gauge)
If a purpose-made intra-osseous needle is not available, a number of alternatives can be used, including bone-marrow needles, short lumbar puncture needles or a large-calibre venepuncture needle. For example, a green needle can be used in a neonate. The disadvantage of venepuncture needles is that they may carry a fragment of bone into the marrow. This is not dangerous, but it may block the needle. Also the bevel of these needles is long, and extravasation of fluid is more likely than with a purpose-made intra-osseous needle.
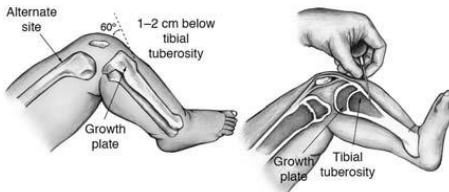
FIGURE 42.1 (a) Intra-osseous needle tibial site. (b) Section through bone.

Other equipment needed
This includes the following:
- a sterile 2-mL syringe containing 1–2% lignocaine to be used whenever the patient is conscious (otherwise the procedure will be very painful)
- two sterile 2 or 5-mL syringes
- sterile 20- or 50-mL syringes and ideally a three-way tap.

Procedure
1 Place padding under the infant's knee so that it is bent at 30 degrees from the straight (180-degree) position, with the heel resting on the table.
2 Locate the correct position (described above and shown in Figure 42.1).
3 Wash your hands and put on sterile gloves. (To avoid osteomyelitis, the procedure must involve strict asepsis using an antiseptic solution and sterile gauze to clean the

site, with the operator wearing sterile gloves.) Clean the skin over and surrounding the site with an antiseptic solution.

4 Infiltrate with lidocaine down to the periosteum if the infant is conscious.

5 Ask an assistant to stabilise the proximal tibia by grasping the thigh and knee above and lateral to the cannulation site, with the fingers and thumb wrapped around the knee but not directly behind the insertion site.

6 Insert the needle at a 90-degree angle with the bevel pointing towards the foot. Advance the needle slowly using a gentle but firm twisting or drilling motion.

7 Stop advancing the needle when you feel a sudden decrease in resistance or when you can aspirate blood. The needle should now be fixed in the bone and stand up by itself.

8 Remove the stylet.

9 Aspirate the marrow contents (which look like blood), using the 2-5-mL syringe, to confirm that the needle is in the marrow cavity and to provide bone marrow/blood for the following tests when appropriate: blood glucose, haemoglobin, group and cross-matching, blood culture and urea and electrolytes. Hb, glucose and electrolyte measurements may not be accurate after infusions have been previously given. Note that failure to aspirate bone-marrow contents does not mean that the needle is not correctly placed.

10 Attach the second 5-mL syringe filled with Ringer- lactate or Hartmann's solution or 0.9% saline. Stabilise the needle and slowly inject 3 mL while palpating the area for any leakage under the skin. If no infiltration is seen, start the infusion.

11 Attach the 50-mL syringe, usually containing Ringer- lactate or Hartmann's solution or saline, but compatible blood or 10% glucose can be used if hypoglycaemia is suspected, and push in the infusion fluid in boluses. It is not possible to infuse fluid through the intra- osseous needle using a standard IV giving set. The fluid has to be pushed in under light pressure, and if large volumes are needed (e.g. when giving boluses of fluid to treat shock) then 20-mL or 50-mL syringes should be used.

12 Check that the calf does not swell during the injections of fluid.

13 Secure IV access as soon as possible.

14 When the needle has been removed, cover with a sterile dressing.

15 Do not place distal to a major fracture or where there is infection.

16 Give prophylactic antibiotics after the immediate emergency has been managed.

17 All drugs and fluids that are given IV (including 10% glucose) can be given into the bone marrow, and they will reach the heart and general circulation as fast as if they had been given through a central vein.

18 Remove the intra-osseous needle as soon as venous access is available. In any case, it should not be in place for more than 8 hours.

Complications
These include the following:

- dislodgement
- misplacement (penetration through posterior cortex, failure to penetrate cortex), resulting in:
    - haematoma
    - tissue necrosis
    - compartment syndrome

- skin infection
- osteomyelitis
- tibial fracture in babies.

The scalp vein needle as an intra-osseous device
In infants, a green 'butterfly' (scalp vein) needle can be used as an intra-osseous needle with the same precautions as above.

Battery-powered intra-osseous device
The EZ-IO drill is a powered device that enables rapid insertion of an intra-osseous needle.
Unfortunately, the disposable needles are extremely and prohibitively expensive for low resource settings.
Various sizes of needle are available (see Textbook) for different-sized patients.
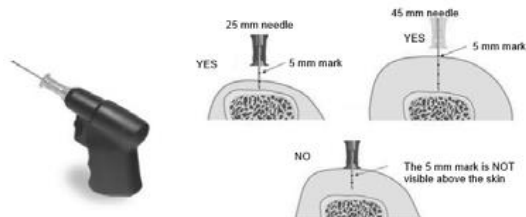
FIGURE 42.2 EZ-IO power drill and needles.

The landmarks are as before, using the upper end of the tibia.

The procedure is less painful for the conscious patient due to its rapidity, the drilling effect and the sharpness of the needles. The EZ-IO needles are available in two sizes, for patients under 40 kg and over 40 kg.

The procedure for insertion is as follows:
Take universal precautions for sterile procedure.
1 Clean the site.
2 Choose an appropriate size of needle and attach it to the drill. It will fix magnetically.
3 Remove the safety cap from the needle.
4 If the patient is conscious, control their movement during insertion.
5 Hold the drill and needle at 90 degrees to the skin surface and push through the skin without drilling, until bone is felt. Ensure that at least 5 mm of the needle is visible at this point.
6 Squeeze the drill button and drill continuously, applying gentle steady downward pressure until there is sudden loss of resistance – there is a palpable 'give' as the needle breaches the cortex. Release the trigger and stop insertion at this point.

7 If the driver stalls and will not penetrate the bone you may be applying too much downward pressure.

8 If the driver fails (this is rare) remove it, grasp the needle kit by hand and twist it into the bone marrow.

9 Remove the drill and unscrew the trochar.

10 Aspirate the bone marrow if possible directly from the needle.

11 Attach the pre-prepared connection tube containing sterile Ringer-lactate or Hartmann's solution or 0.9% saline before any infusion is given.
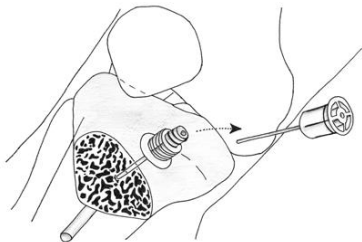
FIGURE 42.3 EZ-IO needle in place, with stylet removed.

12 Do not attach a syringe directly to the EZ-IO catheter hub except when drawing blood with the needle set stabilised by hand (sterile).

13 There is an optional device for securing the needle, but this is not essential.

14 Proceed with the required therapy. It should be noted that rapid infusion of fluid may be painful for the conscious patient.

15 Apply a sterile dressing.

16 When removing the catheter, attach a Luer lock syringe, and continuously rotate it clockwise while slowly and gently applying traction to the catheter. Do not rock or bend the catheter during removal.

17 Do not leave the catheter in place for more than 24 hours.

Defibrillation (see advanced life support section: section 3)

There are two indications for this procedure:
In cardiac arrest when the rhythm is ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) (see Section 1.13). The dose is 4 joules/kg in infants.

In supraventricular tachycardia (SVT) or ventricular tachycardia without shock. The dose is 0.5 joules/kg, rising to 1 joule/kg then 2 joules/kg if the first shocks were unsuccessful.

In any patient who is not in extremis, anaesthesia/sedation must be given before the DC shock is administered.

Safety
A defibrillator delivers enough current to cause cardiac arrest. The user must ensure that other rescuers are not in physical contact with the patient (or the trolley) at the moment when the shock is delivered. The defibrillator should only be charged when the paddles are either in contact with the infant or replaced properly in their storage positions. Oxygen must be discontinued and be moved right away from the patient.

Procedure
1 Basic life support should be interrupted for the shortest possible time (see steps 5 to 9 below).
2 Apply gel pads or electrode gel.
3 Select the correct paddles (paediatric paddles for patients weighing less than 10 kg). If only adult paddles are available for an infant, put one on the front of the chest and one on the back.
4 Select the energy required.
5 Place the electrodes on the pads of gel, and apply firm pressure.
6 Press the charge button.
7 Wait until the defibrillator is charged.
8 Shout 'Stand back!'
9 Check that all of the other rescuers are standing clear.
10 Deliver the shock.

Correct paddle placement
The usual placement is antero-lateral. One paddle is put over the cardiac apex in the mid-axillary line, and the other is placed just to the right of the sternum, immediately below the clavicle.

Good paddle contact
Gel pads or electrode gel should always be used (if the latter is used, care should be taken not to join the two
areas of application). Firm pressure should be applied to the paddles.

Correct energy selection
The recommended level in VF or pulseless VT cardiac arrest is 4 joules/kg (with no patient sedation).

Section 43 Defibrillation

In arrhythmias with a pulse, the dose is 0.5 joules/kg, then 1 joule/kg, then 2 joules/kg if the previous doses were unsuccessful (always with sedation).

Needle pericardiocentesis
Needle pericardiocentesis is a rarely used skill but can be life-saving when indicated. Please see textbook for details.

Section 44 Gastric tube, log roll and abscess incision

Insertion of an orogastric or nasogastric tube

The nasogastric tube is used to feed any infant who is unable to take food by mouth.

Preparation of kit
The following equipment is needed:

- nasogastric tube
- lubricant
- pH indicator paper or litmus paper
- syringe
- stethoscope
- adhesive tape.
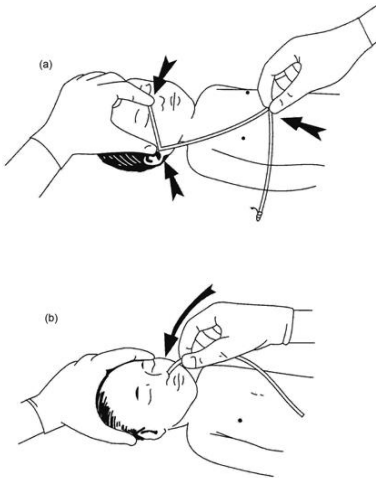
FIGURE 44.1 Inserting a nasogastric tube. (a) The distance from the nose to the ear and then to the epigastrium is measured. (b) The tube is then inserted to the measured distance.

In preterm infants:
4 French gauge tube is used for infants who weigh <1000 grams
6 French gauge tube is used for infants who weigh > 1000 grams (and most neonates)

Section 44 Gastric tube, log roll and abscess incision

8 to 10 French gauge tube is used for abdominal decompression (e.g. in infants with ileus or who are receiving continuous positive airway pressure).

Procedure
1 Place the infant supine with their head in the 'sniffing' position.
2 Measure the length of the tube from the nose via the earlobe to the midpoint between the xiphoid and the umbilicus. Mark the tube at this point with indelible pen.
3 Feed the tube lubricated with KY Jelly or saline through either the nose or the mouth directly backwards. (The neonate is a nose breather, and therefore if there is respiratory distress the oral route may be preferred.) Try to advance the tube as the infant swallows. If a baby has respiratory distress, a gastric tube is best passed through the mouth.
4 Check the position of the tube by very gently aspirating 0.2–0.5 mL of stomach contents using a small (2- or 5-mL) syringe (larger ones can damage the gastric mucosa) and checking the change in the pH indicator paper (the pH should be 5.5 or less, or the litmus paper should change colour from blue to pink), or flush the tube with 2–3 mL of air (only 1 mL in the neonate) and listen over the stomach area with the stethoscope. If in doubt, X-ray the chest and/or abdomen. (Note that the acidity of the gastric fluid may be reduced in preterm infants.)
5 If there is any doubt about the location of the tube, withdraw it and start again. Withdraw immediately if the infant starts coughing, as the tube may then be in the airway.
6 Secure the tube by taping it to the cheek, and record the length of tube outside the nose or mouth.
7 When the tube is in place, fix a 50-mL syringe (without the plunger) to the end of the tube, and pour food or fluid into the syringe, allowing it to flow by gravity.
8 The nasal route is more comfortable and secure, but if the infant has respiratory distress or is receiving CPAP, an orogastric tube is best (if passed through the nose the tube increases upper airway resistance).
9 Never pass a nasogastric tube in a head-injured patient. An orogastric tube is safe. If there is a base-of-skull fracture, a nasal tube could be pushed into brain tissue.

Log roll
When examining the back of the patient with major injury, it is important to minimise the risk associated with unrecognised spinal injury. It is essential to examine the back of the patient at the end of the primary survey (or even during it if there is suspicion of serious injury to the back of the chest or abdomen).

The aim of the log roll is to maintain the orientation of the spine during turning of the patient. It requires three people for an infant. In addition, one person is required for the examination of injuries.

Section 44 Gastric tube, log roll and abscess incision
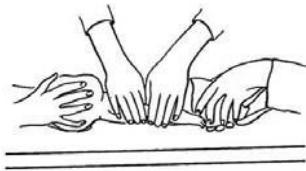
FIGURE 44.2 Log rolling an infant.

Incision and drainage of abscess
Indications
If there is uncertainty whether a hot red mass is an abscess, aspirate for pus before proceeding to incision and drainage.
Multiple/recurrent abscesses may be associated with HIV, TB, malnutrition, diabetes mellitus, anaemia or foreign bodies.

Preparation of kit
The following equipment is needed:
- skin preparation materials
- scalpel
- microbiology swab
- curette
- sterile gauze.

Procedure
1 If the patient is systemically unwell, take blood cultures (before giving antibiotics).
2 Antibiotics are only indicated if the patient is systemically unwell or if spreading cellulitis is present.
3 Use general anaesthesia for certain sites. Regional blocks may be used for limbs. (Note that local infiltration produces poor anaesthesia in inflamed tissue.)
4 Clean the skin.
5 Incise over the most superficial tender point in the direction of skin creases. Take a sample of pus for culture and staining, including the Ziehl–Neelsen stain if indicated. The commonest error is to make the incision too small.
6 Insert a curette spoon or finger to break down any loculi. Send a sample of the wall of the abscess for TB if indicated.
7 Irrigate the cavity with 0.9% saline to flush out necrotic material.
8 If a large cavity exists, loosely pack it with sterile gauze. For a small cavity place a 'wick' (e.g. a piece of rolled gauze) into the wound, forming a track. Cover the wound loosely with absorbent dressing. Change the gauze packing after 24 hours, giving analgesia beforehand if needed. Remove the wick after 48 hours.
9 As the cavity discharges pus it should heal from a depth to superficially through the open skin incision.

Section 46 Lumbar puncture and suprapubic aspiration

Lumbar puncture

Preparation of kit

The following equipment is needed:

- iodine
- sterile gloves
- sterile dressings pack
- spinal needle with stylet
- collodion
- small adhesive dressing
- local anaesthetic

Indications

- As part of septic screen in case meningitis is present.
- For investigating the possible cause of seizures.
- For investigating the possible cause of apnoeic episodes due to meningitis.
- As therapy in post-haemorrhagic hydrocephalus.
- For administration of drugs in leukaemia.

Contraindications

- Signs of raised intracranial pressure, such as deep coma (P or U on the AVPU scale), unequal pupils, rigid posture or paralysis in any of the limbs or the trunk, or irregular breathing.
- Skin infection in the area through which the needle will have to pass.
- Significant bleeding disorder.

If contraindications are present, the potential value of the information gained from a lumbar puncture should be carefully weighed against the risk of the procedure. If in doubt, it might be better to start treatment for suspected meningitis, and delay performing a lumbar puncture.

Precautions

Do not perform a lumbar puncture in the very sick patient (it may precipitate apnoea in an infant)

Excessive neck flexion when positioning can lead to hypoxaemia and acute respiratory deterioration.

If a spinal needle is unavailable and a normal (non- stylet) needle is used, the needle bore may become blocked with skin on insertion and therefore obstruct flow. There is also the risk of tissue implantation leading to a dermoid cyst.

Procedure

The infant lying down on the left side

Section 46 Lumbar puncture and suprapubic aspiration

FIGURE45.1 Holding an infant lying on their left side for a lumbar puncture. Note that the spine is curved to open up the spaces between the vertebrae.

1 When the infant is lying on their side a hard surface should be used.
2 Place the infant on their side so that the vertebral column is parallel to this surface and the transverse axis of the back is vertical (see Figure 45.1).
3 It is helpful to have an experienced assistant present to hold the patient. Flex the spine maximally, but avoid excessive neck flexion. Make sure that the airway is not obstructed and the infant can breathe normally. Take particular care when holding young infants. The assistant should not hold a young infant by the neck or flex the neck to avoid airway obstruction.

4 *Prepare the site*
- Use aseptic technique. Scrub your hands and wear sterile gloves.
- Prepare the skin around the site with an antiseptic solution.
- Sterile towels should be used.

5 *Identify site of insertion*
  Locate the space between the third and fourth lumbar vertebrae or between the fourth and fifth lumbar vertebrae. (The third lumbar vertebra is at the junction of the line between the iliac crests and the vertebral column.)
6 Use an LP needle with a stylet (22 gauge for a young infant, and 20 gauge for an older infant; if these are not available, routine hypodermic needles may be used). Insert the needle into the middle of the inter-vertebral space and aim the needle towards the umbilicus.
7 Advance the needle slowly. The needle will pass easily until it encounters the ligament between the vertebral processes. More pressure is needed to penetrate this ligament, and less resistance is felt as the dura is penetrated. In young infants this decrease in resistance is not always felt, so advance the needle very carefully.
8 Stop advancing when a 'give' or puncture sensation is felt on entering the subarachnoid space (this is often not felt in neonates). Frequent stylet withdrawals

during the procedure should be undertaken to see if the CSF flows, indicating that the subarachnoid space has been success- fully entered. The subarachnoid space is only 0.5–0.7 cm below the skin in premature infants and 1 cm below it in term infants, so it is easy to over-penetrate by mistake. Over-penetration leads to puncturing of the anterior vertebral venous plexus and a bloody sample, so that CSF microscopy is less informative or perhaps impossible. The needle should be withdrawn and the procedure repeated in another disc space.

9 Withdraw the stylet. Obtain a sample of 0.5–1 mL of CSF and place it in sterile containers, allowing six drops of CSF to drip into each sample container.

10 Replace the stylet.

11 Withdraw the needle and stylet completely and apply pressure to the site for a few seconds. Put a sterile dressing over the needle puncture site, and cover the whole site with adhesive dressing.

12 Send samples for the following: microscopy, cell type and counts, Gram and Ziehl-Neelson staining, culture and sensitivity (including for TB) and virology: biochemistry (glucose, protein).

Suprapubic aspiration of urine
Indications
Usually in sick infants where urgent diagnosis is required and there is a palpable bladder that does not respond to manual expression for a clean catch.

Procedure
1 Use a sterile technique throughout. Advance a 23- to 24- gauge needle attached to a syringe to a depth of 3 cm in the midline at the proximal transverse crease above the pubis. Withdraw the urine into a sterile syringe and transfer it to a sterile urine container,

2 Do this only in an infant with a bladder containing sufficient urine, which can be demonstrated by percussion. Do not use urine bags to collect urine, as the specimens may become contaminated.

3 Have a clean urine jar ready in case the infant passes urine during the procedure.
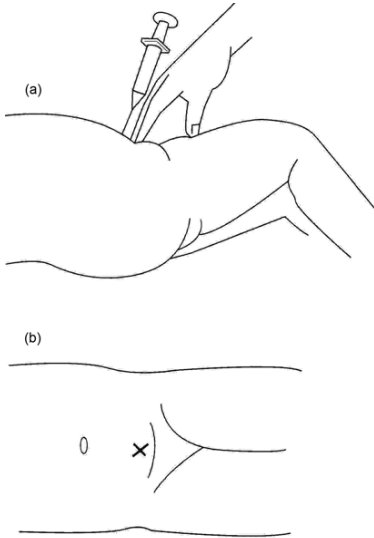
FIGURE 45.2 Position for carrying out suprapubic aspiration of urine in an infant. (a) Side view. (b) Abdominal view. Note the angle of insertion of the needle.

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