Advanced Paediatric Life Support

FIFTH EDITION

Advanced Paediatric Life Support

The Practical Approach

FIFTH EDITION

Advanced Life Support Group

EDITED BY

Martin Samuels

Susan Wieteska

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Preface to the fifth edition

The *Advanced Paediatric Life Support* (APLS) concept and courses have aimed from inception 18 years ago to bring a structured approach and simple guidelines to the emergency management of seriously ill and injured children. The manual was and continues to be an important part of the course, but it has also come to be used as a handbook in clinical practice. This has been a real tribute to the contributors of this text, both current and past editions.

The course has changed since the last edition as a result of changes in medical education and the demands on busy health professionals' time. The course has moved in the UK and some overseas centres from a 3-day face-to-face course, supported by prior learning from the manual, to a 1-day virtual learning environment (VLE) followed by a 2-day face-to-face course. This has meant transferring a substantial amount of the learning process to a web-based format, with increasing use of video clips and interactivity. To complement this, we have moved the manual in to colour and kept it in a loose-bound format for updates.

The fifth edition of the manual reflects the pace of change of medical science and practice, the international nature of APLS and the increasing recognition of the importance of human factors in providing the best emergency care. This edition benefits from the latest guidelines for resuscitation from cardiac arrest by the International Liaison Committee on Resuscitation (ILCOR), published in October 2010.

APLS is established in the United Kingdom, Australasia, the Caribbean, mainland Europe, the Middle and Far East, Scandinavia and South Africa. In addition, the Advanced Life Support Group (ALSG) has collaborated with many other agencies so that the course is now available in a number of resource-poor countries, either in its original form or modified for local use. To ensure this, ALSG has had to be responsive to the different styles, languages, cultures and clinical facilities found in many different countries. It is with the help of so many enthusiastic and dedicated local health professionals that APLS has flourished.

We hope that new as well as current providers of emergency paediatric practice appreciate the changes – continuing professional development is expected of us all and reinforcing your learning on the VLE and with the APLS CD will help achieve this. The material found in these sources, as well as in this manual, is all brought together by the increasing numbers of experts that have contributed to this update. We thank them and all our instructors, who have provided helpful feedback. We ask that this process does not stop, so that we can begin the process that will support the development of the next edition.

Martin Samuels
Sue Wieteska
Manchester 2011

Preface to the first edition

Advanced Paediatric Life Support: The Practical Approach was written to improve the emergency care of children, and has been developed by a number of paediatricians, paediatric surgeons, emergency physicians and anaesthetists from several UK centres. It is the core text for the APLS (UK) course, and will also be of value to medical and allied personnel unable to attend the course. It is designed to include all the common emergencies, and also covers a number of less common diagnoses that are amenable to good initial treatment. The remit is the first hour of care, because it is during this time that the subsequent course of the child is set.

The book is divided into six parts. Part I introduces the subject by discussing the causes of childhood emergencies, the reasons why children need to be treated differently and the ways in which a seriously ill child can be recognised quickly. Part II deals with the techniques of life support. Both basic and advanced techniques are covered, and there is a separate section on resuscitation of the newborn. Part III deals with children who present with serious illness. Shock is dealt with in detail, because recognition and treatment can be particularly difficult. Cardiac and respiratory emergencies, and coma and convulsions, are also discussed. Part IV concentrates on the child who has been seriously injured. Injury is the most common cause of death in the 1–14-year age group and the importance of this topic cannot be overemphasised. Part V gives practical guidance on performing the procedures mentioned elsewhere in the text. Finally, Part VI (the appendices) deals with other areas of importance.

Emergencies in children generate a great deal of anxiety – in the child, the parents and in the medical and nursing staff who deal with them. We hope that this book will shed some light on the subject of paediatric emergency care, and that it will raise the standard of paediatric life support. An understanding of the contents will allow doctors, nurses and paramedics dealing with seriously ill and injured children to approach their care with confidence.

Kevin Mackway-Jones
Elizabeth Molyneux
Barbara Phillips
Susan Wieteska
Editorial Board
1993

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We are greatly indebted to Helen Carruthers, MMAA, Mary Harrison, MMAA and Kate Wieteska for producing the excellent line drawings that illustrate the text. Thanks to the Status Epilepticus Working Party for the status epilecticus protocol and the Child's Glasgow Coma Scale. The information in Table 9.1 is taken from *Lessons from Research for Doctors in Training* produced by the Meningitis Research Foundation. We would also like to thank Neal Jones, NW Simulation Education Network Manager for his input into the Human factors chapter.

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British Society of Paediatric Gastroenterology, Hepatology and Nutrition Dr N. Croft, Paediatric Gastroenterologist, *London*
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Finally, we would like to thank, in advance, those of you who will attend the Advanced Paediatric Life Support course and other courses using this text; no doubt, you will have much constructive criticism to offer.

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Clinicians practising in tropical and under-resourced health care systems are advised to read *International Child Health Care: A Practical Manual for Hospitals Worldwide* (978-0-7279-1476-7) published by Blackwell Publishing Ltd, which gives details of additional relevant illnesses not included in this text.

UPDATES

The material contained within this book is updated on a 5-yearly cycle. However, practice may change in the interim period. We will post any changes on the ALSG website, so we advise that you visit the website regularly to check for updates (url: www.alsg.org – go to the APLS page). The website will provide you with a new page to download and replace the existing page in your book.

REFERENCES

All references are available on the ALSG website www.alsg.org – go to the APLS page.

ON-LINE FEEDBACK

It is important to ALSG that the contact with our providers continues after a course is completed. We now contact everyone 6 months after their course has taken place asking for on-line feedback on the course. This information is then used whenever the course is updated to ensure that the course provides optimum training to its participants.

PART 1

Introduction

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

CHAPTER 1

Introduction

1.1 INTRODUCTION

Each year many millions of children around the world die from potentially preventable and treatable causes. Whilst the majority of these deaths would be prevented by attention to living conditions and public health measures, an improvement in the recognition of serious illness and delivery of initial medical treatment would undoubtedly save lives.

The training of health care practitioners and the resources available for health care delivery varies enormously among countries. It is possible however to improve the outcome of serious illness and injury in children with modest resources if the basic principles of resuscitation are adhered to. The structured sequential approach to the recognition and treatment of the seriously ill and injured child followed in this manual is applicable in many situations and circumstances.

1.2 MORTALITY RATES IN CHILDHOOD

> The infant mortality rate is defined as the number of deaths of children under 1 year of age in one calendar year per 1000 live births in the same calendar year.

Worldwide mortality rates in children have fallen substantially and consistently over the last 100 years. The World Health Organisation has estimated that the global infant mortality rate has fallen from 180 in 1950 to around 50 in 2010. In some developed countries the fall has been even more dramatic. For example in Australia the infant mortality rate in 1902 was 107; 100 years later in 2002 the figure had reduced to 5.0 where it stayed for the next 5 years. Even with figures at such low levels, the rates in developed countries have recently continued to fall. In England and Wales the infant mortality rates have more than halved in the last 28 years, falling from 12 in 1980 down to 4.5 in 2008: the lowest on record.

These dramatic improvements in infant mortality are due largely to improvements in living conditions such as sanitation, shelter, quality of drinking water and better nutrition. Some medical measures such as better obstetric and neonatal care and the advent of mass vaccination have also played substantial roles. The delivery of better acute care for seriously ill and injured children is likely to assist in reducing mortality rates further.

The mortality rate decreases significantly with the increasing age of the child, with the highest death rate occurring in the first 28 days, and indeed most deaths occur on the first day of life. Male children are more likely to die than females in all age groups, a trend which is not reversed until much later in life.

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1.3 CAUSES OF DEATH IN CHILDHOOD

The causes of death in childhood in any country vary with age. Table 1.1 shows the top worldwide causes of death for children under 6 years of age. Table 1.2 shows the causes of death for children in the United Kingdom and illustrates the patterns found in many developed countries. In the newborn period the most common causes are congenital abnormalities, antepartum infections and factors associated with prematurity, such as respiratory immaturity, cerebral haemorrhage and infection due to immaturity of the immune response.

In children aged 1–12 months, congenital abnormalities, conditions related to prematurity and sudden unexplained death each contribute around 20% to mortality. This is in contrast to a number of years ago when sudden infant death syndrome (as it was known then) was much more prevalent.

Congenital abnormalities contribute significantly to mortality rates during all stages of childhood. Complex congenital heart disease, central nervous system malformations, metabolic disorders and chromosomal anomalies are the commonest lethal disorders.

After 1 year of age trauma is a frequent cause of death and remains so until well into adult life. Deaths from trauma have been described as falling into three groups. In the first group there is overwhelming damage at the time of trauma, and the injury caused is incompatible with life; children with such massive injuries will die within minutes whatever is done. Those in the second group die because of progressive respiratory failure, circulatory insufficiency or raised intracranial pressure secondary to the effects of injury; death occurs within a few hours if no treatment is administered, but may be avoided if treatment is prompt and effective.

Table 1.1 Top causes of death worldwide in children under 6 years of age

Neonates aged 0–27 days		Children aged 1–59 months	
Preterm birth complications	12%	Diarrrhoea	14%
Birth asphyxia	9%	Pneumonia	14%
Sepsis	6%	Other infections	9%
Other	5%	Malaria	8%
Pneumonia	4%	Other non communicable disease	4%
Congenital abnormalities	3%	Injury	3%
Diarrhoea	1%	AIDS	2%
Tetanus	1%	Pertussis	2%

Source: www.thelancet.com Vol. 375, 5 June 2010.

Table 1.2 Number and common causes of death by age group in England and Wales, 2008

	0–4 weeks	1–12 months	1–4 years	5–14 years
Number of deaths	3918	1023	506	590
Perinatal conditions and prematurity	62%	22%	3%	1%
Congenital abnormalities	25%	20%	15%	7%
Sudden unexplained deaths	1%	19%	3%	1%
Respiratory infections*		6%	11%	8%
Other infections	1%	7%	11%	3%
Trauma including asphyxia	10%	4%	13%	19%
Other		6%	2%	1%

*Figure for 0–4-week group is included in 'Other infections'.

Source: Office of National Statistics, 2010.

The final group consists of late deaths due to raised intracranial pressure, infection or multiple organ failure. Appropriate management in the first few hours will decrease mortality in this group also.

In developing countries, infectious diseases are still major causes of death. Seven out of 10 childhood deaths can be attributed to just five main causes: pneumonia, diarrhoea, measles, malaria and malnutrition. Three out of every four children seen by health services are suffering from at least one of these conditions. HIV/AIDS has contributed to this and also been associated with increasing deaths from tuberculosis in countries affected. As these societies become more urbanised the mortality from trauma, especially from motor vehicle accidents, increases. In South Africa, a country which, although developing rapidly, has large areas of severe poverty, the under-fives mortality rate has recently been shown to include 40% (42,749) of deaths from HIV/AIDS, 11% (11,876) from low birth weight, 21% (22,680) from infections and 3% (3506) from trauma. In older South African children, trauma, especially road traffic accidents, homicide and suicide are leading causes of death. In Trinidad, children under 1 year of age accounted for 4% of deaths in 1997, with infant mortality at 17 per 1000 live births. In Trinidadian school children, the foremost cause of death was injury, with infections causing one-fifth of deaths.

In developed countries, many children with diseases that were once invariably fatal, such as complex congenital heart disease, inborn errors of metabolism, haematological malignancies or cystic fibrosis, are now treated or 'cured' by drugs, operations, diet, transplant or, soon, even gene therapy. In these children, common acute illnesses such as varicella or chest infections have potentially lethal consequences. They require a low threshold for rapid aggressive treatment delivered by a team with an understanding of their underlying disease.

Only a minority of childhood deaths, such as those due to end-stage neoplastic disease, are expected and 'managed'. There should be timely discussions among child, family and health carers to identify whether and in what manner resuscitation should be carried out to prevent unwanted and inappropriate resuscitation and interventions.

1.4 PATHWAYS LEADING TO CARDIORESPIRATORY ARREST

As the outcome from cardiorespiratory arrest in children is poor the only effective way to prevent death and permanent disability is to understand its antecedent events, and be able to recognise and treat them vigorously.

Cardiac arrest in children is rarely due to primary cardiac disease. This differs from the situation in an adult where the primary arrest is often cardiac, and circulatory and respiratory function may remain near-normal until the moment of arrest.

In children, most cardiorespiratory arrests are secondary to hypoxia caused by respiratory pathology, including birth asphyxia, inhalation of foreign bodies, bronchiolitis and asthma. Respiratory arrest also occurs secondary to neurological dysfunction caused by such events as convulsion or poisoning. Raised intracranial pressure (ICP) due to head injury or acute encephalopathy eventually leads to respiratory arrest, but severe neuronal damage has already been sustained before the arrest occurs.

Whatever the cause, by the time of cardiac arrest the child has had a period of respiratory insufficiency, which will have caused hypoxia and respiratory acidosis. The combination of hypoxia and acidosis causes cell damage and death (particularly in more sensitive organs such as the brain, liver and kidney) before myocardial damage is severe enough to cause cardiac arrest.

Most other cardiac arrests in children are secondary to circulatory failure. This will have resulted often from fluid or blood loss, or from fluid maldistribution within the circulatory system. The former may be due to gastroenteritis, burns or trauma, whilst the latter is often caused by sepsis or anaphylaxis. Because all organs are deprived of essential nutrients and oxygen as shock progresses to cardiac arrest, circulatory failure, like respiratory failure, causes tissue hypoxia and acidosis. In fact, both pathways may occur in the same condition. The pathways leading to cardiac arrest in children are summarised in Figure 1.1.

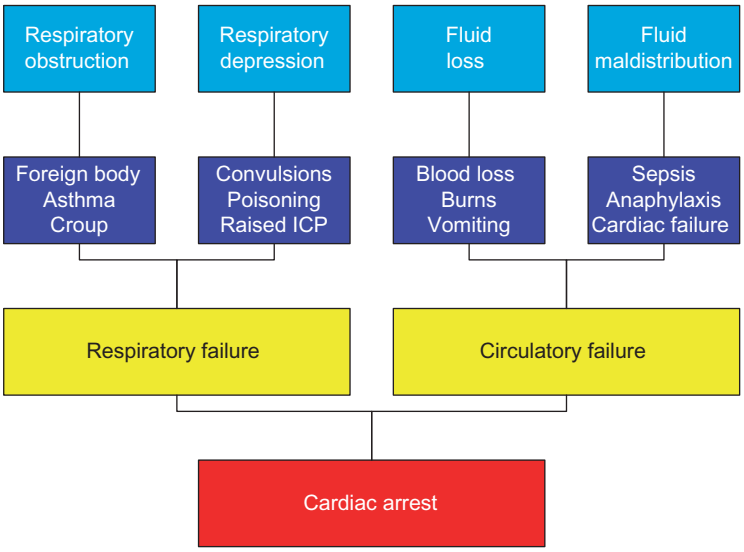

Figure 1.1 Pathways leading to cardiac arrest in childhood (with examples of underlying causes). ICP, intracranial pressure

1.5 OUTCOME FROM CARDIAC ARREST IN CHILDREN

The outcome of cardiac arrest in children is poor. Of those who survive, many are left with permanent neurological deficits. The worst outcome is in children who have had an out-of-hospital arrest and arrive at hospital apnoeic and pulseless. These children have almost no chance of intact neurological survival, especially if cardiopulmonary resuscitation has been in progress for 20 minutes or longer. There has often been a prolonged period of hypoxia and ischaemia before the start of adequate cardiopulmonary resuscitation.

Earlier recognition of seriously ill children and paediatric cardiopulmonary resuscitation training for the public could improve the outcome for these children.

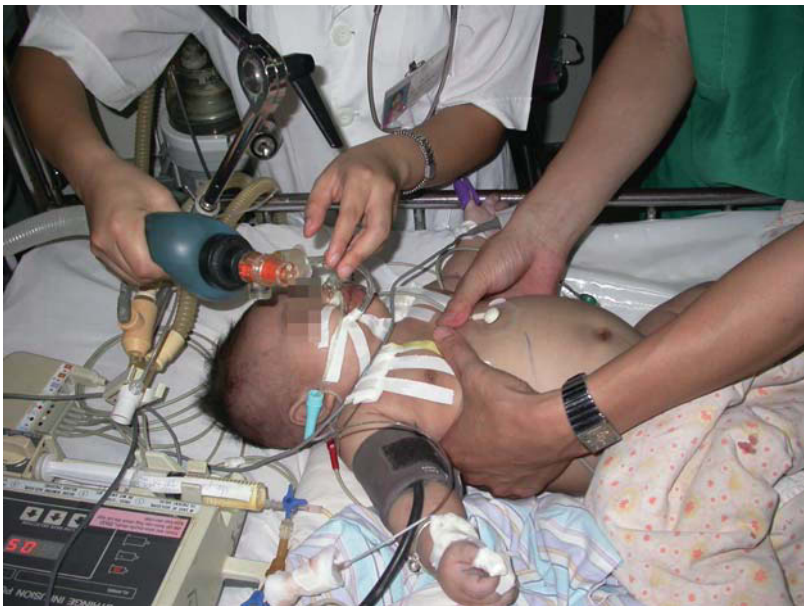

Figure 1.2 Advanced paediatric life support (APLS) in action

CHAPTER 2

Why treat children differently?

2.1 INTRODUCTION

Children are a diverse group varying enormously in weight, size, shape, intellectual ability and emotional responses. At birth a child is, on average, a 3.5 kg, 50 cm long individual with small respiratory and cardiovascular reserves and an immature immune system. They are capable of limited movement, exhibit limited emotional responses and are dependent upon adults for all their needs. Fourteen or more years later at the other end of childhood, the adolescent is a 50 kg, 160 cm tall person who looks physically like an adult and is often exhibiting a high degree of independent behaviour.

Competent management of a seriously ill or injured child who may fall anywhere between these two extremes requires a knowledge of these anatomical, physiological and emotional differences and a strategy of how to deal with them.

Key differences to consider in children

- Weight
- Anatomical – size and shape
- Physiological – cardiovascular, respiratory and immune function
- Psychological – intellectual ability and emotional response

2.2 WEIGHT

The most rapid changes in weight occur during the first year of life. An average birth weight of 3.5 kg will have increased to 10 kg by the age of 1 year. After that time weight increases more slowly until the pubertal growth spurt. This is illustrated in the weight charts shown in Figure 2.1.

As most drugs and fluids are given as the dose per kilogram of body weight, it is important to determine a child's weight as soon as possible. Clearly the most accurate method for achieving this is to weigh the child on scales; however, in an emergency this may be impracticable. Very often, especially with infants, the child's parents or carer will be aware of a recent weight. If this is not possible, various formula or measuring tapes are available. The Broselow or Sandell tapes use the height (or length) of the child to estimate weight. The tape is laid alongside the child and the estimated weight read from the calibrations on the tape. This is a quick, easy and relatively accurate method. Various formulae may also be used although they should be validated to the population in which they are being used.

If a child's age is known the formulae given in Table 2.1 may be useful.

The formula method has the added advantage of allowing an estimation of the weight to be made before the child arrives in hospital so that the appropriate equipment and drugs may be arranged for. Whatever the method, it is essential that the carer is sufficiently familiar with it to be able to use it quickly and accurately under pressure.

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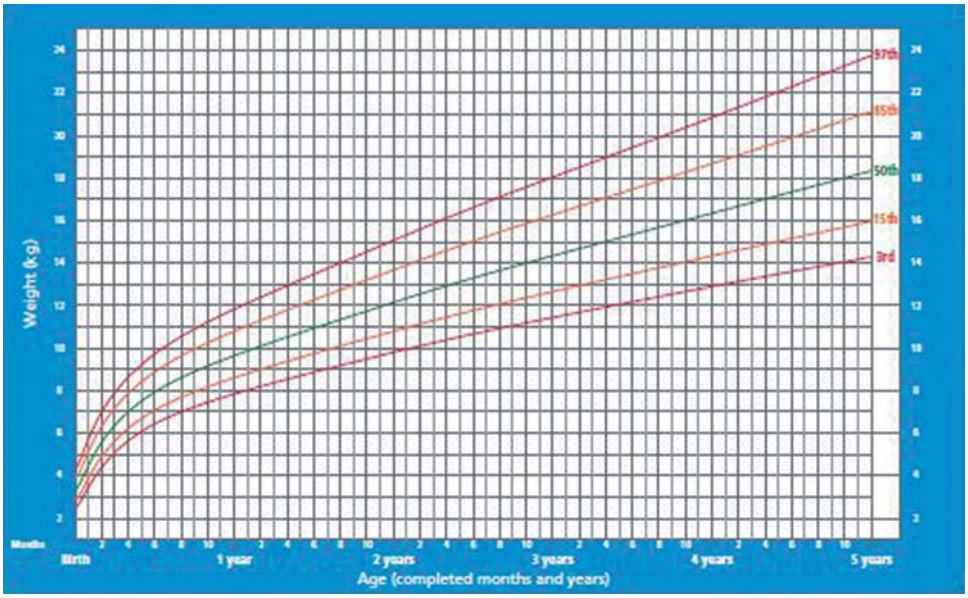
(a)



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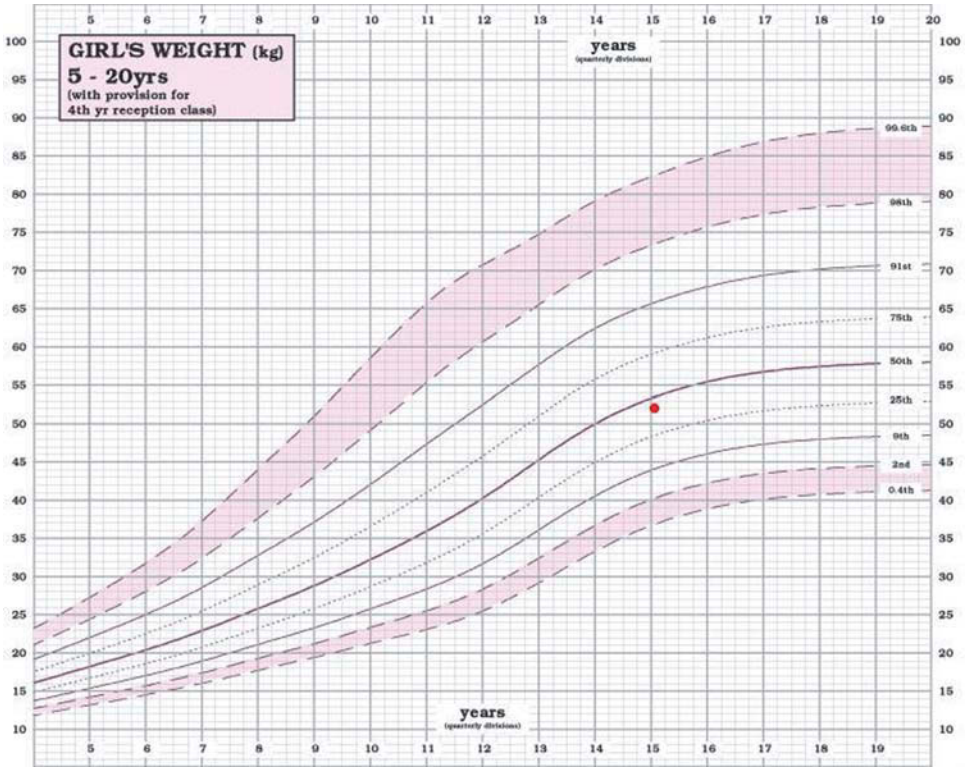


Figure 2.1 Centile chart for weight in (a) boys (0–5 years) and (b) girls (5–20 years)

Table 2.1 Weight formulae in different age groups

Works best for …	Formula
0–12 months	Weight (in kg) = (0.5 × age in months) + 4
1–5 years	Weight (in kg) = (2 × age in years) + 8
6–12 years	Weight (in kg) = (3 × age in years) + 7

2.3 ANATOMICAL

As the child's weight increases with age the size, shape and proportions of various organs also change. Particular anatomical changes are relevant to emergency care.

Airway

The airway is influenced by anatomical changes in the tissues of the mouth and neck. In a young child the head is large and the neck short, tending to cause neck flexion and airway narrowing. The face and mandible are small, and teeth or orthodontic appliances may be loose. The tongue is relatively large and not only tends to obstruct the airway in an unconscious child, but may also impede the view at laryngoscopy. Finally, the floor of the mouth is easily compressible, requiring care in the positioning of fingers when holding the jaw for airway positioning. These features are summarised in Figure 2.2.

The anatomy of the airway itself changes with age, and consequently different problems affect different age groups. Infants less than 6 months old are obligate nasal breathers. As the narrow nasal passages are easily obstructed by mucous secretions, and as upper respiratory tract infections are common in this age group, these children are at particular risk of airway compromise. In 3–8-year-olds, adenotonsillar hypertrophy may be a problem. This not only tends to cause obstruction, but also causes difficulty when the nasal route is used to pass pharyngeal, gastric or tracheal tubes.

In all young children the epiglottis is horseshoe-shaped, and projects posteriorly at 45°, making tracheal intubation more difficult. This, together with the fact that the larynx is high and anterior (at the level of the second and third cervical vertebrae in the infant, compared with the fifth and sixth vertebrae in the adult), means that it is easier to intubate an infant using a straight-blade laryngoscope. The cricoid ring is the narrowest part of the upper airway (as opposed to the larynx in an adult). The narrow cross-sectional area at this point, together with the fact that the cricoid ring is lined by pseudo-stratified ciliated epithelium loosely bound to areolar tissue, makes it particularly susceptible to oedema. As tracheal tube cuffs tend to lie at this level, uncuffed tubes are preferred in emergencies and for use by non-experts in prepubertal children.

The trachea is short and soft. Overextension of the neck as well as flexion may therefore cause tracheal compression. The short trachea and the symmetry of the carinal angles mean that not only is tube displacement more likely, but a tube or a foreign body is also just as likely to be displaced into the left as the right main-stem bronchus.

Breathing

The lungs are relatively immature at birth. The air–tissue interface has a relatively small total surface area in the infant (less than 3 m^2). In addition, there is a 10-fold increase in the number of small airways from birth to adulthood. Both the upper and lower airways are relatively small, and are consequently more easily obstructed. As resistance to flow is inversely proportional to the fourth power of the airway radius (halving the radius increases the resistance 16-fold), seemingly small obstructions can have significant effects on air entry in children.

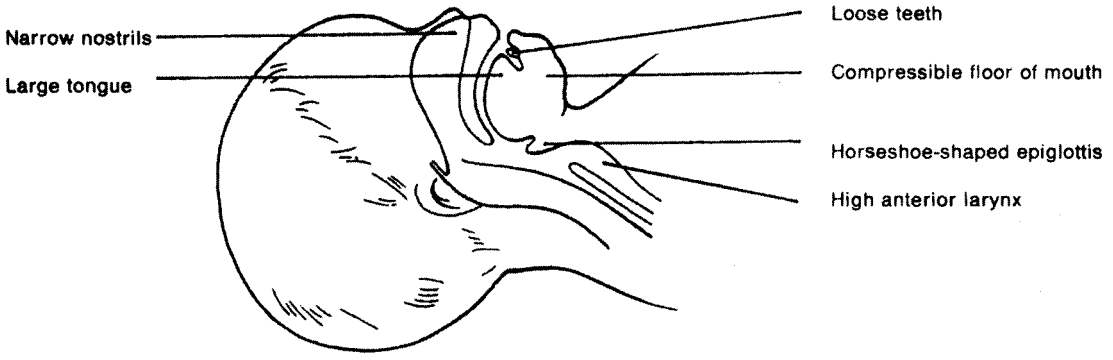


Figure 2.2 Summary of significant upper airway anatomy

Infants rely mainly on diaphragmatic breathing. Their muscles are more likely to fatigue as they have fewer type I (slow-twitch, highly oxidative, fatigue-resistant) fibres compared with adults. Pre-term infants' muscles have even less type I fibres. These children are consequently more prone to respiratory failure.

The ribs lie more horizontally in infants, and therefore contribute less to chest expansion. In the injured child, the compliant chest wall may allow serious parenchymal injuries to occur without necessarily incurring rib fractures. For multiple rib fractures to occur the force must be very large; the parenchymal injury that results is consequently very severe and flail chest is tolerated badly.

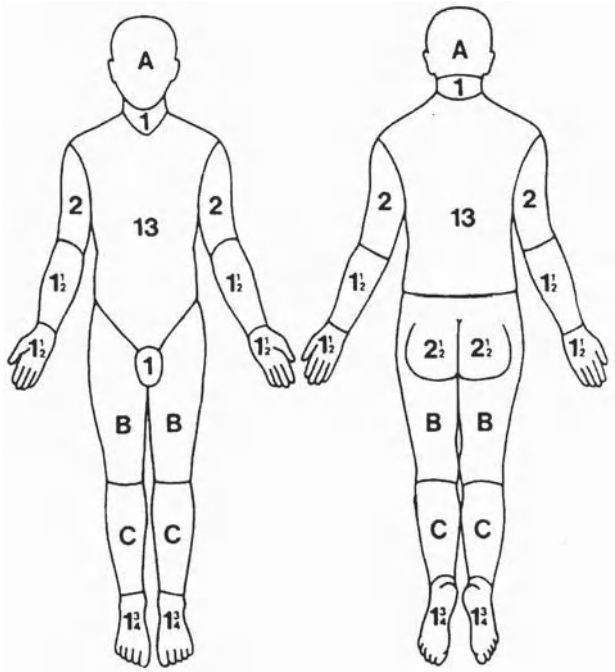
Circulation

At birth the two cardiac ventricles are of similar weight; by 2 months of age the RV : LV weight ratio is 0.5. These changes are reflected in the infant's electrocardiogram (ECG). During the first months of life the right ventricle (RV) dominance is apparent, but by 4–6 months of age the left ventricle (LV) is dominant. As the heart develops during childhood, the sizes of the P wave and QRS complex increase, and the P–R interval and QRS duration become longer.

The child's circulating blood volume per kilogram of body weight (70–80 ml/kg) is higher than that of an adult, but the actual volume is small. This means that in infants and small children, relatively small absolute amounts of blood loss can be critically important.

Body surface area

The body surface area (BSA) to weight ratio decreases with increasing age. Small children, with a high ratio, lose heat more rapidly and consequently are relatively more prone to hypothermia. At birth the head accounts for 19% of BSA; this falls to 9% by the age of 15 years. Figure 2.3 shows these changes.



Area indicated	Surface area at				
	0 year	1 year	5 years	10 years	15 years
A	9.5	8.5	6.5	5.5	4.5
B	2.75	3.25	4.0	4.5	4.5
C	2.5	2.5	2.75	3.0	3.25

Figure 2.3 Body surface area (%). (Reproduced courtesy of Smith & Nephew Pharmaceuticals)

2.4 PHYSIOLOGICAL

Respiratory

The infant has a relatively greater metabolic rate and oxygen consumption. This is one reason for an increased respiratory rate. However, the tidal volume remains relatively constant in relation to body weight (5–7 ml/kg) through to adulthood. The work of breathing is also relatively unchanged at about 1% of the metabolic rate, although it is increased in the pre-term infant.

In the adult, the lung and chest wall contribute equally to the total compliance. In the newborn, most of the impedance to expansion is due to the lung, and is critically dependent on the surfactant. The lung compliance increases over the first week of life as fluid is removed from the lung. The child's compliant chest wall leads to prominent sternal recession and rib space indrawing when the airway is obstructed or lung compliance decreases. It also allows the intrathoracic pressure to be less 'negative'. This reduces small-airway patency. As a result, the lung volume at the end of expiration is similar to the closing volume (the volume at which small-airway closure starts to take place).

At birth, the oxygen dissociation curve is shifted to the left and P_{50} (Po_2 at 50% oxygen saturation) is greatly reduced. This is due to the fact that 70% of the haemoglobin (Hb) is in the form of HbF; this gradually declines to negligible amounts by the age of 6 months.

The immature infant lung is also more vulnerable to insult. Following prolonged ventilation of a pre-term infant, bronchopulmonary dysplasia may cause oxygen dependence for up to a year. Many infants who have suffered from bronchiolitis remain 'chesty' for a year or more.

Table 2.2 shows respiratory rate by age at rest.

Table 2.2 Respiratory rate by age at rest

Age (years)	Respiratory rate (breaths per minute)
<1	30–40
1–2	25–35
2–5	25–30
5–12	20–25
>12	15–20

Cardiovascular

The infant has a relatively small stroke volume (1.5 ml/kg at birth) but has the highest cardiac index seen at any stage of life (300 ml/min/kg). Cardiac index decreases with age and is 100 ml/min/kg in adolescence and 70–80 ml/min/kg in the adult. At the same time the stroke volume increases as the heart gets bigger. As cardiac output is the product of stroke volume and heart rate, these changes underlie the heart rate changes seen during childhood (Table 2.3).

Table 2.3 Heart rate by age

Age (years)	Heart rate (beats per minute)
<1	110–160
1–2	100–150
2–5	95–140
5–12	80–120
>12	60–100

Normal systolic pressures are shown in Table 2.4. Expected systolic blood pressure (BP) can be estimated by the following formula: BP = 85 + (age in years × 2) for the 50th centile; and the 5th centile for blood pressure can be estimated from: BP = 65 + (age in years × 2). BP varies within any age group by height and these values are for the 50th height centile, with an 8–9 mmHg difference between the 5th and 95th height centiles at any age for boys, and a 6–7 mmHg difference for girls. Thus for boys/girls on the 25th centile for height, you remove 2 or 1.5 mmHg from 85 mmHg for the mean/50th centile respectively.

Table 2.4 Systolic blood pressure by age

Age (years)	Systolic BP (mmHg) 5th centile	Systolic BP (mmHg) 50th centile
<1	65–75	80–90
1–2	70–75	85–95
2–5	70–80	85–100
5–12	80–90	90–110
>12	90–105	100–120

As the stroke volume is small and relatively fixed in infants, cardiac output is directly related to heart rate. The practical importance of this is that the response to volume therapy is blunted because stroke volume cannot increase greatly to improve cardiac output. By the age of 2 years myocardial function and response to fluid are similar to those of an adult.

Systemic vascular resistance rises after birth and continues to do so until adulthood is reached. This is reflected in the changes seen in blood pressure (Table 2.4).

Immune function

At birth the immune system is immature and, consequently, babies are more susceptible than older children to many infections such as bronchiolitis, septicaemia, meningitis and urinary tract infections. Maternal antibodies acquired across the placenta provide some early protection but these progressively decline during the first 6 months. These are replaced slowly by the infant's antibodies as he or she grows older. Breastfeeding provides some protection against respiratory and gastrointestinal infections.

2.5 PSYCHOLOGICAL

Children vary enormously in their intellectual ability and their emotional response. A knowledge of child development assists in understanding a child's behaviour and formulating an appropriate management strategy. Particular challenges exist in communicating with children and as far as possible easing their fear of the circumstances they find themselves in.

Communication

Infants and young children either have no language ability or are still developing their speech. This causes difficulty when symptoms such as pain need to be described. Even children who are usually fluent may remain silent. Information has to be gleaned from the limited verbal communication, and from the many non-verbal cues (such as facial expression and posture) that are available. Older children are more likely to understand aspects of their illness and treatment and so be reassured by adequate age-appropriate communication.

Fear

Many emergency situations, and many other situations that adults would not classify as emergencies, engender fear in children. This causes additional distress to the child and adds to parental anxiety. Physiological parameters, such as pulse rate and respiratory rate, are often raised because of it, and this in turn makes clinical assessment of pathological processes such as shock, more difficult.

Fear is a particular problem in the pre-school child who often has a 'magical' concept of illness and injury. This means that the child may think that the problem has been caused by some bad wish or thought that he or she has had. School-age children and adolescents may have fearsome concepts of what might happen to them in hospital because of ideas they have picked up from adult conversation, films and television.

Knowledge allays fear and it is therefore important to explain things as clearly as possible to the child. Explanations must be phrased in a way that the child can understand. Play can be used to do this (e.g. applying a bandage to a teddy first), and also helps to maintain some semblance of normality in a strange and stressful situation. Finally, parents must be allowed to stay with the child at all times; their absence from the child's bedside will only add further fears, both to the child and to the parents themselves.

2.6 SUMMARY

- Absolute size and relative body proportions change with age.
- Observations on children must be related to their age.
- Therapy in children must be related to their age and weight.
- The special psychological needs of children must be considered.

CHAPTER 3

Structured approach to emergency paediatrics

3.1 INTRODUCTION

The reception of a child with a life-threatening condition into the emergency department or the collapse of a child on the ward or in a GP clinic presents a major challenge to staff. The infrequency and, the often unforeseen, nature of the events adds to the anxiety for all. The structured approach will enable a clinician to manage emergencies to the best extent possible and assist in ensuring that vital steps are not forgotten.

The structured approach focuses initially on identifying and treating any *immediate threats to life*: that is a closed or obstructed airway, absent or distressed respiration, or pulselessness or shock. Clinical interventions to reverse these immediate threats comprise *resuscitation*.

After resuscitation is commenced the next step is to identify the *key features* that in any serious illness or injury give the clinician a signpost to the likeliest working diagnosis. From this, the best *emergency treatment* can be identified to start to treat the child's illness or injury.

The final phase of the structured approach is to *stabilise* the child, focusing on achieving homeostasis and system control and leading onto *transfer* to a definitive care environment, which will often be the paediatric intensive care unit.

Figure 3.1 shows the structured approach in diagrammatic form. Throughout this text the same structure will be used so the clinician will become familiar with the approach and be able to apply it to any clinical emergency situation.

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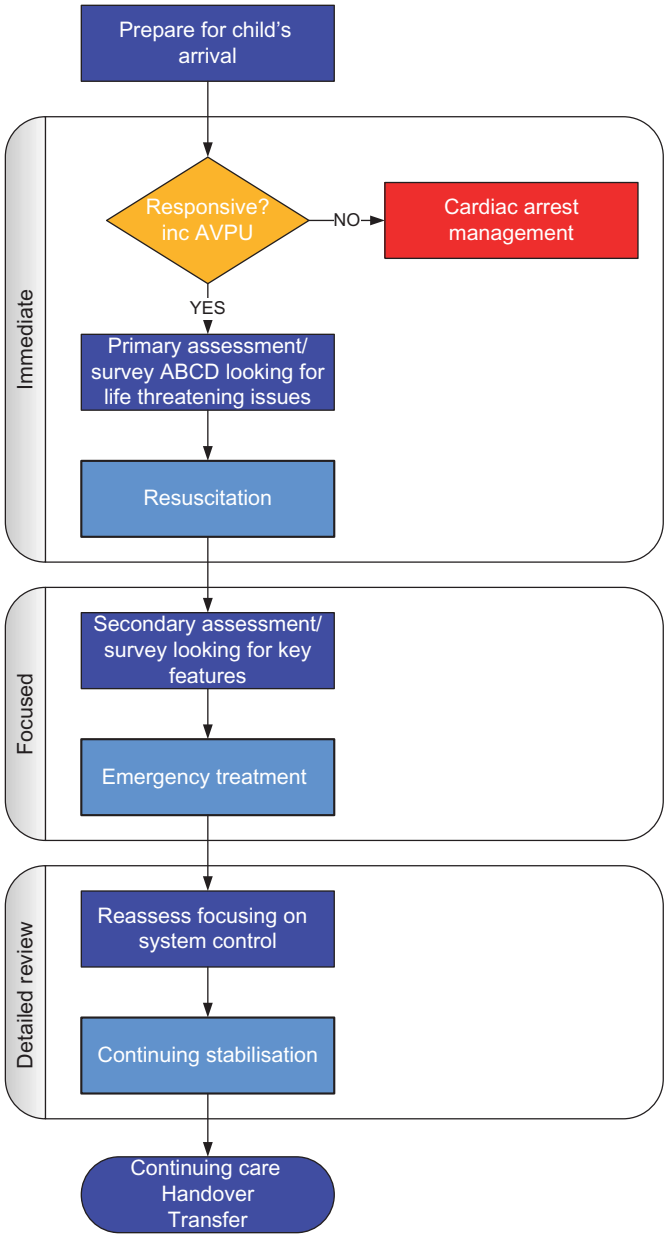

Figure 3.1 The structured approach to emergency paediatrics

3.2 PREPARATION

If warning has been received of the child's arrival then preparations can be made:

- Ensure that appropriate help is available: critical illness and injury need a team approach.
- Work out the likely drug, fluid and equipment needs.

For unexpected emergencies, ensure that all areas where children may be treated are stocked with the drugs, fluid and equipment needed for any childhood emergencies.

3.3 TEAMWORK

Nowhere is a well-functioning team more vital than in the emergency situation. Success depends on each team member carrying out his or her own tasks and being aware of the tasks

and the skills of other team members. The whole team must be under the direction of a team leader. Scenario practice by teams who work together is an excellent way to keep up skills, knowledge and team coordination in preparation for the 'real thing'.

3.4 COMMUNICATION

In the previous chapter, issues about communication with the ill or injured child were highlighted. Communication is no less important with families and with clinical colleagues. When things have gone wrong a fault in communication has often been involved. Contemporaneous recording of clinical findings, of the child's history and of test results and management plans seems obvious but in the emergency situation may be overlooked. A template for note keeping can be found in Chapter 13.

3.5 CONSENT

Consent legislation and practice are complex areas: different jurisdictions have different rulings. The general approach is that in an emergency where you consider that it is in the child's best interests to proceed, you may treat the child, provided it is limited to that treatment which is reasonably required in that emergency.

In the UK, the General Medical Council provides guidance for doctors, and hospitals will have internal policies with which you should be familiar.

PART 2

Life support

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CHAPTER 4

Basic life support

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to assess the collapsed patient and perform basic life support

4.1 INTRODUCTION

Paediatric basic life support (BLS) is not simply a scaled-down version of that provided for adults, although, where possible, guidelines are the same for all ages to aid teaching and retention. Some of the techniques employed need to be varied according to the size of the child. A somewhat artificial line is generally drawn between infants (less than 1 year old) and children (between 1 year and puberty), and this chapter follows that approach. The preponderance of hypoxic causes of paediatric cardiorespiratory arrest means that oxygen delivery rather than defibrillation is the critical step in children. This underlines the major differences with the adult algorithm.

By applying the basic techniques described, a single rescuer can support the vital respiratory and circulatory functions of a collapsed child with no equipment.

Basic life support is the foundation on which advanced life support is built. Therefore it is essential that all advanced life support providers are proficient at basic techniques, and that they are capable of ensuring that basic support is provided continuously and well during resuscitation.

4.2 PRIMARY ASSESSMENT AND RESUSCITATION

Once the child has been approached safely and a simple test for unresponsiveness has been carried out, assessment and treatment follow the familiar ABC pattern. The overall sequence of BLS in paediatric cardiopulmonary arrest is summarised in Figure 4.1. Note: this guidance is for one or more health professionals. BLS guidance for lay people can be found in a later section (see p. 29).

The initial approach: safety, stimulate, shout (SSS)

In the external environment, it is essential that the rescuer does not become a second victim, and that the child is removed from continuing danger as quickly as possible. These considerations should precede the initial airway assessment. Within a health care setting the likelihood of risk is decreased and help should be summoned as soon as the victim is found to be unresponsive. The steps are summarised in Figure 4.2.

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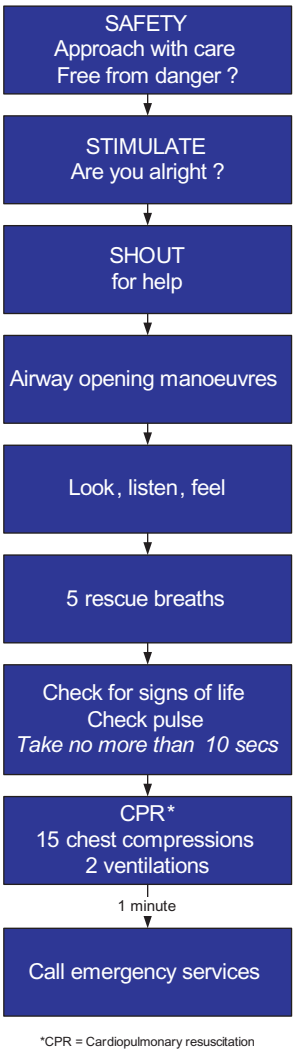

Figure 4.1 The overall sequence of basic life support in cardiopulmonary arrest

Figure 4.2 The initial (SSS) approach

When more than one rescuer is present one starts BLS while another activates the emergency medical services (EMS) system and then returns to assist in the BLS effort. If there is only one rescuer and no help has arrived after 1 minute of cardiopulmonary resuscitation (CPR) then the rescuer must activate the EMS system him- or herself. In the case of a baby or small child the rescuer will probably be able to take the victim with him or her to a telephone whilst attempting to continue CPR on the way.

Phone first

In a few instances the sequence in the above paragraph is reversed. As previously described, in children, respiratory and circulatory causes of cardiac arrest predominate, and immediate respiratory and circulatory support as provided by the breaths and chest compressions of BLS can be life saving. However, there are circumstances in which early defibrillation may be life saving, i.e. cardiac arrests caused by arrhythmia. On these occasions, where there is more than one rescuer, one may start BLS and another summons the EMS as above. But if there is a lone rescuer then he or she should activate the EMS system first on witnessing the collapse and then start BLS afterwards.

The clinical indication for EMS activation before BLS by a lone rescuer include:

- Witnessed sudden collapse with no apparent preceding morbidity.
- Witnessed sudden collapse in a child with a known cardiac condition and in the absence of a known or suspected respiratory or circulatory cause of arrest.

The increasingly wide availability of public access defibrillation programmes with automatic external defibrillators (AEDs) may result in a better outcome for this small group (see below, p. 29).

Are you alright?

The initial simple assessment of responsiveness consists of asking the child 'Are you alright?' and *gently* applying a stimulus such as holding the head and shaking the arm. This will avoid exacerbating a possible neck injury whilst still waking a sleeping child. Infants and very small children who cannot talk yet, and older children who are very scared, are unlikely to reply meaningfully, but may make some sound or open their eyes to the rescuer's voice or touch.

Airway (A)

An obstructed airway may be the primary problem, and correction of the obstruction can result in recovery without further intervention. If a child is not breathing it may be because the airway has been blocked by the tongue falling back and obstructing the pharynx. An attempt to open the airway should be made using the head tilt/chin lift manoeuvre. The rescuer places the hand nearest to the child's head on the forehead and applies pressure to tilt the head back gently. The desirable degrees of tilt are *neutral* in the infant and *sniffing* in the child. These are shown in Figures 4.3 and 4.4.

If a child is having difficulty breathing, but is conscious, then transport to hospital should be arranged as quickly as possible. A child will often find the best position to maintain his or her own airway, and should not be forced to adopt a position that may be less comfortable. Attempts to improve a partially maintained airway in a conscious child in an environment where immediate advanced support is not available can be dangerous, because total obstruction may occur.

Place the hand nearest to the child's head on the forehead and apply pressure to tilt the head back gently. The fingers of your other hand should then be placed under the chin and the chin should be lifted upwards. Care should be taken not to injure the soft tissue by gripping too hard. As this action can close the child's mouth, it may be necessary to use the thumb of the same hand to part the lips slightly. In the infant, the head is placed in the neutral position; in the child, the head should be in the 'sniffing' position.

The patency of the airway should then be assessed. This is done by:

LOOKing	for chest and/or abdominal movement
LISTENing	for breath sounds
FEELing	for breath

and is best achieved by the rescuer placing his or her face above the child's, with the ear over the nose, the cheek over the mouth and the eyes looking along the line of the chest for up to 10 seconds.

If the head tilt/chin lift manoeuvre is not possible or is contraindicated because of suspected neck injury, then the jaw thrust manoeuvre can be performed. This is achieved by placing two

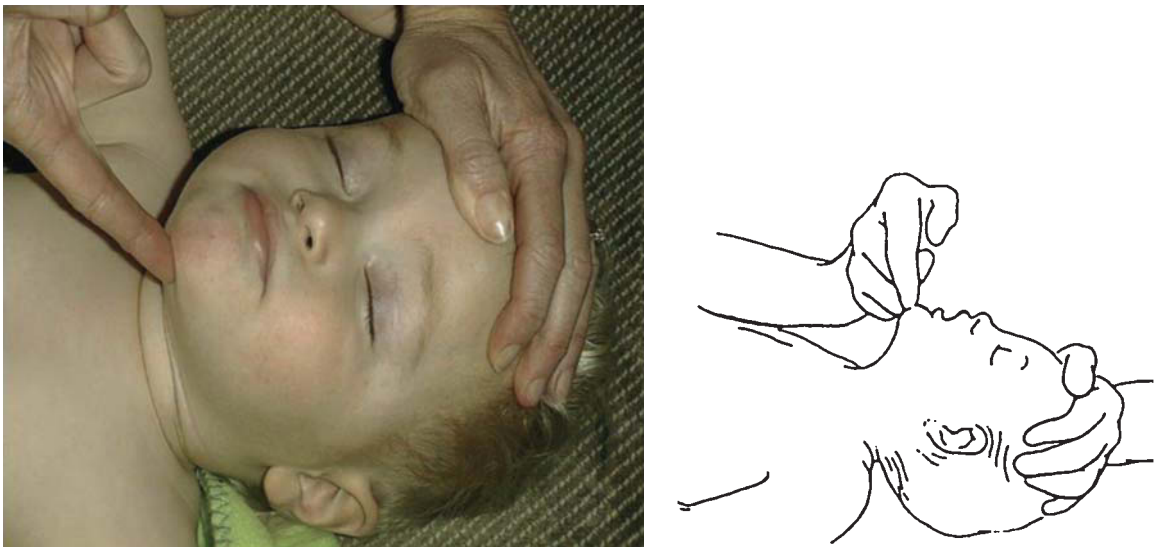

Figure 4.3 Head tilt and chin lift in infants

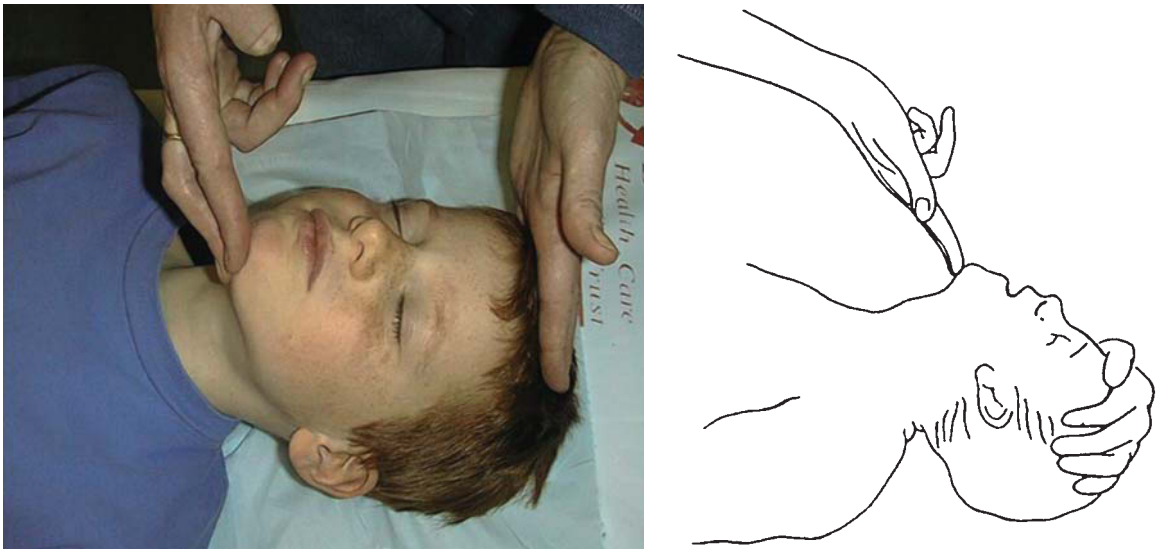

Figure 4.4 Head tilt and chin lift in children

or three fingers under the angle of the mandible bilaterally and lifting the jaw upwards. This technique may be easier if the rescuer's elbows are resting on the same surface as the child is lying on. A small degree of head tilt may also be applied if there is no concern about neck injury. This is shown in Figure 4.5.

As before, the success or failure of the intervention is assessed using the technique described above:

LOOK
LISTEN
FEEL

It should be noted that, if there is a history of trauma, then the head tilt/chin lift manoeuvre may exacerbate cervical spine injury. In general, the safest airway intervention in these circumstances is the jaw thrust without head tilt. However, on rare occasions, it may not be possible to control the airway with a jaw thrust alone in trauma. In these circumstances, an open

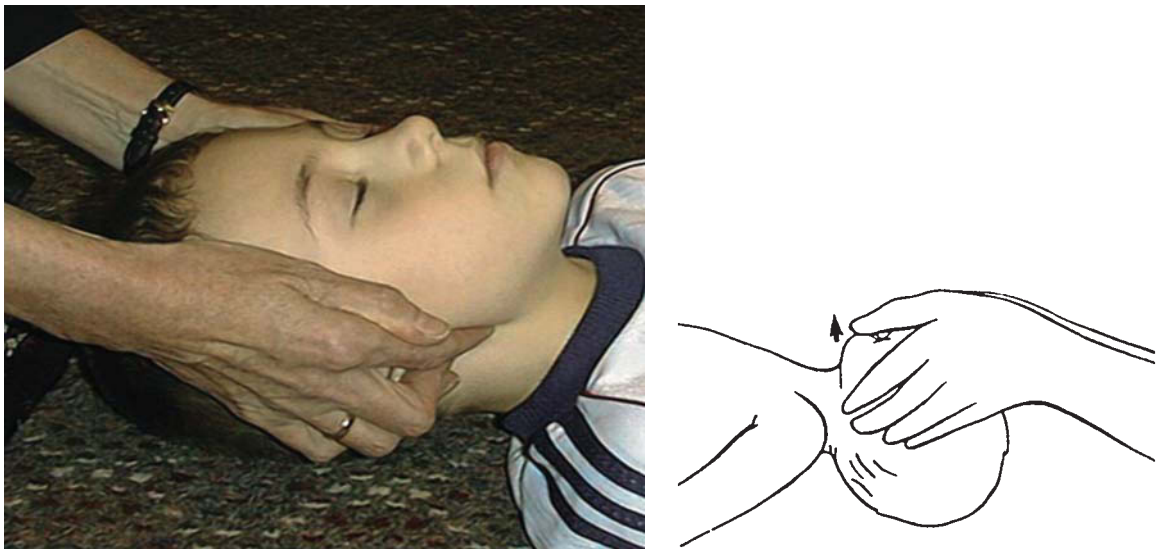

Figure 4.5 Jaw thrust

airway takes priority over cervical spine risk and a gradually increased degree of head tilt may be tried. Cervical spine control should be achieved by a second rescuer maintaining in-line cervical stabilisation throughout.

The blind finger sweep technique should not be used in children. The child's soft palate is easily damaged, and bleeding from within the mouth can worsen the situation. Furthermore, foreign bodies may be forced further down the airway; they can become lodged below the vocal cords (vocal folds) and be even more difficult to remove. In the child with a tracheostomy, additional procedures are necessary (see Section 20.7).

Breathing (B)

If normal breathing starts after the airway is open, turn the child onto his side in the recovery position (see later), maintaining the open airway. Send or go for help and continue to monitor the child for normal breathing. If the airway opening techniques described above do not result in the resumption of adequate breathing within 10 seconds, exhaled air resuscitation should be commenced. The rescuer should distinguish between adequate breathing and ineffective, gasping or obstructed breathing. If in doubt, attempt rescue breathing.

Five initial rescue breaths should be given.

While the airway is kept open as described above, the rescuer breathes in and seals his or her mouth around the victim's mouth (for a child), or mouth and nose (for an infant, as shown in Figure 4.6). If the mouth alone is used then the nose should be pinched closed using the

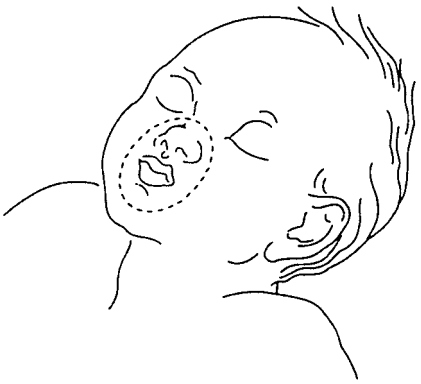

Figure 4.6 Mouth to mouth-and-nose in an infant

thumb and index fingers of the hand that is maintaining the head tilt. Slow exhalation (1–1.5 seconds) by the rescuer should make the victim's chest rise as much as normal – too vigorous a breath will cause gastric inflation and increase the chance of regurgitation of stomach contents into the lungs. The rescuer should take a breath between rescue breaths to maximise oxygenation of the victim.

If the rescuer is unable to cover the mouth and nose in an infant, he or she may attempt to seal only the infant's nose or mouth with his or her mouth and should close the infant's lips or pinch the nose to prevent air escape.

General guidance for exhaled air resuscitation
- The chest should be seen to rise
- Inflation pressure may be higher because the airway is small
- Slow breaths at the lowest pressure reduce gastric distension
- Firm, gentle pressure on the cricoid cartilage may reduce gastric insufflation

If the chest does not rise then the airway is not clear. The usual cause is failure to apply correctly the airway opening techniques discussed above. Thus, the first thing to do is to readjust the head tilt/chin lift position, and try again. If this does not work a jaw thrust should be tried. It is quite possible for a single rescuer to open the airway using this technique and perform exhaled air resuscitation; however, if two rescuers are present one should maintain the airway whilst the other breathes for the child. Five rescue breaths are given. While performing rescue breaths, note any gag or cough response to your action. These responses, or their absence, will form part of your assessment of 'signs of life' described below.

Failure of both head tilt/chin lift and jaw thrust should lead to the suspicion that a foreign body is causing the obstruction, and appropriate action should be taken (see Section 4.4).

Circulation (C)

Once the rescue breaths have been given as above, attention should be turned to the circulation.

Assessment

Failure of the circulation is recognised by the absence of signs of circulation ('signs of life'), i.e. no normal breaths or cough in response to rescue breaths and no spontaneous movement. In addition, the absence of a central pulse for up to 10 seconds or the presence of a pulse at an insufficient rate may be detected.

Even experienced health professionals can find it difficult to be certain that the pulse is absent within 10 seconds. Therefore, the absence of 'signs of life' is the primary indication to start chest compressions. Signs of life include: movement, coughing or normal breathing (not agonal gasps – these are irregular, infrequent breaths).

In children the carotid artery in the neck or the femoral artery in the groin can be palpated. In infants the neck is generally short and fat and the carotid artery may be difficult to identify. Therefore the brachial artery in the medial aspect of the antecubital fossa (Figure 4.7), or the femoral artery in the groin can be felt.

If the pulse is absent for up to 10 seconds or is inadequate (less than 60 beats per minute, with signs of poor perfusion) then cardiac compression is required. Signs of poor perfusion include pallor, lack of responsiveness and poor muscle tone.

Start chest compressions if
- There are no signs of life.
- There is no pulse.
- There is a slow pulse (less than 60 beats per minute with poor perfusion).

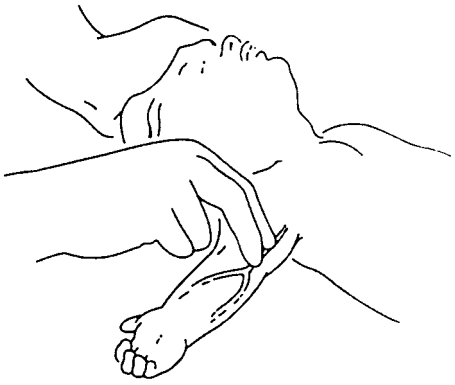

Figure 4.7 Feeling for the brachial pulse

In the absence of signs of life, chest compressions must be started unless you are **certain** that you can feel a pulse of more than 60 beats per minute within 10 seconds. 'Unnecessary' chest compressions are almost never damaging and it is important not to waste vital seconds before starting them. If 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 effect the child must be placed lying flat on his or her back, on a hard surface.

Children vary in size, and the exact nature of the compressions given should reflect this. In general, infants (less than 1 year old) require a technique different from children up to puberty in whom the method used in adults can be applied with appropriate modifications for their size. Compressions should be at least one-third of the depth of the child's or infant's chest.

Position for chest compressions

Chest compressions should compress the lower half of the sternum. Ensure that the chest wall fully recoils before the next compression starts.

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 carried out, as shown in Figure 4.8. This method is only possible when there are two rescuers, as the time needed to reposition the airway precludes its use 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, employing the other hand to maintain the airway position as shown in Figure 4.9.

Children Place the heel of one hand over the lower half of the sternum. Lift the fingers to ensure that pressure is not applied over the child's ribs. Position yourself vertically above the child's chest and, with your arm straight, compress the sternum to depress it by at least one-third of the depth of the chest (Figure 4.10). For larger children, or for small rescuers, this may be achieved most easily by using both hands with the fingers interlocked (Figure 4.11). The rescuer may choose one or two hands to achieve the desired compression of at least one-third of the depth of the chest.

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

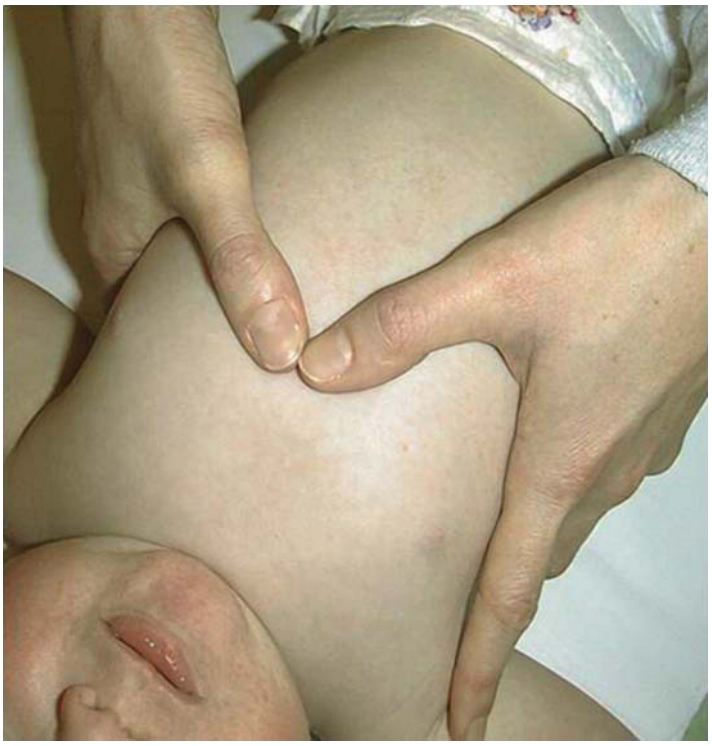

Figure 4.8 Infant chest compression: hand-encircling technique

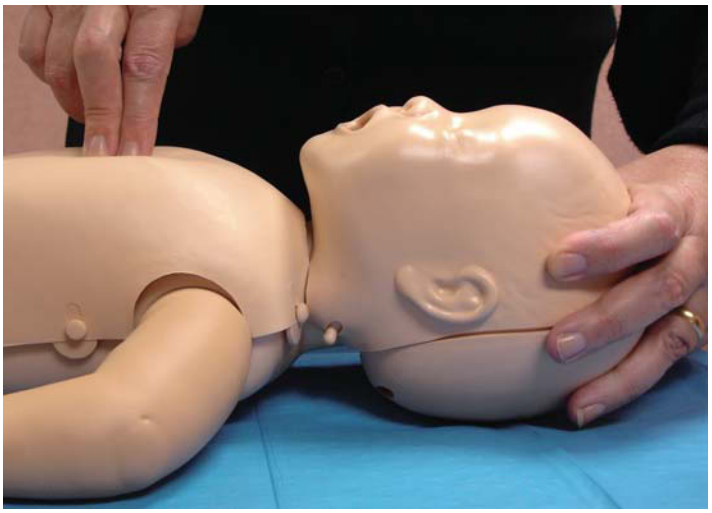

Figure 4.9 Infant chest compression: two-finger technique

Compression : ventilation ratios

Experimental work has shown that coronary perfusion pressure in resuscitation increases if sequences of compressions are prolonged rather than curtailed. Equally, ventilations are a vital part of all resuscitation and are needed early especially in the hypoxic/ischaemic arrests characteristic of childhood. Once basic life support has started interruptions to chest compressions should only be for ventilations. Pausing compressions will decrease coronary perfusion pressure to zero and several compressions will be required before adequate coronary perfusion recurs. There is no experimental evidence to support any particular ratio in childhood but a 15:2 ratio has been validated by experimental and mathematical studies and is the recommended ratio for health care professionals.

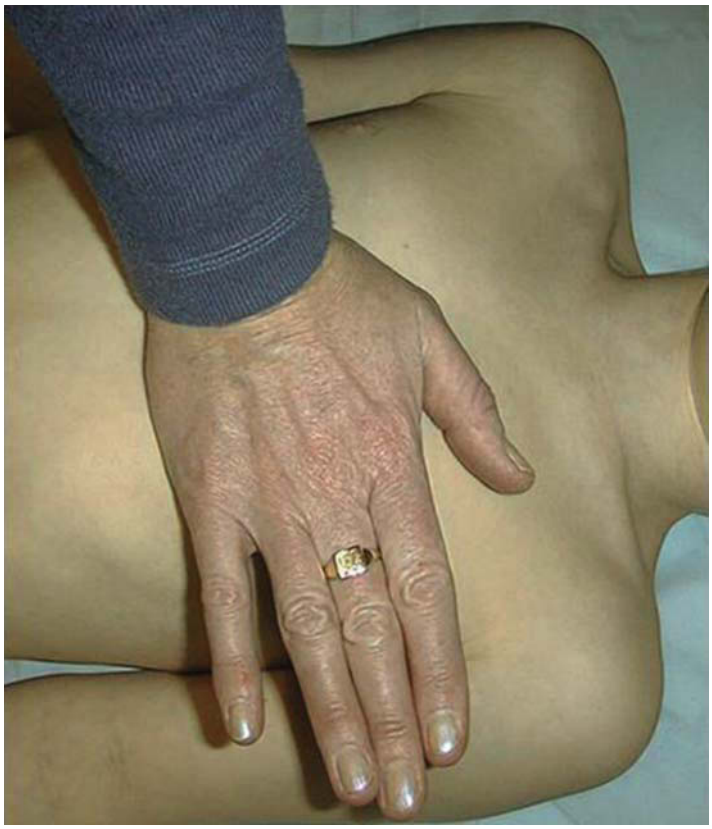

Figure 4.10 Chest compression: one-handed technique

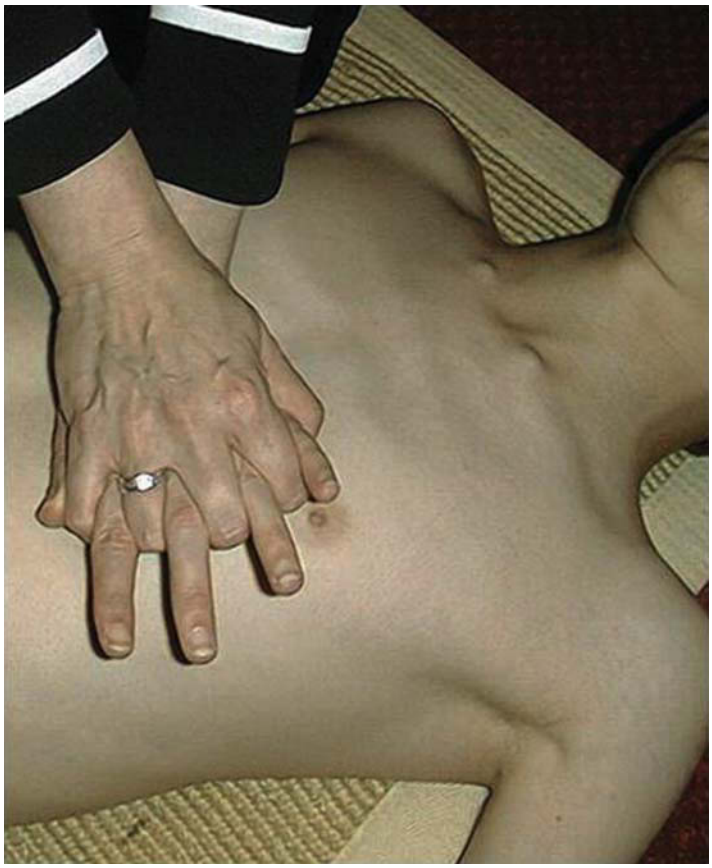

Figure 4.11 Chest compression: two-handed technique

Table 4.1 Summary of basic life support techniques in infants and children

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

Continuing cardiopulmonary resuscitation

The compression rate at all ages is 100–120 per minute. A ratio of 15 compressions to two ventilations is maintained whatever the number of rescuers. If no help has arrived the emergency services must be contacted after 1 minute of CPR. 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. *Apart from this interruption to summon help, basic life support must not be interrupted unless the child moves or takes a breath*.

Research on the delivery of CPR has shown that rescuers tend to compress too slowly and too gently. So the current emphasis is on CPR which is 'hard and fast' with compressions of at least one-third of the victim's anteroposterior chest diameter and a rate of between 100 and 120 compressions a minute, minimising interruptions as completely as possible. 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 very real problem for the solo rescuer, and there is no easy solution. In the infant and small child, the free hand can maintain the head position. The correct position for compressions does not need to be remeasured after each ventilation.

The CPR manoeuvres recommended for infants and children are summarised in Table 4.1.

Age definitions

As the techniques of CPR have been simplified there is now no need to distinguish between different ages of children but only between infants (under 1 year) and children (from 1 year to puberty). It is clearly inappropriate and also unnecessary to establish the physical evidence for puberty at CPR. The rescuer should use paediatric guidelines if he or she believes the victim to be a child. If the victim is, in fact, a young adult, no harm will be caused as the aetiology of cardiac arrest is, in general, similar in this age group to that in childhood, i.e. hypoxic/ischaemic rather than cardiac in origin.

Recovery position

No specific recovery position has been identified for children. The child should be placed in a stable, lateral position that ensures maintenance of an open airway with free drainage of fluid from the mouth, the ability to monitor and gain access to the patient, security of the cervical spine and attention to pressure points.

The following is a description of the technique for adults and is suitable for children:

- Kneel beside the victim and make sure that both his legs are straight.
- Place the arm nearest to you out at right angles to his body, elbow bent with the hand palm-up.
- Bring the far arm across the chest, and hold the back of the hand against the victim'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 his hand pressed against his cheek, pull on the far leg to roll the victim towards you on to his side.
- Adjust the upper leg so that both the hip and knee are bent at right angles.
- Tilt the head back to make sure that the airway remains open.
- If necessary, adjust the hand under the cheek to keep the head tilted and facing downwards to allow liquid material to drain from the mouth.
- Check breathing regularly.
- If the victim has to be kept in the recovery position for more than 30 minutes turn him to the opposite side to relieve the pressure on the lower arm.

Lay rescuers

It has become clear that bystanders often do not undertake BLS because they are afraid to do it wrongly and because of an anxiety about performing mouth-to-mouth resuscitation on strangers. For lay rescuers, therefore, the adult compression : ventilation ratio of 30 compressions to two ventilations is recommended for children as well as adults, thus simplifying the guidance. To increase the appropriateness for children, lay rescuers can be advised to precede their efforts by five rescue breaths if the victim is a child. If lay rescuers are unable or unwilling to perform mouth-to-mouth resuscitation they may perform compression-only CPR. Single health care professional rescuers are also encouraged to perform a ratio of 30 compressions to two ventilations for children if they find difficulty in the transition from compressions to ventilations.

Automatic external defibrillators (AEDs) in children

The use of the AED is now included in basic life support teaching for adults because early defibrillation is the most effective intervention for the large majority of unpredicted cardiac arrests in adults. As has been stated, in children and young people circulatory or respiratory causes of cardiac arrest predominate. However, in certain circumstances (described in Section 4.2) children may suffer a primary cardiac cause for cardiac arrest, and the use of an AED may be life saving. Recently there has been a large increase in the number of AEDs, together with trained operators, made available in public places such as airports, places of entertainment and shops, so the opportunity for their use will correspondingly increase.

In this text, the discussion of the use of AEDs with regard to children will be found in Chapter 6.

4.3 BASIC LIFE SUPPORT AND INFECTION RISK

There have been a few reports of transmission of infectious diseases from casualties to rescuers during mouth-to-mouth resuscitation. The most serious concern in children is meningococcus, and rescuers involved in the resuscitation of the airway in such patients should take standard prophylactic antibiotics (rifampicin or ciprofloxacin). Tuberculosis can be transmitted during CPR and appropriate precautions should be taken when this is suspected.

There have been no reported cases of transmission of human immunodeficiency virus (HIV) through mouth-to-mouth ventilation. Blood-to-blood contact is the single most important route of transmission of such viruses, and in non-trauma resuscitations 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, semen, vaginal secretions, cerebrospinal fluid, pleural and peritoneal fluids and amniotic fluid. Precautions are also recommended if any bodily secretion contains visible blood. Devices that prevent direct contact between the rescuer and the victim (such as resuscitation masks) can be used to lower risk; gauze swabs or any other porous material placed over the victim's mouth is of no benefit in this regard.

The number of children in the UK with acquired immune deficiency syndrome (AIDS) or HIV-1 infection is less than the number of adults similarly affected. If transmission of HIV-1 does occur in the UK, it is therefore much more likely to be from the adult rescuer to the child rather than the other way around.

In countries where HIV/AIDS is more prevalent, the risk to the rescuer will be greater. In South Africa, in a medical ward 25–40% of children may be HIV-positive but the prevalence is lower in trauma cases. In the Caribbean, HIV prevalence is second only to sub-Saharan Africa. The situation may change, as effective antiretroviral agents are made available to resource-poor countries.

Although practice manikins have not been shown to be a source of infection, regular cleaning is recommended and should be carried out as shown in the manufacturer's instructions. Infection rates vary from country to country and rescuers must be aware of the local risk.

4.4 THE CHOKING CHILD

The vast majority of deaths from foreign body airway obstruction (FBAO) occur in pre-school children. Virtually anything may be inhaled, foodstuffs predominating. The diagnosis may not be clear-cut, but should be suspected if the onset of respiratory compromise is sudden and is associated with coughing, gagging and stridor.

Airway obstruction also occurs with infections such as acute epiglottitis and croup. In these cases, attempts to relieve the obstruction using the methods described below are dangerous. Children with known or suspected infectious causes of obstruction, and those who are still breathing and in whom the cause of obstruction is unclear, should be taken to hospital urgently. The treatment of these children is dealt with in Chapter 8.

If a foreign body is easily visible and accessible in the mouth then remove it, but while attempting that take great care not to push it further into the airway. Do not perform blind finger sweeps of the mouth or upper airway as these may further impact a foreign body and damage tissues without removing the object.

The physical methods of clearing the airway, described below, should therefore only be performed if:

1 The diagnosis of FBAO is clear-cut (witnessed or strongly suspected) and ineffective coughing and increasing dyspnoea, loss of consciousness or apnoea have occurred.
2 Head tilt/chin lift and jaw thrust have failed to open the airway of an apnoeic child. (The sequence of instructions is shown in Figure 4.12.)

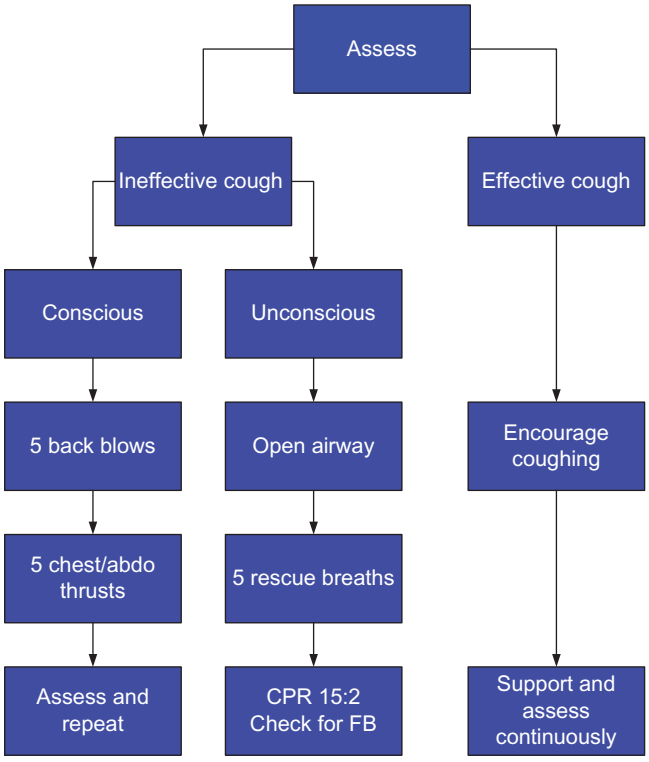

Figure 4.12 The sequence of actions in a choking child. FB, foreign body

If the child is coughing he should be encouraged. A spontaneous cough is more effective at relieving an obstruction than any externally imposed manoeuvre. An effective cough is recognised by the victim's ability to speak or cry and to take a breath between coughs. The child should be continually assessed and not left alone at this stage. No intervention should be made unless the cough becomes ineffective, that is quieter or silent, and the victim cannot cry, speak or take a breath or if he becomes cyanosed or starts to lose consciousness. Then call for help and start the intervention.

These manoeuvres are then alternated with each other and with examination of the mouth and attempted breaths as shown in Figure 4.12.

Infants

Abdominal thrusts may cause intra-abdominal injury in infants. Therefore a combination of back blows and chest thrusts is recommended for the relief of foreign body obstruction in this age group.

The baby is placed along one of the rescuer's arms in a head-down position, with the rescuer's hand supporting the infant's jaw in such a way as to keep it open, in the neutral position. The rescuer then rests his or her arm along the thigh, and delivers five back blows with the heel of the free hand.

If the obstruction is not relieved the baby is turned over and laid along the rescuer's thigh, still in a head-down position. Five chest thrusts are given – using the same landmarks as for cardiac compression but at a rate of one per second. If an infant is too large to allow use of the single-arm technique described above, then the same manoeuvres can be performed by laying the baby across the rescuer's lap. These techniques are shown in Figures 4.13 and 4.14.

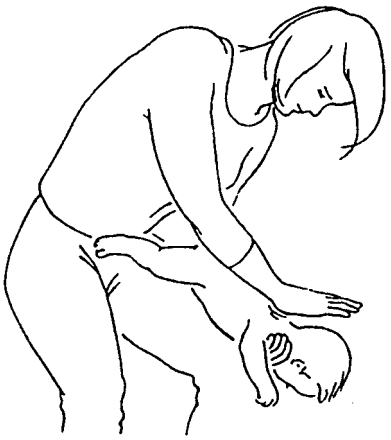

Figure 4.13 Back blows in an infant

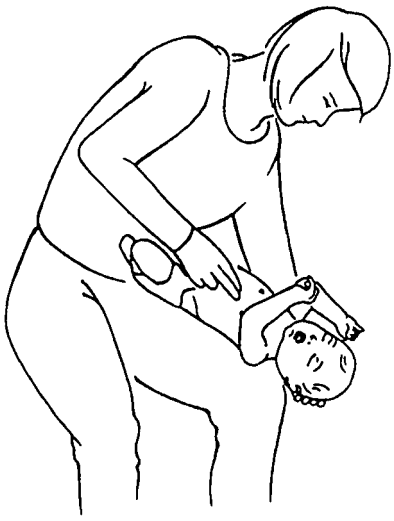

Figure 4.14 Chest thrusts in an infant

Children

Back blows can be used as in infants (Figure 4.15) or, in the case of a larger child, with the child supported in a forward leaning position. In the child the abdominal thrust (Heimlich manoeuvre) can also be used. This can be performed with the victim either standing or lying, but the former is usually more appropriate.

If this is to be attempted with the child standing, the rescuer moves behind the victim and passes his or her arms around the victim's body. Owing to the short height of children, it may be necessary for an adult to raise the child or kneel behind them to carry out the standing manoeuvre effectively. One hand is formed into a fist and placed against the child's abdomen above the umbilicus and below the xiphisternum. The other hand is placed over the fist, and both hands are thrust sharply upwards into the abdomen. This is repeated five times unless

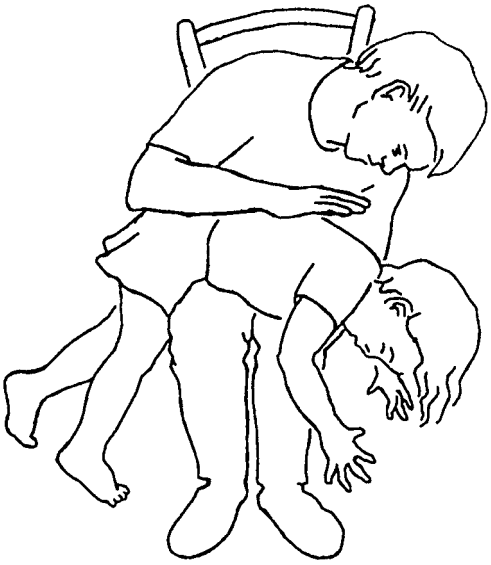

Figure 4.15 Back blows in a small child

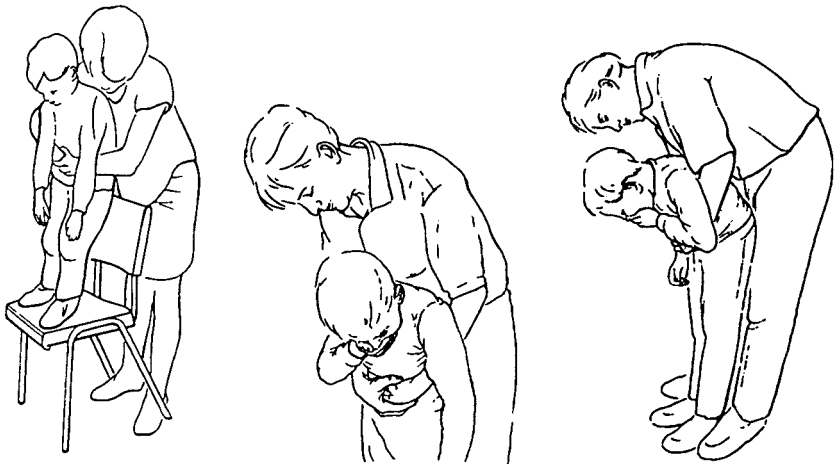

Figure 4.16 Heimlich manoeuvre in a standing child

the object causing the obstruction is expelled before then. This technique is shown in Figure 4.16.

To carry out the Heimlich manoeuvre in a supine child, the rescuer kneels at his or her feet. If the child is large it may be necessary to kneel astride him or her. The heel of one hand is placed against the child's abdomen above the umbilicus and below the xiphisternum. The other hand is placed on top of the first, and both hands are thrust sharply upwards into the abdomen, with care being taken to direct the thrust in the midline. This is repeated five times unless the object causing the obstruction is expelled before that.

Following successful relief of the obstructed airway, assess the child clinically. There may be still some part of the foreign material in the respiratory tract. If abdominal thrusts have been performed, the child should be assessed for possible abdominal injuries.

Each time breaths are attempted, look in the mouth for the foreign body and remove it if visible. Take care not to push the object further down and avoid damaging the tissues. If the obstruction is relieved the victim may still require either continued ventilations if not breathing but is moving or gagging or both ventilations and chest compressions if there are no signs of life. Advanced life support may also be needed.

If the child breathes effectively then place him in the recovery position and continue to monitor him.

Unconscious infant or child with foreign body airway obstruction

- Call for help.
- Place the child supine on a flat surface.
- Open the mouth and attempt to remove any visible object.
- Open the airway and attempt five rescue breaths, repositioning the airway with each breath if the chest does not rise.
- Start chest compressions even if the rescue breaths were ineffective.
- Continue the sequence for single rescuer CPR for about a minute then summon help again if none is forthcoming.

Each time breaths are attempted, look in the mouth for the foreign body and remove it if visible. Take care not to push the object further down and avoid damaging the tissues.

If the obstruction is relieved the victim may still require either continued ventilations if not breathing but is moving or gagging or both ventilations and chest compressions if there are no signs of life. Advanced life support may also be needed.

If the child breathes effectively then place him in the recovery position and continue to monitor him.

4.5 SUMMARY

The teaching in this chapter is consistent with the ILCOR guidelines, Resuscitation 2010, and there are an enormous number of references which have informed this process. These are available on the ALSG website. See details on p. xv.

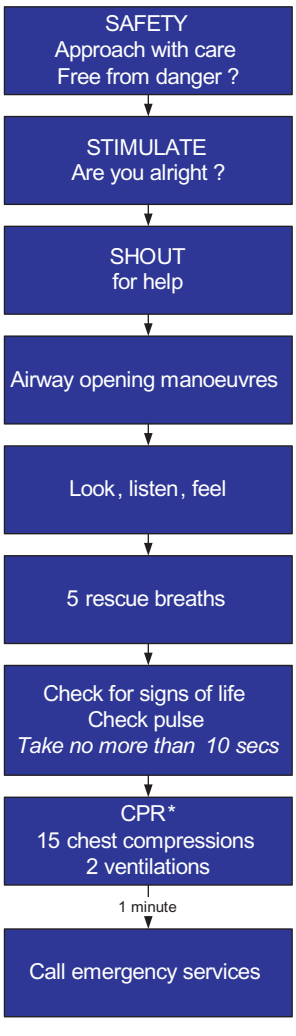

Figure 4.17 The overall sequence of basic life support in cardiopulmonary arrest

CHAPTER 5

Advanced support of the airway and ventilation

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to assess and manage the airway with adjuncts and equipment
- How to support breathing with simple equipment
- How to respond to an airway/breathing problem with a structured approach

5.1 INTRODUCTION

Management of airway and breathing has priority in the resuscitation of patients of all ages; however, the rate at which respiratory function can deteriorate in children is particularly high. Effective resuscitation techniques must be applied quickly and in order of priority. The differences between adults and children must be realised and familiarity with equipment assured. Techniques for obtaining a patent and protected airway, and for achieving adequate ventilation and oxygenation, must be learned and practised. These techniques must be integrated into a prioritised system of care. It should be stressed that basic, simple techniques are often effective and life saving.

5.2 EQUIPMENT FOR MANAGING THE AIRWAY

Necessary airway equipment
- Face masks
- Airways including laryngeal mask airways (LMAs)
- Self-inflating bag–valve–mask devices
- Tracheal tubes, introducers and connectors
- Suction devices
- Cricothyroidotomy cannulae

The airway equipment indicated in the above box should be available in all resuscitation areas. It is crucial that familiarity with it is gained before an emergency situation occurs.

Pharyngeal airways

There are two main types of pharyngeal airway: oropharyngeal and nasopharyngeal.

1 The *oropharyngeal or Guedel airway* is used in the unconscious or obtunded patient to provide a patent airway channel between the tongue and the posterior pharyngeal wall. It may also

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

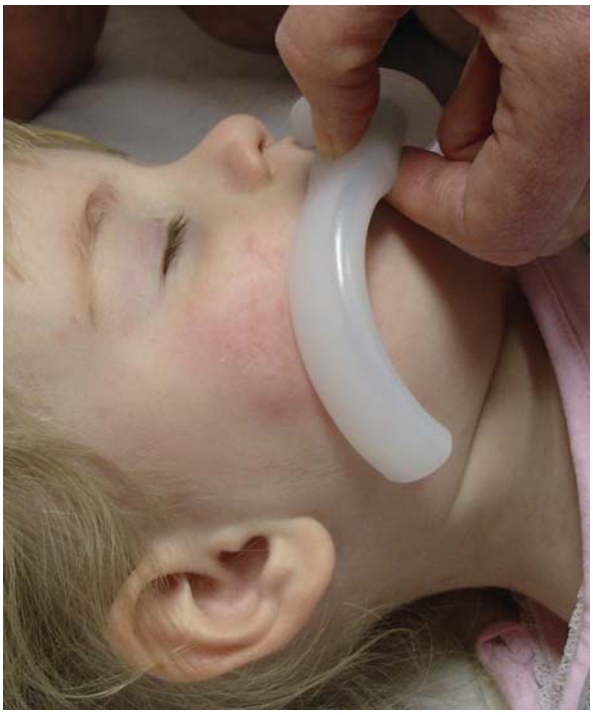

Figure 5.1 Sizing an orophyaryngeal airway

be used to stabilise the position of an oral endotracheal tube. In the awake 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 (Figure 5.1). Too small an airway may be ineffective, 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. Techniques for insertion are described in Chapter 20.

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

The acid test of success, as in all therapeutic interventions, is that insertion of one or other of these devices should result in improvement in the patient's condition. If it does not occur then a reappraisal of the choice or size of airway is urgently required.

Laryngoscopes

Two principal designs of laryngoscope for use in children exist: straight bladed and curved bladed.

1 The *straight-bladed laryngoscope* is usually employed to directly lift the epiglottis, thereby uncovering the vocal folds (Figure 5.2). The advantage of this approach is that the epiglottis is moved sufficiently so that it does not obscure the cords. The disadvantage potentially is vagal stimulation causing laryngospasm or bradycardia.

2 The *curved-bladed laryngoscope* is designed to move the epiglottis forward by lifting it from in front (Figure 5.3). 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 at obtaining a view of the cords and has the

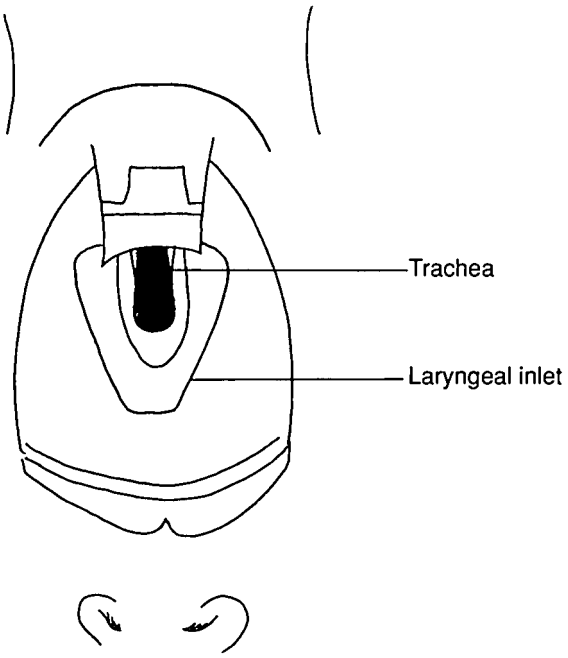

Figure 5.2 View of the larynx using a straight-bladed laryngoscope

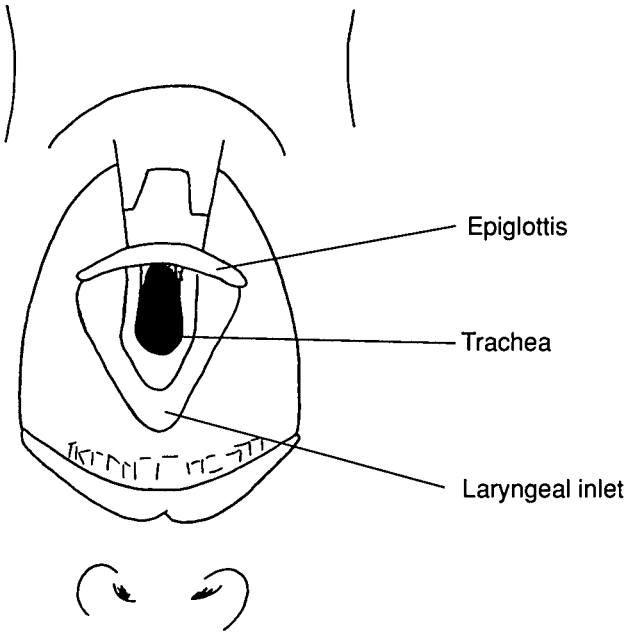

Figure 5.3 View of the larynx using a curved-blade laryngoscope

advantage that less vagal stimulation ensues, as the mucosa of the vallecula is innervated by the glossopharyngeal nerve.

A laryngoscope blade should be chosen appropriate for age. It is possible to intubate with a blade that is too long but not one that is too short.

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

Tracheal tubes

Uncuffed tubes should be used during resuscitation, by those who do not have much paediatric anaesthetic experience, for children up until approximately 10 years of age. If familiar with cuffed tube placement, both cuffed and uncuffed tubes are acceptable in infants and children undergoing emergency intubation, but not neonates. Until the age of about 10 years, 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 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.

Estimating the appropriate size of an uncuffed tracheal tube is carried out as follows:

Internal diameter (mm) = (Age/4) + 4

Length (cm) = (Age/2) + 12 for an oral tube

Length (cm) = (Age/2) + 15 for a nasal tube

These formulae are appropriate for ages over 1 year. Neonates usually require a tube of internal diameter 3–3.5 mm, although pre-term infants may need one of diameter 2.5 mm. Cuffed tubes should not be used in neonates.

For cuffed tracheal tubes, an estimate of the appropriate internal diameter for children from 2 years is carried out as follows:

Internal diameter (mm) = (Age/4) + 3.5

For infants of more than 3 kg and up to 1 year, a size 3 cuffed tube, and from 1 to 2 years a size 3.5 cuffed tube, are usually acceptable. Tracheal tubes are measured in sizes by their internal diameter in millimetres. They are provided in whole- and half-millimetre sizes. The clinician should select a tube of appropriate size but also prepare one a size smaller and one a size larger.

In the circumstance of resuscitation in a young child where the lungs are very 'stiff', for example 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, placed through the lumen of the tracheal tube. There are two types: soft and flexible or firm and malleable. The former can be allowed to project out of the tip of the tube, as long as it is handled very gently. The latter 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.

Tracheal tube connectors

In adults, the proximal end of the tube connectors is of standard size, based on the 15–22 mm system, ensuring that they can be connected to a standard self-inflating bag.

The same standard Portex system exists for children, including neonates. Smaller connectors may be used later in intensive care units (in infants) but should be avoided in the resuscitation setting.

Magill's forceps

The Magill's forceps is angled to allow a view around the forceps when in the mouth. It may be useful to help position a tube through the cords by lifting it anteriorly, or to remove pharyngeal or supraglottic foreign bodies.

Suction devices

In the resuscitation room, the usual suction device is the pipeline vacuum unit. It 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.

The most useful suction ending is the Yankauer sucker, which 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. In small infants, a suction catheter and a Y-piece are often preferred, but are less capable of removing vomit.

Portable suction devices are required for resuscitation in remote locations, and for transport to and from the resuscitation room. These are usually battery powered.

Tracheal suction catheters

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 French gauge 6.

Cricothyroidotomy cannulae and ventilation systems

Purpose-made cricothyroidotomy cannulae are available, usually in three sizes: 12 gauge for an adult, 14 gauge for a child and 18 gauge for a baby. They are less liable to kinking than intravenous cannulae and have a flange for suturing or securing to the neck.

In an emergency, an intravenous cannula can be inserted through the cricothyroid membrane and oxygen insufflated at 2 l/min to provide some oxygenation (but no ventilation). A side hole can be cut in the oxygen tubing or a Y-connector can be placed between the cannula and the oxygen supply, to allow intermittent occlusion and achieve partial ventilation, as described in Chapter 20.

5.3 EQUIPMENT FOR PROVIDING OXYGEN AND VENTILATION

The equipment for oxygenation and ventilation indicated in the box should be readily available.

Necessary equipment for oxygenation and ventilation

- Oxygen source and masks for spontaneous breathing
- Face masks (for artificial ventilator)
- Self-inflating bags
- T-piece and open-ended bag
- Mechanical ventilators
- Chest tubes
- Gastric tubes

Oxygen source and masks for spontaneous breathing

A wall oxygen supply (at a pressure of 4 atmospheres, 400 kPa or 60 p.s.i.) is provided in most resuscitation rooms. A flow meter capable of delivering at least 15 l/min should be fitted.

A mask with a reservoir bag should be used in the first instance so that a high concentration of oxygen is delivered. A simple mask or other device such as a head box may be used later on if a high oxygen concentration is no longer required. Nasal prongs are often well tolerated in the pre-school age, but they cause drying of the airway, may cause nasal obstruction in infants and provide an unreliable oxygen concentration.

Younger children are more susceptible to the drying effect of a non-humidified oxygen supply.

Face masks (for artificial ventilation)

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 child's face and have a low deadspace. Circular soft plastic masks give an excellent seal and are preferred by many. Children's masks should be clear to allow the child's colour or the presence of vomit to be seen.

The Laerdal pocket mask is a single-size, clear plastic mask with an air-filled cushion rim designed for mouth-to-mask resuscitation. It can be supplied with a port for attaching to the oxygen supply and can be used in adults and children. By using it upside down it may be used to ventilate an infant.

Self-inflating bags

Self-inflating bags come in three sizes: 250, 500 and 1500 ml. The smallest bag is ineffective except in very small babies. The two smaller sizes usually have a pressure-limiting valve set at 4.5 kPa (45 cmH_2O), which may (rarely) need to be overridden for high resistance/low compliance lungs, but which protects the normal lungs from inadvertent barotrauma. The patient end of the bag connects to a one-way valve of a fish-mouth or leaf-flap design. The opposite end has a connection to the oxygen supply and to a reservoir attachment. The reservoir enables high oxygen concentrations to be delivered. Without a reservoir bag, it is difficult to supply more than 50% oxygen to the patient whatever the fresh gas flow, whereas with one an inspired oxygen concentration of 98% can be achieved.

T-piece and open-ended bag

This equipment should only be used in children weighing up to about 20 kg. It is used frequently by anaesthetists, but is difficult to use in inexperienced hands and should not be used in a resuscitation environment. It is entirely ineffective unless a pressurised supply of fresh gas is available.

Mechanical ventilators

A detailed discussion of individual mechanical ventilators is beyond the scope of this book. If a ventilator is used, continual re-evaluation with monitoring of expired CO_2 is mandatory, as is measurement of arterial blood gases.

Chest tubes

These are included because haemothorax or pneumothorax may severely limit ventilation. They are described elsewhere (Chapter 22).

Gastric tubes

Children are prone to air swallowing and vomiting. Air may also be inadvertently forced into the stomach during bag-and-mask ventilation. This may cause vomiting, vagal stimulation and diaphragmatic splinting. A gastric tube will decompress the stomach and significantly improve both breathing and general well-being. Withholding the procedure 'to be kind to the child' may cause more distress than performing it.

5.4 PRACTICAL SKILLS

The following practical skills are described in detail in Chapter 20:

- Oropharyngeal airway insertion in the:
  - small child, and
  - older child.
- Nasopharyngeal airway insertion.
- Ventilation without intubation in:
  - mouth-to-mask ventilation, and
  - bag-and-mask ventilation.

- Orotracheal intubation (including rapid sequence induction) in the:
  - infant/small child, and
  - older child.
- Laryngeal mask airway insertion.
- Surgical airway:
  - needle cricothyroidotomy, and
  - surgical cricothyroidotomy.

Tracheal tube placement check

Following the placement of the tracheal tube in the trachea its position must be verified by:

- Observing bilateral and symmetrical movement of the chest.
- Auscultation of the chest and abdomen.
- Monitoring expired carbon dioxide in the exhaled air by either colour change capnometry or end tidal capnography.

The continued monitoring of expired carbon dioxide is a good indicator of effective ventilation in a perfusing patient, but it must be remembered that CO_2 will not be detected in the absence of any circulation or where the lungs have not yet been inflated (at birth).

Capnography is now seen to be of use during resuscitation from cardiac arrest providing information on the efficiency of chest compressions. A sudden rise in exhaled CO_2 may indicate return of spontaneous circulation (ROSC).

Laryngeal mask airway

The laryngeal mask airway (LMA) is an airway device that is widely used in adult and paediatric anaesthesia. It is also commonly used in adult resuscitation. It is an excellent device for ventilation but does not offer the protection against regurgitation and aspiration that an endotracheal tube does.

The smaller sizes used in children whilst easy to position are also easy to dislodge, and may provide a false sense of security to the clinician.

The place of the laryngeal mask airway in resuscitation of infants and children is still uncertain, although for those proficient in its use, it may be life saving in the situation of 'can't intubate, can't ventilate'. For this reason if for no other, it should be available in any area where intubation might be carried out.

A variety of other, new airway devices have become available in recent years but none of these are currently recommended for use in paediatrics.

5.5 PUTTING IT TOGETHER: AIRWAY AND BREATHING MANAGEMENT

In order to respond urgently and yet retain thoroughness, effective emergency management demands a systematic, prioritised approach. Care can be structured into the following phases.

Primary assessment

This consists of a rapid 'physiological' examination to identify immediately life-threatening emergencies. It should be completed in less than a minute. It is prioritised as shown in the box:

Airway
Breathing
Circulation
Disability (nervous system)
Exposure

From the respiratory viewpoint, do the following:
- Look, listen and feel for airway obstruction, respiratory arrest, depression or distress.
- Assess the effort of breathing.
- Count the respiratory rate.
- Listen for stridor and/or wheeze.
- Auscultate for breath sounds.
- Assess skin colour.

If a significant problem is identified, management should be started immediately. After appropriate interventions have been performed, primary assessment can be resumed or repeated.

Resuscitation

During this phase, life-saving interventions are performed. These include such procedures as intubation, ventilation, cannulation and fluid resuscitation. At the same time, oxygen is provided, vital signs are recorded and essential monitoring is established.

From the respiratory viewpoint, do the following.

Airway

- Perform basic airway-opening manoeuvres.
- Give oxygen.
- Provide suction.
- Place airway adjuncts.
- Proceed to intubation if required.

Breathing

- Establish adequate ventilation via a bag–valve mask.
- Intubate if necessary.
- Perform chest decompression if necessary.
- Consider needle cricothyroidotomy if unable to oxygenate by alternative means.
- Initiate pulse oximetry and other monitoring at this time.

Secondary assessment

This consists of a thorough physical examination, together with appropriate investigations. Before embarking on this phase, it is important that the resuscitative measures are fully under way.

From the respiratory viewpoint, do the following:
- Perform a detailed examination of the airway, neck and chest.
- Identify any swelling, bruising or wounds.
- Re-examine for symmetry of breath sounds and movement.
- Do not forget to inspect and listen to the back of the chest.

Emergency treatment

All other urgent interventions are included in this phase. If at any time the patient deteriorates, return to the primary assessment and recycle through the system.

In the very sick or critically injured child, the primary assessment and resuscitation phases become integrally bound together. As a problem is identified, care shifts to the relevant intervention, before returning to the next part of the primary assessment. The simplified airway and breathing management protocol illustrates how this integration can be achieved.

Airway and breathing management protocol

Begin airway assessment	
Assess the airway and give oxygen	
If evidence of blunt trauma:	
protect cervical spine from the outset	
If evidence of obstruction or altered consciousness:	
perform airway-opening manoeuvre	common
consider suction, foreign body removal	common
If obstruction persists:	
consider oro- or nasopharyngeal airway or LMA	common
If obstruction still persists:	
consider intubation, and if carried out check position of tracheal tube	rare
If intubation is impossible or unsuccessful:	
consider cricothyroidotomy	extremely rare
If stridor but relatively alert:	
allow self-ventilation whenever possible	
encourage oxygen but do not force to wear mask	
do not force to lie down	
do not inspect the airway (except as a definitive procedure under controlled conditions)	
assemble expert team and equipment	
Assess the breathing	
If respiratory arrest or depression:	
administer oxygen by bag–valve–mask	
consider intubation and check the position of tube if inserted	
If sedative or paralysing drugs possible:	
administer reversal agent	
If respiratory distress or tachypnoea:	
administer oxygen	
If lateralised ventilatory deficit:	
consider haemopneumothorax or inhaled foreign body	
lung consolidation, collapse or effusion	
If chest injury:	
consider tension pneumothorax and massive haemothorax	
flail segment, open pneumothorax	
If evidence of tension pneumothorax:	
perform immediate needle decompression	
follow up with chest drain	
If evidence of massive haemothorax:	
insert chest drain	
commence blood volume replacement (simultaneously if possible)	
If wheeze or crackles:	
consider asthma, bronchiolitis, pneumonia, heart failure	
remember inhaled foreign body	
If evidence of acute severe asthma:	
give inhaled or intravenous -agonists	
give steroids and consider aminophylline	
Continue primary assessment:	
proceed to assess the circulation and nervous system	
If deterioration from any cause:	
reassess airway and breathing	
be prepared to intubate and ventilate if not already done	

CHAPTER 6

The management of cardiac arrest

LEARNING OBJECTIVES
In this chapter you will learn:
- How to assess the cardiac arrest rhythm and perform advanced life support

6.1 INTRODUCTION

Cardiac arrest has occurred when there is no effective cardiac output. Before any specific therapy is started, effective basic life support (BLS) must be established as described in Chapter 4. The cardiac arrest algorithm is shown in Figure 6.1.

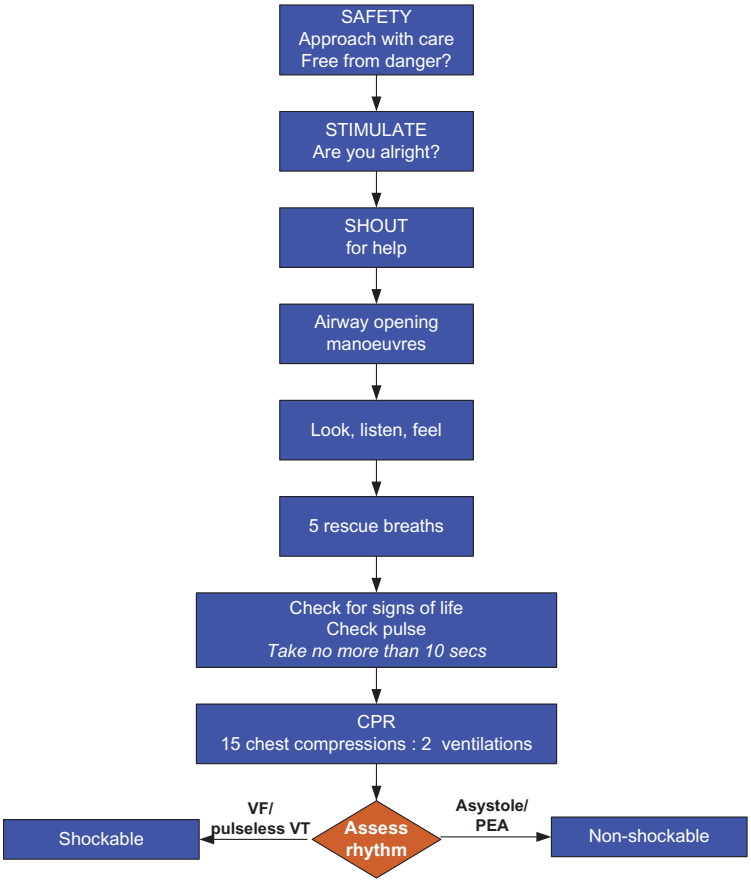

Figure 6.1 Cardiac arrest algorithm. CPR, cardiopulmonary resuscitation; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Four cardiac arrest rhythms will be discussed in this chapter:

1 Asystole.
2 Pulseless electrical activity (including electromechanical dissociation).
3 Ventricular fibrillation.
4 Pulseless ventricular tachycardia.

The four are divided into two groups: two that do not require defibrillation (called 'non-shockable') and two that do require defibrillation ('shockable').

6.2 NON-SHOCKABLE RHYTHMS

This includes asystole and pulseless electrical activity.

Asystole

This is the most common arrest rhythm in children, because the response of the young heart to prolonged severe hypoxia and acidosis is progressive bradycardia leading to asystole (Figure 6.2). The electrocardiogram (ECG) will distinguish asystole from ventricular fibrillation, ventricular tachycardia and pulseless electrical activity (PEA). The ECG appearance of ventricular asystole is an almost straight line; occasionally P-waves are seen. Check that the appearance is not caused by an artifact, e.g. a loose wire or disconnected electrode. Turn up the gain on the ECG monitor.

Pulseless electrical activity

This is the absence of signs of life or a palpable pulse despite the presence on the ECG monitor of recognisable complexes that normally produce perfusion (Figure 6.3). PEA is treated in the same way as asystole and is often a pre-asystolic state.

Pulseless electrical activity may be due to an identifiable and reversible cause. In children, trauma is most often associated with a reversible cause of PEA. This may be severe hypovolaemia, tension pneumothorax or pericardial tamponade. PEA is also seen in hypothermic patients and in patients with electrolyte abnormalities, including hypocalcaemia from calcium channel blocker overdose. Rarely in children it may be seen after massive pulmonary thromboembolus.

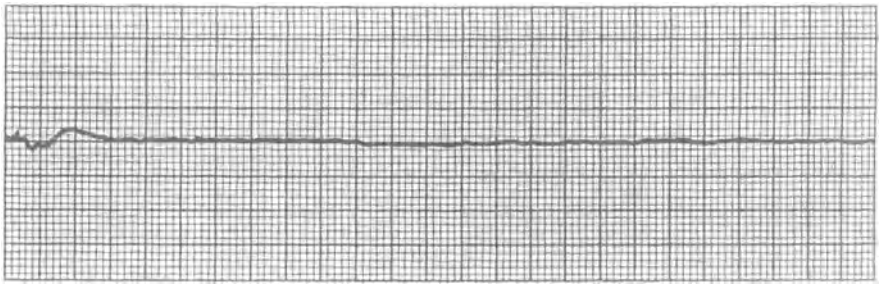

Figure 6.2 Asystole

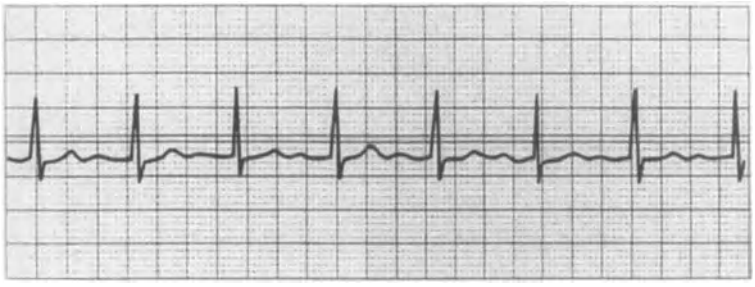

Figure 6.3 Pulseless electrical activity

Management of asystole/PEA

The first essential is to establish ventilations and chest compressions effectively. Ventilations are provided initially by bag-and-mask with high concentration oxygen. Ensure a patent airway, initially using an airway manoeuvre to open the airway and stabilising it with an airway adjunct.

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 anteroposterior diameter of the chest. The child should have a cardiac monitor attached and the *heart's rhythm assessed.*

Although the procedures to stabilise the airway and gain circulatory access are now described sequentially, they should be undertaken simultaneously under the direction of a resuscitation team leader.

If asystole or PEA is identified give *adrenaline 10 micrograms/kg intravenously or intraosseously.* Adrenaline is the first-line drug for asystole. Through α-adrenergic-mediated vasoconstriction, its action is to increase aortic diastolic pressure during chest compressions and thus coronary perfusion pressure and the delivery of oxygenated blood to the heart. It also enhances the contractile state of the heart and stimulates spontaneous contractions. The intravenous or intraosseous (IO) dose is 10 micrograms/kg (0.1 ml/kg of 1:10,000 solution). Whenever venous access is not readily attainable, intraosseous access should be considered early. Central lines provide more secure long-term access, but compared to IO or peripheral IV access, offer no advantages. In each case the adrenaline is followed by a normal saline flush (2–5 ml).

If circulatory access cannot be gained, the tracheal tube may be used but this is the least satisfactory route because of highly variable absorption and should be avoided. It may also cause transient β-adrenergic effects, decreasing coronary perfusion. If the route is used, ten times the intravenous dose (100 micrograms/kg) should be given. The drug should be injected quickly down a narrow-bore suction catheter beyond the tracheal end of the tube and then flushed in with 1 or 2 ml of normal saline.

As soon as is feasible a skilled and experienced operator should *intubate the child's airway.* This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Both cuffed and uncuffed tracheal tubes are acceptable for infants and children undergoing emergency intubation (see Chapters 5 and 20). Once the child has been intubated and compressions are uninterrupted, the ventilation rate should be 10–12 per minute. It is important for the team leader to assess that the ventilations remain adequate when chest compressions are continuous.

The protocol for asystole and PEA is shown in Figure 6.4.

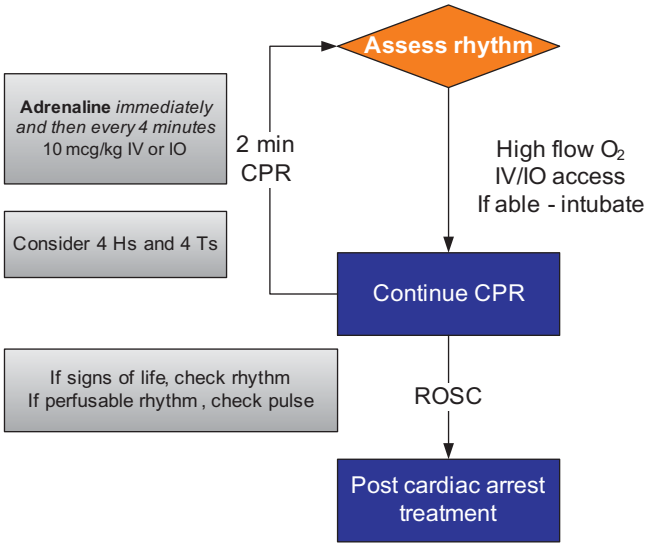

Figure 6.4 Protocol for asystole and PEA. CPR, cardiopulmonary resuscitation; IO, intraosseous; IV, intravenous; ROSC, return of spontaneous circulation

During and following the administration of adrenaline, chest compressions and ventilations 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 to interrupt BLS is to shock the patient if needed and to check the rhythm. It may be necessary to briefly interrupt during difficult intubation. Giving chest compressions is tiring for the operator so the team leader should change the operator every few minutes and continuously ensure that the compressions 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 anteroposterior diameter of the chest.

At intervals of 2 minutes briefly pause in the delivery of chest compressions to assess the rhythm on the monitor. If asystole remains, continue cardiopulmonary resuscitation (CPR) while again checking the electrode position and contacts. If there is an organised rhythm check for signs of life and for a pulse. If there a return of spontaneous circulation (ROSC), continue post-resuscitation care, increasing ventilations to 12–20 breaths per minute. If there are no signs of life and no pulse continue the protocol. Give adrenaline every 4 minutes at a dose of 10 micrograms/kg.

Reversible causes

Continually during CPR consider and correct reversible causes of the cardiac arrest based on the history of the event, known underlying illness in the child and any clues that are found during resuscitation. The causes of cardiac arrest in infancy and childhood are multifactorial but the two commonest pathways are through *hypoxia* and *hypovolaemia*.

All factors are conveniently remembered as the *4Hs and 4Ts*:

- *Hypoxia* is a prime cause of cardiac arrest in childhood and is key to successful resuscitation.
- *Hypovolaemia* may be significant in arrests associated with trauma, anaphylaxis and sepsis and requires infusion of crystalloid (see Chapter 13).
- *Hyperkalaemia*, hypokalaemia, hypocalcaemia and other metabolic abnormalities may be suggested by the patient's underlying condition (e.g. renal failure), tests taken during the resuscitation or clues given in the ECG (see Appendices A and B). Intravenous calcium (0.3 ml/kg of 10% calcium gluconate) is indicated in hyperkalaemia, hypocalcaemia and calcium channel blocker overdose.
- *Hypothermia* is associated with drowning incidents and requires particular care: a low reading thermometer must be used to detect it (see Chapter 19).
- *Tension* pneumothorax and cardiac *tamponade* are especially associated with PEA and found in trauma cases (see Chapter 14).
- *Toxic* substances either as a result of accidental or deliberate overdose or from an iatrogenic mistake may require specific antidotes (see Appendix H).
- *Thromboembolic* phenomena are much less common in children than in adults.

Adrenaline dosage

Adrenaline has been used for many years although its place has never been subjected to trial against placebo in children. In adults a prospective randomised study of drugs, including adrenaline, showed an improvement in ROSC but no increase in long-term neurologically intact survival. Its use is supported by animal studies and its known effects in improving relative coronary and cerebral perfusion. There was a trend to the use of higher doses of adrenaline in past years but evidence now links high dosage to poorer outcome, especially in asphyxial arrests. High-dose (100 micrograms/kg) adrenaline should only be used in very specific circumstances, e.g. if necessary after cardiac arrest associated with β-blocker overdose.

Alkalising agents

Children with asystole will be acidotic as cardiac arrest has usually been preceded by respiratory arrest or shock. However, the routine use of alkalising agents has not been shown to be

of benefit. Sodium bicarbonate therapy increases intracellular carbon dioxide levels so administration, if used at all, should follow assisted ventilation with oxygen and effective basic life support (BLS). Once ventilation is ensured and adrenaline plus chest compressions are provided to maximise circulation, use of sodium bicarbonate may be considered for the patient with prolonged cardiac arrest. These agents should be administered only in cases where profound acidosis is likely to adversely affect the action of adrenaline. In addition, sodium bicarbonate is recommended in the treatment of patients with hyperkalaemia (Appendix B) and tricyclic antidepressant overdose (Appendix H).

In the arrested patient, arterial pH does not correlate well with tissue pH. Mixed venous or central venous pH should be used to guide any further alkalising therapy and it should always be remembered that good BLS is more effective than alkalising agents at raising myocardial pH.

Bicarbonate is the most common alkalising agent currently available, the dose being 1 mmol/kg (1 ml/kg of an 8.4% solution). If it must be used:

- Bicarbonate must not be given in the same intravenous line as calcium because precipitation will occur.
- Sodium bicarbonate inactivates adrenaline and dopamine and therefore the line must be flushed with saline if these drugs are subsequently given.
- Bicarbonate must not be given by the intratracheal route.

Calcium

In the past, calcium was recommended in the treatment of PEA and asystole, but there is no evidence for its efficacy and there is evidence for harmful effects as calcium is implicated in cytoplasmic calcium accumulation in the final common pathway of cell death. This results from calcium entering cells following ischaemia and during reperfusion of ischaemic organs. Administration of calcium in the resuscitation of asystolic patients is not recommended. Calcium is indicated only for treatment of documented hypocalcaemia and hyperkalemia, and for the treatment of hypermagnesaemia and of calcium channel blocker overdose.

Atropine

Atropine has no place in the management of cardiac arrest. Its use is to combat excessive vagal tone causing bradycardia in the perfusing patient.

6.3 SHOCKABLE RHYTHMS

Ventricular fibrillation and pulseless ventricular tachycardia

ECGs showing ventricular fibrillation (VF) and ventricular tachycardia (VT) are shown in Figures 6.5 and 6.6, respectively.

These arrhythmias are less common in children but either may be expected in sudden collapse, those suffering from hypothermia, poison by tricyclic antidepressants and those with cardiac disease. The protocol for VF and pulseless VT is the same and is shown in Figure 6.7.

There is little direct evidence for the best approach to cardiac arrest from VF/pVT in children. The guidance is based on that developed for adults, although it is recognised that the pathology causing VF/pVT arrest in children is both less common and more varied than that in adults. Recognised causes of VF/pVT in children include underlying cardiac disease, usually congenital, hypothermia and some drug overdoses. A sudden witnessed collapse is also suggestive of a VF/pVT episode.

The guidance here is for non-experts in paediatric cardiology. In the Paediatric Cardiac Intensive Care unit, theatre or catheter laboratory, patient treatment should be individualised appropriately. If the patient is being monitored, the rhythm can be identified before significant deterioration. With immediate identification of VF/pulseless VT, asynchronous electrical

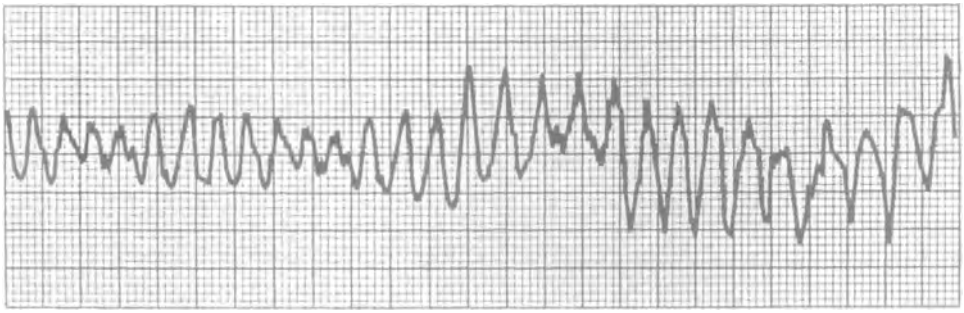

Figure 6.5 Ventricular fibrillation

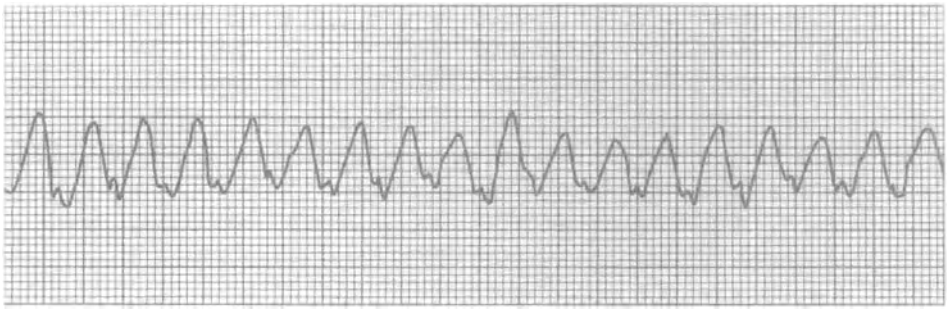

Figure 6.6 Ventricular tachycardia

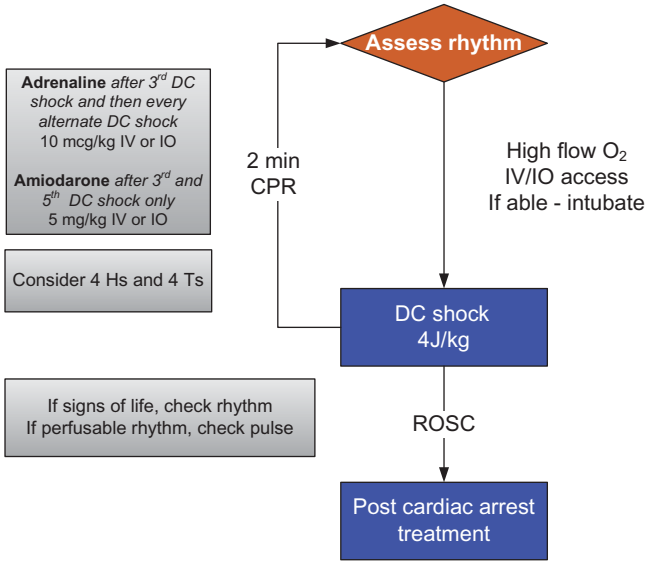

Figure 6.7 Protocol for ventricular fibrillation and ventricular tachycardia. CPR, cardiopulmonary resuscitation; DC, direct current; IO, intraosseous; IV, intravenous; ROSC, return of spontaneous circulation

defibrillation of 4 joules per kilogram (J/kg) should be carried out immediately and the protocol continued as below.

In unmonitored children, BLS will have been started in response to the collapse and the identification of VF/pulseless VT will occur when the cardiac monitor is put in place.

An *asynchronous shock of 4 J/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 chance of a successful outcome if a further shock is needed. No harm accrues from 'unnecessary' compressions.

Paediatric paddles (4.5 cm) should be used for children weighing less than 10 kg. One electrode is placed over the apex in the mid-axillary line, whilst the other is put immediately below the clavicle just to the right of the sternum. If paddles are too large, one should be placed on the upper back, below the left scapula and the other on the front, to the left of the sternum.

Automated external defibrillators (AEDs) are now commonplace. The standard adult shock is used for children over 8 years. For children under 8 years, attenuated paediatric paddles should be used with the AED.

For the infant of less than 1 year, a manual defibrillator that 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. The order of decreasing preference for defibrillation in the under 1-year-olds is as follows:

1 Manual defibrillator.
2 AED with dose attenuator.
3 AED without dose attenuator.

Many AEDs can detect VF/VT in children of all ages and differentiate 'shockable' from 'non-shockable' rhythms with a high degree of sensitivity and specificity.

If the shock fails to defibrillate, attention must revert to supporting coronary and cerebral perfusion as in asystole. Although the procedures to stabilise the airway and gain 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, a compression depth of at least one-third of the anteroposterior diameter of the chest and a ratio of 15 compressions to two ventilations. As soon as is feasible, a skilled and experienced operator should *intubate the child's airway.* This will both control and protect the airway and enable chest compressions to be given continuously, thus improving coronary perfusion. Once the child has been intubated and compressions are uninterrupted, the ventilation rate should be 10–12 per minute. It is important for the team leader to assess that the ventilations remain adequate when chest compressions are continuous. *Gain circulatory access.* Whenever venous access is not readily attainable, intraosseous access should be considered early as it is rapid and effective. Central lines provide more secure long-term access, but compared to IO or peripheral IV access, offer no advantages. In each case any drug is followed by a normal saline flush (2–5 ml).

Two minutes after the first shock, pause chest compressions briefly to check the monitor. If VF/VT is still present, give a *second shock of 4 J/kg* and *immediately resume CPR* commencing with chest compressions.

Consider and correct reversible causes (4Hs and 4Ts) while continuing CPR for a further 2 minutes. Pause briefly to check the monitor.

If the rhythm is still VF/VT give a *third shock of 4 J/kg.*

Resume chest compressions immediately and, once established, give *adrenaline 10 micrograms/kg* 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 J/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 J/kg*.

Resume chest compressions immediately and, once established, give a second dose of *adrenaline 10 micrograms/kg* and a second dose of *amiodarone 5 mg/kg* intravenously or intraosseously.

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 time there are signs of life, such as regular respiratory effort,

coughing, eye opening or a sudden increase in end tidal CO_2 (see below) stop CPR and check the monitor:

- If still VF/VT, continue with the sequence as above.
- If 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 below 60 beats per minute with poor perfusion) and no other signs of life continue the asystole/PEA sequence.

Precordial thump

A precordial thump may be given in monitored children in whom the onset of the arrhythmia is witnessed, there are several clinicians present and if the defibrillator is not immediately to hand. However, it is rarely effective and early activation of emergency services and getting an AED is more appropriate; start CPR as soon as possible.

Antiarrhythmic drugs

Amiodarone is the treatment of choice in shock-resistant VF and pulseless VT. This is based on evidence from adult cardiac arrest and experience with the use of amiodarone in children in the catheterisation laboratory setting. The dose of amiodarone for VF/pulseless VT is 5 mg/kg via rapid intravenous bolus.

There may be circumstances where the routine use of amiodarone should be omitted. This includes VF/pulseless VT caused by an overdose of an arrhythmogenic drug. Expert advice should be obtained from a poisons centre. Amiodarone is likely to be unhelpful in the setting of VF caused by hypothermia but may be used, nevertheless.

Lidocaine (lignocaine) is an alternative to amiodarone if the latter is unavailable. The dose is 1 mg/kg IV or IO. It is the DC shock that converts the heart back to a perfusing rhythm, not the drug. The purpose of the antiarrhythmic 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 of defibrillation.

Magnesium 25–50 mg/kg (maximum of 2 g) is indicated in children with hypomagnesaemia or with polymorphic VT (torsades de pointes), regardless of cause.

Reversible causes

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

These factors are remembered as the *4Hs and 4Ts*:

- *Hypoxia* is a prime cause of cardiac arrest in childhood and is key to successful resuscitation.
- *Hypovolaemia* may be significant in arrests associated with trauma, anaphylaxis and sepsis and requires infusion of crystalloid (see Chapter 13).
- *Hyperkalaemia*, hypokalaemia, hypocalcaemia and other metabolic abnormalities may be suggested by the patient's underlying condition (e.g. renal failure), tests taken during the resuscitation or clues given in the ECG (see Appendices A and B). Intravenous calcium chloride is indicated in hyperkalaemia, hypocalcaemia and calcium channel blocker overdose. Hyperkalaemia is then treated with bicarbonate, insulin and glucose (see Appendix A and B).
- *Hypothermia* is associated with drowning incidents and requires particular care: a low reading thermometer must be used to detect it and defibrillation may be resistant until core temperature is increased. Active rewarming should be commenced (see Chapter 19).
- *Tension* pneumothorax and cardiac *tamponade* are especially associated with PEA and found in trauma cases (see Chapter 14).
- *Toxic* substances either as a result of accidental or deliberate overdose or from an iatrogenic mistake may require specific antidotes. If the VF/VT has been caused by an overdose of tri-

cyclic antidepressants then the patient should be alkalised and antiarrhythmic drugs avoided except under expert guidance (see Appendix H).

- *Thromboembolic* phenomena are much less common in children than in adults.

If there is still resistance to defibrillation, different paddle positions or another defibrillator may be tried. In the infant in whom paediatric paddles have been used, larger paddles applied to the front and back of the chest may be an alternative.

If the rhythm initially converts and then deteriorates back to VF or pulseless VT then the sequence should continue to cycle omitting a further dose of amiodarone if two have already been given. If further amiodarone is thought necessary an infusion should be given of 300 micrograms/kg/h to a maximum of 1.5 mg/kg/h to a maximum of 1.2 g in 24 hours.

Automatic external defibrillators

The introduction of automated external defibrillators in the pre-hospital setting and especially for public access has significantly improved the outcome for VF/VT cardiac arrest in adults in some settings. In the pre-hospital setting, automatic external defibrillators (AEDs) are commonly used in adults to assess cardiac rhythm and to deliver defibrillation. Many AEDs can detect VF/VT in children of all ages and differentiate 'shockable' from 'non-shockable' rhythms with a high degree of sensitivity and specificity. Thus, if an AED is the only defibrillator available its use should be considered (preferably with the paediatric pads) as described earlier.

These devices have paediatric attenuation pads that decrease the energy to a level more appropriate for the child (1–8 years) or leads reducing the total energy to 50–80 joules. For the infant of less than 1 year, a manual defibrillator that 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. The order of decreasing preference for defibrillation in the under 1-year-olds is as follows:

1 Manual defibrillator.
2 AED with dose attenuator.
3 AED without dose attenuator.

Modern defibrillators now use biphasic wave forms. Defibrillation appears to be as effective at lower energy doses as conventional wave forms in adults and the energy appears to cause less myocardial damage than monophasic shocks. Both monophasic and biphasic wave form defibrillators are acceptable for use in childhood.

Capnography

Monitoring of end-tidal CO_2 ($ETCO_2$) can be helpful in managing cardiac arrest as long as the operator appreciates that the absence of a waveform is more likely to be due to absent or very poor pulmonary perfusion than to tube misplacement. The presence of exhaled CO_2 during CPR is encouraging evidence of efficacy of CPR or even ROSC. Adrenaline will decrease and bicarbonate increase the measured CO_2. Levels of less than 2 kPa should prompt attention to chest compression adequacy.

Oxygen use

While 100% oxygen, when available, remains the recommendation for use during the resuscitation process outside the delivery room, once there is return of spontaneous circulation (ROSC) there can be detriment to recovering tissues from hyperoxia. Pulse oximetry should be used to monitor and adjust for oxygen requirement after a successful resuscitation. Saturations should be maintained between 94% and 98%.

Therapeutic hypothermia

Recent data suggest that there is some evidence that post-arrest hypothermia (core temperatures of 32–34°C) may have beneficial effects on neurological recovery in adults and newborns although there is no direct evidence in children. Current recommendations are that post-arrest patients may benefit from being cooled to 32–34°C for at least 24 hours, when they should be

warmed to 34°C. Conversely, increased core temperature increases metabolic demand by 10–13% for each degree centigrade increase in temperature above normal. Therefore, in the post-arrest patient, hyperthermia should be treated with active cooling to achieve a normal core temperature. Shivering should be prevented since it will increase metabolic demand. Sedation may be adequate to control shivering, but neuromuscular blockade may be needed. See also Chapter 19 on drowning.

Hypoglycaemia

All children, especially infants, can become hypoglycaemic when seriously ill. Blood glucose should be checked frequently and hypoglycaemia corrected carefully (see Chapter 11). It is important not to cause hyperglycaemia as this will promote an osmotic diuresis. Both hypoglycaemia and hyperglycaemia are associated with a worse neurological outcome in animal models of cardiac arrest.

Resuscitation for the newborn outside the delivery room

As there are some significant differences in the recommendations for resuscitation at birth and resuscitation of the infant and child, a dilemma presents itself to the clinician confronted with the collapsed neonate outside the delivery room. There is no research in this area, so current guidance is to recommend that providers use the resuscitation protocol with which they are familiar, i.e the newborn protocol in the NICU and the infant and child protocol in other areas (the PICU, emergency department, etc.). The exception is the neonate with a probable cardiac aetiology for the arrest who should be resuscitated using the infant and child protocol.

6.4 WHEN TO STOP RESUSCITATION

Resuscitation efforts are unlikely to be successful and cessation can be considered if there is no return of spontaneous circulation at any time with up to 20 minutes of cumulative life support and in the absence of recurring or refractory VF/VT. Exceptions are patients with a history of poisoning or a primary hypothermic insult in whom prolonged attempts may occasionally be successful. Seek expert help from a toxicologist or paediatric intensivist.

6.5 PARENTAL PRESENCE

In general, family members should be offered the opportunity to be present during the resuscitation of their child. Evidence also suggests that the presence of parents at the child's side during resuscitation enables them to gain a realistic understanding of the efforts made to save their child and they subsequently may show less anxiety and depression.

Important points:

- A staff member must be designated to be the parents' support and interpreter of events at all times.
- The team leader, not the parents, decides when it is appropriate to stop the resuscitation. If the presence of the parents is impeding the progress of the resuscitation, they should sensitively be asked to leave.
- The team needs a debriefing session to support staff and reflect on practice.

6.6 SUMMARY

The teaching in this chapter is consistent with the ILCOR guidelines, Resuscitation 2010, and there are an enormous number of references that have informed this process. These are available on the ALSG website. See details on p. xv.

PART 3

The seriously ill child

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

CHAPTER 7

The structured approach to the seriously ill child

LEARNING OBJECTIVES

In this chapter you will learn:

- How to recognise the seriously ill child
- A structured approach to the assessment of the seriously ill child
- A structured approach to resuscitation and treatment of the seriously ill child

7.1 INTRODUCTION

As described in Chapter 1, the outcome for children following cardiac arrest is, in general, poor. Earlier recognition and management of potential respiratory, circulatory or central neurological failure will reduce mortality and secondary morbidity. Sections 7.2–7.5 outline the physical signs that should be used for the rapid assessment of children. It is divided systematically into looking for signs of potential respiratory, circulatory and central neurological failure and constitutes the primary and secondary assessment.

7.2 PRIMARY ASSESSMENT OF AIRWAY AND BREATHING

Recognition of potential respiratory failure

The effort, efficacy and effect of breathing need to be assessed bearing in mind the effects of respiratory inadequacy on other organs in the child's body.

Effort of breathing

The degree of increase in the effort of breathing allows clinical assessment of the severity of respiratory disease. It is important to assess the following.

Respiratory rate

Normal resting respiratory rates at differing ages are shown in Table 7.1. Rates are higher in infancy, and fall with increasing age. Care should be taken in interpreting single measurements: infants can show rates of between 30 and 90 breaths per minute depending on their state of activity. The World Health Organisation (WHO) uses a cut-off of 60 breaths per minute for pneumonia in infants and young children. Most useful are trends in the measurement as an indicator of improvement or deterioration. At rest, tachypnoea indicates that increased ventilation is needed because of either lung or airway disease, or metabolic acidosis. A slow respiratory rate indicates fatigue, cerebral depression or a pre-terminal state.

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Table 7.1 Respiratory rate by age at rest

Age (years)	Respiratory rate (breaths/min)
<1	30–40
1–2	25–35
2–5	25–30
5–12	20–25
>12	15–20

Recession
Intercostal, subcostal, sternal or suprasternal (tracheal tug) recession shows increased effort of breathing. This sign more readily develops in younger infants as they have a more compliant chest wall. Its presence in older children (i.e. over 6 or 7 years) suggests severe respiratory problems. The degree of recession gives an indication of the severity of respiratory difficulty. In the child who has become exhausted through increased effort of breathing, recession decreases.

Inspiratory or expiratory noises
An inspiratory noise while breathing (stridor) is a sign of laryngeal or tracheal obstruction. In severe obstruction the stridor may also occur in expiration, but the inspiratory component is usually more pronounced. Wheezing indicates lower airway narrowing and is more pronounced in expiration. A prolonged expiratory phase also indicates lower airway narrowing. The volume of the noise is not an indicator of severity, as it may disappear in the pre-terminal state.

Grunting
Grunting is produced by exhalation against a partially closed glottis. It is an attempt to generate a positive end-expiratory pressure and prevent airway collapse at the end of expiration in children with 'stiff' lungs. This is a sign of severe respiratory distress and is characteristically seen in infants with pneumonia or pulmonary oedema. It may also be seen with raised intracranial pressure, abdominal distension or peritonism.

Accessory muscle use
As in adult life, the sternomastoid muscle may be used as an accessory respiratory muscle when the effort of breathing is increased. In infants, this is ineffectual and just causes the head to bob up and down with each breath.

Flaring of the nostrils
Flaring of the nostrils is seen especially in infants with respiratory distress.

Gasping
This is a sign of severe hypoxia and may be pre-terminal.

Exceptions

There may be absent or decreased evidence of increased effort of breathing in three circumstances:

1. In the infant or child who has had severe respiratory problems for some time, fatigue may occur and the signs of increased effort of breathing will decrease. *Exhaustion is a pre-terminal sign.*
2. Children with cerebral depression from raised intracranial pressure, poisoning or encephalopathy will have respiratory inadequacy without increased effort of breathing. The respiratory inadequacy in this case is caused by decreased respiratory drive.
3. Children who have neuromuscular disease (such as spinal muscular atrophy or muscular dystrophy) may present in respiratory failure without increased effort of breathing.

The diagnosis of respiratory failure in such children is made by observing the efficacy of breathing, and looking for other signs of respiratory inadequacy. These are discussed in the text.

Efficacy of breathing

Observations of the degree of *chest expansion* (or, in infants, abdominal excursion) provide an indication of the amount of air being inspired and expired. Similarly, important information is given by *auscultation* of the chest. Listen for reduced, asymmetrical or bronchial breath sounds. *A silent chest is an extremely worrying sign*.

Pulse oximetry can be used to measure the arterial oxygen saturation (Spo_2). A good plethysmographic (pulse) waveform is important to help confirm the accuracy of measurements. In severe shock and hypothermia, there may be poor or absent pulse detection. Measurements are also less accurate when the Spo_2 is less than 70%, with motion artefact, high levels of ambient light and in the presence of carboxyhaemoglobin. Oximetry in air gives a good indication of the efficacy of breathing, although to assess the adequacy of ventilation, some measure of carbon dioxide should be obtained. Supplemented oxygen will mask low Spo_2 due to ineffective breathing unless the hypoxia is severe. Normal Spo_2 in an infant or child at sea level is 97–100%.

Effects of respiratory inadequacy on other organs

Heart rate

Hypoxia produces tachycardia in the older infant and child. Anxiety and a fever will also contribute to tachycardia, making this a non-specific sign. *Severe or prolonged hypoxia leads to bradycardia. This is a pre-terminal sign*.

Skin colour

Hypoxia (via catecholamine release) produces vasoconstriction and skin pallor. *Cyanosis is a late and pre-terminal sign of hypoxia* as it usually appears when Spo_2 falls to <70%, and only in the absence of anaemia. By the time central cyanosis is visible in acute respiratory disease, the patient is close to respiratory arrest. In the anaemic child, cyanosis may never be visible despite profound hypoxia. A few children will be cyanosed because of cyanotic heart disease, but may have adequate oxygen delivery. Their cyanosis will be largely unchanged by O_2 therapy.

Mental status

The hypoxic or hypercapnic child will be agitated and/or drowsy. Gradually drowsiness increases and eventually consciousness is lost. These extremely useful and important signs are often more difficult to detect in small infants. The parents may say that the infant is just 'not himself'. The doctor must assess the child's state of alertness by gaining eye contact and noting the response to voice and, if necessary, to painful stimuli. A generalised muscular hypotonia also accompanies hypoxic cerebral depression.

7.3 PRIMARY ASSESSMENT OF THE CIRCULATION

Recognition of potential circulatory failure

The cardiovascular status needs to be assessed bearing in mind the effects of circulatory inadequacy on other organs.

Cardiovascular status

Heart rate

Normal rates are shown in Table 7.2. The heart rate initially increases in shock due to catecholamine release and as compensation for decreased stroke volume. The rate, particularly in small infants, may be extremely high (up to 220 per minute).

An abnormally slow pulse rate, or bradycardia, is defined as less than 60 beats per minute or a rapidly falling heart rate associated with poor systemic perfusion. This is a pre-terminal sign.

Table 7.2 Heart rate by age

Age (years)	Heart rate (beats/min)
<1	110–160
1–2	100–150
2–5	95–140
5–12	80–120
>12	60–100

Pulse volume

Although blood pressure is maintained until shock is severe, an indication of perfusion can be gained by comparative palpation of both peripheral and central pulses. Absent peripheral pulses and weak central pulses are serious signs of advanced shock, and indicate that hypotension is already present. Bounding pulses may be caused by an increased cardiac output (e.g. septicaemia), arteriovenous systemic shunt (e.g. patent arterial duct) or hypercapnia.

Capillary refill

Following cutaneous pressure on the centre of the sternum or on a digit for 5 seconds, capillary refill should occur within 2 seconds (Figure 7.1). A slower refill time than this can indicate poor skin perfusion, a sign which may be helpful in early septic shock, when the child may otherwise appear well, with warm peripheries.

The presence of fever does not affect the sensitivity of delayed capillary refill in children with hypovolaemia but a low ambient temperature reduces its specificity, so the sign should be used with caution in trauma patients who have been in a cold environment. Poor capillary refill and differential pulse volumes are neither sensitive nor specific indicators of shock in infants and children, but are useful clinical signs when used in conjunction with the other signs described. They should not be used as the only indicators of shock nor as quantitative measures of the response to treatment.

In children with pigmented skin, the sign is more difficult to assess. In these cases the nail beds are used and additionally the sole of the feet in young babies.

Blood pressure

Normal systolic pressures are shown in Table 7.3. Expected systolic blood pressure (BP) for the 50th centile can be estimated by the following formula: BP= 85 + (age in years × 2); and the 5th centile for BP can be estimated from the formula: BP= 65 + (age in years × 2). BP varies within any age group by height and these values are for the 50th height centile, with a 8–9 mmHg difference between 5th and 95th height centiles for any age for boys and 6–7 mmHg difference for girls. Thus for boys/girls on the 25th centile for height, you remove 2 or 1.5

Table 7.3 Systolic blood pressure by age

Age (years)	Systolic BP (mmHg) 5th centile	Systolic BP (mmHg) 50th centile
<1	65–75	80–90
1–2	70–75	85–95
2–5	70–80	85–100
5–12	80–90	90–110
>12	90–105	100–120

mmHg from 85 mmHg for the mean/50th centile respectively. In septic shock, the target is a higher systolic BP and the formula for estimation in that case is: BP= 90 + (age in years × 2). Use of the correct cuff size is crucial if an accurate blood pressure measurement is to be obtained. This caveat applies to both auscultatory and oscillometric devices. The width of the cuff should be more than 80% of the length of the upper arm and the bladder more than 40% of the arm's circumference.

If the BP is less than the median systolic you should check for other signs of circulatory failure. *Hypotension (less than the 5th centile) is a late and pre-terminal sign of circulatory failure.* Once a child's blood pressure has fallen, cardiac arrest is imminent. Hypertension can be the cause or result of coma and raised intracranial pressure.

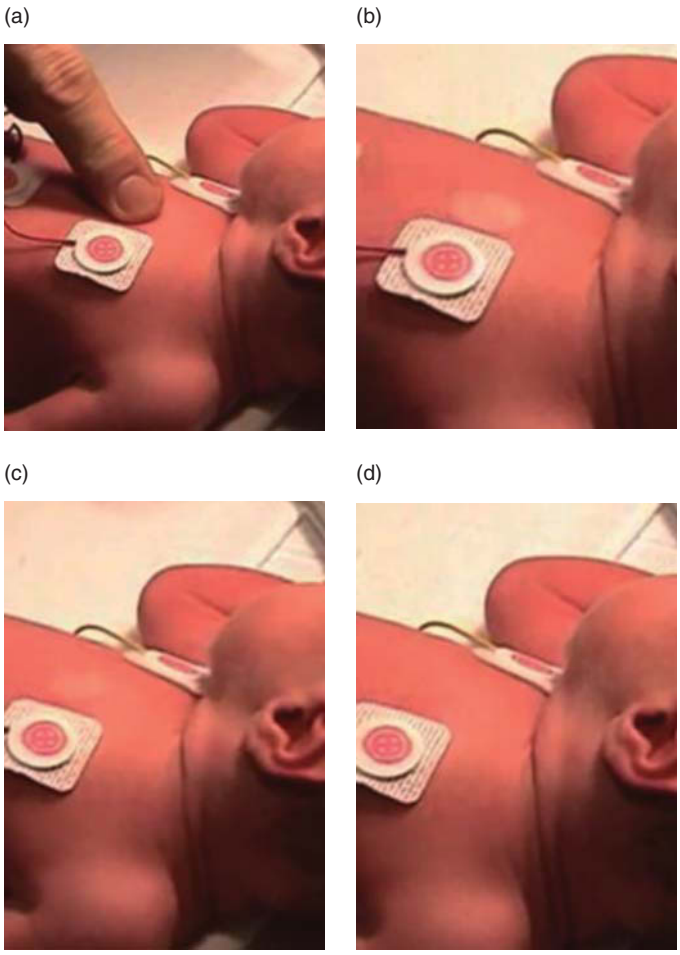


Figure 7.1 (a–d) Capillary refill assessment: apply pressure for 5 seconds and then release. Count in seconds how long it takes for the skin colour to return to normal

Effects of circulatory inadequacy on other organs

Respiratory system

A rapid respiration rate with an increased tidal volume, but without recession, may be caused by the metabolic acidosis resulting from circulatory failure.

Skin

Mottled, cold, pale skin peripherally indicates poor perfusion. A line of coldness may be felt to move centrally as circulatory failure progresses.

Mental status

Agitation and then drowsiness leading to unconsciousness are characteristic of circulatory failure. These signs are caused by poor cerebral perfusion. In an infant, parents may say that he is 'not himself'.

Urinary output

A urine output of less than 1 ml/kg/h in children and less than 2 ml/kg/h in infants indicates inadequate renal perfusion during shock. A history of oliguria or anuria should be sought.

Cardiac failure

The following features suggest a cardiac cause of respiratory inadequacy:

- Cyanosis, not correcting with oxygen therapy.
- Tachycardia out of proportion to respiratory difficulty.
- Raised jugular venous pressure.
- Gallop rhythm/murmur.
- Enlarged liver.
- Absent femoral pulses.

7.4 PRIMARY ASSESSMENT OF DISABILITY

Recognition of potential central neurological failure

Neurological assessment should only be performed after airway (A), breathing (B) and circulation (C) have been assessed and treated. There are no neurological problems that take priority over ABC.

Both respiratory and circulatory failure will have central neurological effects. Conversely, some conditions with direct central neurological effects (such as meningitis, raised intracranial pressure from trauma, and status epilepticus) may also have respiratory and circulatory consequences.

Neurological function

Conscious level

A rapid assessment of conscious level can be made by assigning the patient to one of the categories shown in the box.

A	ALERT
V	responds to VOICE
P	responds only to PAIN
U	UNRESPONSIVE to all stimuli

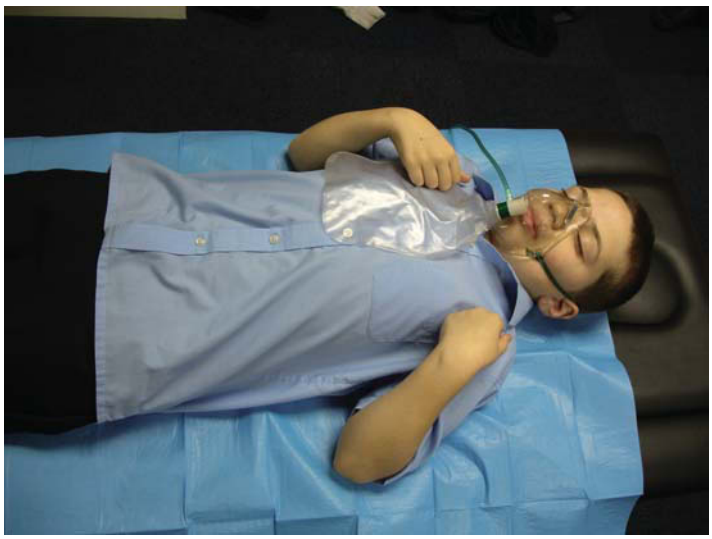
If the child does not respond to voice, it is important that assessment follows of the response to pain. The painful central stimulus can be delivered by sternal pressure, by supraorbital ridge pressure or by pulling frontal hair. A child who is unresponsive or who only responds to pain has a significant degree of coma, equivalent to 8 or less on the Glasgow Coma Scale (GCS).

If the child responds to pain, it is best to note what the eyes and limbs did and what sounds or words were uttered, rather than simply categorising the child as 'P'. Simple descriptions that will form the basis of a subsequent formal GCS, such as 'opening eyes to pain' or 'localising to pain' are much more informative than 'P' alone. A child who does not open his eyes to pain, utters no sounds and extends his limbs has a GCS score of 4 and is likely to need prompt airway protection. A child who opens her eyes to pain, shouts recognisable words inappropriately and localises to the stimulus has a GCS of 10 and is at much less immediate risk. Both are classified as 'P'.

Posture

Many children who are suffering from 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) children is a sign of serious brain dysfunction (Figure 7.2). *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 opisthotonos that occurs with meningeal irritation. A stiff neck and full fontanelle in infants are signs which suggest meningitis.

(a)



(b)

Figure 7.2 (a) Decorticate posturing, and (b) decerebrate posturing

Pupils

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

Respiratory effects of central neurological failure

There are several recognisable breathing pattern abnormalities with raised intracranial pressure. However, they are often changeable and may vary from hyperventilation to Cheyne–Stokes breathing to apnoea. The presence of any abnormal respiratory pattern in a patient with coma suggests mid- or hind-brain 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*.

7.5 EXPOSURE

The examination of the seriously ill child will involve examination for markers of illness that will help provide specific emergency treatment.

Temperature

A fever suggests an infection as the cause of the illness, but may also be the result of prolonged convulsions or shivering. In young infants, infection may present with a low body temperature.

Rash and bruising

Examination is made for rashes, such as urticaria in allergic reactions, purpura, petechiae and bruising in septicaemia and child abuse, or maculopapular and erythematous rashes in allergic reactions and some forms of sepsis.

Reassessment

Single observations on respiratory and heart rates, degree of recession, blood pressure, conscious level, pupils, etc. are useful but much more information can be gained by frequent repeated observations to detect a trend in the patient's condition. These are commonly now combined into a scoring system to provide an early warning of deterioration, e.g. the Paediatric Early Warning System (PEWS).

Summary: the rapid clinical assessment of an infant or child

Airway and **B**reathing
- Effort of breathing
- Respiratory rate/rhythm
- Stridor/wheeze
- Auscultation
- Skin colour

Circulation
- Heart rate
- Pulse volume
- Capillary refill
- Skin temperature

Disability
- Mental status/conscious level
- Posture
- Pupils

Exposure
- Fever
- Rash and bruising

The whole assessment should take less than a minute

Once airway (A), breathing (B) and circulation (C) are clearly recognised as being stable or have been stabilised, then definitive management of the underlying condition can proceed. During definitive management reassessment of ABCDE at frequent intervals will be necessary to assess progress and detect deterioration

7.6 STRUCTURED APPROACH TO THE SERIOUSLY ILL CHILD

Treatment of a child in an emergency requires rapid assessment and urgent intervention. The structured approach includes:
- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

Primary assessment and *resuscitation* involve management of the vital ABC functions and assessment of disability (central nervous system function). This assessment and stabilisation occurs before any illness-specific diagnostic assessment or treatment takes place. Once the patient's vital functions are supported, secondary assessment and emergency treatment begins. Illness-specific pathophysiology is sought and emergency treatments are instituted. During the secondary assessment vital signs should be checked frequently to detect any change in the child's condition. If there is deterioration then primary assessment and resuscitation should be repeated.

Stabilisation and transfer to definitive care are covered in Chapter 24.

7.7 PRIMARY ASSESSMENT AND RESUSCITATION

In a severely ill child, a rapid examination of vital functions is required. The physical signs described in sections 7.2–7.4 are used in an ABC approach. This primary assessment and any

necessary resuscitation must be completed before the more detailed secondary assessment is performed.

Airway

Primary assessment

- Assess patency by:
  - *looking* for chest and/or abdominal movement,
  - *listening* for breath sounds, and
  - *feeling* for expired air.
- Vocalisations, such as crying or talking, indicate ventilation and some degree of airway patency.
- If there is obvious spontaneous ventilation, note other signs which may suggest upper airway obstruction:
  - the presence of stridor, and
  - evidence of recession.
- If there is no evidence of air movement then chin lift or jaw thrust manoeuvres must be carried out. *Reassess the airway after any airway opening manoeuvres.*
- If there continues to be no evidence of air movement then airway patency can be assessed by performing an airway opening manoeuvre while giving rescue breaths (see Chapter 4).

Resuscitation

- If the airway is not patent, then this can be secured by:
  - a chin lift or jaw thrust,
  - the use of an airway adjunct, or
  - tracheal intubation.

Breathing

Primary assessment

A patent airway does not ensure adequate ventilation. The latter requires an intact respiratory centre and adequate pulmonary function augmented by coordinated movement of the diaphragm and chest wall. The adequacy of breathing can be assessed as described in Section 7.2.

Resuscitation

- Give high-flow oxygen (flow rate 15 l/min) through a non-rebreathing mask with a reservoir bag to any child with respiratory difficulty or hypoxia.
- In the child with inadequate breathing, this should be supported either with bag–valve–mask ventilation or intubation and intermittent positive pressure ventilation.

Circulation

Primary assessment

The assessment of circulation has been described in Section 7.3. It is more difficult to assess than breathing and individual measurements must not be overinterpreted.

Resuscitation

In every child with an inadequate circulation:

- give high-flow oxygen through either a non-rebreathing mask with a reservoir bag or an endotracheal tube if intubation has been necessary for airway control or inadequate breathing;
- venous or intraosseous access should be gained and an immediate infusion of crystalloid (20 ml/kg) given. Urgent blood samples, especially blood glucose, may be taken at this point.

Disability (neurological evaluation)

Primary assessment

Both hypoxia and shock can cause a decrease in conscious level. Any problem with ABC must be addressed before assuming that a decrease in conscious level is due to a primary neurological problem. The rapid assessment of central neurological failure has been described in Section 7.4. In addition, any patient with a decreased conscious level or convulsions must have an initial glucose stick test performed.

Resuscitation

- Consider intubation to stabilise the airway in any child with a conscious level recorded as P or U (only responding to painful stimuli or unresponsive).
- If hypoglycaemia has been found, treat hypoglycaemia with a bolus of glucose (2 ml/kg of 10% glucose) followed by an IV infusion of glucose, after having taken blood for glucose measurement in the laboratory and a sample for further studies.
- Intravenous lorazepam, buccal midazolam or rectal diazepam should be given for prolonged or recurrent fits (see Chapter 12 for further details).
- Manage raised intracranial pressure if present (see Chapter 11 for further details).

7.8 SECONDARY ASSESSMENT AND EMERGENCY TREATMENT

The secondary assessment takes place once vital functions have been assessed and the initial treatment of life threat to those vital functions has been started. It includes a medical history, a 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 child. 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 affecting the child and may have formulated a differential diagnosis. Emergency treatments will be appropriate at this stage – either to treat specific conditions (such as asthma) or processes (such as raised intracranial pressure). The establishment of a definite diagnosis is part of definitive care.

The history often provides the vital clues that help the practitioner identify the disease process and provide the appropriate emergency care. In the case of children, the history is often obtained from an accompanying parent, although a history should be sought from the child if possible. Do not forget to ask the paramedic about the child's initial condition and about treatments and response to treatments that have already been given.

Some children will present with an acute exacerbation of a known condition such as asthma or 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 prevents this problem. Unlike trauma (which is dealt with later), 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 other systems should be assessed and any additional emergency treatments commenced as appropriate.

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

The following gives an outline of a structured approach in the first hour of emergency management. It is not exhaustive but addresses the majority of emergency conditions that are amenable to specific emergency treatments in this time period.

The symptoms, signs and treatments relevant to each emergency condition are elaborated in the relevant chapters that follow.

Respiratory

Secondary assessment

The box below gives common symptoms and signs that should be sought in the respiratory system. Emergency investigations are suggested.

Symptoms	Signs
Breathlessness	Cyanosis
Coryza	Tachypnoea
Cough	Recession
Noisy breathing – grunting, stridor, wheeze	Grunting
Drooling and inability to drink	Stridor
Abdominal pain	Wheeze
Chest pain	Chest wall crepitus
Apnoea	Tracheal shift
Feeding difficulties	Abnormal percussion note
Hoarseness	Crepitations on auscultation
	Acidotic breathing

Investigations
Oxygen saturation
Peak flow *if asthma suspected*
End-tidal/transcutaneous carbon dioxide *if hypoventilation suspected*
Blood culture *if infection suspected*
Chest X-ray (selective)
Arterial blood gases (selective)

Emergency treatment

- If 'bubbly' noises are heard, the airway is full of secretions, which may require clearance by suction.
- If there is a harsh stridor associated with a barking cough and severe respiratory distress, upper airway obstruction due to severe croup should be suspected and the child given nebulised adrenaline (400 micrograms/kg 0.4 ml/kg of 1:1000 nebulised in oxygen).
- If there is a quiet stridor, drooling and a short history in a sick-looking child, consider epiglottitis or tracheitis. Intubation is likely to be urgently required, preferably by a senior anaesthetist. Do not jeopardise the airway by unpleasant or frightening interventions. Give intravenous cefotaxime or ceftriaxone.
- With a sudden onset and significant history of inhalation, consider a laryngeal foreign body. If the 'choking child' procedure has been unsuccessful, the patient may require laryngoscopy. Do not jeopardise the airway by unpleasant or frightening interventions but contact a senior anaesthetist/ENT surgeon urgently. However, in extreme cases of life threat, immediate direct laryngoscopy to remove a visible foreign body with a Magill's forceps may be necessary.
- Stridor following ingestion/injection of a known allergen suggests anaphylaxis. Children in whom this is likely should receive IM adrenaline (10 micrograms/kg or 150 micrograms (<6 years), 300 micrograms (6–12 years) or 500 micrograms (>12 years)).
- Children with a history of asthma or with wheeze and significant respiratory distress, decreased peak flow and/or hypoxia should receive inhaled β_2-agonists and oxygen therapy. Infants with wheeze and respiratory distress are likely to have bronchiolitis and require only oxygen.
- In acidotic breathing, take an arterial blood sample for acid–base balance and blood sugar. Treat diabetic ketoacidosis with IV normal (physiological) saline and insulin.

Cardiovascular (circulation)

Secondary assessment

The box below gives common symptoms and signs that should be sought in the cardiovascular system. Emergency investigations are suggested.

Symptoms	Signs
Breathlessness	Tachy- or bradycardia
Fever	Hypo- or hypertension
Palpitations	Abnormal pulse volume or rhythm
Feeding difficulties	Abnormal skin perfusion or colour
Drowsiness	Cyanosis/pallor
Pallor	Hepatomegaly
Fluid loss	Crepitations on auscultation
Poor urine output	Cardiac murmur
	Peripheral oedema
	Raised jugular venous pressure
	Hypotonia
Investigations	Purpuric rash
Urea and electrolytes	
Full blood count	
Arterial blood gas	
Coagulation studies	
Blood culture	
ECG	
Chest X-ray (selective)	

Emergency treatment

- Further boluses of fluid should be given to shocked children who have not had a sustained improvement to the first bolus given at resuscitation.
- Consider inotropes, intubation and central venous pressure monitoring with the third bolus.
- Consider IV cefotaxime/ceftriaxone in shocked children with no obvious fluid loss, as sepsis is likely.
- If a patient has a cardiac arrhythmia the appropriate protocol should be followed.
- If anaphylaxis is suspected, give IM adrenaline (10 micrograms/kg or 150 micrograms (<6 years), 300 micrograms (6–12 years) or 500 micrograms (>12 years)), in addition to fluid boluses.
- Give Prostin (alprostadil or dinoprostone) if duct-dependent congenital heart disease is suspected, e.g. in neonates with unresponsive shock.
- Surgical advice and intervention may be needed for gastrointestinal emergencies. The following symptoms and signs may suggest this.

Symptoms	Signs
Vomiting	Abdominal tenderness
Blood PR	Abdominal mass
Abdominal pain	

Neurological (disability)

Secondary assessment

The box below gives common symptoms and signs that should be sought in the nervous system.

Symptoms	Signs
Headache	Altered conscious level
Convulsions	Convulsions
Change in behaviour	Altered pupil size and reactivity
Change in conscious level	Abnormal posture
Weakness	Abnormal oculocephalic reflexes
Visual disturbance	Meningism
Fever	Papilloedema or retinal haemorrhage
	Altered deep tendon reflexes
	Hypertension
	Slow pulse

Investigations

Urea and electrolyte
Blood sugar
Liver function tests
Ammonia
Blood culture
Arterial blood gas
Coagulation studies
Blood and urine toxicology
CT brain scan

Emergency treatment

- For convulsions follow the status epilepticus protocol. (see Chapter 12).
- If there is evidence of raised intracranial pressure (decreasing conscious level, abnormal posturing and/or abnormal ocular motor reflexes) then the child should undergo:
  - intubation and ventilation (to maintain a PCO_2 of 4.5–5.5 kPa),
  - nursing with head in-line and 20° head-up position (to help cerebral venous drainage),
  - IV infusion with mannitol 250–500 mg/kg (1.25–2.5 ml of mannitol 20%) over 15 minutes, and repeated if needed and provided serum osmolality remains below 325 mOsm/l, or IV hypertonic (2.7) 3% saline 3 ml/kg, especially if haemodynamically unstable, and
  - consideration of dexamethasone 0.5 mg/kg twice daily (for oedema surrounding a space-occupying lesion).
- In a child with a depressed conscious level or convulsions, consider meningitis/encephalitis. Give cefotaxime/aciclovir.
- In drowsiness with sighing respirations, check blood sugar, acid–base balance and salicylate level. Treat diabetic ketoacidosis with IV normal saline and insulin.
- In unconscious children with pinpoint pupils, consider opiate poisoning. A trial of naloxone should be given.

External (exposure)

Secondary assessment

The box below gives common symptoms and signs that should be sought externally.

Symptoms	Signs
Rash	Purpura
Swelling of lips/tongue	Urticaria
Fever	Angio-oedema

Emergency treatment

- In a child with circulatory or neurological symptoms and signs, a purpuric rash suggests septicaemia/meningitis. The patient should receive cefotaxime preceded by a blood culture.
- In a child with respiratory or circulatory difficulty, the presence of an urticarial rash or angio-oedema suggests anaphylaxis. Give adrenaline (10 micrograms/kg or 150 micrograms (<6 years), 300 micrograms (6–12 years) or 500 micrograms/kg (>12 years)) IM.

Further history

Developmental and social history

Particularly in a small child or infant, knowledge of the child's developmental progress and immunisation status may be useful. The family circumstances may also be helpful – it may be worth prompting parents to remember other details of the family's medical history.

Drugs and allergies

Any medication that the child is currently on or has been on should be recorded. If poisoning is a possibility, it is important to document any medication in the home that the child might have had access to, as even relatively benign over-the-counter medications for adults may cause serious toxicity in small children. A history of allergies should be sought.

7.9 SUMMARY

The structured approach to the seriously ill child outlined here allows the practitioner to focus on the appropriate level of diagnosis and treatment during the first hour of care. Primary assessment and resuscitation are concerned with the maintenance of vital functions, while secondary assessment and emergency treatment allow more specific urgent therapies to be started. This latter phase of care requires a system-by-system approach and this minimises the chances of significant conditions being missed.

The following chapters and appendices discuss in more detail the recognition, resuscitation and emergency management of children with the following:

- Chapter 8: Breathing difficulties.
- Chapter 9: Shock.
- Chapter 10: Abnormalities of pulse rate or rhythm.
- Chapter 11: Decreased conscious level.
- Chapter 12: Convulsions.
- Appendix H: Poisoning.

CHAPTER 8

The child with breathing difficulties

LEARNING OBJECTIVES

In this chapter, you will learn:

- Why infants and young children are susceptible to respiratory failure
- How to assess children with breathing difficulties
- How to resuscitate the child with life-threatening breathing difficulties

8.1 INTRODUCTION

There is a wide range of problems that may cause apparent difficulties in breathing in children (Table 8.1). Most children with breathing difficulties will have an upper or lower respiratory

Table 8.1 Causes of breathing difficulty in children, according to mechanism

Mechanism	Cause
Upper airway obstruction	Croup/epiglottitis
	Foreign body
Lower airway obstruction	Tracheitis
	Asthma
	Bronchiolitis
Disorders affecting lungs	Pneumonia
	Pulmonary oedema (e.g. in cardiac disease)
Disorders around the lungs	Pneumothorax
	Pleural effusion or empyema
	Rib fractures
Disorders of the respiratory muscles	Neuromuscular disorders
Disorders below the diaphragm	Peritonitis
	Abdominal distension
Increased respiratory drive	Diabetic ketoacidosis
	Shock
	Poisoning (e.g. salicylates)
	Anxiety attack and hyperventilation
Decreased respiratory drive	Coma
	Convulsions
	Raised intracranial pressure
	Poisoning

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tract illness. These are the commonest causes of acute benign conditions in children but are also the most likely causes of life-threatening illness, especially in the very young. However, there are disorders outside the respiratory system that may cause apparent breathing difficulties, such as cardiac disease, poisoning and metabolic and neurological disorders. This chapter will provide the student with an approach to the assessment, resuscitation and emergency management of such children.

Disorders of the respiratory tract are the commonest illnesses of childhood. They are the most frequent reason for children to be seen by their general practitioner, and they account for 30–40% of acute medical admissions to hospital in children. Despite advances in the management of respiratory illnesses, they still resulted in almost 200 deaths in children between the ages of 4 weeks and 14 years in England and Wales in 2009. Globally, pneumonia alone accounts for nearly 20% of deaths in children under 5 years old.

Most respiratory illnesses are self-limiting minor infections, but a few present as potentially life-threatening emergencies. In these, accurate diagnosis and prompt initiation of appropriate treatment are essential if unnecessary morbidity and mortality are to be avoided.

8.2 SUSCEPTIBILITY TO RESPIRATORY FAILURE

Severe respiratory illness may result in the development of respiratory failure, defined as an inability of physiological compensatory mechanisms to ensure adequate oxygenation and carbon dioxide clearance, resulting in either arterial hypoxia, or hypercapnia, or both. Young children and infants may develop respiratory failure more readily than older children and adults, reflecting important differences in the immune status, and the structure and function of the lungs and the chest wall of children and adults.

- Children, and particularly infants, are susceptible to infection with many organisms to which adults have acquired immunity.
- The upper and lower airways in children are smaller and are more easily obstructed by mucosal swelling, secretions or a foreign body. Airway resistance is inversely proportional to the fourth power of the radius of the airway: a reduction in the radius by a half causes a 16-fold increase in airway resistance. Thus, 1 mm of mucosal oedema in an infant's trachea of 5 mm diameter results in a much greater increase in resistance than the same degree of oedema in a trachea of 10 mm diameter. From 2 months of age, airway resistance begins to decrease.
- The thoracic cage of young children is much more compliant than that of adults. When there is airways obstruction and increased inspiratory effort, this increased compliance results in marked chest wall recession and a reduction in the efficiency of breathing. The more compliant chest wall also provides less support for the maintenance of lung volume.
- The lung volume at end expiration is similar to the closing volume in infants, increasing the tendency to small airway closure and hypoxia. This may be exacerbated by an increased tendency to bronchoconstriction from alveolar or airway hypoxia.
- The number of alveoli is fewer in early childhood and this may increase the susceptibility to ventilation–perfusion mismatch.
- The respiratory muscles of young children are relatively inefficient. In infancy, the diaphragm is the principal respiratory muscle, and the intercostal and accessory muscles make relatively little contribution. Respiratory muscle fatigue can develop rapidly and result in respiratory failure and apnoea.
- The pulmonary vascular bed is relatively muscular in infancy, increasing the tendency with which pulmonary vasoconstriction occurs. In turn, this can lead to right to left shunting, ductal opening (in the early neonatal period), ventilation–perfusion mismatch and further hypoxia.
- In the first 1–2 months of life there may be a paradoxical inhibition of respiratory drive, with the result that infections can present with apnoea or hypoventilation, rather than the usual respiratory distress.

- Fetal haemoglobin is present up until 4–6 months of age, so oxygen is given up less readily to the tissues; this results in the oxygen dissociation curve being shifted to the left. Thus at any given Po_2, the Spo_2 is higher in early infancy.

8.3 CLINICAL PRESENTATIONS OF THE CHILD WITH BREATHING DIFFICULTY

Respiratory conditions do not always present with respiratory symptoms, and may include:

Respiratory	Breathlessness
	Cough
	Noisy breathing (stridor or wheeze)
	Chest pain
Non-respiratory	Poor feeding
	Abdominal pain
	Meningism
	Changes in tone: hypotonia
	Change in colour or conscious level

Noisy breathing may be normal or pathological. Parents and carers commonly understand different meanings from those understood by doctors and nurses for the terms used to describe breathing noises, or they may have their own terms. Useful historical features include relieving or aggravating factors (e.g. sleep, crying, feeding, position, exercise) and whether the voice or usual vocalisations are normal. Stridor is usually a high-pitched sound on inspiration from obstruction of the larynx or trachea and should be distinguished from stertor or snoring, which are lower pitched inspiratory noises suggestive of poor airway positioning or pharyngeal obstruction. Bubbly or gurgly noises suggest pharyngeal secretions, often seen in the child with cerebral palsy, who may have noises permanently from poor airway control and inability to spontaneously clear secretions. Wheeze is a predominantly expiratory noise from lower airway obstruction, but may be termed a variety of other names by parents. An expiratory grunt may suggest small airway closure or alveolar filling, such as found in pneumonia or pulmonary oedema.

Chest pain is an unusual symptom in children, and does not usually reflect cardiac disease, as it so often does in adults.

While parents are usually alert to breathing difficulties in toddlers and older children, abnormal respiration may be more difficult for them to detect in infants. Infants with breathing difficulties may present with acute feeding problems. Feeding for an infant is one of the most strenuous activities, and parents are accustomed to seeing feeding as a gauge of their infant's well-being.

8.4 PRIMARY ASSESSMENT OF THE CHILD WITH BREATHING DIFFICULTY

This is dealt with in Chapter 7. Below is a summary.

Assess response

If no response, proceed to life support. If response, assess ABC.

Airway

- Assess vocalisations – crying or talking indicates ventilation and some degree of airway patency.

- Assess airway patency by
  - *looking* for chest and/or abdominal movement, symmetry and recession,
  - *listening* for breath sounds and stridor, and
  - *feeling* for expired air.
- Reassess after any airway-opening manoeuvres.

Breathing

- Effort of breathing:

respiratory rate	recession	
stridor	wheeze	grunting
accessory muscle use	flaring of nostrils	gasping

Exceptions

Increased effort of breathing *does not* occur in three circumstances:

1 Exhaustion (with imminent respiratory arrest).
2 Central respiratory depression.
3 Neuromuscular disease.

- Efficacy of breathing:
  - chest expansion/abdominal excursion,
  - breath sounds – reduced or absent, and symmetry on auscultation, and
  - Spo_2 in air.
- Effects of respiratory failure on other physiology:
  - heart rate,
  - skin colour, and
  - mental status.

Circulation

- Heart rate.
- Pulse volume.
- Capillary refill.
- Skin temperature.

Disability

- Mental status/conscious level.
- Posture.
- Pupils.

Exposure

- Rash or fever.

Features suggesting cardiac cause of respiratory inadequacy

- Cyanosis, not correcting with oxygen therapy
- Tachycardia out of proportion to respiratory difficulty
- Raised jugular venous pressure
- Gallop rhythm/murmur
- Enlarged liver
- Absent femoral pulses

8.5 RESUSCITATION OF THE CHILD WITH BREATHING DIFFICULTY

Airway

- A patent airway is the first requisite. If the airway is not patent, an airway-opening manoeuvre should be used.
- The airway should then be secured with a pharyngeal airway device or by intubation with experienced senior help.

Breathing

- All children with breathing difficulties should receive high-flow oxygen through a face mask with oxygen as soon as the airway has been demonstrated to be adequate.
- Use a flow of 10–15 l/min via a face mask and reservoir bag to provide the patient with 100% oxygen. If lower flows maintain adequate Spo_2 (i.e. 94–98%), then nasal cannulae or nasopharyngeal catheters may be used (at rates of <2 l/min).
- If the child is hypoventilating with a slow respiratory rate or weak effort, respiration should be supported with oxygen via a bag–valve–mask device and experienced senior help summoned.

Circulation

Fluid intake may have been reduced, particularly in infants presenting with breathing difficulties. Consider a fluid bolus (20 ml/kg of 0.9% saline) if there are signs of circulatory failure and particularly when intubation and positive pressure ventilation is initiated. But be aware that respiratory illnesses can cause inappropriate antidiuretic hormone secretion, leading to fluid retention, so maintenance fluids may need to be reduced to two-thirds normal.

8.6 SECONDARY ASSESSMENT AND LOOKING FOR KEY FEATURES OF THE CHILD WITH BREATHING DIFFICULTIES

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours and any significant previous illness should be gained.

All children with breathing difficulties will have varying degrees of respiratory distress and cough, so these are not useful diagnostic discriminators.

Certain key features, which will be identified clinically in the above assessment and from the focused history, can point the clinician to the likeliest working diagnosis for emergency treatment.

- Inspiratory noises, i.e. *stridor*, point to upper airway obstruction — Section 8.7
- Expiratory noises, i.e. *wheeze*, point to lower airway obstruction — Section 8.8
- Fever without stridor suggests *pneumonia* — Section 8.9
- Signs of heart failure point to congenital or acquired *heart disease* — Section 8.10
- Short history, exposure to allergen and urticarial rash point to *anaphylaxis* — Sections 9.6 and 9.10
- Suspicion of ingestion and absence of cardiorespiratory pathology point to *poisoning* — Section 8.12

8.7 APPROACH TO THE CHILD WITH STRIDOR

Obstruction of the upper airway (larynx and trachea) is potentially life threatening. The small cross-sectional area of the upper airway renders the young child particularly vulnerable to obstruction by oedema, secretions or an inhaled foreign body (Table 8.2).

Reassess airway

Is the airway partially obstructed or narrowed and what is the likely cause? Note the presence of inspiratory noises.

Table 8.2 Causes of stridor

Incidence (UK)	Diagnosis	Clinical features
Very common	Croup – viral laryngotracheitis	Coryzal, barking cough, mild fever, hoarse voice
Common	Croup – recurrent or spasmodic croup	Sudden onset, recurrent, history of atopy
Uncommon	Laryngeal foreign body	Sudden onset, history of choking
Rare	Epiglottitis	Drooling, muffled voice, septic appearance
	Croup – bacterial tracheitis	Harsh cough, chest pain, septic appearance
	Trauma	Neck swelling, crepitus, bruising
	Retropharyngeal abscess	Drooling, septic appearance
	Inhalation of hot gases	Facial burns, peri-oral soot
	Infectious mononucleosis	Sore throat, tonsillar enlargement
	Angioneurotic oedema	Itching, facial swelling, urticarial rash
	Diphtheria	Travel to endemic area, unimmunised

- If 'bubbly' noises are heard, the airway is full of secretions requiring clearance. This also suggests that the child is either very *fatigued*, or has a *depressed conscious level* and cannot clear the secretions him- or herself by coughing.
- If stertorous (snoring) respiratory noises are heard, consider partial obstruction of the airway due to a *depressed conscious level*.
- If there is a harsh stridor associated with a barking cough, upper airway obstruction due to *croup* should be suspected.
- If a quiet stridor in a sick-looking child is present, consider *epiglottitis*.
- With a very sudden onset, no prodromal symptoms and a history suggestive of inhalation, consider a laryngeal *foreign body*.

Reassess the breathing: what degree of *effort* is needed for breathing and what is its *efficacy* and *effect*? The answer to this question will inform the clinician as to the severity of the upper airway obstruction. A pulse oximeter should be put in place and the oxygen saturation noted both on breathing air and high-flow oxygen.

Airway emergency treatment

In the child with a compromised but functioning airway, an important principle in all cases is to avoid worsening the situation by upsetting the child. Crying and struggling may quickly convert a partially obstructed airway into a completely obstructed one. Administration of oxygen, nebulised adrenaline or the performance of a radiograph may all require skill. Parents' help should be enlisted.

Partial obstruction from secretions or a depressed conscious level

- Use suction to clear an airway partially obstructed by secretions as long as there is no stridor.
- Support the airway with the chin lift or jaw thrust manoeuvre in a child with stertorous breathing due to a depressed conscious level or extreme fatigue and ask an anaesthetist to attend urgently.
- Further maintenance of the airway can be accomplished with an oro- or nasopharyngeal airway, but the child may require intubation.

- Whilst help is summoned, continuous positive airway pressure can be given to patients with a reduced conscious level using a face mask, oxygen flow and breathing circuit, e.g. an anaesthetic breathing circuit (Ayre's T-piece).

Croup syndromes

- Give nebulised adrenaline (400 micrograms/kg 0.4 ml/kg of 1:1000) with oxygen through a face mask to patients with severe respiratory distress in association with harsh stridor and a barking cough. This will produce a transient improvement beginning within minutes and lasting for up to 2 hours. It may need to be repeated, and if so, additional measures should be taken to ensure the airway is managed by senior staff.
- Children who require adrenaline for the emergency treatment of croup should also receive nebulised or oral steroids, such as oral dexamethasone 150 micrograms/kg (see below).
- Adrenaline reduces the clinical severity of obstruction, but does not improve arterial blood gases, reduce the duration of hospitalisation or eliminate the need for intubation. Children who have received adrenaline will appear improved for a short while only and need to be observed very closely with continuous ECG and oxygen saturation monitoring. They may later require tracheal intubation. A marked tachycardia is usually produced by the adrenaline, but other side effects are uncommon. This treatment is best used to 'buy time' in which to assemble an experienced team to treat a child with severe croup.
- Give humidified oxygen through a face mask, and monitor the oxygen saturation. Many children admitted to hospital with croup have hypoxia as a result of alveolar hypoventilation secondary to airways obstruction and ventilation perfusion imbalance. Whilst the respiratory rate and the degree of sternal recession are valuable clinical indicators of severity and response to treatment, the degree of hypoxia is the best assessment. However, hypoventilation may be masked when the child is receiving high ambient oxygen. The oxygen saturation with the child breathing air should be checked intermittently.
- Inhalation of warm moist air is widely used but is of unproven benefit.

Foreign body

- Laryngoscopy will be needed for children with severe respiratory distress and a significant history of foreign body inhalation if the 'choking child' procedure has been unsuccessful.
- Do not jeopardise the airway by unpleasant or frightening interventions, but contact a senior anaesthetist/ENT surgeon urgently.
- In extreme cases of life threat, immediate direct laryngoscopy with Magills forceps to remove a visible foreign body (Figure 8.1) may be necessary.

(a) (b)

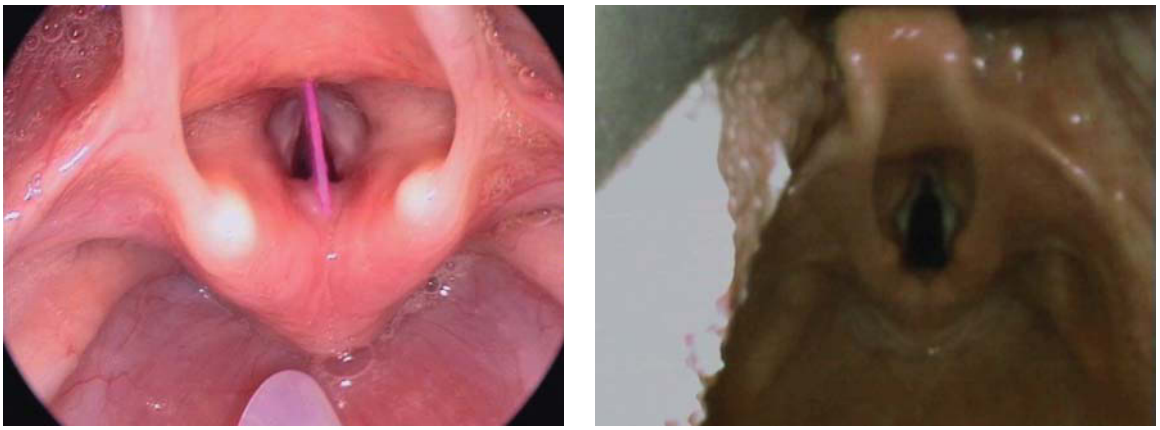

Figure 8.1 (a) Larynx with foreign body obstruction, and (b) normal larynx

(a) (b)

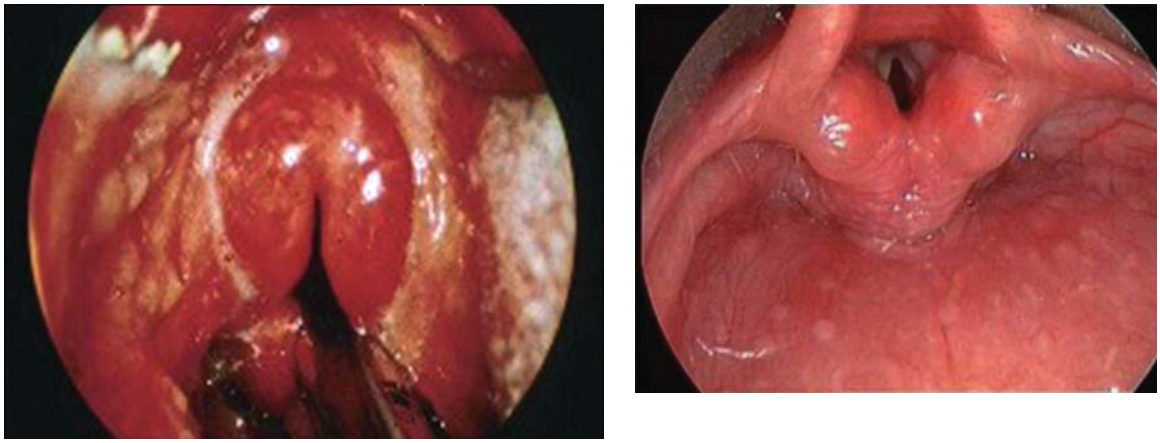

Figure 8.2 (a) Larynx epiglottitis, and (b) normal larynx

Epiglottitis

The diagnosis of acute epiglottitis is made from the characteristic history and clinical findings, as described above.

- Intubation is likely to be required. Contact a senior anaesthetist urgently, who will perform a careful gaseous induction of anaesthesia. When deeply anaesthetised the child can be laid on his back to allow laryngoscopy and intubation. Tracheal intubation may be difficult because of the intense swelling and inflammation of the epiglottis ('cherry red epiglottis') (Figure 8.2). A smaller tube than the one usually required for the child's size will be necessary.
- Do not jeopardise the airway by unpleasant or frightening interventions. Do not lie the child down if he or she prefers sitting up.
- Interventions, such as lateral radiographs of the neck and venepuncture, should be avoided as they disturb the child and have precipitated fatal total airway obstruction.
- There is no evidence that nebulised adrenaline or steroids are beneficial.

Anaphylaxis

See Section 9.6 for details of emergency treatment of anaphylaxis.

Specific upper airway conditions

Most cases of upper airway obstruction in children are the result of infection, but inhalation of a foreign body or hot gases (house fires), angioneurotic oedema and trauma can all result in obstruction and the normal airway will become obstructed in the unconscious, supine patient.

Croup

Background

Croup is defined as an acute clinical syndrome with inspiratory stridor, a barking cough, hoarseness and variable degrees of respiratory distress. This definition embraces several distinct disorders. Acute viral laryngotracheobronchitis (viral croup) is the commonest form of croup and accounts for over 95% of laryngotracheal infections. Parainfluenza viruses are the commonest pathogens but other respiratory viruses, such as respiratory syncytial virus and adenoviruses, produce a similar clinical picture. The peak incidence of viral croup is in the second year of life and most hospital admissions are in children aged between 6 months and 5 years.

The typical features of a barking cough, harsh stridor and hoarseness are usually preceded by fever and coryza for 1–3 days. The symptoms often start, and are worse, at night. Many children have stridor and a mild fever (<38.5°C), with little or no respiratory difficulty. If tracheal narrowing is minor, stridor will be present only when the child hyperventilates or is

upset. As the narrowing progresses, the stridor becomes both inspiratory and expiratory, and is present even when the child is at rest. Some children, and particularly those below the age of 3 years, develop the features of increasing obstruction and hypoxia with marked sternal and subcostal recession, tachycardia, tachypnoea and agitation. If the infection extends distally to the bronchi, wheeze may also be audible.

Some children have repeated episodes of croup without preceding fever and coryza. The symptoms are often of sudden onset at night, and usually persist for only a few hours. This recurrent or spasmodic croup may be associated with atopic disease (asthma, eczema, hay fever). The episodes can be severe, but are more commonly self-limiting. They are difficult to distinguish clinically from infectious croup and appear to respond identically to treatment, so there is a case for considering both conditions as part of one spectrum of disease.

Treatment

Steroids modify the natural history of croup: they give rise to some clinical improvement within 30 minutes and may lead to a reduction in hospital stay. Current treatments are either systemic dexamethasone 150 micrograms/kg or inhaled nebulised budesonide 2 mg. Dexamethasone and budesonide are equally effective and may be repeated after 12 hours if clinically indicated. The choice will depend on which route is most appropriate for the individual child, but oral dexamethasone is the treatment of choice in general.

Fewer than 5% of children admitted to hospital with croup require tracheal intubation. The decision to intubate is a clinical one based on increasing tachycardia, tachypnoea and chest retraction, or the appearance of cyanosis, exhaustion or confusion. Ideally, the procedure should be performed under general anaesthetic by an experienced paediatric anaesthetist, unless there has been a respiratory arrest. A much smaller gauge tracheal tube than usual is often required. If there is doubt about the diagnosis, or difficulty in intubation is anticipated, an ENT surgeon capable of performing a tracheotomy should be present. The median duration of intubation in croup is 3 days: the younger the child, the longer the intubation is usually required. Prednisolone (1 mg/kg every 12 hours) or dexamethasone (0.15 mg/kg 12–24 hourly) reduces the duration of intubation and the need for re-intubation in children with severe croup. All intubated children must have continuous CO_2 and SpO_2 monitoring.

Bacterial tracheitis

Bacterial tracheitis, or pseudomembranous croup, is an uncommon but life-threatening form of croup. Infection of the tracheal mucosa with *Staphyloccocus aureus*, streptococci or *Haemophilus influenzae* B (Hib) results in copious, purulent secretions and mucosal necrosis. The child appears toxic, with a high fever and the signs of progressive upper airway obstruction. The croupy cough and the absence of drooling help distinguish this condition from epiglottitis. Over 80% of children with this illness need intubation and ventilatory support to maintain an adequate airway, as well as intravenous antibiotics (a combination of flucloxacillin and cefotaxime).

Epiglottitis

Background

Acute epiglottitis shares some clinical features with croup but it is a quite distinct entity. Although much less common than croup, its importance is that unless the diagnosis is made rapidly and appropriate treatment commenced, total obstruction and death are likely to ensue. This is far less commonly seen in countries where Hib immunisation is routine, but may still occur in cases of vaccine failure and in unimmunised children.

Infection with Hib causes intense swelling of the epiglottis and the surrounding tissues and obstruction of the larynx. Epiglottitis is most common in children aged 1–6 years, but it can occur in infants and in adults.

The onset of the illness is usually acute with high fever, lethargy, a soft inspiratory stridor and rapidly increasing respiratory difficulty over 3–6 hours. In contrast to croup, cough is minimal or absent. Typically the child sits immobile, with the chin slightly raised and the mouth

open, drooling saliva. He looks very toxic and pale, and has poor peripheral circulation (most are septicaemic). There is usually a high fever (>39°C). Because the throat is so painful, the child is reluctant to speak and unable to swallow drinks or saliva.

> Disturbance of the child, and particularly attempts to lie the child down, to examine the throat with a spatula, or to insert an intravenous cannula, can precipitate total obstruction and death, and must be avoided.

Treatment

After securing the airway, blood should be sent for culture and treatment with intravenous cefotaxime or ceftriaxone commenced. With appropriate treatment, most children can be extubated after 24–36 hours and they recover fully within 3–5 days. Complications such as hypoxic cerebral damage, pulmonary oedema and other serious *Haemophilus* infections are rare. In countries where the Hib vaccine is in use, there should be an investigation into vaccine failure.

Foreign bodies

Background

The inquisitive and fearless toddler and the infant with toddler siblings is at risk of inhaling a *foreign body*. If an inhaled foreign body lodges in the larynx or trachea the outcome is often fatal at home, unless measures such as those discussed in Chapter 4 are performed. In 2008, fifteen children in England and Wales died from choking. Should a child present to hospital with very sudden onset of stridor and other signs of acute upper airway obstruction, especially during waking hours, and particularly if there is no fever or preceding illness, then a laryngeal foreign body is the likely diagnosis. A history of eating or of playing with small objects immediately prior to the onset of symptoms is strong supportive evidence. Foodstuffs (nuts, sweets, meat) are the commonest offending items.

In some instances, objects may compress the trachea from their position of lodgement in the upper oesophagus, producing a similar but less severe picture of airway obstruction. The object may pass through the larynx into the bronchial tree, where it produces a persistent cough of very acute onset, and unilateral wheezing. Examination of the chest may reveal decreased air entry on one side or evidence of a collapsed lung. Inspiratory and expiratory chest radiographs may show mediastinal shift on expiration due to gas trapping distal to the bronchial foreign body.

Treatment

Removal through a bronchoscope under general anaesthetic should be performed as soon as possible because there is a risk that coughing will move the object into the trachea and cause life-threatening obstruction. In the case of a stridulous child with a relatively stable airway and a strong suspicion of foreign body inhalation, careful gaseous induction of anaesthesia should be induced by an experienced anaesthetist, with the presence of an ENT surgeon to perform a tracheotomy in case of deterioration. The foreign body can then be removed under controlled conditions. In some cases, prior to anaesthesia, it may be appropriate to perform a careful lateral neck radiograph in the emergency room (taking extreme care not to distress the child, which might provoke complete obstruction) to ascertain the position and nature of the object.

Anaphylaxis

Background

Anaphylaxis is a potentially life-threatening, immunologically mediated reaction with respiratory or circulatory effects that develop over minutes, often associated with skin or mucosal changes. Laryngeal oedema causing upper airway obstruction is one manifestation. Food, especially nuts, drugs (including contrast media and anaesthetic drugs) and venom are the

commonest causes of this. Prodromal symptoms of flushing, itching, facial swelling and urticaria usually precede stridor. Abdominal pain, diarrhoea, wheeze and shock may be additional or alternative manifestations of anaphylaxis (see Section 9.10 for further details).

A severe episode of anaphylaxis can be predicted in patients with a previous severe episode or a history of increasingly severe reaction, a history of asthma or treatment with β-blockers. Measurement of blood levels of mast cell tryptase at presentation can be helpful in confirming the diagnosis.

Treatment

See Section 9.10 for treatment of anaphylaxis.

Other causes of upper airways obstruction

Although croup accounts for the large majority of cases of acute upper airway obstruction, several other uncommon conditions need to be considered in the differential diagnosis. *Diphtheria* is seen only in children who have not been immunised against the disease. Always ask about immunisations in any child with fever and signs of upper airways obstruction, particularly if they have been to endemic areas recently. Specific treatment of diphtheric croup includes penicillin, steroids and antitoxin.

Marked tonsillar swelling in *infectious mononucleosis* or *acute tonsillitis* can rarely compromise the upper airway. The passage of a nasopharyngeal tube may give instant relief and steroids are often helpful.

Retropharyngeal abscess is uncommon nowadays, but can present with fever and the features of upper airway obstruction together with feeding difficulties. Treatment is by surgical drainage and intravenous antibiotics.

8.8 APPROACH TO THE CHILD WITH WHEEZE

The two common causes of lower respiratory obstruction are:

1. *Acute severe asthma*: see below.
2. *Bronchiolitis*: see later in this section.

Almost without exception, bronchiolitis is confined to the under 1-year-olds and asthma is much more commonly diagnosed in the over 1-year-olds.

Acute severe asthma

It can be difficult to assess the *severity* of an acute exacerbation of asthma. Clinical signs correlate poorly with the severity of airway obstruction. Some children with acute severe asthma do not appear distressed, and young children with severe asthma are especially difficult to assess.

Historical features associated with more severe or life-threatening airway obstruction include:

- A long duration of symptoms and symptoms of regular nocturnal awakening.
- Poor response to treatment already given in this episode.
- A severe course of previous attacks, including the use of intravenous therapy, and those who have required admission to an intensive care unit.

After resuscitation and before progressing to specific treatment for acute asthma in any setting, it is essential to assess accurately the severity of the child's condition. The following clinical signs should be recorded regularly, e.g. every 30–60 minutes, or before and after each dose of bronchodilator:

- Pulse rate.
- Respiratory rate and degree of recession.
- Use of accessory muscles of respiration.
- Degree of agitation and conscious level.
- Spo_2.
- Peak flow.

Table 8.3 Symptoms of severe and life-threatening asthma

Acute severe asthma	Life-threatening asthma
Too breathless to feed or talk	Exhaustion
Respiratory rate: >30/min (>5 years) >50/min (2–5 years)	Poor respiratory effort Silent chest Hypotension
Pulse rate: >120 beats/min (>5 years) >130 beats/min (2–5 years)	Conscious level depressed/agitated *Consider whether this could be anaphylaxis (see Section 9.10 for further details)*

Two characteristic levels are described to indicate the appearance of asthmatic children at the most severe end of the spectrum. These are *severe* and *life-threatening* asthma (Table 8.3).

Arterial oxygen saturation as measured non-invasively by a pulse oximeter (Spo_2) is useful in assessing severity, monitoring progress and predicting outcome in acute asthma. More intensive inpatient treatment is likely to be needed for children with $Spo_2 < 92\%$ on air after initial bronchodilator treatment.

The peak expiratory flow (PEF) can be a valuable measure of severity, but children under 6 years old and those who are very dyspnoeic are usually unable to produce reliable readings.

Examination features that are poor signs of severity include the degree of wheeze, respiratory rate and pulsus paradoxus. A chest radiograph is indicated only if there is severe dyspnoea, uncertainty about the diagnosis, asymmetry of chest signs or signs of severe infection.

Asthma emergency treatment

- Assess ABC.
- Give high-flow oxygen via a face mask with reservoir bag.
- Attach pulse oximeter; always aim to keep Spo_2 94–98%.
- Give a β_2-agonist, such as salbutamol:
  - In those with mild to moderate asthma and maintaining $Spo_2 > 92\%$ in air, use a pressurised aerosol 1000 micrograms (10 sprays) via a valved holding chamber (spacer) with/without a face mask. Children with mild to moderate asthma are less likely to have tachycardia and hypoxia if given β_2-agonists via a pressurised aerosol and spacer. Children aged <3 years are likely to require a face mask connected to the mouthpiece of a spacer for successful drug delivery. Inhalers should be sprayed into the spacer in individual puffs and inhaled immediately by tidal breathing.
  - In those with severe or life-threatening asthma, or when oxygen is needed, use nebulised salbutamol 2.5 mg (<5 years) or 5 mg (>5 years) (with ipratropium bromide, see below) with oxygen at a flow of 6–8 l/min in order to provide small enough particle sizes. Higher flows may be used, but more of the nebulised drug may be lost from the face mask.
  - When there is uncertainty about reliable inhalation or where the inhaled drug produces no effective response, give a bolus of IV salbutamol (see below), and consider a continuous infusion (on high dependency unit (HDU)/paediatric intensive care unit (PICU)).
- Give oral prednisolone 1 mg/kg or, if vomiting, IV hydrocortisone 4 mg/kg.
- If receiving nebulised salbutamol, mix with ipratropium bromide 250 micrograms (< 2 years: 125 micrograms) driven with oxygen. This may be given every 20–30 minutes initially, reducing the dose as improvement occurs. In severe asthma, the nebulisers should be continuous as breaks between them can lead to a rebound of symptoms.

- If an infant or child is clearly in respiratory failure with poor respiratory effort, a depressed conscious level and poor saturation despite maximum oxygen therapy, attempt to support ventilation with a bag–valve–mask and arrange for urgent intubation; give an IV salbutamol infusion (e.g. a bolus of 15 micrograms/kg; under 2 years: 5 micrograms/kg).

Reassess ABC and monitor the response to treatment carefully. Assessment is based on physical signs and oxygen saturation measurements performed immediately before and 15–30 minutes after inhaled treatment. This should be accompanied by improved peak flow measurement.

If not responding, or deteriorating condition

- Give salbutamol IV 15 micrograms/kg over 10 minutes in patients over 2 years old (5 micrograms/kg under 2 years). The latter may be followed by IV infusion of 1–5 micrograms/kg/min, whilst monitoring ECG and serum potassium regularly to allow for detection and treatment of hypokalaemia.
- Give magnesium sulphate 25–40 mg/kg over 20 minutes.
- Contact the PICU and senior anaesthetic support.
- Consider intubation for mechanical ventilation: either rapid sequence induction with IV ketamine or halothane induction may help bronchodilatation.
- If the child is not on oral theophylline or other methylxanthines, give a loading dose of IV aminophylline 5 mg/kg over 20 minutes, monitoring the ECG for arrhythmias, followed by an infusion of 1 mg/kg/h.
- If respiratory effort is poor or deteriorating, or conscious level is depressed, or Spo_2 is low and falling despite maximum oxygen therapy, attempt to support ventilation with a bag–valve–mask, or with a mask, T-piece and bag with high-flow oxygen, whilst arranging for urgent intubation.

Indications for intubation

- Increasing exhaustion
- Progressive deterioration in:
  - clinical condition
  - Spo_2 – decreasing and/or oxygen requirement increasing
  - Pco_2 – increasing

Mechanical ventilation is rarely required. There are no absolute criteria, as the decision to intubate is usually based on the clinical condition of the child and response to previous treatment. In cases of acute severe asthma that respond to treatment, there is usually little value to be gained from routine blood gas measurement. However, in those responding poorly, repeat blood gases help in the decision to intubate. For example, ventilation should be considered if the Pco_2 is rising above normal (>6 kPa), if there is persistent hypoxia (Po_2 < 8 kPa in inspired oxygen of 60%) and with increasing exhaustion, despite intensive drug therapy. In skilled hands, the prognosis is good but complications such as air leak and lobar collapse are common. Children with acute asthma who require mechanical ventilation should be transferred to the PICU. All intubated children must have frequent or continuous CO_2 monitoring.

If responding and improving

- If there has been considerable improvement (Spo_2 > 92% in air, minimal recession, PEF > 50% of normal value) intravenous treatment can be discontinued.
- Change from a nebulised bronchodilator to the use of 8–10 aerosol sprays of a β_2-agonist inhaler, such as salbutamol or terbutaline, giving one spray at a time during tidal breathing through a spacer with mouthpiece or face mask – this can usually be done when additional oxygen is no longer needed.

- Reduce the frequency of inhaled therapy from half-hourly to 4-hourly, reducing frequency as improvement occurs.

The child's maintenance treatment should be reviewed and altered if inadequate. Inhaler technique should be checked.

Other measures

- Reassure the child and avoid upset.
- Monitor the ECG and Spo_2.
- Ensure that there is avoidance of any identifiable trigger.
- Intravenous fluids: restrict to two-thirds of the normal requirements.
- Antibiotics: do not give routinely, as most asthma attacks are triggered by viral infections.

Drug notes (Table 8.4)

- *Corticosteroids* expedite recovery from acute asthma. Although a single dose of oral prednisolone is effective, many paediatricians use a 3–5-day course. There is no need to taper off the dose for courses lasting up to 10–14 days, unless the child is on maintenance treatment with oral or high-dose inhaled steroids. Unless the child is vomiting, there is no advantage in giving steroids parenterally.
- Intravenous *salbutamol* has been shown to offer an advantage over inhaled delivery. Although inhaled drugs should be given first as they are accessible and more acceptable to the child, intravenous salbutamol has a place in severe or life-threatening episodes that do not respond promptly to inhaled therapy. Important side effects include sinus tachycardia and hypokalaemia: serum potassium levels should be checked 12-hourly, and supplementation may be needed.
- Intravenous *magnesium sulphate* is a safe treatment for acute asthma. Doses of up to 40 mg/kg/day (maximum 2 g) by slow infusion have been used. Studies of efficacy for severe childhood asthma unresponsive to more conventional therapies have shown some evidence of benefit although its place in management is not yet widely established.
- Intravenous *aminophylline* still has a role in the child who fails to respond adequately to nebulised therapy. A loading dose is given over 20 minutes, followed by a continuous infusion. Seizures, severe vomiting and fatal cardiac arrhythmias may follow rapid infusion.

Table 8.4 Drug treatment of severe acute asthma

Oxygen	High flow
Nebulised β_2-bronchodilator	Salbutamol 2.5–5 mg as required according to severity and response
	Terbutaline 2.5–10 mg
Nebulised ipratropium	250 mcg (< 2 years 125 mcg) every 20–30 minutes
Prednisolone	1 mg/kg/day for 3 days (max. dose/day 40 mg) *or* Intravenous hydrocortisone succinate: loading dose 4 mg/kg continuous infusion 1 mg/kg/h
Intravenous salbutamol	Loading dose 15 mcg/kg in children aged over 2 years Continuous infusion 1–5 mcg/kg/min
IV magnesium	25–40 mg/kg over 20 minutes
Aminophylline	Loading dose 5 mg/kg IV over 20 minutes* Continuous infusion 1 mg/kg/h

*Omit if the child has received oral theophylline in the previous 12 hours.

Continuous ECG monitoring should therefore be undertaken during infusion of the loading dose. If the child has received slow-release theophylline in the previous 12 hours the loading dose should be omitted.
- There is no evidence to support the routine use of inhaled steroids, heliox or leukotriene receptor antagonists for the treatment of acute asthma in childhood.

Background information on asthma

Asthma affects over 1 million children in the UK and resulted in about 28,000 emergency admissions in 2006–2007. Acute exacerbation of *asthma* is the commonest reason for a child to be admitted to hospital in the UK. In 2008, asthma caused 29 deaths in under 15-year-olds; reviews of such cases often identify preventable factors in both the recognition and management of the condition.

Except in the young infant, there is rarely any problem in making a diagnosis of acute asthma. An inhaled foreign body, bronchiolitis, croup and acute epiglottitis should be considered as alternative diagnoses. The classic features of acute asthma are cough, wheeze and breathlessness. An increase in these symptoms and difficulty in walking, talking or sleeping all indicate worsening asthma. Decreasing relief from increasing doses of a bronchodilator always indicates worsening asthma.

Upper respiratory tract infections (URTIs) are the commonest precipitant of symptoms of asthma in the pre-school child. Viruses cause 90% of these infections. Exercise-induced symptoms are more frequent in the older child. Emotional upset, laughing or excitement may also precipitate acute exacerbations. It is hard to assess the importance of allergen exposure to the onset of acute symptoms in an individual asthmatic, partly because of the ubiquitous nature of the common allergens (house dust mite, grass pollens, moulds) and partly because delay in the allergic response makes a cause and effect relationship difficult to recognise. A rapid fall in air temperature, exposure to a smoky atmosphere and other chemical irritants such as paints and domestic aerosols may trigger an acute attack.

Bronchiolitis emergency treatment

Management is usually supportive, as although there is specific antiviral treatment for respiratory syncitial virus (RSV, the commonest cause of bronchiolitis), this is not frequently used.
- Assess ABC.
- Ensure that the airway is patent and clear: use of a Yankauer suction catheter applied to the nares can help to ensure that the nose and nasopharynx are cleared, which can have a significant impact on an infant's respiratory distress.
- Give a high concentration of oxygen via a mask with reservoir bag. Monitor the Spo_2 and keep at 94–98%. Milder and improving cases may use oxygen via nasal cannulae at <2l/min.
- Consider using humidity, prone positioning and high-flow systems.
- Maintain hydration and nutrition. In infants with significant respiratory distress, maintain hydration by feeding via a nasogastric tube, or intravenously at two-thirds the usual maintenance. Remember, nasogastric tubes may partially occlude the airway. Breastfeeding may be too stressful, in which case breast milk should be expressed and given via a gastric tube.
- Monitor for apnoea/hypoventilation in those <2 months old:
  - Spo_2,
  - respiratory frequency/apnoea monitor, and
  - Pco_2 – transcutaneous, capillary or end-tidal.
- Mechanical ventilation is required in 2% of infants admitted to hospital. Whilst non-invasive methods of respiratory support (e.g. continuous positive airway pressure (CPAP)) have been used, there are no randomised controlled data to show that this avoids intubation. In severe cases, infants with the following may need intubation and mechanical ventilation:
  - recurrent apnoea,
  - exhaustion, or
  - severe hypercapnia and hypoxia.
- All intubated infants must have continuous Spo_2 and CO_2 monitoring.

- Both nebulised 3% saline and adrenaline have been shown to have some benefit, but their place in current management is unclear. The same applies to the nebulised antiviral agent ribavirin – it is indicated for children with pre-existing lung disease, those with impaired immunity and infants with congenital heart disease, although these groups commonly will have received palivizumab, which may lessen the severity of the RSV disease.
- Bronchodilators, steroids and antibiotics are of no proven value.

Most children recover from the acute infection within 2 weeks. However, as many as half will have recurrent episodes of cough and wheeze over the next 3–5 years. Rarely there is severe permanent damage to the airways (bronchiolitis obliterans).

Background information on bronchiolitis

Bronchiolitis is the most common serious respiratory infection of childhood: it occurs in 10% of all infants and 2–3% are admitted to hospital with the disease each year. Ninety per cent of patients are aged 1–9 months; it is rare after 1 year of age. There is an annual winter epidemic. RSV is the pathogen in 60–70% of cases, the remainder of cases being caused by other respiratory viruses, such as parainfluenza, influenza and adenoviruses. Acute bronchiolitis is not a primary bacterial infection, and secondary bacterial involvement is uncommon.

Fever and a clear nasal discharge precede a dry cough and increasing breathlessness. Wheezing is often, but not always, present. Feeding difficulties associated with increasing dyspnoea are often the reason for admission to hospital. Recurrent apnoea is a serious and potentially fatal complication, and is seen particularly in infants born prematurely. Children with pre-existing chronic lung disease (e.g. cystic fibrosis, bronchopulmonary dysplasia in premature infants) and children with congenital heart disease or immune deficiency syndromes are at particularly high risk of developing severe respiratory failure with bronchiolitis.

The findings on examination are characteristic (Table 8.5).

The chest radiograph shows hyperinflation with downward displacement and flattening of the diaphragm due to small airways obstruction and gas trapping. In one-third of infants there is also evidence of collapse or consolidation, particularly in the upper lobes (Figure 8.3). RSV and other viruses can be cultured or identified with a fluorescent antibody technique on nasopharyngeal secretions. Blood gas analysis, which is required in only the most severe cases, shows lowered oxygen and raised carbon dioxide levels.

Risk factors for severity in bronchiolitis

- Age under 6 weeks
- Premature birth
- Chronic lung disease
- Congenital heart disease
- Immunodeficiency

Table 8.5 Bronchiolitis: characteristic findings on examination

Sign	Findings
Tachypnoea	50–100 breaths/min
Recession	Subcostal and intercostal
Cough	Sharp, dry
Hyperinflation of the chest	Sternum prominent, liver depressed
Tachycardia	140–200 beats/min
Crackles	Fine end-inspiratory
Wheezes	High-pitched expiratory > inspiratory
Colour	Cyanosis or pallor
Breathing pattern	Irregular breathing/recurrent apnoea

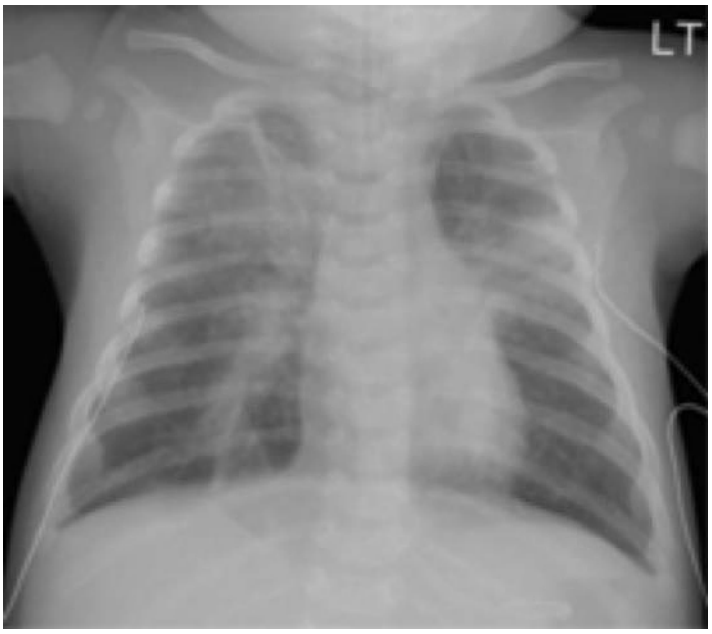

Figure 8.3 Chest X-ray of bronchiolitis

Bronchiolitis may trigger heart failure in an infant with a previously undiagnosed cardiac lesion. Distinguishing features are listed in Table 8.6.

Table 8.6 Features of heart failure due to bronchiolitis

Heart failure	Bronchiolitis
Feeding difficulty with growth failure	
Restlessness, sweating	
Tachycardia and tachypnoea	Coryzal and harsh cough
Pallor, sweating and cool peripheries	
Large heart with displaced apex beat	Normal or apparently small heart
Large liver	Liver lower than normal
Gallop rhythm	
Murmur	No murmur
Chest X-ray shows pulmonary congestion and large heart	Hyperinflation on chest X-ray

Although many causes of breathing difficulties are associated with infection, a high fever is usually associated only with pneumonia, epiglottitis and bacterial tracheitis. Although many cases of asthma are precipitated by an URTI, the asthmatic child is rarely febrile and a low-grade fever is characteristic of bronchiolitis. Therefore, in the absence of stridor and wheeze, breathing difficulties in association with a significant fever are likely to be due to pneumonia.

8.9 APPROACH TO THE CHILD WITH FEVER

Pneumonia emergency treatment

- Assess ABC.
- Provide a high concentration of oxygen via a face mask with reservoir bag. Attach a pulse oximeter; if a low flow maintains SpO_2 at 94–98%, then nasal cannulae may be used with a flow <2l/min.

- As it is not possible to differentiate reliably between bacterial and viral infection on clinical, haematological or radiological grounds, all children diagnosed as having significant pneumonia should receive antibiotics. The preferred antibiotics in the seriously ill child are:
  - cefuroxime – effective against most bacteria,
  - cefotaxime – if there is a septic component,
  - flucloxacillin – if *Staphylococcus aureus* is suspected, or
  - macrolide antibiotic – if atypical pneumonia or pertussis (unimmunised infant) is suspected.
- Maintain hydration: extra fluid may be needed to compensate for loss from fever, and restriction may be needed because of inappropriate antidiuretic hormone (ADH) secretion. Fluid overload can contribute to worsening breathlessness.
- Clinical examination and the chest radiograph may reveal a pleural effusion (Figure 8.4). This should be confirmed with ultrasound and, if large, it should be drained to relieve breathlessness, aid diagnosis and allow the instillation of intrapleural fibrinolytic agents. Ultrasound may guide the positioning of an intrapleural drain. Details of the procedure can be found in Chapter 22.
- Airway and breathing support may especially be needed in children with neurological handicap who may have poor airway control and weak respiratory muscles even when well.

Background to pneumonia

Pneumonia in childhood was responsible for 70 deaths of children aged under 15 years in England and Wales in 2009 (Office of National Statistics). Infants, and children with congenital abnormalities or chronic illnesses, are at particular risk. In adults, two-thirds of cases of pneumonia are caused by either *Streptococcus pneumoniae* or *Haemophilus influenzae*. A much wider spectrum of pathogens causes pneumonia in childhood, and different organisms are important in different age groups.

In the newborn, organisms from the mother's genital tract, such as *Escherichia coli* and other Gram-negative bacilli, group B β-haemolytic *Streptococcus* and, increasingly, *Chlamydia trachomatis*, are the most common pathogens. In infancy, respiratory viruses, particularly respiratory syncytial virus, are the most frequent cause, but *Streptococcus pneumoniae*, *Haemophilus* and, less commonly, *Staphylococcus aureus* are also important. In older children, viruses become less frequent pathogens and bacterial infection is more important. *S. pneumoniae* remains the

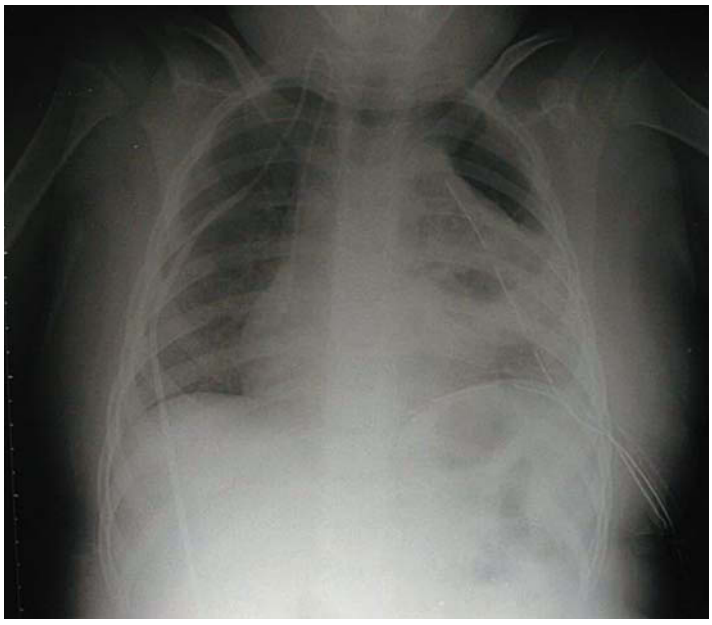

Figure 8.4 Chest X-ray of pneumonia

commonest cause in this age group but *Mycoplasma pneumonia* is also a cause of pneumonia in the school-age child. *Bordetella pertussis* can present with pneumonia as well as with classic whooping cough, even in children who have been fully immunised. It can cause a severe pneumonitis, leading to respiratory failure in unimmunised infants.

Fever, cough, breathlessness and chest recession in the younger child and lethargy are the usual presenting symptoms. The cough is often dry initially but then becomes loose. Older children may produce purulent sputum but in those below the age of 5 years it is usually swallowed. Pleuritic chest pain, neck stiffness and abdominal pain may be present if there is pleural inflammation. Classic signs of consolidation such as impaired percussion, decreased breath sounds and bronchial breathing are often absent, particularly in infants, and a chest radiograph is needed. This may show lobar consolidation, widespread bronchopneumonia or, less commonly, cavitation of the lung. Pleural effusions are quite common, particularly in bacterial pneumonia. An ultrasound of the chest will delineate a pleural effusion and be helpful in the placing of a chest drain. Blood cultures, swabs for viral isolation and a full blood count should also be performed. It can be useful to save an acute serum for further microbiological diagnosis.

As it is not possible to differentiate reliably between bacterial or viral infection on clinical or radiological grounds, all children diagnosed as having significant pneumonia should receive antibiotics. Oral antibiotics are sufficient in most cases, unless there is vomiting or severe respiratory distress. The initial choice of antibiotics depends on the age of the child. Antibiotics should be given for 7–10 days, although complicated pneumonias, e.g. with empyema, may require several weeks' duration.

In children with no respiratory difficulty, treatment will occur at home with penicillin, a cephalosporin or macrolide. Infants, and children who look toxic, have definite dyspnoea, an Spo_2 below 93%, grunting or signs of dehydration should be admitted and usually require intravenous treatment initially (see above). Oxygen (if $Spo_2 < 93\%$) and an adequate fluid intake (70% maintenance, as inappropriate ADH secretion occurs in pneumonia) are also required. Chest physiotherapy is not beneficial in previously healthy children with community-acquired pneumonia, but may help children who are known to have problems with secretion clearance.

Mechanical ventilation is rarely required unless there is a serious underlying condition. Transfer to the PICU should be considered with the following: an $Fio_2 > 0.6$ to keep the Spo_2 at 94–98%, shock, exhaustion, rising CO_2, apnoea or irregular breathing. If a child has recurrent or persistent pneumonia, investigations to exclude underlying conditions such as cystic fibrosis or immunodeficiency should be performed.

8.10 APPROACH TO THE CHILD WITH HEART FAILURE

Infants and children with serious cardiac pathology may present with breathlessness, cyanosis or cardiogenic shock. The immediate management of shock is described in Chapter 9.

Causes of heart failure that may present as breathing difficulties

Left ventricular volume overload or excessive pulmonary blood flow:	
Ventricular septal defect	Atrioventricular septal defect
Common arterial trunk	Persistent arterial duct
Left heart obstruction:	
Hypertrophic cardiomyopathy	Critical aortic stenosis
Aortic coarctation	Hypoplastic left heart syndrome
Primary 'pump' failure:	
Myocarditis	Cardiomyopathy
Dysrhythmia:	
Supraventricular tachycardia	Complete heart block

Heart failure emergency treatment

Reassess ABC

- If there are signs of shock – poor pulse volume or low blood pressure with extreme pallor and depressed conscious level – treat the child for cardiogenic shock (see Chapter 9).
- If circulation is adequate and oxygen saturation is normal or improves significantly with oxygen by face mask but there are signs of heart failure, then the breathing difficulty is due to pulmonary congestion secondary to a large left to right shunt. The shunt may be through a ventricular septal defect (VSD), atrioventricular septal defect (AVSD), patent ductus arteriosus (PDA) or, more rarely, a truncus arteriosus. In many cases a heart murmur will be heard. A chest radiograph will also give confirmatory evidence, with a large, usually globular heart and radiological signs of pulmonary congestion. Give:
  - high-flow oxygen by face mask with a reservoir, and
  - diuretics such as frusemide (furosemide) (1 mg/kg IV followed by initial maintenance dose of 1–2 mg/kg/day in one to three divided doses); if there is no diuresis within 2 hours, the intravenous bolus can be repeated.
- Babies in the first few days of life who present with breathlessness and increasing cyanosis largely unresponsive to oxygen supplementation are likely to have duct-dependent congenital heart disease such as tricuspid or pulmonary atresia. See Section 9.11 for further details and treatment.
- Children of all ages who present with breathlessness from heart failure may have myocarditis. This is characterised by a marked sinus tachycardia and the absence of signs of structural abnormality. The patients should be treated with oxygen and diuretics.

A full blood count, and measurements of serum urea and electrolytes, calcium, glucose and arterial blood gases should be performed on all patients in heart failure. A routine infection screen including blood cultures is recommended, especially in infants. A full 12-lead ECG and a chest radiograph are essential. All patients suspected of having heart disease should be discussed with a paediatric cardiologist, as transfer to a tertiary centre will usually be required. Echocardiography will establish the diagnosis in most cases.

Background to heart failure in infancy and childhood

In infancy, heart failure is usually secondary to structural heart disease, and medical treatment is directed at improving the clinical condition prior to definitive surgery. With modern obstetric management many babies are now discharged from the maternity unit only hours after birth. Therefore babies with serious congenital neonatal heart disease may present to paediatric or accident and emergency departments. See Section 9.11 for further details.

Duct-dependent congenital heart disease

There are also several rarer and more complex congenital heart defects in which the presence of a PDA is essential to maintain pulmonary or systemic flow. The normal PDA closes functionally in the first 24 hours of life. Infants with duct-dependent right or left heart lesions present in the first few days of life as the ductus arteriosus starts closing in response to transition from fetal to postnatal life (see Section 9.11 for further details).

In the older child, myocarditis and cardiomyopathy are the usual causes of the acute onset of heart failure and remain rare (see box on p. 88). Presenting features include fatigue, effort intolerance, anorexia, abdominal pain and cough. On examination a marked sinus tachycardia, hepatomegaly and raised jugular venous pressure are found with inspiratory crackles on auscultation.

8.11 APPROACH TO THE CHILD WITH ANAPHYLAXIS

This is covered in detail in Section 9.10.

8.12 APPROACH TO THE CHILD WITH METABOLIC AND POISONING PROBLEMS

Diabetes

As hyperventilation is a feature of the severe acidosis produced by diabetes, occasionally a child may present with a primary breathing difficulty. The correct diagnosis is usually easy to establish and management is described in Appendix B.

Poisoning

There may be apparent breathing difficulties following the ingestion of a number of poisons.

The respiratory rate may be increased by poisoning with:

- Salicylates.
- Ethylene glycol (antifreeze).
- Methanol.
- Cyanide.

However, usually only poisoning with salicylates causes any diagnostic dilemma. Poisoning with drugs that cause a depression of ventilation will present as a diminished conscious level. The management of the poisoned child is dealt with in Appendix H.

8.13 SUMMARY

You should use the structured approach in the assessment and management of the child with breathing difficulties:

- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

CHAPTER 9

The child in shock

LEARNING OBJECTIVES

In this chapter, you will learn:

- The causes of shock in infants and children
- About the pathophysiology of shock
- How to assess children with shock
- How to resuscitate the child with life-threatening shock
- The emergency treatment of the different causes of shock
- The properties of different resuscitation fluids

9.1 INTRODUCTION

Shock is an acute, complex clinical syndrome of circulatory dysfunction resulting in inadequate delivery of oxygen and other nutrients to meet tissue metabolic demands. The final pathway is a failure of both substrate delivery and removal of metabolites leading to a state of acute cellular oxygen deficiency; irrespective of the initial insult triggering shock, which can be any severe disease or injury. This in turn leads to anaerobic metabolism and tissue acidosis culminating in loss of normal cellular function, cell death, organ dysfunction and eventually death, if not recognised and appropriately treated.

Maintenance of adequate tissue perfusion and oxygen supply depends on blood volume, cardiac output and arterial oxygen content. Cardiac output is defined by heart rate and stroke volume, and is directly proportional to adequate preload (venous return), afterload (systemic vascular resistance) and cardiac contractility; oxygen-carrying capacity is defined by haemoglobin content and arterial oxygenation. Therefore, an insult affecting any of these can lead to a shock state.

Inadequate tissue perfusion resulting in impaired cellular respiration (i.e. shock) may result from defects of the heart pump (cardiogenic), loss of fluid (hypovolaemic), abnormalities of vessels (distributive), flow restriction (obstructive) or inadequate oxygen-releasing capacity of blood (dissociative) or blockage to oxygen utilisation (septic). In many causes of shock, several mechanisms may coexist, therefore the clinician must consider which of several alternative emergency treatments will be effective for any individual patient.

9.2 THE PATHOPHYSIOLOGY OF SHOCK

Shock results from an acute failure of circulatory function. Inadequate amounts of nutrients, especially oxygen, are delivered to body tissues and there is inadequate removal of tissue waste products. Other than underlying illnesses, age-dependent maturation of different organ systems and the body's defense mechanisms define the response to shock.

Shock is a progressive state which can be divided into three phases: compensated, uncompensated and irreversible. Although artificial, this division is useful because each phase has characteristic clinicopathological manifestations and outcome.

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Compensated shock

In this early phase, physiological neurohormonal compensatory mechanisms maintain vital (i.e. brain, heart and kidneys) organ perfusion. Sympathetic nervous system reflexes increase systemic arterial resistance, divert blood away from non-essential tissues, constrict the venous reservoir and increase the heart rate to maintain cardiac output. The systolic blood pressure remains normal whereas the diastolic pressure may be elevated due to the increased systemic arterial resistance. Increased secretion of angiotensin and vasopressin allows the kidneys to conserve water and salt, while reduced renal perfusion leads to reduced urine output and intestinal fluid is reabsorbed from the digestive tract. The clinical signs characteristic of this stage are mild agitation or confusion, skin pallor, increased heart rate, cold peripheral skin with decreased capillary return and reduced urine output. Early recognition is crucial as appropriate therapeutic interventions at this stage can completely reverse shock. In older patients, raised cardiac output with decreased systemic resistance may give rise to warm extremities and wide pulse pressure.

Uncompensated shock

If shock is unrecognised or untreated in the early stage, it progresses further and the compensatory mechanisms fail to support the circulatory system. Poorly perfused tissue beds can no longer sustain aerobic metabolism and comparatively inefficient anaerobic metabolism becomes their major source of energy production. Anaerobic metabolism produces lactate; the acidosis is further compounded by intracellular carbonic acid formed because of the inability of the circulation to remove CO_2. Acidosis reduces myocardial contractility and impairs the response to circulating catecholamines. A further result of anaerobic metabolism is the failure of the energy-dependent sodium–potassium pump, which maintains the normal homeostatic environment for optimum cellular function.

Lysosomal, mitochondrial and membrane functions deteriorate without this homeostasis. Sluggish blood flow and chemical changes in small vessels lead to platelet adhesion and may produce damaging chain reactions in the kinin and coagulation systems, heralding the onset of disseminated intravascular coagulation (DIC).

If recognised in this stage, shock may still be reversible if appropriately treated. Clinical manifestations of this stage are a normal or falling blood pressure, tachycardia, prolonged capillary refill, cold peripheries, acidotic breathing, depressed cerebral state and severely reduced or absent urine output. Blood gases reveal metabolic acidosis and blood lactate is increased.

Irreversible shock

If the shock goes untreated, it progresses to an irreversible stage where the cellular damage cannot be reversed even if cardiovascular function is restored to adequate levels. Despite haemodynamic correction, multiple organ failure occurs. This underlies the clinical observation that during shock progression a point is reached where death of the patient becomes inevitable, despite appropriate therapeutic intervention. Hence early recognition and appropriate treatment is vital.

9.3 CLASSIFICATION OF THE CAUSES OF SHOCK

The causes of shock are listed in Table 9.1, with the more common in bold. It can be seen that the most common cause in paediatric patients is hypovolaemia from a number of different conditions. The cause of shock is often multifactorial. For example, in septic shock, hypovolaemia, cardiac dysfunction, abnormal vascular tone and dissociative shock due to impaired mitochondrial function occur simultaneously.

Table 9.1 Causes of shock

Causes of shock		Chapter
Hypovolaemic	**Haemorrhage**	13
	Gastroenteritis, stomal losses	9
	Intussusception, volvulus	9
	Burns	18
	Peritonitis	
Distributive	**Septicaemia**	9
	Anaphylaxis	9
	Vasodilating drugs	
	Spinal cord injury	17
Cardiogenic	Arrhythmias	10
	Cardiomyopathy	10
	Heart failure	8
	Valvular disease	
	Myocardial contusion	14
Obstructive	Congenital cardiac (coarctation, hypoplastic left heart, aortic stenosis)	10
	Tension pneumothorax	14
	Haemopneumothorax	14
	Flail chest	14
	Cardiac tamponade	14
	Pulmonary embolism	
Dissociative	Profound anaemia	9
	Carbon monoxide poisoning	18
	Methaemoglobinaemia	

9.4 APPROACH TO THE CHILD IN SHOCK

The child may present with fever, rash, pallor, poor feeding, drowsiness, history of trauma or poisoning. Other than certain obvious causes of shock like external haemorrhage, signs and symptoms of early compensated shock can be easily missed. Early recognition of shock is crucial and requires a high index of suspicion and knowledge of the conditions that predispose children of different ages and co-morbidities to shock. For example, it is important to know a history of congenital heart disease, immunodeficiency, trauma, surgery, toxin ingestion or allergies. The initial clinical assessment should identify the child in shock.

9.5 PRIMARY ASSESSMENT

This is dealt with in Chapter 7. Below is a summary.

Airway

- Assess vocalisations – crying or talking indicate ventilation and some degree of airway patency.
- Assess airway patency by:
  - *looking* for chest and/or abdominal movement, symmetry and recession,
  - *listening* for breath sounds and stridor, and
  - *feeling* for expired air.

- *Reassess after any airway-opening manoeuvres*. If there is still no evidence of air movement then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see Chapter 4).

Breathing

- Effort of breathing:

respiratory rate	recession	
stridor	wheeze	grunting
accessory muscle use	flaring of the nostrils	gasping

Exceptions

Increased effort of breathing *does not* occur in three circumstances:

1 Exhaustion.
2 Central respiratory depression.
3 Neuromuscular disease.

- Efficacy of breathing:
  - chest expansion/abdominal excursion,
  - breath sounds – reduced or absent, and symmetry on auscultation, and
  - Spo_2 in air.
- Effects of respiratory failure on other physiology:
  - heart rate,
  - skin colour, and
  - mental status.

Circulation

- Vital signs:
  - heart rate,
  - pulse volume, and
  - blood pressure.
- Skin and mucous membrane perfusion:
  - capillary refill time (central and peripheral),
  - temperature, and
  - colour.
- Organ perfusion:
  - effects on breathing,
  - mental status, and
  - urine output.

Monitor the heart rate/rhythm, blood pressure, capillary refill time, core–toe temperature difference and urine output. If the heart rate is above 200 in an infant or above 150 in a child, or if the rhythm is abnormal, perform a standard electrocardiogram (ECG). It is also important to check the blood gas for lactate, base excess and gas exchange.

Disability

- Mental status/conscious level (AVPU).
- Pupillary size and reaction.
- Posture: children in shock are usually hypotonic.

Exposure

- *Rash*: this is often a key clinical indicator for the cause of shock (see Sections 9.9 and 9.10). There is a pathognomonic haemorrhagic rash associated with meningococcal septicaemia. In

a child with non-specific symptoms there should be a thorough examination for a petechial/purpuric rash.
- *Fever*: suggests an infective cause.
- *Injury*: evidence of trauma.
- Consider evidence for poisoning.

9.6 RESUSCITATION

Airway

- Use an airway-opening manoeuvre, if not patent, or partially obstructed. If there is improvement, use airway adjuncts to support the airway.
- Suction.
- The airway may need to be secured by tracheal intubation.

Breathing

- All children in shock should receive high-flow oxygen through a face mask with a reservoir as soon as the airway has been demonstrated to be adequate.
- If the child is hypoventilating, respiration should be supported with oxygen via a bag–valve–mask device and experienced, senior help should be summoned for early tracheal intubation and mechanical ventilation.

Circulation

- Gain intravenous or intraosseous access:
  - insert two short, wide-bore IV cannulae if possible,
  - insert an intraosseous line if peripheral venous access is difficult due to poor perfusion,
  - femoral venous access can be used in situations where peripheral or intraosseous access is impossible; long saphenous vein cut down may also be considered in difficult circumstances,
  - using neck veins for access should ideally be reserved for experienced personnel, as there is a risk of pneumothorax, haemorrhage or exacerbation of an unsuspected neck injury, and
  - techniques for vascular access are described in Chapter 21.
- Take blood for blood gas (including lactate and ionised calcium), glucose stick test and laboratory tests including full blood count (FBC), urea and electrolytes (U&Es), renal and liver function, C-reactive protein (CRP), blood culture, cross-match and coagulation studies.
- Give a 20 ml/kg rapid bolus of crystalloid to all patients (caution in those with primary cardiogenic shock and in those with signs of raised intracranial pressure). Patients with cardiac aetiology (i.e. myocarditis) leading to shock may still benefit from a cautious fluid bolus to optimise preload and, instead of 20 ml/kg, a 10 ml/kg fluid bolus should be considered in patients where a cardiac cause is suspected. In patients with signs suggestive of raised intracranial pressure (i.e. relative bradycardia and hypertension, posturing or seizures), hypotension is detrimental for cerebral perfusion, but excessive fluids carry the theoretical risk of worsening cerebral oedema; hence fluids should be given cautiously in 10 ml/kg aliquots with careful reassessment of clinical signs after each fluid bolus. Apart from trauma, septic shock and the acute abdomen, it is uncommon to need more than one or two 20 ml/kg fluid boluses for resuscitation. Reassess the patient after each fluid bolus to look for signs of improvement such as: fall in heart rate, improvement in skin perfusion and urine output, improved conscious level, increase in blood pressure and improvement in metabolic acidosis and lactate.

  Early tracheal intubation and mechanical ventilation should be considered in patients who have received more than 40 ml/kg and have signs of ongoing shock. Mechanical ventilation decreases energy requirements of the heart and respiratory muscles, allows delivery of adequate concentrations of oxygen and helps reduce the risk of development of pulmonary

oedema. It also facilitates placement of indwelling catheters for arterial and central venous access.

Renal perfusion should be monitored with a urinary catheter and hourly urine output measurement as it is an important marker of renal perfusion.

- Give an antibiotic such as ceftriaxone or cefotaxime 80 mg/kg for those with an obvious or suspected diagnosis of septicaemia, e.g. in the presence of a purpuric rash or in those where the aetiology is unknown.
  - In paediatric practice, septicaemia is the commonest cause of shock. Unless an alternative diagnosis is very obvious (such as trauma, anaphylaxis or poisoning), antibiotics should be given as soon as possible, preferably after doing a blood culture.
  - A third-generation cephalosporin, such as cefotaxime or ceftriaxone, is usually used, but an antistaphylococcal antibiotic (flucloxacillin, vancomycin) should be considered in possible toxic shock syndrome (suggested by high fever, diffuse erythema, mucous membrane changes such as conjunctivitis, strawberry tongue, cracked lips and shock – findings may also include a trivial injury such as infected wound, cut, scratch, minor burn or scald, surgical wound infection or coexistent deep-seated infection such as pneumonia or bone/joint infection).
- In patients with trauma, haemorrhage must be looked for and controlled for effective management of shock. If there are no obvious wounds or fractures accounting for blood loss, thoracic, abdominal and pelvic cavities need to be assessed thoroughly for any signs of internal bleeding. If there are no signs of bleeding anywhere, spinal shock must be considered in patients with ongoing shock. A rapid ultrasound scan (focused abdominal with sonography for trauma (FAST) scan) can rule out intra-abdominal injury and an emergency computed tomography (CT) scan can be performed once the patient is stabilised.
- If a tachyarrhythmia is identified as the cause of shock, up to three synchronous electric shocks at 1.0, 2.0 and 2.0 J/kg should be given (see Chapter 10):
  - If the arrhythmia is broad-complex and the synchronous shocks are not activated by the defibrillator then attempt an asynchronous shock.
  - A conscious child should be anaesthetised first if this can be done in a timely manner.
  - If the shocked child's tachyarrhythmia is supraventricular tachycardia (SVT) then this can be treated with intravenous/intraosseous adenosine as this can often be administered more quickly than a synchronous electric shock.
- If anaphylaxis is obvious give adrenaline 10 micrograms/kg IM or 150 micrograms (<6 years), 300 micrograms (6–12 years) or 500 micrograms (>12 years).

Disability

If there is coexistent evidence of raised intracranial pressure, manage as in Chapter 11.

'Don't ever forget glucose' (DEFG)

Hypoglycaemia may give a similar clinical picture to that of compensated shock. This must always be excluded by urgent glucose stick test and blood glucose estimation. Shock and hypoglycaemia may coexist due to limited glycogen reserves and the fact that ill children may not have had adequate nutritional intake.

9.7 KEY FEATURES OF THE CHILD IN SHOCK

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours and any significant previous illness should be gained. Certain key features which will be identified from this – and the initial blood test results – can point the clinician to the likeliest working diagnosis for emergency treatment.

• A history of vomiting and/or diarrhoea points to *fluid loss* either externally (e.g. gastroenteritis) or into the abdomen (e.g. volvulus, intussusception)	Section 9.8
• The presence of fever and/or rash points to *septicaemia*	Section 9.9
• The presence of urticaria, angioneurotic oedema or history of allergen exposure points to *anaphylaxis*	Section 9.10
• The presence of cyanosis unresponsive to oxygen or a grey colour with signs of heart failure in a baby under 4 weeks points to *duct-dependent congenital heart disease*	Section 9.11
• The presence of heart failure in an older infant or child points to *cardiomyopathy or myocarditis*.	Section 9.12
• A history of sickle cell disease or a recent diarrhoeal illness and a very low haemoglobin points to *acute haemolysis*. A history of sickle cell disease, abdominal pain and enlarged spleen points to acute splenic sequestration	Sections 9.13 and 9.14
• An immediate history of major trauma points to blood loss and, more rarely, *tension pneumothorax*, *haemothorax*, *cardiac tamponade* or *spinal cord transection*	Part 4
• The presence of severe tachycardia and an abnormal rhythm on the ECG points to a cardiac cause for shock	Chapter 10
• A history of polyuria and the presence of acidotic breathing and a very high blood glucose points to *diabetes ketoacidosis*	Appendix B
• A history of drug ingestion points to *poisoning*	Appendix H

9.8 APPROACH TO THE CHILD WITH FLUID LOSS

Infants are more likely than older children to present with shock due to sudden and rapid fluid losses due to gastroenteritis or with concealed fluid loss secondary to a 'surgical abdomen' such as a volvulus. This is due both to the infant's low physiological reserve and increased susceptibility to these conditions.

In infants, gastroenteritis may occasionally present as circulatory collapse with little or no significant history of vomiting or diarrhoea. This is due to sudden massive loss of fluid from the bowel wall into the gut lumen, causing depletion of intravascular volume. The infecting organism can be any of the usual diarrhoeal pathogens, of which viruses are the most common.

Having completed the primary assessment and resuscitation and identified by means of the key features that fluid loss is the most likely diagnosis, the child is reassessed to identify the response to the first fluid bolus.

Emergency treatment of fluid loss

Reassess ABC

- The child has had one fluid bolus of 20 ml/kg IV.
- If signs of shock persist after the first bolus, give a second fluid bolus of crystalloid or colloid:
  - in gastroenteritis, one to two boluses usually restore circulating volume, and
  - in gastroenteritis, initiate enteral or gastric tube oral rehydration solution (see Appendix B).
- Recheck acid–base status and electrolytes:
  - acidosis will usually be corrected by treatment of shock; bicarbonate losses need to be corrected only in patients who could have had large bicarbonate losses in the stool, and
  - sodium imbalance may occur, and this may cause convulsions (see Appendix B).
- Consider diagnostic possibilities:
  - abdominal X-ray or ultrasound scan to detect distended bowel, intra-abdominal air or fluid,
  - consider urgent surgical referral especially if bile-stained vomiting or abdominal guarding is present. Ensure that the patient has had antibiotics as sepsis or toxic shock syndrome may mimic an acute abdomen, and

  - consider sepsis (secondarily to the surgical abdomen) and give appropriate IV antibiotics.
- Consider tracheal intubation and mechanical ventilation, particularly if more than 40 ml/kg of fluid are required.
- Consider a third fluid bolus if still shocked; at this stage, colloid (4.5% human albumin solution) is commonly used.
- Consider the need for inotropes and monitoring of central venous pressure (CVP) – these will usually be essential if a third fluid bolus is given.
- The child's bladder should have been catheterised in order to assess accurately the urinary output.

9.9 APPROACH TO THE CHILD WITH SEPTIC SHOCK

The incidence of septic shock varies with age and is highest in infants. It carries significant mortality and morbidity. Septic shock is the classic example of a combination of several factors contributing to the shock. These include hypovolaemia (fever, often associated diarrhoea, vomiting and anorexia, together with alterations in capillary permeability leading to capillary leakage), cardiogenic (impaired cardiac function due to hypovolaemia and direct myocardial suppressive factors from infecting organisms and the host inflammatory response), distributive (alterations in vascular tone with vasoconstriction in some vascular beds and vasodilatation in others) and dissociative (there is a non-specific sepsis-induced mitochondrial dysfunction impairing cellular oxygen utilisation) elements. Septic shock is defined as sepsis with cardiovascular organ dysfunction.

Infection with *Neisseria meningitidis* (the meningococcus) is the commonest cause of community-acquired septicaemia in infants and children. In countries where a vaccine against meningococcus C has been introduced, there has been a significant fall in the number of cases of infection due to this organism. Other causes of septicaemia in children include group B streptococcal infection in young infants, Gram-negative sepsis in relation to underlying urinary tract or gut problems and group A streptococcal sepsis. In children with underlying comorbidities, respiratory or neurological infection is important, and infection of long-term indwelling devices (such as venous catheters) is becoming increasingly prevalent.

The cardinal sign of meningococcal septicaemia is a purpuric rash in an ill child. At the onset, however, the rash may be absent, or mistaken for viral exanthems and a careful search should be made for purpura in any unwell child. In about 15% of patients with meningococcal septicaemia, a blanching erythematous rash replaces or precedes a purpuric one, and in 7% of cases no rash occurs.

In toxic shock syndrome, the initial clinical picture includes a high fever, headache, confusion, conjunctival and mucosal hyperaemia, scarlatiniform rash, subcutaneous oedema, vomiting and watery diarrhoea. Early administration of antistaphylococcal and antistreptococcal antibiotics, concurrent with initial resuscitation, is vital. Intravenous immunoglobulin should be considered along with urgent drainage of any localised pus. Early adequate fluid resuscitation is the key to survival in children with septic shock; however, indiscriminate or overaggressive fluid resuscitation can be harmful. Choice of fluid is still debated; septic children requiring multiple fluid boluses should be given 4.5% human albumin after the initial 20–40 ml/kg of crystalloid fluid if available.

Having completed the primary assessment and resuscitation and identified by means of the key features that septicaemia is the most likely diagnosis, the child is reassessed.

Emergency treatment of septicaemia

Reassess ABC

- Give fluid bolus(es) of 0.9% saline:
  - Subsequent boluses should be either 0.9% saline or 4.5% human albumin.
  - Children often require repeated boluses of fluid to achieve relative stability (up to 200 ml/kg in the first 24 hours has been used to treat severe shock, i.e. 2.5 times the blood volume).

  - After 40 ml/kg, if there are signs of ongoing shock and continuing need for fluid resuscitation, urgent tracheal intubation and mechanical ventilation should be instituted. It will be necessary to consider inotropes and ideally to monitor CVP.
  - Central venous access will be valuable, particularly in shock needing ≥40 ml/kg. It should ideally be achieved using a multilumen catheter. The femoral approach is most commonly used, and if expertise is available, the internal jugular vein can also be used. Adult data suggest that optimal CVP is 8 cmH_2O. Optimising CVP can improve cardiac output with less risk of inducing heart failure. Cardiac failure may be induced by excessive IV fluids, especially if severe anaemia, malnutrition or a primary cardiac disorder is present. Inotropic support will be required. Recent data suggest that measurement of central venous oxygen saturation ($SCVO_2$) from a central vein in the neck is useful to determine oxygen delivery and utilisation in order to guide resuscitation, aiming for a $SCVO_2$ of >70%.
- Ensure that an antibiotic such as cefotaxime or ceftriaxone has been given.
- Consider tracheal intubation by rapid sequence induction of anaesthesia and provide assisted ventilation:
  - Positive pressure ventilation can improve oxygenation, and prevents/treats pulmonary oedema. It can improve cardiac output.
  - All intubated children must have continuous Spo_2 and capnography, with frequent blood gas monitoring.
- Consider an intravenous infusion of dopamine:
  - This is considered if a third bolus of fluid is required. Start at a dose of 10 micrograms/kg/min and increase to 20 micrograms/kg/min if there is a poor response.
  - Dopamine can initially be given through a peripheral vein until central venous access or intraosseous access is obtained. Do not hesitate to increase the infusion rapidly in the face of a poor response.
  - Adrenaline by IV infusion at 0.05–2 micrograms/kg/min or other vasoactive agents may be required if there is no response to dopamine. Ideally, these should be given centrally but it may be necessary to infuse adrenaline peripherally if no other access is immediately available.

It is difficult to manage a seriously ill patient requiring mechanical ventilation and inotropic support without intensive care facilities and invasive monitoring. If these treatments are required, a paediatric intensive care unit must be involved early to give advice and to retrieve the patient.

Further investigations

In addition to the blood tests taken during resuscitation, the following blood tests are needed in the septic child: calcium, magnesium, phosphate, coagulation screen and arterial blood gas. Electrolyte and acid–base abnormalities can have a deleterious effect on myocardial function. They should be sought and corrected early (Table 9.2)

Table 9.2 Corrective measures for electrolyte and acid–base derangements in shock

Result	Treat if less than	Correct with
Glucose	3 mmol/l	2 ml/kg 10% glucose followed by maintenance glucose
Metabolic acidosis	pH < 7.2	Half correction $NaHCO_3$ (avoid in diabetic ketoacidosis) Ensure adequate ventilation
Potassium	3.5 mmol/l	0.25 mmol/kg KCl over 30 min: ECG monitoring
Calcium	Ionised <1	0.3 ml/kg 10% Ca gluconate over 30 min (max. 20 ml)
Magnesium	0.75 mmol/l	0.2 ml/kg 50% $MgSO_4$ over 30 min (max. 10 ml)

Reassess disability

Meningitis may accompany septicaemia. Assess neurological status (conscious level, using the Glasgow Coma Score, pupillary size and reaction, and posture), particularly looking for signs of raised intracranial pressure. If, despite effective treatment of shock, there is decreasing conscious level or abnormal posturing or focal neurological signs, treat for raised intracranial pressure (ICP).

Treatment of disability in shock

The priority in patients with a mixed picture of shock and meningitis is management of shock, as adequate brain perfusion is dependent on adequate cardiac output. If signs of raised ICP persist, tracheal intubation and mechanical ventilation should be initiated urgently.

- Monitor CO_2 levels by capnography and blood gases, and keep within a normal range (4.5–5.5 kPa).
- Insert a urinary catheter early, and monitor urine output.
- Nurse the child with 20° head elevation and midline position.
- Maintenance of a normal blood pressure to ensure an adequate cerebral perfusion pressure is mandatory. Treatment of the shocked state takes priority over treatment of increased ICP. An adequate blood pressure is necessary to maintain cerebral perfusion.
- Lumbar puncture must be avoided as its performance may cause death through coning of the brainstem through the foramen magnum.

Paediatric intensive care skills and monitoring are paramount in these patients. Seek advice early.

9.10 APPROACH TO THE CHILD WITH ANAPHYLAXIS

Anaphylaxis is a potentially life-threatening, immunologically mediated reaction to ingested, inhaled or topical substances, which may present as either shock or respiratory distress. Common triggers include certain foods, especially nuts and shellfish, and drugs, such as penicillin, anaesthetic agents and radiographic contrast media. The life-threatening features include breathing difficulties with stridor or wheeze, or shock, due to acute vasodilatation and fluid loss from the intravascular space caused by increased capillary permeability. Any of these may lead to collapse and respiratory or cardiac arrest.

Prodromal symptoms of flushing, itching, facial swelling, urticaria, abdominal pain, diarrhoea, wheeze and stridor may precede shock or may be the only manifestations of anaphylaxis. The presence of these additional symptoms confirms anaphylaxis as the cause of breathlessness and/or shock in a child. Most patients will have a history of previous, less severe allergic reactions, some may have a ‘medic-alert’ bracelet or carry their own adrenaline. Confirmation of the diagnosis may be aided by measurement of blood mast cell tryptase levels.

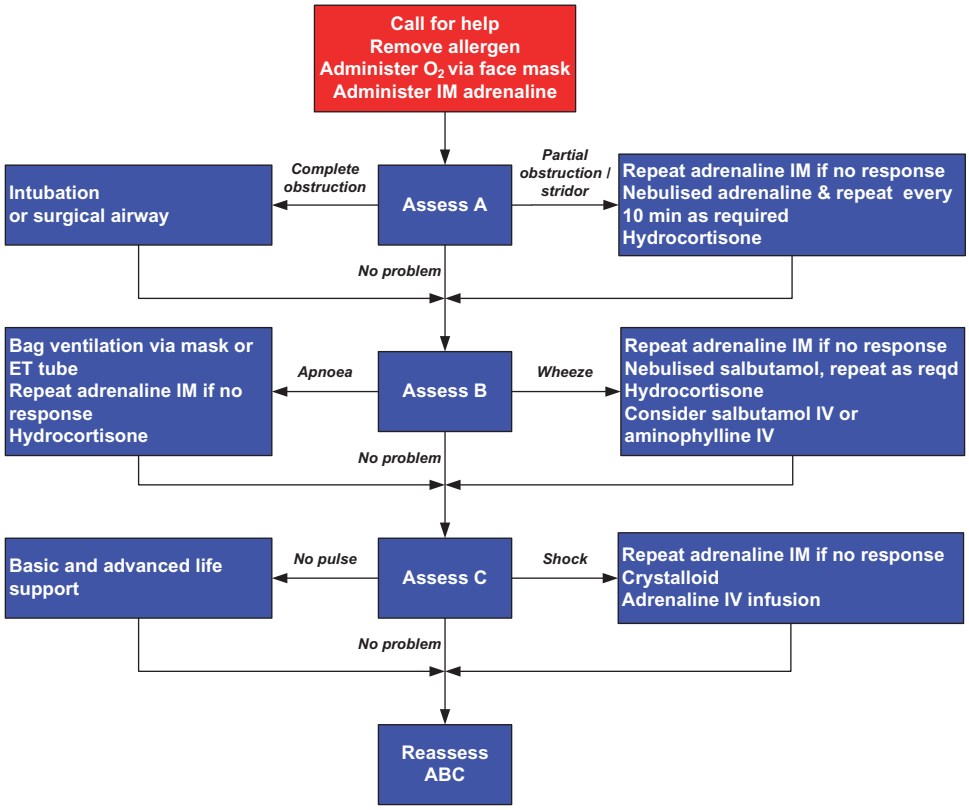
Key points in the history may point to a severe reaction. These are shown in the box below.

Previous severe reaction
History of increasingly severe reaction
History of asthma
Treatment with β-blockers

Symptoms and signs vary according to the body’s response to the allergen. These are shown in Table 9.3.

Table 9.3 Symptoms and signs in allergic reaction

	Symptoms	Signs
Allergic reactions	Burning sensation in mouth, itching of lips, mouth and throat, coughing, feeling of warmth, nausea, abdominal pain, loose bowel motions, sweating	Urticarial rash, angio-oedema, conjunctivitis
Anaphylaxis	Difficulty breathing, noisy breathing, cyanosis, agitation, collapse	Wheeze, stridor, tachycardia with hypotension, poor pulse volume and pallor, respiratory arrest or cardiac arrest



Drugs in anaphylaxis	Dosage by age			
	Less than 6 months	6 months to 6 years	6 – 12 years	More than 12 years
Adrenaline IM – pre-hospital practitioners	150 micrograms (0.15 ml of 1:1000)		300 micrograms (0.3 ml of 1:1000)	500 micrograms (0.5 ml of 1:1000)
Adrenaline IM – in-hospital practitioners	10 micrograms/kg *0.1ml/kg of 1:10,000 (infants and young children) OR 0.01ml/kg of 1:1000 (older children)*[1]			
Adrenaline IV	Titrate 1 microgram/kg*			
Crystalloid	20 ml/kg			
Hydrocortisone (IM or slow IV)	25 mg	50 mg	100 mg	200 mg

* 1 microgram/kg given over 1 minute (range 30 seconds to 10 minutes), e.g. 0.5 ml/kg of 1:10,000 adrenaline made up to 50 ml saline 0.9% and run at 1ml/min is 1 microgram/kg/min

[1] The strength of IM adrenaline is not intended to be prescriptive, 1:1000 or 1:10,000 could be used depending on what is practicable. The problem with sticking solely to 1:1000 is that when used in infants and small children, you are then drawing up very small volumes

Figure 9.1 Emergency treatment of anaphylaxis

Emergency treatment of anaphylaxis (Figure 9.1)

Specific treatment includes:

- Oxygen.
- Intramuscular adrenaline: either 10 micrograms/kg *or* 150 micrograms (<6 years), 300 micrograms (6–12 years) or 500 micrograms (>12 years).

- Nebulised adrenaline 400 micrograms/kg, 0.4 ml/kg ml of 1:1000, as for croup.
- Nebulised bronchodilator.

The management of anaphylactic shock requires airway management and ventilation, administration of adrenaline and aggressive fluid resuscitation. Intubation will be required for severe cases.

Note that the intramuscular route is the preferred route for the delivery of adrenaline. Intravenous/intraosseous adrenaline should be reserved for children with life-threatening shock or airway obstruction, for whom an intramuscular injection has had to be given in repeated doses or been ineffective or for those in cardiac arrest. The patient must be carefully monitored.

Having completed the primary assessment and resuscitation and identified by means of the key features that anaphylaxis is the most likely diagnosis, the child is reassessed.

Further emergency management of anaphylaxis

For ongoing shock, and shock resistant to treatment, continue with boluses of crystalloid or colloid and ventilatory support and give further doses of adrenaline intramuscularly every 5 minutes if the symptoms are not reversed. Additional inotropes will not be needed because the adrenaline used for the treatment of anaphylaxis is a powerful inotrope. However, in the face of shock resistant to intramuscular adrenaline and one or two boluses of fluid, an infusion of intravenous adrenaline may be life saving. The dose is 0.1–5.0 micrograms/kg/min and the patient should be closely monitored for pulse and blood pressure.

In addition to the above treatment, it is also customary to give patients with anaphylaxis an antihistamine and steroids. The role these drugs play in management is limited, as their onset of action is too delayed to be of much benefit in the first hour.

9.11 APPROACH TO THE INFANT WITH A DUCT-DEPENDENT CONGENITAL HEART DISEASE

Features suggesting a cardiac cause of circulatory inadequacy

- Cyanosis, not correcting with oxygen therapy
- Tachycardia out of proportion to respiratory difficulty
- Raised jugular venous pressure
- Gallop rhythm, murmur
- Enlarged heart on chest X-ray
- Enlarged liver
- Absent femoral pulses

The ductus arteriosus connects the systemic and pulmonary circulations in fetal life. Infants with duct-dependent right or left heart lesions present in the first few days of life as the ductus arteriosus starts closing in response to transition from fetal to postnatal life.

Neonates with duct-dependent pulmonary circulation (e.g. critical pulmonary stenosis, pulmonary atresia, tricuspid atresia) present in the first few days of life with increasing cyanosis unresponsive to supplemental oxygen and signs of severe hypoxaemia with little respiratory distress before collapsing with cardiogenic shock. A high index of suspicion is required to diagnose these conditions as frequently there is no audible murmur. Patients may have tachycardia, tachypnoea and an enlarged liver.

Neonates with duct-dependent systemic circulation (e.g. transposition of great arteries, aortic stensosis/atresia, hypoplastic left heart syndrome, coarctation of aorta) also present in the first few days of life with inability to feed, breathlessness, a grey appearance and collapse with poor peripheral circulation and cardiogenic shock. These infants have signs of poor organ perfusion

with severe metabolic acidosis, poor urine output and decreased conscious level. Infants with left heart obstructive lesions such as critical aortic stenosis or coarctation can be severely ill; pulses can be difficult to feel in these patients because of left-sided obstruction to cardiac output and a difference may be noticed in the upper and lower limb pulses and blood pressure depending on the site of the lesion.

Having completed the primary assessment and resuscitation and identified by means of the key features that duct-dependent congenital heart disease is the most likely diagnosis, the child is reassessed.

Emergency treatment of duct-dependent congenital heart disease

Reassess ABC

- Oxygen therapy will usually have little beneficial effect, and may accelerate duct closure, therefore use oxygen judiciously. Keep a low threshold for tracheal intubation and mechanical ventilation in patients with cardiogenic shock. This decreases metabolic demands of the body and assists cardiac function.

> The most experienced personnel should be summoned for support in these situations as use of anaesthetic-induction agents can worsen the situation. Frequent discussion with a paediatric cardiologist and intensivist is mandatory.

- Give an intravenous infusion of Prostin (e.g. for prostaglandin E2 (PGE2)):
  - an initial dose of 5 nanograms/kg/min (may be increased to 20 nanograms/kg/min in 5 nanograms/kg/min increments until side effects develop),
  - this will reopen and keep the arterial duct patent which will help in stabilising the patient before definitive surgical intervention, and
  - prostaglandins can cause apnoea in some infants; frequent assessment is necessary to identify those who need ventilatory support.

Prostaglandins can also cause vasodilatation and subsequent drop in blood pressure. Such patients benefit from a fluid bolus to optimise preload.

Investigations

- Chest X-ray.
- ECG.
- Full blood count.
- Arterial blood gases.
- Urea and electrolytes, and calcium.
- Glucose.
- Lactate.
- Blood cultures.

Discuss with and transfer to a paediatric cardiology unit. Monitor pre- and post-ductal saturations.

9.12 APPROACH TO THE CHILD WITH CARDIOMYOPATHY

Cardiomyopathy/myocarditis is uncommon, but may rarely be found in an infant or child presenting with shock, arrhythmias and signs of heart failure with no history of congenital heart disease. It may be difficult to differentiate these patients from septic patients and treatment is dictated by the management of shock.

If such a patient were in the first few weeks of life, a trial of Prostin (PGE1 or 2) would be appropriate and would be beneficial for duct-dependent circulations as discussed above.

Having completed the primary assessment and resuscitation and having identified by means of the key features that cardiomyopathy/myocarditis is the most likely diagnosis, the child is reassessed.

Emergency treatment of cardiomyopathy

Reassess ABC

- Give high-flow oxygen.
- Give fluid resuscitation in 10 ml/kg aliquots, assessing response after each bolus. Children may be fluid depleted and have cardiac dysfunction, so judicious use of fluid would not be harmful.
- Careful assessment and consideration is required of the cardiac haemodynamics, namely, preload, contractility and afterload. Treatment then needs to be titrated according to the clinical picture. Consider a diuretic, if the child is not shocked, to offload the heart, such as frusemide (furosemide) 1 mg/kg IV. Start an intravenous infusion of dobutamine 5–20 micrograms/kg/min. Afterload-reducing agents may eventually be required in the majority of patients.

Investigations

- Chest X-ray.
- ECG.
- Full blood count.
- Arterial blood gases.
- Urea and electrolytes, and calcium.
- Glucose.
- Lactate.
- Blood cultures.

Urgent cardiology advice should be sought. Echocardiography should establish the diagnosis in almost all cases. Consider early liaison and transfer to a paediatric cardiac centre as these children can be extremely difficult to manage.

9.13 APPROACH TO THE CHILD WITH PROFOUND ANAEMIA

Severe anaemia exists if the haemoglobin level is less than 50 g/l. If acute haemolysis is the cause of anaemia, urine will usually be dark brown in colour, the child will be lethargic with palms and soles near white, and there may be signs of heart failure. The most usual situation in which a child develops sudden severe haemolysis is in the case of septicaemia associated with sickle cell disease. In children returning from endemic areas, severe malaria may present with severe anaemia, with or without haemolysis.

Emergency treatment of profound anaemia

- Transfusion should in general be considered when the haemoglobin level is less than 50 g/l.
- The presence of heart failure affects the decision to transfuse; diuretics will be required or an exchange transfusion may be safer.
- Overhydration may exacerbate or lead to cardiogenic shock and pulmonary oedema.
- Treatment may also be required as for sepsis with volume support, intubation and inotropes.
- Fresh blood should be used where possible.
- Management of these children includes early paediatric intensive care advice and transfer.

9.14 APPROACH TO THE CHILD WITH SICKLE CELL CRISIS

Sickle cell disease is characterised by episodic clinical events called ‘crises’. Vaso-occlusive crisis is the most common and occurs when abnormal red cells clog small vessels, causing tissue ischaemia. The other crises are acute chest syndrome, sequestration crisis (severe anaemia and hypotension, resulting from pooling of blood in the spleen and liver), aplastic crisis and hyperhaemolytic crisis. Factors that precipitate or modulate the occurrence of sickle cell crises are

not fully understood, but infections, hypoxia, dehydration, acidosis, stress and cold are believed to play some role.

Oxygen therapy, rehydration, antibiotics and analgesia are considered standard treatment in sickle cell crises. Parenteral morphine is also considered essential for relieving pain in severe vaso-occlusive crises and acute chest syndrome.

9.15 AFTER RESUSCITATION AND EMERGENCY TREATMENT OF SHOCK

Following successful restoration of adequate circulation, varying degrees of organ dysfunction may remain, and should be actively sought and managed. The problems are similar but of a lesser degree than those expected following resuscitation from cardiac arrest. Thus after the initial resuscitation and emergency treatment, the patient should have a review of ABC, as well as a full systems review to ensure stabilisation for safe and effective transfer (see Chapter 24).

9.16 USE OF FLUIDS IN RESUSCITATION

Which fluid?

Crystalloid and colloid fluids are available for volume replacement. Dextrose infusions are not appropriate fluids for resuscitation and can be dangerous, for example, by lowering serum sodium and predisposing to hyponatraemic seizures. For further details of the composition of different fluids, see Appendix B.

- Compared with colloids, crystalloid fluids diffuse more readily into the interstitial space.
- Compared with colloids, crystalloid fluids may be associated with more peripheral oedema.
- Where capillary leak exists, crystalloid fluids allow more water to enter the interstitial space, because of a lower osmotic pressure.
- Crystalloid fluids need 2–3 times the volume of colloids to expand the vascular space.

There has been a longstanding debate about whether crystalloid or colloid solutions should be used in resuscitation. No definitive answer can be given. Where small volumes of fluid are needed the choice of fluid is probably not important.

In acute collapse, a smaller volume of colloid fluid is needed than crystalloid to produce a given increase in intravascular volume, and so a more rapid correction of haemodynamic derangement may be possible with colloid solutions if these are readily available.

When larger volumes of fluid are used, the choice of fluid becomes more important. As the circulating volume of a child is approximately 80 ml/kg, if more than 40 ml/kg of fluid is used for resuscitation, one-half of the child's circulating volume will have been given. If much more fluid resuscitation is needed, significant haemodilution may result, and consideration should be given to using blood for fluid resuscitation with measurements of the central venous pressure (effectively cardiac preload) to guide fluid resuscitation and the haematocrit to guide the need for blood transfusion. Where large volumes are used, 4.5% or 5% human albumin solution is generally preferred in paediatric practice, although most adult patients are resuscitated with synthetic colloids, crystalloid or hypertonic solutions.

If blood is needed, a full cross-match is to be undertaken, which takes about 1 hour to perform. For urgent need, type-specific non-cross-matched blood (which is ABO Rhesus compatible but has a higher incidence of transfusion reactions) takes about 15 minutes to prepare. In dire emergencies O-negative blood must be given.

How much fluid?

The volume of fluid needed will depend on clinical assessment. However, the clinical situation may dictate the rapidity with which repeat boluses are given. For example, in a retrospective review of children with septic shock, early administration of large volumes of fluid (>40 ml/kg in the first hour) was associated with better outcome than smaller volume resuscitation, encouraging an aggressive approach in septicaemia. In contrast, where shock is caused by

penetrating trauma requiring definitive surgical management, maximal fluid resuscitation may be best delayed until surgery, as improving perfusion without improving oxygen-carrying capacity results in a worse outcome.

If large volumes are needed, resuscitation is best guided by measurement of the CVP and invasive blood pressure and urine output. Patients requiring large-volume resuscitation need early involvement from and transfer to a paediatric intensive care unit. When large volumes are used, fluids should be warmed.

In conclusion, there is no definitive evidence demonstrating which fluid is best for resuscitation. Other important questions – how much and when should fluids be used – also remain to be answered. Clinical trials will be needed to answer these questions, though they are likely to be difficult to perform. At present, optimal management should be guided by knowledge of the pathophysiology underlying the disease, and of the different roles of the different fluids.

9.17 SUMMARY

You should use the structured approach in the assessment and management of the child with shock:

- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

CHAPTER 10

The child with an abnormal pulse rate or rhythm

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to assess children with an abnormal pulse rate or rhythm
- How to resuscitate the child with life-threatening brady- or tachyarrhythmia

10.1 INTRODUCTION

In tachyarrhythmias in children, the rate is fast but the rhythm is largely regular. Causes include:
- Re-entrant congenital conduction pathway abnormality (common).
- Poisoning.
- Metabolic disturbance.
- After cardiac surgery.
- Cardiomyopathy.
- Long QT syndrome.

In bradyarrhythmias in children, the rate is slow and the rhythm usually irregular. Causes include:
- Pre-terminal event in hypoxia or shock.
- Raised intracranial pressure.
- After conduction pathway damage during cardiac surgery.
- Congenital heart block (rare).
- Long QT syndrome.

Presentations include:
- History of palpitations (verbal child).
- Poor feeding (pre-verbal child).
- Heart failure or shock.

10.2 PRIMARY ASSESSMENT

This is dealt with in Chapter 7. Below is a summary.

Airway
- Assess vocalisations: crying or talking indicate ventilation and some degree of airway patency.
- Assess airway patency by:

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  - *looking* for chest and/or abdominal movement, symmetry and recession,
  - *listening* for breath sounds and stridor, and
  - *feeling* for expired air.
- *Reassess after any airway-opening manoeuvres.* If there continues to be no evidence of air movement then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see Chapter 4).

Breathing

- Effort of breathing:

respiratory rate	recession	
stridor	wheeze	grunting
accessory muscle use	flaring of nostrils	gasping

Exceptions

Increased effort of breathing *does not* occur in three circumstances:

1 Exhaustion.
2 Central respiratory depression.
3 Neuromuscular disease.

- Efficacy of breathing:
  - chest expansion/abdominal excursion,
  - breath sounds – reduced or absent, and symmetry on auscultation, and
  - Spo_2 in air.
- Effects of respiratory failure on other physiology:
  - heart rate,
  - skin colour, and
  - mental status.

Circulation

- Heart rate: this is the defining observation for this presentation. An abnormal pulse rate is defined as one falling outside the normal range given in Chapter 2.

  In practice, most serious disease or injury states are associated with a sinus tachycardia. In infants this may be as high as up to 220 beats per minute (bpm) and in children up to 180 bpm. Rates over these figures are highly likely to be tachyarrhythmias, but in any case of significant tachycardia, i.e. 200 bpm in an infant and 150 bpm in a child, an electrocardiogram (ECG) rhythm strip should be examined and, if in doubt, a full 12-lead ECG performed. Very high rates may be impossible to count manually and the pulse oximeter is often unreliable in this regard. Again, a rhythm strip is advised.

  An abnormally slow pulse rate is defined as one less than 60 bpm or a rapidly falling heart rate associated with poor systemic perfusion. This will almost always be in a child who requires major resuscitation.
- Pulse volume.
- Blood pressure.
- Capillary refill.
- Skin temperature.

Disability

- Mental status/conscious level.
- Posture.
- Pupils.

Exposure
- Rash or fever.

10.3 RESUSCITATION

Airway
If the airway is not open, use one of the following:
- An airway-opening manoeuvre.
- An airway adjunct.
- Urgent induction of anaesthesia followed by intubation to secure the airway.

Breathing
- Give high-flow oxygen through a face mask with a reservoir as soon as the airway has been shown to be adequate.
- If the child is hypoventilating or has bradycardia, respiration should be supported with oxygen via a bag–valve–mask device and consideration given to intubation and ventilation.

Circulation
- If there is shock and the heart rate is <60/min, start chest compressions.
- If there is shock and the ECG shows ventricular tachycardia (VT), give up to one or two synchronous electric shocks at 1 and 2 J/kg.
  - The child who is responsive to pain should be anaesthetised or sedated first.
  - If the synchronous shocks for VT are ineffectual (because the defibrillator cannot recognise the abnormally shaped QRS complex), then the shocks may have to be given asynchronously, recognising that this is a more risky procedure – because without conversion the rhythm may deteriorate to ventricular fibrillation (VF) or asystole.
  - Synchronisation relies on the ability of the defibrillator to recognise the QRST complex, and is designed to avoid shock delivery at a point in the cardiac cycle likely to precipitate VF.
- Gain intravenous or intraosseous access.
- If the tachyarrhythmia is supraventricular tachycardia (SVT) then give intravenous/intraosseous adenosine (see Appendix J for more on adenosine) 100 micrograms/kg to a maximum single dose of 500 micrograms/kg (300 micrograms/kg if the baby is aged under 1 month) if this can be administered more quickly than a synchronous electric shock.
- Take blood for full blood count, renal function, glucose stick test and glucose laboratory test.
- Give a bolus of 20 ml/kg IV of crystalloid to a patient with bradycardia who is in shock.

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours should be gained. Certain key features that will be identified clinically in the primary assessment, from the focused history, from the initial blood tests and from the rhythm strip and 12-lead ECG can point the clinician to the likeliest working diagnosis for emergency treatment.

From the ECG the arrhythmia can be categorised by the following simple questions:

1 Is the *rate*:
too fast?
too slow?

2 Is the *rhythm*:
regular?
irregular?

3 Are the QRS *complexes*:
narrow?
broad?

Bradycardia

- Bradycardia is usually a pre-terminal rhythm. It is seen as the final response to profound *hypoxia* and *ischaemia* and its presence is ominous.
- Bradycardia is precipitated by *vagal stimulation* as occurs in tracheal intubation and suctioning and may be found in postoperative cardiac patients. The rhythm is usually irregular.
- Bradycardia may be seen in patients with *raised intracranial pressure*. These patients will have presented with coma and their management can be found in Chapters 11 and 16.
- Bradycardia can be a side effect of *poisoning* with *digoxin* or *β-blockers* and the management can be found in Appendix H.

Tachyarrhythmia

- Tachyarrhythmia with a narrow QRS complex on the ECG is *supraventricular tachycardia*. The rhythm is usually regular.
- Tachyarrhythmia with a wide QRS complex on the ECG is *ventricular tachycardia*; this can be provoked by:
  - *hyperkalaemia*, or
  - poisoning with *tricyclic antidepressant*s. Additional details on the management of the poisoned child with ventricular tachycardia can be found in Appendix H.

10.4 APPROACH TO THE CHILD WITH BRADYCARDIA

In paediatric practice bradycardia is almost always a pre-terminal finding in patients with respiratory or circulatory insufficiency. Airway, breathing and circulation should always be assessed and treated if needed before pharmacological management of bradycardia.

Emergency treatment of bradycardia (Figure 10.1)

Reassess ABC

- If there is hypoxia and shock, treat with:
  - High concentration oxygen, bag–mask ventilation, intubation and intermittent positive pressure ventilation.

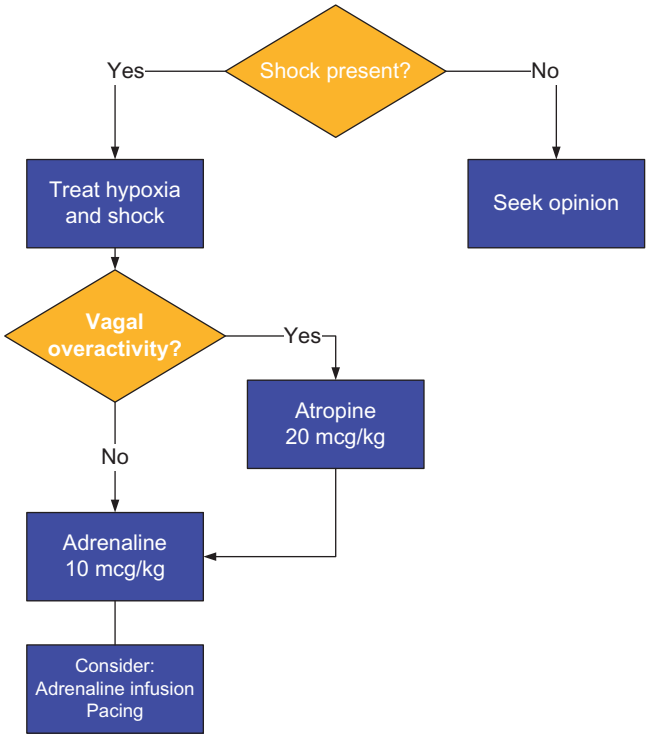

Figure 10.1 Algorithm for the management of bradycardia

  - Volume expansion (20 ml/kg of 0.9% saline repeated as recommended in the treatment of shock).
  - If the above is ineffective give a bolus of adrenaline 10 micrograms/kg IV.
  - If the above is ineffective try an infusion of adrenaline 0.05–2 micrograms/kg/min IV.
- If there has been vagal stimulation:
  - Treat with adequate ventilation.
  - Give atropine 20 micrograms/kg IV/IO (minimum dose 100 micrograms; maximum dose 600 micrograms).
  - The dose may be repeated after 5 minutes (maximum total dose of 1 mg in a child and 2 mg in an adolescent).
  - If IV/IO access is unavailable, atropine (0.04 mg/kg) may be administered tracheally, although absorption into the circulation may be unreliable.
- If there has been poisoning, seek expert toxicology help.

10.5 APPROACH TO THE CHILD WITH SUPRAVENTRICULAR TACHYCARDIA

Supraventricular tachycardia is the most common non-arrest arrhythmia during childhood and is the most common arrhythmia that produces cardiovascular instability during infancy. SVT in infants generally produces a heart rate >220 bpm, and often 250–300 bpm. Lower heart rates occur in children during SVT. The QRS complex is narrow, making differentiation between marked sinus tachycardia due to shock and SVT difficult, particularly because SVT may also be associated with poor systemic perfusion.

The following characteristics may help to distinguish between sinus tachycardia and SVT (Figures 10.2 and 10.3):

- Sinus tachycardia is typically characterised by a heart rate less than 200 bpm in infants and children whereas infants with SVT typically have a heart rate greater than 220 bpm.

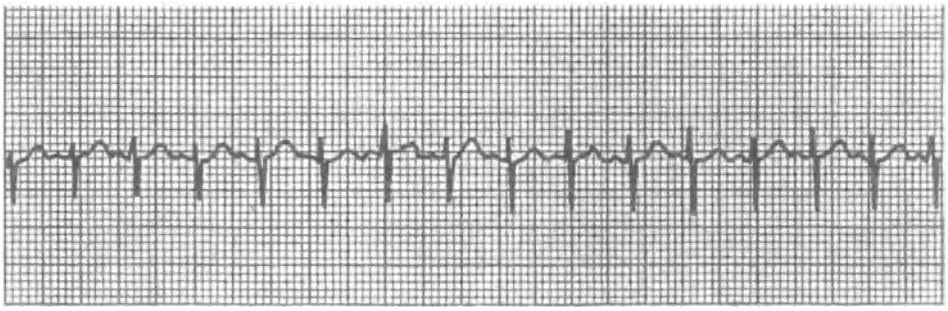

Figure 10.2 Sinus tachycardia

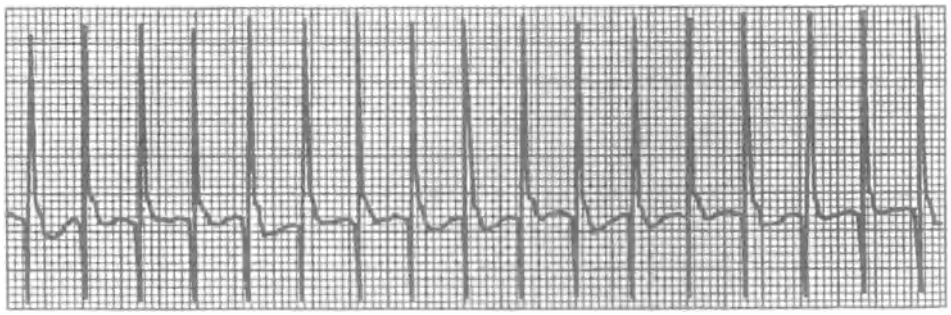

Figure 10.3 Supraventricular tachycardia

- P-waves may be difficult to identify in both sinus tachycardia and SVT once the ventricular rate exceeds 200 bpm. If P-waves are identifiable, they are usually upright in leads I and AVF in sinus tachycardia while they are negative in leads II, III and AVF in SVT.
- In sinus tachycardia, the heart rate varies from beat to beat and is often responsive to stimulation, but there is no beat to beat variability in SVT.
- Termination of SVT is abrupt whereas the heart rate slows gradually in sinus tachycardia in response to treatment.
- A history consistent with shock (e.g. gastroenteritis or septicaemia) is usually present with sinus tachycardia.

Cardiopulmonary stability during episodes of SVT is affected by the child's age, duration of SVT and prior ventricular function and ventricular rate. Older children usually complain of lightheadedness, dizziness or chest discomfort or they note the fast heart rate, but very rapid rates may be undetected for long periods in young infants until they develop a low cardiac output state and shock. This deterioration in cardiac function occurs because of increased myocardial oxygen demand and limitation in myocardial oxygen delivery during the short diastolic phase associated with very rapid heart rates. If baseline myocardial function is impaired (e.g. in a child with a cardiomyopathy), SVT can produce signs of shock in a relatively short time.

Emergency treatment of supraventricular tachycardia (Figure 10.4)

Reassess ABC

- Try vagal stimulation while continuing ECG monitoring. The following techniques can be used:
  - Elicit the 'diving reflex', which produces an increase in vagal tone, slows atrioventricular conduction and interrupts the tachycardia. This can be done by the application of a rubber

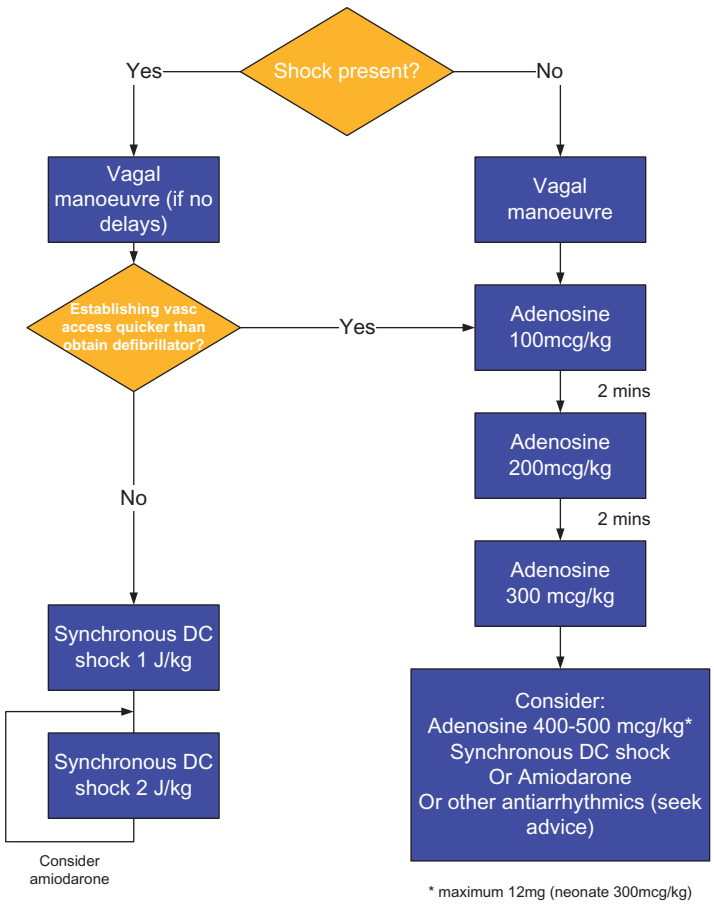

Figure 10.4 Algorithm for the management of supraventricular tachycardia

glove filled with iced water over the face, or if this is ineffectual, wrapping the infant in a towel, and immersing the face in iced water for 5 seconds.
  - One-sided carotid sinus massage.
  - Older children can try a Valsalva manoeuvre. Some children know that a certain position or action will usually effect a return to sinus rhythm. Blowing hard through a straw may be effective for some children.
- Do not use ocular pressure in an infant or child, because ocular damage may result.

If these manoeuvres are unsuccessful, give:

- *Intravenous adenosine*: start with a bolus dose of 100 micrograms/kg intravenously and increase the dose to 200 micrograms/kg after 2 minutes if success is not achieved. The next dose should be 300 micrograms/kg. The maximum single dose that should be given is 500 micrograms/kg (300 micrograms/kg in a child under 1 month) up to a maximum of 12 mg. Adenosine is a very rapidly acting drug with a half-life of less than 10 seconds. This means that side effects (flushing, nausea, dyspnoea, chest tightness) are short lived. It also means, however, that the effect may be short lasting and the SVT may recur.

  For the same reason, if the drug is given through a small peripheral vein, an insufficiently high concentration may reach the heart and therefore a larger dose may need to be given. Preferably, the drug should be injected into a large peripheral vein and rapidly followed by a saline flush. Adenosine is the drug of choice for SVT because of its efficacy and safety record.

If the stable SVT of a child has not been converted to a normal rhythm with intravenous adenosine, it is essential to seek the advice of a paediatric cardiologist before further treatment. One of the following may be suggested:

- *Amiodarone*: this drug can be used in refractory atrial tachycardia. The dose is 5 mg/kg over 30 minutes.
- *Flecainide* (2 mg/kg over at least 10 minutes): this is a membrane stabiliser but can be proarrhythmic and has a negative inotropic effect.

DC cardioversion under general anaesthetic is preferable, but if used, only one drug should be given and further cardiological advice sought:

- *Digoxin*: dosage schedules vary with age and underlying condition. Seek advice.
- *Verapamil*: this drug has been associated with irreversible hypotension and asystole when given to infants. It therefore should *not be used in children under 1 year of age*. The dose is 100–300 micrograms/kg, to a maximum of 5 mg. The drug should be terminated when sinus rhythm is seen, even if the calculated dose has not been given. *Do not use if a patient has received β-blockers, flecainide or amiodarone*.
- *Propranolol* (25–50 micrograms/kg slowly intravenously): only use if pacing is available because asystole may occur. *Do not give propranolol if the patient has been given verapamil*.

It is unsafe to give verapamil and propranolol to the same patient because they both have negative inotropic actions. It is, however, safe to give propranolol and digoxin.

10.6 APPROACH TO THE CHILD WITH VENTRICULAR TACHYCARDIA

In the haemodynamically stable child with ventricular tachycardia a history should be carefully obtained to identify an underlying cause for the tachycardia because this will often determine ancillary therapy.

- Consider the following underlying causes:
  - congenital heart disease and surgery,
  - poisoning with tricyclic antidepressants, procainamide, quinidine,
  - renal disease or another cause of hyperkalaemia, or
  - long QT syndrome.
- Look for characteristics of the ECG indicative of torsade de pointes: polymorphic VT with QRS complexes that change in amplitude and polarity so that they appear to rotate around an isoelectric line. This is seen in conditions characterised by a long QT interval or poisoning

with quinine, quinidine, disopyramide, amiodarone, tricyclic antidepressants, digoxin and cisapride with erythromycin.
- Check serum potassium, magnesium and calcium levels.
- Analysis of the ECG should be done in consultation with a paediatric cardiologist, who should be sent a copy urgently.

Emergency treatment of ventricular tachycardia (Figure 10.5)

Reassess ABC

- The treatment of the haemodynamically stable child with ventricular tachycardia should always include early consultation with a paediatric cardiologist. They may suggest:
  - amiodarone (5 mg/kg over 20 minutes; 30 minutes in neonates), or
  - intravenous procainamide (15 mg/kg over 30–60 minutes, monitor ECG and blood pressure).
- Both can cause hypotension, which should be treated with volume expansion.
- In cases where the ventricular arrhythmia has been caused by drug toxicity, sedation/anaesthesia and DC shock may be the safest approach. Use synchronous shocks initially, as these are less likely to produce ventricular fibrillation than an asynchronous shock. If synchronous shocks are ineffectual, subsequent attempts will have to be asynchronous if the child is in shock.
- The treatment of torsade de pointes VT is magnesium sulphate in a rapid IV infusion (several minutes) of 25–50 mg/kg (up to 2 g).
- Amiodarone 5 mg/kg may be given over a few minutes in VT if the child is in severe shock.

It is important not to delay a safe therapeutic intervention for longer than necessary in VT as the rhythm often deteriorates quite quickly into pulseless VT or VF.

Wide QRS SVT (i.e. SVT with aberrant conduction) is uncommon in infants and children. Correct diagnosis and differentiation from ventricular tachycardia depends on careful analysis of at least a 12-lead ECG that may be supplemented by information from an oesophageal lead. The patient and family history should be evaluated to help identify the presence of an underly-

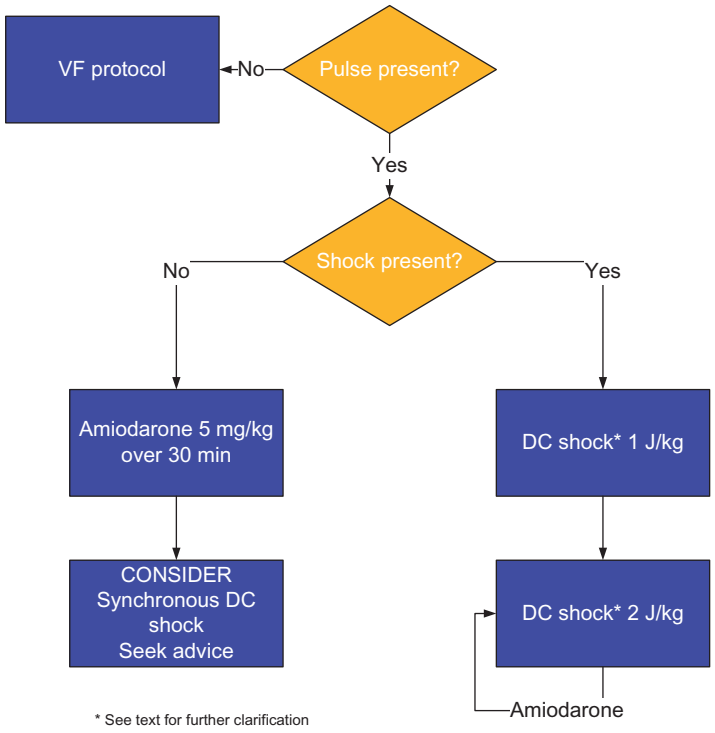

Figure 10.5 Algorithm for the management of ventricular tachycardia

ing condition predisposing to stable VT. Because either SVT or VT can cause haemodynamic instability, assumptions about the mechanism (i.e. ventricular versus supraventricular) should not be based solely on the haemodynamic status of the patient.

A dose of adenosine may help identify the underlying aetiology of the arrhythmia, but should be used with extreme caution in haemodynamically stable children with wide-complex tachycardia because acceleration of the tachycardia and significant hypotension are known risks and should not delay definitive treatment in children with shock. Seek advice.

10.7 SUMMARY

You should use the structured approach in the assessment and management of the child with an abnormal pulse rate or rhythm:

- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

CHAPTER 11

The child with a decreased conscious level

LEARNING OBJECTIVES

In this chapter, you will learn:

- The causes of a decreased conscious level in infants and children
- About the pathophysiology of raised intracranial pressure
- How to assess children with a decreased conscious level
- How to resuscitate the child with a decreased conscious level

11.1 INTRODUCTION

The conscious level may be altered by disease, injury or intoxication. The level of awareness decreases as a child passes through stages from drowsiness (mild reduction in alertness and increase in hours of sleep) to unconsciousness (unrousable, unresponsive). Because of variability in the definition of words describing the degree of coma, the Glasgow and the Children's Coma Scales (Table 11.1) have been developed as semiquantitative measures and, more importantly, as an aid to communication between carers. The Glasgow Coma Scale was developed and validated for use in the head-injured patient but has come to be used as an unvalidated tool for the description of conscious states from all pathologies.

In children, coma is caused by a diffuse metabolic insult (including cerebral hypoxia and ischaemia) in 95% of cases, and by structural lesions in the remaining 5%. Metabolic disturbances can produce diffuse, incomplete and asymmetrical neurological signs falsely suggestive of a localised lesion. Early signs of metabolic encephalopathy may be subtle, with reduced attention and blunted affect. The conscious level in metabolic encephalopathies is often quite variable from minute to minute. The most common causes of coma are summarised in the box below.

Disorders causing coma in children

- Hypoxic ischaemic brain injury following respiratory or circulatory failure
- Epileptic seizures
- Trauma:
  - intracranial haemorrhage
  - brain swelling
- Infections:
  - meningitis
  - encephalitis
  - cerebral and extracerebral abscesses
  - malaria

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- Intoxication
- Metabolic:
  - renal or hepatic failure
  - hypo- or hypernatraemia
  - hypoglycaemia
  - hypothermia
  - hypercapnia
  - inherited metabolic disease
- Cerebrovascular event, secondary to arteriovascular malformation or tumour
- Cerebral tumour
- Hydrocephalus, including blocked intraventricular shunts

Table 11.1 Glasgow Coma Scale and Children's Glasgow Coma Scale

Glasgow Coma Scale (4–15 years)		**Children's Glasgow Coma Scale (<4 years)**	
Response	**Score**	**Response**	**Score**
Eye opening		*Eye opening*	
Spontaneously	4	Spontaneously	4
To verbal stimuli	3	To verbal stimuli	3
To pain	2	To pain	2
No response to pain	1	No response to pain	1
Best motor response		*Best motor response*	
Obeys verbal command	6	Spontaneous or obeys verbal command	6
Localises to pain	5	Localises to pain or withdraws to touch	5
Withdraws from pain	4	Withdraws from pain	4
Abnormal flexion to pain (decorticate)	3	Abnormal flexion to pain (decorticate)	3
Abnormal extension to pain (decerebrate)	2	Abnormal extension to pain (decerebrate)	2
No response to pain	1	No response to pain	1
Best verbal response		*Best verbal response*	
Orientated and converses	5	Alert; babbles, coos words to usual ability	5
Disorientated and converses	4	Less than usual words, spontaneous irritable cry	4
Inappropriate words	3	Cries only to pain	3
Incomprehensible sounds	2	Moans to pain	2
No response to pain	1	No response to pain	1

Children with a decreased conscious level are usually presented by parents who are very aware of the seriousness of the symptom. They may also have noted other features such as fever, headache or exposure to poisoning, which may aid the clinician in making a presumptive diagnosis.

11.2 PATHOPHYSIOLOGY OF RAISED INTRACRANIAL PRESSURE

In very young children, before the cranial sutures are closed, considerable intracranial volume expansion may occur if the process is slow (i.e. hydrocephalus). However, if the process is rapid and in children with a fixed volume cranium, increase in volume due to brain swelling, haematoma or cerebrospinal fluid (CSF) blockage will cause raised intracranial pressure (ICP). Initially CSF and venous blood within the cranium decrease in volume. Soon, this compensating mechanism fails and as the ACP continues to rise the cerebral perfusion pressure (CPP) falls and cerebral arterial blood flow is reduced.

$$CPP = MAP - ICP$$

where MAP is mean arterial pressure. Reduced CPP reduces cerebral blood flow (CBF). Normal CBF is over 50 ml/100 g brain tissue/min. If the CBF falls below 20 ml/100 g brain tissue/min, the brain suffers ischaemia. The aim is to keep CPP above 50–60 mmHg depending on age.

Increasing intracranial pressure will push brain tissue against more rigid intracranial structures. Two clinical syndromes are recognisable by the site of localised brain compression (Figure 11.1).

Central syndrome

The whole brain is pressed down towards the foramen magnum and the cerebellar tonsils herniate through it ('coning'). Neck stiffness may be noted. A slow pulse, raised blood pressure and irregular respiration leading to apnoea are seen terminally, usually preceded by significant tachycardia.

Uncal syndrome

The intracranial volume increase is mainly in the supratentorial part of the intracranial space. The uncus, which is part of the hippocampal gyrus, is forced through the tentorial opening and compressed against the fixed free edge of the tentorium. If the pressure is unilateral (e.g. from a subdural or extradural haematoma), this leads to third nerve compression and an ipsilateral

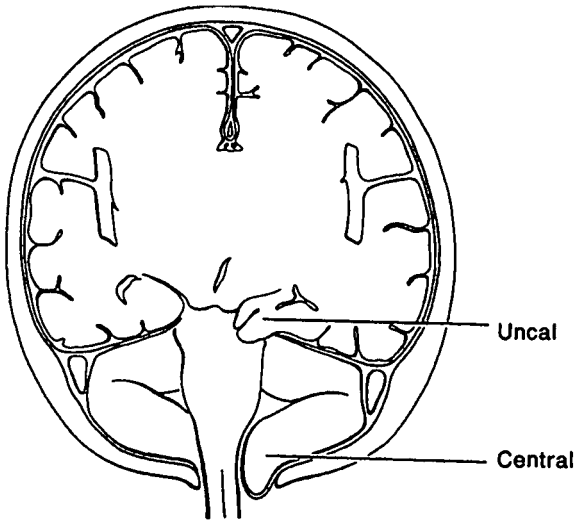

Figure 11.1 Herniations of the brain

dilated pupil. Next, an external oculomotor palsy appears, so the eye cannot move laterally. Hemiplegia may then develop on either or both sides of the body, depending on the progression of the herniation.

11.3 PRIMARY ASSESSMENT OF THE CHILD WITH A DECREASED CONSCIOUS LEVEL

The first steps in the management of the patient with a decreased conscious level are to assess and if necessary support airway, breathing and circulation. This will ensure that the diminished conscious level is not secondary to hypoxia and/or ischaemia and that whatever the cerebral pathology it will not be worsened by lack of oxygenated blood supply to the brain. This is dealt with in Chapter 7. Below is a summary.

Airway

- Assess vocalisations: crying or talking indicate ventilation and some degree of airway patency.
- Assess airway patency by:
  - *looking* for chest and/or abdominal movement, symmetry and recession,
  - *listening* for breath sounds and stridor, and
  - *feeling* for expired air.
- *Reassess after any airway-opening manoeuvres*. If there is still no evidence of air movement, then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see Chapter 4).

Breathing

- Effort of breathing:

respiratory rate	recession	
stridor	wheeze	grunting
accessory muscle use	flaring of nostrils	gasping

Exceptions

Increased effort of breathing *does not* occur in three circumstances:

1. Exhaustion.
2. Central respiratory depression.
3. Neuromuscular disease.

- Efficacy of breathing:
  - chest expansion/abdominal excursion,
  - breath sounds – reduced or absent, and symmetry on auscultation, and
  - Spo_2 in air.
- Effects of respiratory failure on other physiology:
  - heart rate,
  - skin colour, and
  - mental status.

Circulation

- Heart rate: the presence of an inappropriate bradycardia will suggest raised intracranial pressure.
- Pulse volume.
- Blood pressure: significant hypertension indicates a possible cause for the coma or may be a result of it.
- Capillary refill time.

Table 11.2 Summary of pupillary changes

Pupil size and reactivity	Cause
Small reactive pupils	Metabolic disorders
	Medullary lesion
Pinpoint pupils	Metabolic disorders
	Narcotic/organophosphate ingestions
Fixed midsize pupils	Midbrain lesion
Fixed dilated pupils	Hypothermia
	Severe hypoxia
	Barbiturates (late sign)
	During and post seizure
	Anticholinergic drugs
Unilateral dilated pupil	Rapidly expanding ipsilateral lesion
	Tentorial herniation
	Third nerve lesion
	Epileptic seizures

- Skin temperature and colour.
- Effects on breathing and mental status: acidotic sighing respirations may suggest metabolic acidosis from diabetes, an inborn error of metabolism, or salicylate or ethylene glycol poisoning as a cause for the coma.
- Heart rate/rhythm and core–toe temperature difference.

Disability

- Mental status/conscious level (AVPU).
- Pupillary size and reaction (Table 11.2).
- Posture: decorticate or decerebrate posturing in a previously normal child should suggest raised intracranial pressure.
- Look for neck stiffness in a child and a full fontanelle in an infant, which suggest *meningitis*.
- The presence of convulsive movements should be sought: these may be subtle.
- There should be a specific assessment for raised intracranial pressure. There are very few absolute signs of raised ICP, these being papilloedema, a bulging fontanelle and absence of venous pulsation in retinal vessels. All three signs are often absent in acutely raised ICP.
- In a previously well, unconscious child (Glasgow Coma Scale score <9) who is not in a postictal state, the signs in the box below are suggestive of raised ICP.

Signs of raised intracranial pressure

1 Abnormal oculocephalic reflexes (avoid in patients with neck injuries):
- when the head is turned to the left or right a normal response is for the eyes to move away from the head movement; an abnormal response is no (or random) movement
- when the head is flexed, a normal response is deviation of the eyes upward; a loss of conjugate upward gaze is a sign suggestive of raised ICP

2 Abnormal posture (see Figure 7.2 on p. 61):
- decorticate (flexed arms, extended legs)
- decerebrate (extended arms, extended legs)

Posturing may need to be elicited by a painful stimulus

3 Abnormal pupillary responses: unilateral or bilateral dilatation suggests raised ICP
4 Abnormal breathing patterns: there are several recognisable breathing pattern abnormalities in raised ICP. However, they are often changeable and may vary from hyperventilation to Cheyne–Stokes breathing to apnoea
5 Cushing's triad: slow pulse, raised blood pressure and breathing pattern abnormalities are a late sign of raised ICP

Exposure

- *Rash*: if one is present, ascertain if it is purpuric as an indicator of meningococcal disease or non-accidental injury.
- *Fever*: a fever is suggestive evidence of an infectious cause (but its absence does not exclude it) or poisoning with ecstasy, cocaine or salicylates. Hypothermia suggests poisoning with barbiturates or ethanol.
- Look for evidence of poisoning.

11.4 RESUSCITATION (Figure 11.2)

Airway

- A patent airway is the first requisite. If the airway is not patent it should be opened and maintained with an airway manoeuvre and the child ventilated by bag–valve–mask oxygenation. An airway adjunct can be used. The airway should then be secured with intubation by experienced senior help.
- If the child has an AVPU score of 'P' or 'U', or the gag or cough reflex is absent, the airway is at risk. It should be maintained by an airway manoeuvre or adjunct and senior help requested to secure it.

Breathing

- All children with a decreased conscious level should receive high-flow oxygen through a face mask with a reservoir as soon as the airway has been demonstrated to be adequate.
- If the child is hypoventilating, respiration should be supported with oxygen via a bag–valve–mask device and consideration given to intubation and ventilation. Inadequate breathing in coma can lead to a rise in arterial P_{CO_2}, which can cause a dangerous rise in intracranial pressure.

Circulation

Circulation needs to be optimised; if ICP is high, then cerebral perfusion will be compromised. However, overenthusiastic fluid administration should be avoided.

- Establish IV access quickly or use the intraosseous route.
- Check blood glucose – **DEFG: 'Don't ever forget glucose'**.
  - Take blood for a glucose stick test and laboratory test. If in doubt or the test is unavailable, it is safer to treat as if hypoglycaemia is present (<3 mmol/l): give a bolus IV 2 ml/kg 10% glucose, followed by an infusion containing glucose, e.g. 5 ml/kg/h of 10% glucose with 0.45% saline. Without the follow-on infusion, there is a risk of rebound hypoglycaemia, which may also occur with larger bolus doses of glucose.
  - If hypoglycaemia is a new condition for the patient take 5 ml of lithium heparin blood before giving the glucose and send it to the laboratory for the plasma to be frozen. This will allow later investigation of the cause of the hypoglycaemic state.
- Take blood samples for blood culture, full blood count, renal and liver function tests, plasma ammonia (send rapidly to the laboratory on ice), group and save/cross-match, and blood gas analysis.

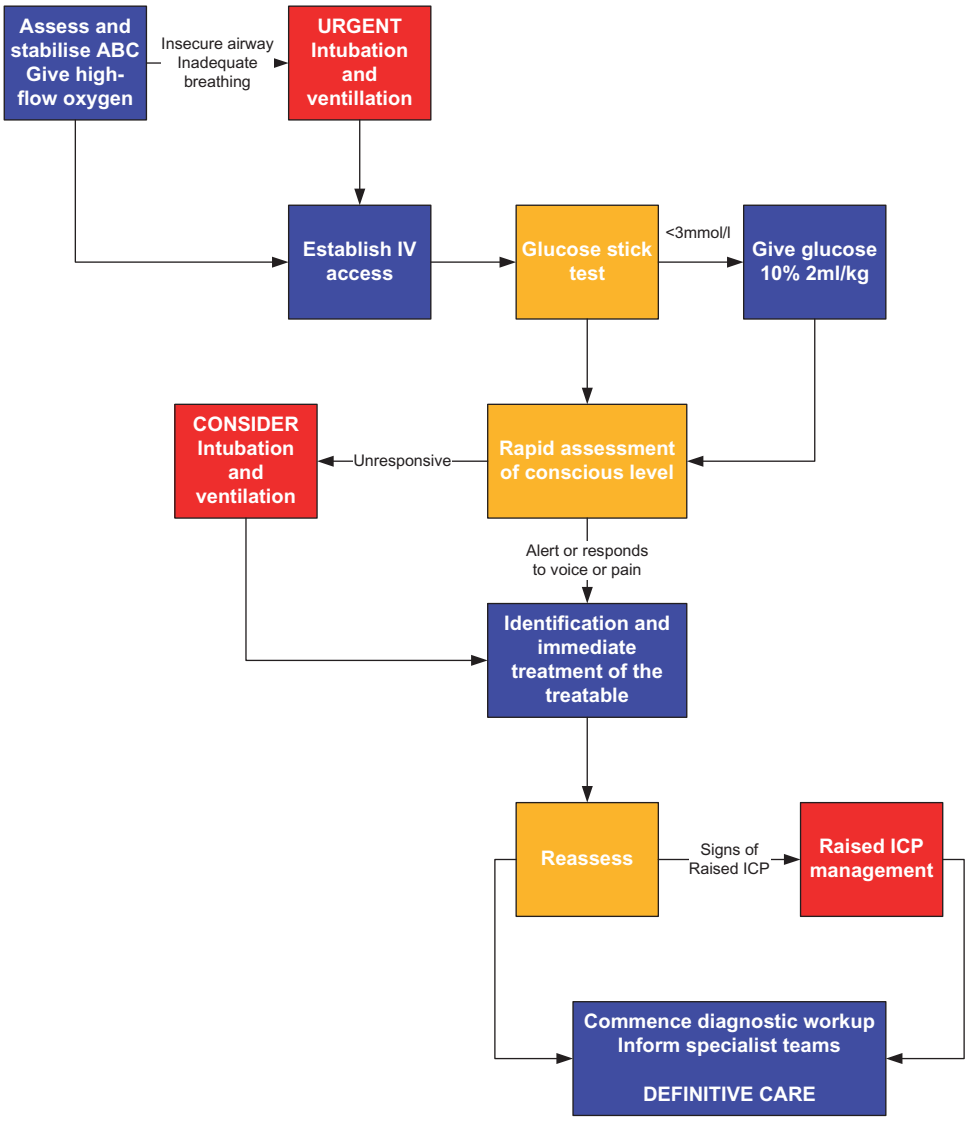

Figure 11.2 Algorithm of the initial management of coma. ICP, intracranial pressure

- Give a broad-spectrum antibiotic, e.g. cefotaxime 80 mg/kg or ceftriaxone, to any child in whom sepsis is suspected, e.g. meningococcal disease or other meningitis.
- Give a 20 ml/kg rapid bolus of crystalloid to any patient with signs of shock (and see treatment of coexistent shock and meningitis in meningococcal disease, Section 9.9).

Disability

Undertake appropriate medical management of raised intracranial pressure, if noted:

- Intubate and support ventilation (maintain a PCO_2 of 4.5–5.5 kPa or 30–34 mmHg).
- Nurse with the head in-line in a 20° head-up position (to help cerebral venous drainage).
- Give mannitol (250–500 mg/kg; i.e. 1.25–2.5 ml of 20% IV over 15 minutes, and give 2-hourly as required, provided serum osmolality is not greater than 325 mOsm/l) or hypertonic saline 3 ml/kg of (2.7) 3% solution.
- Consider dexamethasone (for oedema surrounding a space-occupying lesion) 0.5 mg/kg 6-hourly.

If the situation remains unstable or is deteriorating, further urgent primary assessment and resuscitation must be initiated. If the patient is stable, a further and more detailed neurological examination will reassess the earlier findings, help localise the site of neurological dysfunction, and provide a reference for further examinations.

Lumbar puncture

Lumbar puncture should not be performed in a child in coma.

The purpose of a lumbar puncture is to confirm the diagnosis of meningitis and to identify the organism and its antibiotic sensitivity. There is a risk of coning and death if a lumbar puncture is performed in a child with significantly raised intracranial pressure. Normal fundi do not exclude acutely, severely raised intracranial pressure. The lumbar puncture can be performed some days later when the child's condition allows, to confirm or refute the diagnosis of meningitis or encephalitis if antibiotic treatment or aciclovir, respectively, has been started. In addition, the results of blood cultures and polymerase chain reaction (PCR) are therefore important.

The relative contraindications to a lumbar puncture are shown in the box.

Relative contraindications to lumbar puncture

- Prolonged or focal seizures
- Focal neurological signs, e.g. asymmetry of limb movement and reflexes, ocular palsies
- A widespread purpuric rash in an ill child. In this case intravenous cefotaxime should be given immediately after a blood culture
- Glasgow Coma Scale score of less than 13
- Pupillary dilatation
- Impaired oculocephalic reflexes (doll's eye reflexes)
- Abnormal posture or movement, decerebrate or decorticate posturing, or cycling movements of the limbs
- Inappropriately low pulse, elevated blood pressure and irregular respirations (i.e. signs of impending brain herniation)
- Thrombocytopenia or coagulation disorder
- Papilloedema
- Hypertension

11.5 SECONDARY ASSESSMENT AND LOOKING FOR KEY FEATURES

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours and any significant previous illness should be gained. In a patient in coma, it is often impossible to be certain of the diagnosis in the first hour. The main immediate aims are therefore to maintain homeostasis and 'treat the treatable'.

Specific points for *history taking* include:

- Recent trauma.
- Pre-existing neurological disability.
- History of epilepsy.
- Poison ingestion.
- Known chronic condition (e.g. renal disease, cardiac abnormality, diabetes).
- Last meal.
- Known metabolic disorder or family history of one.
- Previous episodes of encephalopathy with illness.
- Recent trips abroad.

Specific *additional neurological examination* includes:

- Eye examination:
  - pupil size and reactivity (see Table 11.2 on page 120),
  - fundal changes: haemorrhage and papilloedema (trauma, hypertension), and
  - ophthalmoplegia: lateral or vertical deviation.

- Reassess posture and tone: look for lateralisation.
- Assess deep tendon reflexes and plantar responses: look for lateralisation.

Lateralisation suggests a localised rather than a generalised lesion, but this is often a false indicator in childhood. The child will almost certainly need a computed tomography (CT) scan.

A *general physical examination* may add clues to point to a working diagnosis. Specific findings include the following:

- Skin: rash, haemorrhage, trauma, evidence of neurocutaneous syndromes.
- Scalp: evidence of trauma.
- Ears and nose:
  - bloody or clear discharge: base of skull fracture (Chapter 16), and
  - evidence of otitis media or mastoiditis: may accompany meningitis.
- Neck tenderness or rigidity: meningitis, cerebrovascular accident.
- Odour: alcohol intoxication, ketones in diabetic ketoacidosis, metabolic disorders.
- Abdomen: enlarged liver (in conjunction with hypoglycaemia, inherited metabolic disease).

The *key features*, which will be identified clinically, from the history, examination and the initial blood test results, can point the clinician to the likeliest working diagnosis for emergency treatment.

• Coma that develops over several hours, associated with irritability and/or fever and a rash points to *meningitis/encephalitis* (this should also be a lead working diagnosis in the absence of a clear alternative)	Section 11.7
• A history of opiate ingestion and/or pinpoint pupils points to *poisoning with opiates*	Section 11.8
• Coma occurring in the setting of, or just after, a minor illness presenting with vomiting, hepatomegaly and hypoglycaemia points to *metabolic encephalopathy*	Section 11.9
• A history of travel abroad might point to malaria	Section 11.10
• Coma associated with significant hypertension points to *hypertensive encephalopathy*	Section 12.7
• A history of onset of coma over an hour or so in an otherwise well child is suggestive of *poisoning*	Appendix H
• A vague and inconsistent history and/or suspicious bruising in an infant are suggestive of *non-accidental head injury*; the presence of retinal haemorrhage is supportive evidence of this	Chapter 16
• Hyperglycaemia points to *diabetes*	Appendix B
• A history of very sudden onset of coma, sometimes with a preceding headache points to a *cerebrovascular accident* (rare in childhood)	

In addition, unless meningitis can be excluded by the clear identification of another cause for coma, it should be assumed present as the consequence of missed diagnosis is catastrophic and the risk of unnecessary treatment with antibiotics small. This also applies to meningoencephalitis from *Mycoplasma* and herpes, and the use of erythromycin and acyclovir, respectively. Early initiation of treatment is important because these have a worse prognosis when treatment is *seriously* delayed. Senior advice should be sought.

11.6 FURTHER GENERAL TREATMENT OF COMA

- Give normal fluid maintenance to avoid hypoglycaemia and maintain electrolyte balance, unless there is evidence of raised ICP or increased antidiuretic hormone secretion.
- Maintain normoglycaemic state:
  - use 5% glucose solutions (10% in young infants), increasing as needed, and
  - be cautious of administering insulin to hyperglycaemic patients, as this may be stress induced.

- Assess and maintain electrolyte balance:
  - if possible keep serum sodium in the normal range, 135–145 mmol/l, and
  - avoid hyponatraemia by using normal saline or 0.45% saline.
- Treat seizures if present and give prophylactic anticonvulsants if the child has repeated seizures.
- Insert a gastric tube to aspirate stomach contents. Perform gastric lavage in appropriate circumstances (see Appendix H).
- Regulate temperature, ensuring hyperthermia above 37.5°C is avoided.
- Undertake appropriate medical management of raised ICP, if noted:
  - support ventilation (maintain a P_{CO_2} of 4.5–5.5 kPa),
  - maintain a 20° head-up position with the head in-line,
  - give mannitol (250–500 mg/kg, i.e. 1.25–2.5 ml of 20% IV over 15 minutes, and give 2-hourly as required, provided serum osmolality stays <325 mOsm/l) or hypertonic saline 3 ml/kg of (2.7) 3% solution,
  - give dexamethasone (for oedema surrounding a space-occupying lesion) 0.5 mg/kg twice daily, and
  - catheterise the bladder (bladder distension may aggravate raised ICP) and monitor urine output.
- Maintain skin care to prevent bedsores, and eye padding to avoid xerophthalmia.

11.7 APPROACH TO THE CHILD WITH MENINGITIS/ENCEPHALITIS

After the neonatal period, the commonest cause of bacterial meningitis is *Neisseria meningitidis* (meningococcus). There is still a mortality rate of around 5% and a similar rate of permanent serious sequelae. Infection with *Streptococcus pneumoniae* is less common and may follow an upper respiratory infection with or without otitis media. Long-term morbidity and mortality occur in up to 30% of cases. Widespread Hib vaccination has reduced the incidence of *Haemophilus influenzae* infection. A wide range of infections may also cause encephalitis.

Diagnosis of bacterial meningitis

In the 3-year-old child and under

Bacterial meningitis is difficult to diagnose in its early stages in this age group. The classic signs of neck rigidity, photophobia, headache and vomiting are often absent. A bulging fontanelle is a sign of advanced meningitis in an infant, but even this serious and late sign will be masked if the baby is dehydrated from fever and vomiting. Almost all children with meningitis have some degree of raised ICP, so that, in fact, the signs and symptoms of meningitis are primarily those of raised ICP. The following are signs of possible meningitis in infants and young children:

- Coma.
- Drowsiness (often shown by lack of eye contact with parents or doctor).
- High-pitched cry or irritability that cannot be easily soothed by parent.
- Poor feeding.
- Unexplained pyrexia.
- Convulsions with or without fever.
- Apnoeic or cyanotic attacks.
- Purpuric rash.

In older children of 4 years and over

These children are more likely to have the classic signs of headache, vomiting, pyrexia, neck stiffness and photophobia. Some present with coma or convulsions. In all unwell children, and children with an unexplained pyrexia, a careful search should be made for neck stiffness and for a purpuric rash. The finding of such a rash in an ill child is almost pathognomic of meningococcal infection, for which immediate treatment is required (see Section 9.9).

Emergency treatment of meningitis

Reassess ABCD

- Specific assessment should be made of the severity of raised intracranial pressure, as many of the clinical signs of meningitis arise from this.
- After the above assessment, give intravenous cefotaxime or another suitable antibiotic if meningitis is suspected and this has not yet been given. Treat a child with possible raised ICP and meningitis without performing a lumbar puncture. Ensure blood cultures and PCR have been taken, as these may help in diagnosis.
- Treat with aciclovir and a macrolide in a febrile, comatose child for the rare respective possibilities of herpes simplex virus and *Mycoplasma* encephalitis.
- Give dexamethasone (150 micrograms/kg, max. 10 mg, four times a day) in suspected or confirmed bacterial meningitis, aiming to start within 4 hours of antibiotics (not later than 12 hours); *do not use in infants younger than 3 months*, but in older infants and children, corticosteroids can reduce the rate of severe hearing loss and possibly other long-term neurological sequelae.

11.8 APPROACH TO THE CHILD POISONED WITH OPIATES

These children are usually toddlers who have drunk the green liquid form of methadone. The sedative effect of the drug may reduce the conscious level sufficiently to put the airway at risk and cause hypoventilation.

Emergency treatment of opiate poisoning

Reassess ABC

Following stabilisation of airway, breathing and circulation, the specific antidote is naloxone. An initial bolus dose of 10 micrograms/kg is used but some children need doses as high as 100 micrograms/kg up to a maximum of 2 mg. Naloxone has a short half-life, relapse often occurring after 20 minutes. Further boluses, or an infusion of 10–20 micrograms/kg/min, may be required.

It is important to normalise CO_2 before the naloxone is given because adverse events such as ventricular arrhythmias, acute pulmonary oedema, asystole or seizures may otherwise occur. This is because the opioid system and the adrenergic system are interrelated. Opioid antagonists and hypercapnia stimulate sympathetic nervous system activity. Therefore, if ventilation is not provided to normalise carbon dioxide prior to naloxone administration, the sudden rise in adrenaline concentration can cause arrhythmias.

11.9 APPROACH TO THE CHILD WITH METABOLIC COMA

Metabolic coma can arise from a variety of conditions, including a number of rare inborn errors of metabolism. These illnesses often present with a rapidly progressive encephalopathy, vomiting, drowsiness and convulsions or coma. There may be associated hepatomegaly (from fatty change), hypoglycaemia, abnormal liver enzymes or hyperammonaemia. In a case of otherwise unexplained coma with a Glasgow Coma Score of <12, a key urgent investigation is a plasma ammonia. Interpretation of the concentration can be difficult, as can specific treatment of the hyperammonaemia. In this event seek advice from a specialist in inherited metabolic disease and the paediatric intensive care unit.

11.10 APPROACH TO THE CHILD WITH MALARIA

Plasmodium falciparum causes 95% of deaths and most severe complications. It is transmitted by the bite of an infected *Anopheles* mosquito, and less commonly by infected blood transfusion, needle stick injuries or by the transplacental route.

The clinical features of severe disease include reduced conscious level, convulsions, acidosis and severe anaemia. Cerebral malaria may produce encephalopathy, rapid-onset coma and raised intracranial pressure. Diagnosis requires microscopy of a thick film (quick diagnosis) and thin film (species identification).

Emergency treatment of cerebral malaria

Reassess ABCD

- Give an intravenous quinine loading dose 20 mg/kg over 4 hours in glucose 5%. Give with ECG monitoring.
- Also give an antibiotic, e.g. intravenous cefotaxime.
- If Hb <5 g/dl consider transfusion, especially if there are signs of heart failure.

11.11 APPROACH TO THE CHILD WITH SYSTEMIC HYPERTENSIVE CRISIS

See Section 12.7.

11.12 STABILISATION AND TRANSFER TO DEFINITIVE CARE

After the child has been stabilised and conditions such as hypoglycaemia, meningitis and opiate poisoning have been treated as indicated, some children will remain undiagnosed. These children and those in whom there is any suggestion of lateralisation or intracranial bleeding should have an urgent CT scan. Children who remain very ill and those in whom the cause of coma is as yet unidentified will require transfer to a paediatric intensive care unit and the involvement of other specialists such as from neurology, inherited metabolic diseases or endocrinology as indicated.

Patients will almost certainly need intubation and ventilation for safe transfer by the retrieval team (see Chapter 24). In such patients neurological assessment cannot be continued, and there should therefore be clear documentation of neurological signs, including their progression before transfer is commenced.

11.3 SUMMARY

You should use the structured approach in the assessment and management of the child with a decreased conscious level:

- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

CHAPTER 12

The convulsing child

LEARNING OBJECTIVES

In this chapter, you will learn:

- The causes of convulsions in infants and children
- How to assess the convulsing child
- How to resuscitate the child with convulsions
- How to terminate a tonic–clonic convulsion
- The emergency treatment of the different causes of convulsions

12.1 INTRODUCTION

Generalised convulsive (tonic–clonic) status epilepticus (CSE) is currently defined as a generalised convulsion lasting 30 minutes or longer or when successive convulsions occur so frequently over a 30-minute period that the patient does not recover consciousness between them. Although the outcome of CSE is mainly determined by its cause, the duration of the convulsion is also relevant. In addition, the longer the duration of the episode, the more difficult it is to terminate it. In general, convulsions that persist beyond 5 minutes may not stop spontaneously, so it is usual practice to institute anticonvulsive treatment when the episode has lasted 5 or more minutes.

Common causes of convulsions in children include fever (<6 years), meningitis, epilepsy, hypoxia and metabolic abnormalities. Tonic–clonic status occurs in approximately 1–5% of patients with epilepsy. Up to 5% of children with febrile seizures will present with status epilepticus.

Status epilepticus can be fatal, but mortality is lower in children than in adults – at about 4%. Death may be due to complications of the convulsion, such as obstruction of the airway, hypoxia and aspiration of vomit, to overmedication, cardiac arrhythmias or to the underlying disease process. Complications of prolonged convulsions include cardiac arrhythmias, hypertension, pulmonary oedema, hyperthermia, disseminated intravascular coagulation and myoglobinuria.

Neurological outcomes (persistent epilepsy, motor deficits, learning and behavioural difficulties) are age dependent, occurring in 6% of those over 3 years but 29% of those under 1 year.

12.2 PATHOPHYSIOLOGY OF PROLONGED CONVULSIONS

A generalised convulsion increases the cerebral metabolic rate at least three-fold. Initially, there is an increased sympathetic activity with the release of catecholamines, which lead to peripheral vasoconstriction and increased systemic blood pressure. There is also a loss of cerebral arterial regulation and, following the increase in systemic blood pressure, there is a resulting increase in cerebral blood flow to provide the necessary oxygen and energy. If convulsions continue,

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the systemic blood pressure falls and this is followed by a fall in cerebral blood flow. Lactic acid accumulates and there is subsequently cell death, oedema and raised intracerebral pressure resulting in further worsening of cerebral perfusion. Cellular metabolism of calcium and sodium is also impaired, with further cell death.

12.3 PRIMARY ASSESSMENT

The first steps in the management of the convulsing patient are to assess and, if necessary, support airway, breathing and circulation. This will ensure that the convulsion is not secondary to hypoxia and/or ischaemia and that whatever the cerebral pathology, it will not be worsened by lack of oxygenated blood supply to the brain. An important early step is to identify and treat any hypoglycaemia.

This is dealt with in Chapter 7. Below is a summary, with additional notes relevant to this presentation:

Airway

- Assess airway patency by:
  - *looking* for chest and/or abdominal movement, symmetry and recession,
  - *listening* for breath sounds and stridor, and
  - *feeling* for expired air.
- *Reassess after any airway-opening manoeuvres*. If there is still no evidence of air movement then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see Chapter 4).

Breathing

- Effort of breathing:

respiratory rate	recession	
stridor	wheeze	grunting
accessory muscle use	flaring of the nostrils	gasping

Grunting may be caused by the convulsion and not be a sign of respiratory distress in this instance.

Exceptions

Increased effort of breathing *does not* occur in three circumstances:

1. Exhaustion.
2. Central respiratory depression.
3. Neuromuscular disease.

- Efficacy of breathing:
  - chest expansion/abdominal excursion,
  - breath sounds – reduced or absent, and symmetry on auscultation, and
  - Spo_2 in air.
- Effects of respiratory failure on other physiology:
  - heart rate,
  - skin colour, and
  - mental status.

Circulation

- Heart rate: the presence of an inappropriate bradycardia will suggest raised intracranial pressure.
- Pulse volume.

- Blood pressure: significant hypertension indicates a possible cause for the convulsion or more likely is a result of it.
- Capillary refill time.
- Skin temperature and colour.
- Effects on breathing and mental status.
- Monitor heart rate/rhythm, blood pressure and core–toe temperature difference.

Disability

- Mental status/conscious level (AVPU).
- Pupillary size and reaction (see Table 11.2 on page 120).
- Posture: decorticate or decerebrate posturing in a previously normal child should suggest raised intracranial pressure. *These postures can be mistaken for the tonic phase of a convulsion.* Consider also the possibility of a drug-induced dystonic reaction or a psychogenic, pseudo-epileptic attack. All these movement disorders are distinguishable from tonic–clonic status epilepticus as long as they are considered.
- Look for neck stiffness in a child and a full fontanelle in an infant, which suggest *meningitis*.

Exposure

- *Rash*: if one is present, ascertain if it is purpuric as an indicator of meningococcal disease or non-accidental injury.
- *Fever*: a fever is suggestive evidence of an infectious cause (but its absence does not suggest the opposite) or poisoning with ecstasy, cocaine or salicylates. Hypothermia suggests poisoning with barbiturates or ethanol.
- Consider the evidence for poisoning: history or characteristic smell (see Appendix H).

12.4 RESUSCITATION

Airway

- A patent airway is the first requisite. If the airway is not patent it should be opened and maintained with an airway manoeuvre or an airway adjunct.
- Even if the airway is open, the oropharynx may need secretion clearance by gentle suction.
- If the child is breathing satisfactorily, the recovery position should be adopted to minimise the risk of aspiration of vomit.

Breathing

- All convulsing children should receive high-flow oxygen through a face mask with a reservoir as soon as the airway has been demonstrated to be adequate.
- If the child is hypoventilating, respiration should be supported with oxygen via a bag–valve–mask device and consideration given to intubation and ventilation.

Circulation

Gain intravenous or intraosseous access.

- Take blood for a glucose stick test and laboratory test. If in doubt or a test is unavailable, it is safer to treat as if hypoglycaemia is present (<3 mmol/l): give a bolus IV 2 ml/kg 10% glucose, followed by an infusion containing glucose, e.g. 5 ml/kg/h of 10% glucose with 0.45% saline. Without the follow-on infusion there is a risk of rebound hypoglycaemia, which may also occur with larger bolus doses of glucose.

  If hypoglycaemia is a new condition for the patient take 5 ml of lithium heparin blood before giving the glucose and send to the laboratory for the plasma to be frozen. This will allow later investigation of the cause of the hypoglycaemic state.
- Give a 20 ml/kg rapid bolus of crystalloid to any patient with signs of shock. Colloid and an antibiotic such as cefotaxime should be used for those in whom a diagnosis of

septicaemia is made obvious by the presence of a purpuric rash after blood has been taken for culture.
- Give an antibiotic such as cefotaxime or ceftriaxone to any child in whom a diagnosis of meningitis is made obvious by a stiff neck or bulging fontanelle after blood has been taken for culture.

12.5 SECONDARY ASSESSMENT AND LOOKING FOR KEY FEATURES

While the primary assessment and resuscitation are being carried out, a focused history of the child's health and activity over the previous 24 hours and any significant previous illness should be gained.

Specific points for history taking include:
- Current febrile illness.
- Recent trauma.
- History of epilepsy.
- Poison ingestion.
- Last meal.
- Known illnesses.

The immediate emergency treatment requirement, after ABC stabilisation and exclusion or treatment of hypoglycaemia is to stop the convulsion.

12.6 EMERGENCY TREATMENT OF THE CONVULSION

Reassess ABC

This evidence-based consensus guideline is not intended to cover all circumstances. There are patients with recurrent convulsive status whose physicians recognise that they respond to certain drugs and not to others and for these children an individual protocol is more appropriate. In addition, seizures in neonates are managed differently to those of infants and children.

The protocol is for the majority of children in CSE who present acutely on wards or in an accident and emergency department (Figure 12.1). It is not applicable to non-convulsive status epilepticus.

Step 1 is undertaken 5 minutes after the seizure has started. If this has started in the pre-hospital environment it may be more than 5 minutes after the seizure started before the child arrives in the department – you would therefore move immediately to step 1 on arrival.

Step 1

- Many children may have already undergone step 1 before arrival in hospital.
- If in a pre-hospital setting or intravenous access is not established and if the seizure has lasted longer than 5 minutes, give buccal midazolam 0.5 mg/kg or rectal diazepam 0.5 mg/kg.
- If intravenous access is already established or can be established quickly, give intravenous lorazepam 100 micrograms/kg.

Step 2

- If the convulsion continues for 10 minutes after step 1 and in the hospital setting, give a second dose of benzodiazepine, and call for senior help.
- If the child has received buccal midazolam or rectal diazepam before or in hospital, and is still convulsing, obtain intravenous access to give one dose of intravenous lorazepam (0.1 mg/kg). *Do not give more than two doses of benzodiazepine, including any pre-hospital medication.* If intravenous access still has not been achieved, obtain IO access.
- Start to prepare phenytoin for step 3.
- Reconfirm it is an epileptic seizure.

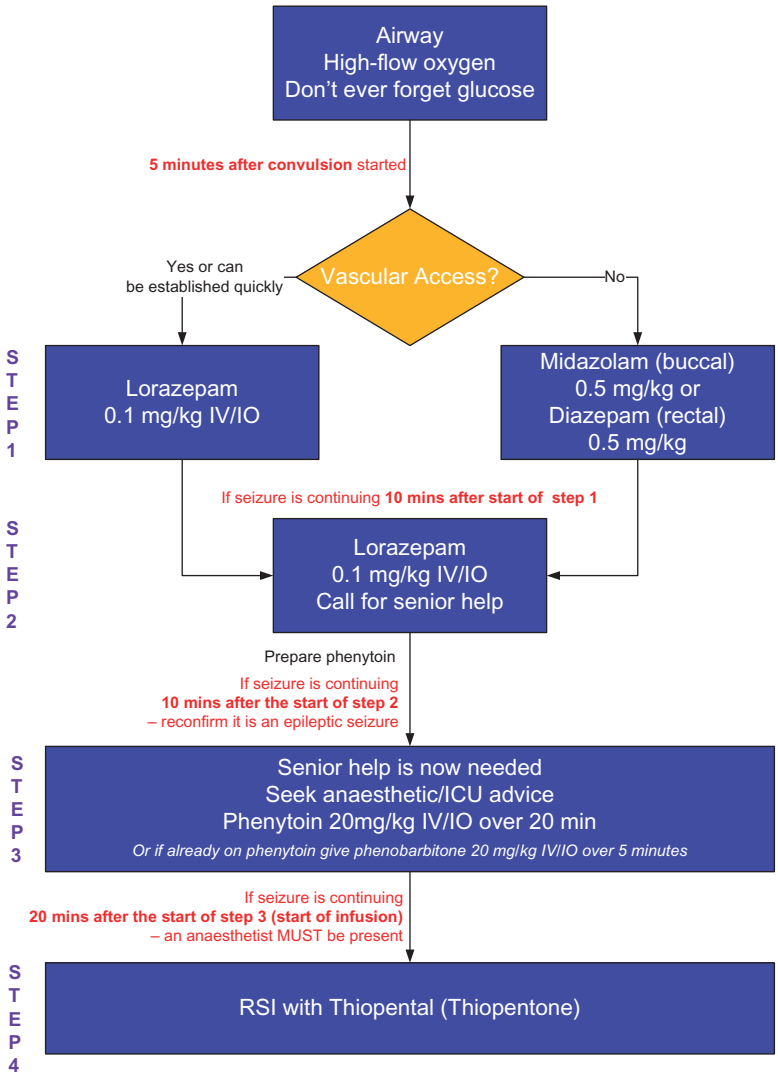

Figure 12.1 Status epilepticus algorithm. ICU, intensive care unit; RSI, rapid sequence induction

In the majority of children, treatment in steps 1 to 2 will be effective.

Step 3

At this stage senior help is needed to reassess the child and advise on management. It is also wise to seek anaesthetic or intensive care advice as the child will need anaesthetising and intubating if this step is unsuccessful.

- Give phenytoin (20 mg/kg by IV infusion over 20 minutes). After the start of the phenytoin infusion a dose of rectal paraldehyde (0.4 ml/kg) mixed with an equal volume of olive oil may be given as directed by senior staff.
- If the convulsion stops before phenytoin is started, the infusion should not be commenced without specialist advice.
- If the convulsion stops after phenytoin has been started, the complete dose should still be given, as this will have an anticonvulsant effect for up to 24 hours.
- In the case of children already receiving phenytoin as maintenance treatment for their epilepsy, phenobarbitone (20 mg/kg IV over 5 minutes) should be used in place of phenytoin. Alternatives that senior staff may recommend include IV sodium valproate or levetiracetam.

Step 4

If 20 minutes after step 3 has started the child remains in CSE, an anaesthetist must be present. Check airway, breathing and circulation. Take blood for glucose, arterial blood gas, urea, electrolytes and calcium. Treat any vital function problem and correct metabolic abnormalities slowly. Treat pyrexia with paracetamol or diclofenac rectally. Consider mannitol (250–500 mg/kg IV over 30–60 minutes) or (2.7) 3% saline (3 ml/kg).

- Rapid sequence induction of anaesthesia is performed with thiopentone and a short-acting paralysing agent and the child should be transferred to the paediatric intensive care unit.
- Further advice on management should be sought from a paediatric neurologist.
- In children under 3 years with a history of chronic, active epilepsy, a trial of pyridoxine should be considered.

Drugs

Lorazepam

Lorazepam is equally or more effective than diazepam and possibly produces less respiratory depression. It has a longer duration of action (12–24 hours) than diazepam (less than 1 hour). It appears to be poorly absorbed from the rectal route. Lorazepam is not available in every country. If this is the case, diazepam can be substituted at a dose of 0.25 mg/kg IV/IO.

Midazolam

This is an effective, quick-acting anticonvulsant, which takes effect within minutes but has a shorter lasting effect than lorazepam. It can be given by the buccal or intravenous routes. Most children do not convulse again once the seizure has been terminated. It has a depressant effect on respiration, but this occurs only in about 5% of patients, is short lived and is usually easily managed with bag–valve–mask ventilatory support.

If using the buccal route, draw up the higher dose (0.5 mg) of the IV preparation using a needle (to avoid any fragments of glass from the ampoule) and after removing the needle, inject the drug into the buccal area between the lower bottom lip and the gum margin at the side of the mouth. Buccal midazolam is twice as effective as rectal diazepam, but both drugs produce the same level and degree of respiratory depression.

Diazepam

This is also an effective, quick-acting anticonvulsant with similar characteristics to midazolam. It is widely used but may now be superseded by the more effective midazolam where the latter is available. The rectal dose is well absorbed.

Paraldehyde

The dose is 0.4 ml/kg per rectum, made up as a 50 : 50 solution in olive oil or physiological saline. Arachis oil should be avoided because children with peanut allergy may react to it. Paraldehyde can cause rectal irritation, but intramuscular paraldehyde causes severe pain and may lead to sterile abscess formation. Paraldehyde causes little respiratory depression. It should not be used in liver disease.

Paraldehyde takes 10–15 minutes to act and its action is sustained for 2–4 hours. *Do not leave paraldehyde standing in a plastic syringe for longer than a few minutes.*

Phenytoin

The dose is 20 mg/kg intravenously with a rate of infusion no greater than 1 mg/kg/min. The infusion should be made up in 0.9% sodium chloride solution to a maximum concentration of 10 mg in 1 ml. Measure the plasma phenytoin levels 60–90 minutes after completion of the infusion.

Phenytoin can cause dysrhythmias and hypotension, therefore monitor the eclectrocardiogram (ECG) and blood pressure (BP). It has little depressant effect on respiration.

Do not use this if the child is known to be on oral phenytoin until the blood level of phenytoin is known. Then only give it if the phenytoin level is less than 5 micrograms/ml. Phenytoin has a peak action within 1 hour but a long half-life that is dose dependent. Its action therefore is more sustained than diazepam.

Fosphenytoin

This is a pro-drug of phenytoin, so is rapidly converted into phenytoin once administered. Because it does not need propylene glycol as a solvent, it can be administered more rapidly than phenytoin over 7–10 minutes and is said to cause fewer cardiac side effects. At present there are no paediatric efficacy data on use in CSE in children. If used it is prescribed in 'phenytoin equivalents', which could cause confusion: 75 mg fosphenytoin is equivalent to 50 mg phenytoin.

Thiopental (thiopentone) sodium

The induction dose is 4–8 mg/kg intravenously. It is an alkaline solution, which will cause irritation if the solution leaks into subcutaneous tissues. It has no analgesic effect and is a general anaesthetic agent. Repeated doses have a cumulative effect. It is a potent drug with marked cardiorespiratory effects and should be used only by experienced staff who can intubate a child.

It is not an effective long-term anticonvulsant and its principal use in status epilepticus is to facilitate ventilation and the subsequent management of cerebral oedema due to the prolonged seizure activity. Other antiepileptic medication must be continued.

The child should not remain paralysed as continued seizure activity cannot universally be adequately monitored by cerebral function analysis monitoring. When the child is stable he or she will need transfer to a paediatric intensive care unit. A paediatric neurologist should continue to give clinical advice and support. There are several regimes for continued drug control of the convulsions but they are outside the scope of this text.

General measures

- Maintain a normoglycaemic state:
  - use 5% glucose solutions (10% in young infants), increasing as needed, and
  - be cautious of administering insulin to hyperglycaemic patients, as this may be stress induced.
- Give normal fluid maintenance to avoid hypoglycaemia and to maintain electrolyte balance, unless there is evidence of raised intracranial pressure or increased antidiuretic hormone secretion, when these should be restricted.
- Assess and maintain electrolyte balance:
  - if possible keep serum sodium in the normal range, 135–145 mmol/l, and
  - avoid hyponatraemia by using normal saline or 0.45% saline.
- Insert a gastric tube to aspirate the stomach contents. Perform gastric lavage if appropriate for specific drug ingestions (see Appendix H).
- Regulate temperature, ensuring temperatures above 37.5°C are avoided.
- Undertake appropriate medical management of raised intracranial pressure, if noted:
  - support ventilation (maintain a PCO_2 of 4.5–5.5 kPa),
  - maintain a 20° head-up position with the head in-line,
  - give mannitol (250–500 mg/kg; that is 1.25–2.5 ml of 20% IV over 15 minutes, and give 2-hourly as required, provided the serum osmolality stays <325 mOsm/l) or hypertonic (2.7) 3% saline 3 ml/kg, and
  - give dexamethasone (for oedema surrounding a space-occupying lesion) 500 micrograms/kg twice daily.
  - Catheterise the bladder (bladder distension may aggravate raised intracranial pressure).

Frequent reassessment of ABC is mandatory as therapy may cause depression of ventilation or hypotension. This particularly applies when treatment with benzodiazepines or barbiturates

has been used to control the fit. Although it is appropriate to continue face mask oxygen treatment, its use may mask hypoventilation if breathing efficacy is being monitored by SpO_2. Either assess carbon dioxide levels or ensure that SpO_2 is assessed regularly while breathing room air. Support ventilation if hypoventilation is present.

The role of cerebral function analysis monitoring is still unclear. Currently, clinical features and standard electroencephalography (EEG) are the preferred methods of assessing seizure activity.

After the fit has been controlled the clinician must consider the underlying cause of the convulsion. In many cases there will be an infectious cause: either a benign, self-limiting infection causing 'febrile status' or possibly meningitis (see Chapter 11). Additional treatments will depend on the clinical situation.

12.7 APPROACH TO THE CHILD WITH SYSTEMIC HYPERTENSIVE CRISIS

Hypertension is uncommon in children. Renal disorders such as dysplastic kidneys, reflux nephropathy or glomerulonephritis account for the majority of children presenting with severe hypertension. Coarctation of the aorta is another important cause. Blood pressure is rarely measured routinely in otherwise healthy children and therefore hypertension usually presents with symptoms that may be diverse in nature. Neurological symptoms are more common in children than in adults. There may be a history of severe headaches, with or without vomiting, suggestive of raised intracranial pressure. Children may also present acutely with convulsions or in coma. Some children will present with a facial palsy or hemiplegia, and small babies may even present with apnoea or cardiac failure.

Blood pressure measurement

This may be difficult in small children and misleading if not done correctly. The following guidelines should be observed:

- Always use the biggest cuff that will fit comfortably on the upper arm. A small cuff will give erroneously high readings.
- The systolic BP may give a more reliable reading than the diastolic because the fourth Korotkoff sound is frequently either not heard or is audible down to zero.
- When using an electronic device, if the result is unexpected recheck it manually before acting on it.
- Raised BP in a child who is fitting, in pain or screaming must be rechecked when the child is calm.
- If the child is very small or uncooperative, using a Doppler device may be helpful. Approximate systolic blood pressures may be obtained by the palpation method.

Blood pressure increases with age – the reading should be checked against normal ranges for the child's age. Any BP over the 95th centile should be repeated and if persistently raised will need treatment. Blood pressures leading to symptomatology will be grossly elevated for the child's age and the diagnosis should not be difficult.

12.8 HYPERTENSION EMERGENCY TREATMENT

Reassess ABC

Initial treatment will be that of the presentation. Airway, breathing and circulation should be assessed and managed in the usual way and neurological status assessed and monitored. Convulsions usually respond to lorazepam, midazolam or diazepam and patients with clinical signs of raised intracranial pressure should be managed with intubation, maintenance of normal PCO_2, a 20° head-up position for nursing and mannitol or hypertonic saline (see Chapter 11).

Once the patient has been resuscitated, management of the hypertension is urgent, but should only be commenced after discussion with a paediatric nephrologist, cardiologist or

intensivist. The aim of treatment is to achieve a safe reduction in BP to alleviate the urgent presenting symptoms whilst avoiding the optic nerve or neurological damage that may occur with too rapid a reduction. Typically the aim is to bring the BP down to the 95th centile for age (or height) over 24–48 hours, with perhaps one-third of the reduction in the first 8 hours. This can be difficult to do without very close BP monitoring and a titratable infusion of the antihypertensive drug.

Monitoring of visual acuity and pupils is crucial during this time as lowering the BP may lead to infarction of the optic nerve heads. Any deterioration must be treated by urgently raising the BP using intravenous saline or colloid. Some children may be anuretic – renal function (serum creatinine, urea and electrolytes) should be analysed promptly.

Some drugs commonly used to achieve BP reduction in children are shown in Table 12.1.

Some specialists may recommend the use of nifedipine as a temporary measure before transfer; if any drug is used, the child should have their blood pressure monitored as above and an intravenous infusion in place.

These children should be cared for in a unit experienced in paediatric hypertension. This will usually be the regional paediatric nephrology (or paediatric cardiology) centre. It is essential that adequate consultation takes place before transfer.

Table 12.1 Drug therapy of severe hypertension

Drug	Dose	Comments
Labetalol	Bolus	Loading dose
	250–500 micrograms/kg	α- and β-blockers
	1–3 mg/k/h	Titratable infusion. *Do not use* in patients with fluid overload or acute heart failure
Sodium nitroprusside	0.2–1 micrograms/kg/min	Vasodilator. Very easy to adjust dose. Titratable infusion. Protect from light. Monitor cyanide levels
Nifedipine	0.25 mg/kg	Fluid can be drawn up from capsules and squirted into mouth sublingually. Better to bite the capsule and swallow. May be difficult to control BP drop because it is given as a bolus

12.9 SUMMARY

You should use the structured approach in the assessment and management of the convulsing child:

- Primary assessment.
- Resuscitation.
- Secondary assessment and looking for key features.
- Emergency treatment.
- Stabilisation and transfer to definitive care.

PART 4

The seriously injured child

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

CHAPTER 13

The structured approach to the seriously injured child

LEARNING OBJECTIVES
In this chapter, you will learn:
- The structured approach to the seriously injured child

13.1 INTRODUCTION

Children and adults are affected differently by major injuries – physically, physiologically and psychologically. A young child cannot describe pain, or even localise, symptoms. The more frightened children become, the 'younger' they behave, and the less they can cooperate with management. Symptoms may be denied vehemently. Their inexperience, lack of awareness of danger and denial of threats posed puts them at particular risk of trauma. The relative elasticity of their tissues allows more energy to be transmitted to other body parts, with less being dissipated at the impact site. Their relatively small size affects the pattern of injuries sustained. For example, the point of impact of a car bumper is higher on the body of a child pedestrian, leading to different anatomical injuries than those sustained in the same incident involving an adult.

Although traumatised children have a number of unique problems, this in no way affects the validity of a structured approach. By following the principles outlined, problems should be identified and treated in the same order of priority as for adults. It should be emphasised from the start that, although assessment and management are discussed separately, this is purely to allow the steps to be shown clearly. In trauma resuscitation, it is essential to intervene immediately, as soon as a problem is found.

The form of the structured approach is shown in Figure 13.1.

13.2 PRIMARY SURVEY

During the primary survey life-threatening conditions are identified. Assessment follows the familiar ABC pattern with significant additions:

<Catastrophic external haemorrhage>
Airway with cervical spine control
Breathing with ventilatory support
Circulation with haemorrhage control
Disability with prevention of secondary insult
Exposure with temperature control

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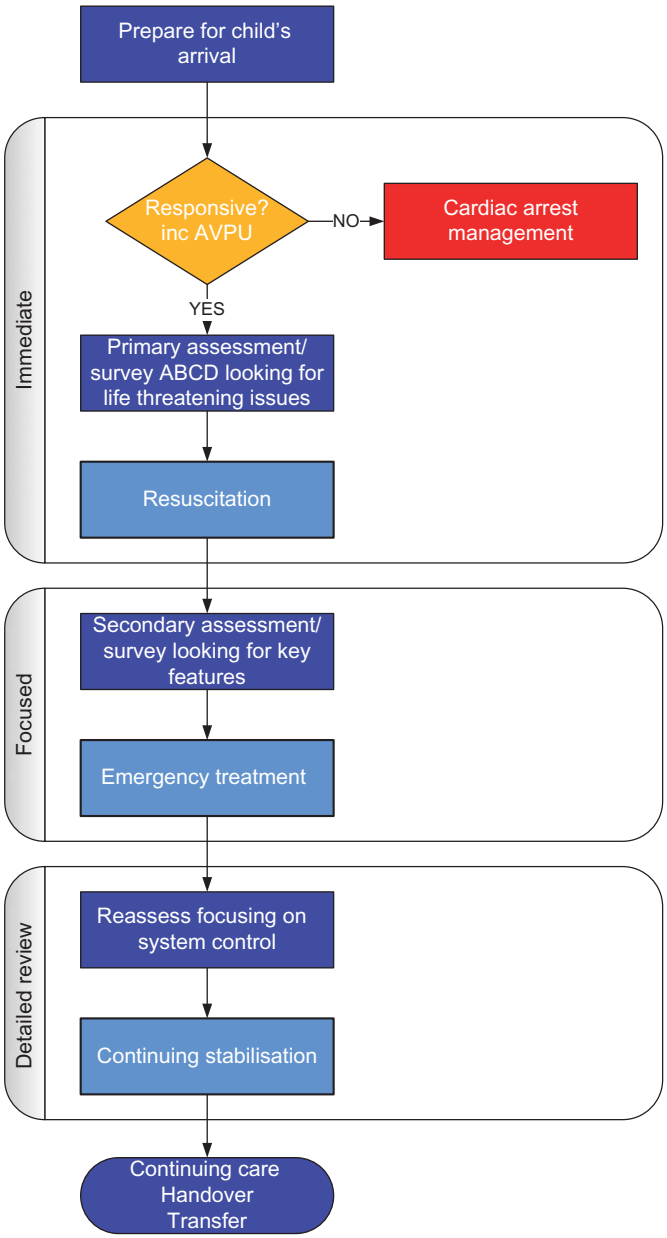

Figure 13.1 The structured approach to emergency paediatrics

Catastrophic external haemorrhage

In military trauma management <C>ABC has become the established approach for casualty care following blast or significant penetrating trauma. For children too, obvious external exsanguinating haemorrhage must be identified and managed immediately.

Airway and cervical spine

Airway assessment following trauma has the highest priority and should follow the standard technique discussed in Chapters 4 and 5.

LOOK
LISTEN
FEEL

If the child is unconscious, uncooperative or has had a significant mechanism of injury that makes it possible to have a spinal injury, the head and neck should be stabilised initially by manual immobilisation. A hard collar should be fitted and applied to all cooperative patients.

Some situations are particularly difficult. An injured child may be uncooperative for many reasons including fear, pain or hypoxia. Manual immobilisation should be maintained and the contributing factors addressed. Too rigid immobilisation of the head in such cases may increase leverage on the neck as the child struggles. The infant or baby who is too small for a hard collar should have manual immobilisation supported throughout.

Breathing

After dealing with any immediate airway problems, breathing should be assessed as the next priority. As discussed in earlier chapters, the adequacy of breathing is checked in three domains – the effort of breathing, the efficacy of breathing and the effects of inadequate respiration on other organ systems. These are summarised in the box. When examining the chest, the *look*, *listen* and *feel* approach is again appropriate, but it is important to remember to *percuss* also to distinguish a tension pneumothorax from a massive haemothorax.

Assessment of the adequacy of breathing

Effort of breathing

- Recession
- Respiratory rate
- Inspiratory or expiratory noises
- Grunting
- Accessory muscle use
- Flaring of the nostrils

Efficacy of breathing

- Breath sounds
- Chest expansion
- Abdominal excursion

Effects of inadequate respiration

- Heart rate
- Skin colour
- Mental status
- *Don't forget to percuss each side of the chest*

The normal resting respiratory rate changes with age. These changes are summarised in Table 13.1 below.

Table 13.1 Vital signs: approximate range of normal

Age (years)	Respiratory rate (breaths/min)	Systolic BP (mmHg) 5th centile	Systolic BP (mmHg) 50th centile	Pulse (beats/min)
<1	30–40	65–75	80–90	110–160
1–2	25–35	70–75	85–95	100–150
2–5	25–30	70–80	85–100	95–140
5–12	20–25	80–90	90–110	80–120
>12	15–20	90–105	100–120	60–100

Circulation

Circulatory assessment in the primary survey involves the rapid assessment of heart rate and rhythm, pulse volume and peripheral perfusion (colour, temperature and capillary return, remembering that exposure to cold prolongs the capillary refill time in healthy people – test on the sternum). In addition, a rapid further check should be made for significant external haemorrhage (and pressure applied if appropriate). Blood pressure takes too long to perform as part of the primary survey itself, but it should be measured as an adjunct (afterwards or in parallel by other personnel). An abnormal respiratory rate and altered mental status in the presence of circulatory compromise indicate the effect of shock on other organ systems. Using these measures, an estimate of the need for fluid replacement can be made (Table 13.2). Again, remember the caveats about heart rate, differential pulse volume and capillary refill time outlined in Chapter 7. It can be difficult to assess the circulatory state of an injured child: in blunt trauma large blood losses are the exception rather than the rule. A single abnormal sign is not predictive of shock, but more than two of the signs tending to the same conclusion is predictive of the requirement for fluid replacement (Table 13.2).

Circulatory assessment must take into account the fact that resting heart rate, blood pressure and respiratory rate vary with age. The normal values are shown in Table 13.2. Note that the systolic blood pressure in children who have been injured is raised above normal and that the degree of hypertension is unrelated to age or trauma severity. The clinician should therefore view with suspicion a systolic pressure in the lower part of the normal range in an injured child.

Table 13.2 Recognition of clinical signs indicating blood loss requiring urgent treatment

Sign	Indicator
Heart rate	Marked or increasing tachycardia or relative bradycardia
Systolic BP	Falling
Capillary refill time (normal <2 s)	Increased to >4–5 s
Respiratory rate	Tachypnoea unrelated to thoracic problem
Mental state	Altered conscious level unrelated to head injury

Disability

The assessment of disability during the primary survey consists of a brief neurological examination to determine the conscious level and to assess pupil size and reactivity. The conscious level is described by the child's response to voice and (where necessary) to pain. The AVPU method describes the child as *alert*, *responding to voice*, *responding to pain* or *unresponsive* and is a rapid, if crude, assessment.

A	**A**lert
V	Responds to **V**oice
P	Responds only to **P**ain
U	**U**nresponsive to all stimuli

If the child does not respond to voice, then a painful stimulus is needed. If the child responds to pain, it is best to note what the eyes and limbs did and what sounds or words were uttered, rather than simply categorising the child as 'P'. Simple descriptions that will form the basis of a subsequent formal Glasgow Coma Score assessment, such as 'opening eyes to pain' or 'localis-

ing to pain', are much more informative than 'P' alone (see examples on p. 61). The score will identify whether or not there is an abnormality of the child's conscious level and alert the clinician to the possible need for airway protection if the level is 'P' or 'U'.

Exposure

In order to assess a seriously injured child fully, it is necessary to remove his or her clothes. Children become cold very quickly, and may be acutely embarrassed when undressed in front of strangers. Although exposure is necessary the duration should be minimised, and a blanket provided at all other times.

Conditions identified

By the end of the primary survey, the following conditions may have been recognised and should be treated as soon as they are found:

- Airway obstruction.
- Tension pneumothorax.
- Open pneumothorax.
- Massive haemothorax.
- Flail chest.
- Cardiac tamponade.
- Shock (haemorrhagic or otherwise).
- Decompensating head injury.

13.3 RESUSCITATION

Life-threatening problems should be treated as they are identified during the primary survey.

Catastrophic external haemorrhage

Catastrophic external haemorrhage must be managed immediately. In this situation, applying direct pressure with a dressing pad, applying a tourniquet to a limb, or packing an open wound with a haemostatic substance must be done instantly.

Airway and cervical spine

Airway

The airway may be compromised by material *in the lumen* (blood, vomit, teeth, a foreign body), by damage to or loss of control of the structures *in the wall* (mouth, tongue, pharynx, larynx, trachea) or by external compression or distortion from *outside the wall* (e.g. compression from a pre-vertebral haematoma in the neck or distortion from a displaced maxillary fracture). The commonest cause is from occlusion by the tongue in an unconscious, head-injured child. Whatever the cause, airway management should follow the sequence described in Chapters 4 and 5 bearing in mind the need to protect the cervical spine. This is summarised in the box.

Airway management sequence

- Jaw thrust
- Suction/removal of foreign body under direct vision
- Oro/nasopharyngeal airways
- Tracheal intubation
- Surgical airway

Head tilt/chin lift is not recommended following trauma, because cervical spine injuries may be made worse.

Cervical spine

For any mechanism of injury capable of causing spinal injury (or in cases with an uncertain history), the cervical spine is *presumed to be at risk*, until it can be *cleared*. Children (and adults) can suffer spinal cord injury despite normal plain radiographs. If ignored, ligamentous instability without radiographic evidence of a fracture can have devastating consequences.

If a cervical spine injury is suspected the child should be immobilised initially by manual in-line stabilisation (Figure 13.2) and then by using an appropriately sized hard collar if tolerated (Figure 13.3). Too rigid immobilisation of the head may increase leverage on the neck as the child struggles (see further details in Sections 17.7–17.12). It is imperative that the child is treated from the outset in a gentle, supportive atmosphere in a way that is appropriate for their age and that parents remain at the bedside, so that anxiety is minimised and unnecessary interventions are avoided.

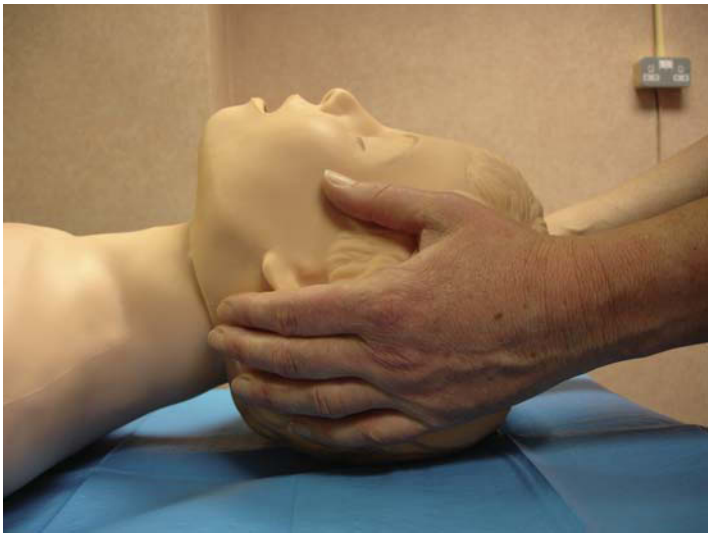

Figure 13.2 Manual in-line stabilisation (MILS)

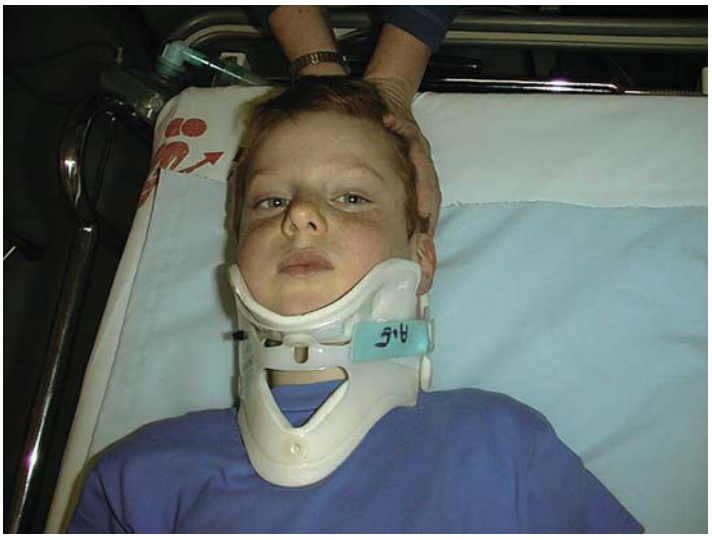

Figure 13.3 Child wearing collar

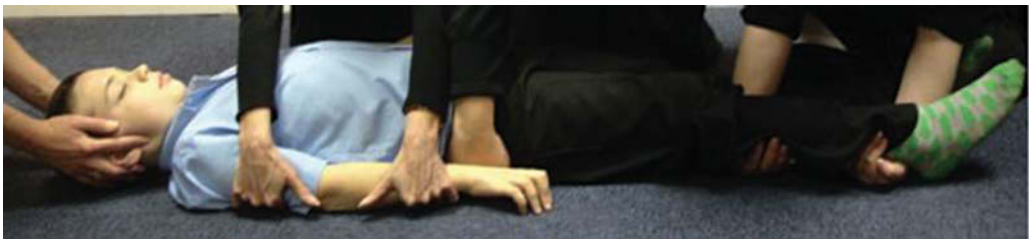

Figure 13.4 Log-roll: four person technique

Vomiting poses an obvious threat to the unprotected airway, especially if there is also a risk of spinal injury. Positioning the child safely and providing airway suction are the key interventions if the child vomits. If the child has only just arrived and the head, chest, pelvis and legs are still strapped securely to a spine board, then it is feasible to tilt the board temporarily while clearing the airway. Once the body straps have been removed, turning the child into the lateral position puts the spine at risk, unless it can be carried out *immediately* by three or four carers as a properly coordinated log-roll (Figure 13.4). It is generally much simpler and more effective to tip the trolley head-down (so that the trachea runs 'uphill') and provide suction to the mouth and pharynx.

If the spine has not been cleared, manual in-line immobilisation will be needed for intubation if indicated. Intubation is significantly harder with the collar left on, as jaw opening is hindered. Having weighed up the risk of failed intubation against any benefit of the collar being left in place, it is generally accepted that *manual in-line immobilisation with the collar off* is by far the best option. Immobilisation with the collar reapplied is necessary after securing the endotracheal tube. The cervical collar may only be removed when radiographs are normal, the child has no neck pain and the neurological examination is normal. Clearly, if the child is paralysed, sedated and ventilated, the neurological examination cannot be done and spinal immobilisation may need to be maintained for prolonged periods. In such cases, a two piece collar should be used for prolonged immobilisation. Computed tomography (CT) or magnetic resonance imaging (MRI) may be necessary to assess the risk to the spinal cord in this event. It is important to ensure that the cervical collar does not restrict venous outflow in the neck as this may increase intracranial pressure. See Figure 13.5 for the management and clearance of the cervical spine. This is discussed in more detail in Chapter 17.

Breathing

If breathing is inadequate, ventilation must be commenced. Initially bag–mask ventilation should be performed. Generally speaking, a child who requires bag–mask ventilation initially following trauma will subsequently require intubation to control the airway. Following intubation, mechanical ventilation can be commenced.

The indications for intubation and mechanical ventilation are summarised in the box below.

Indications for intubation and ventilation

- Persistent airway obstruction
- Predicted airway obstruction, e.g. inhalational burn
- Loss of airway reflexes
- Inadequate ventilatory effort or increasing fatigue
- Disrupted ventilatory mechanism, e.g. severe flail chest
- Persistent hypoxia despite supplemental oxygen
- Controlled ventilation as part of the management of raised ICP

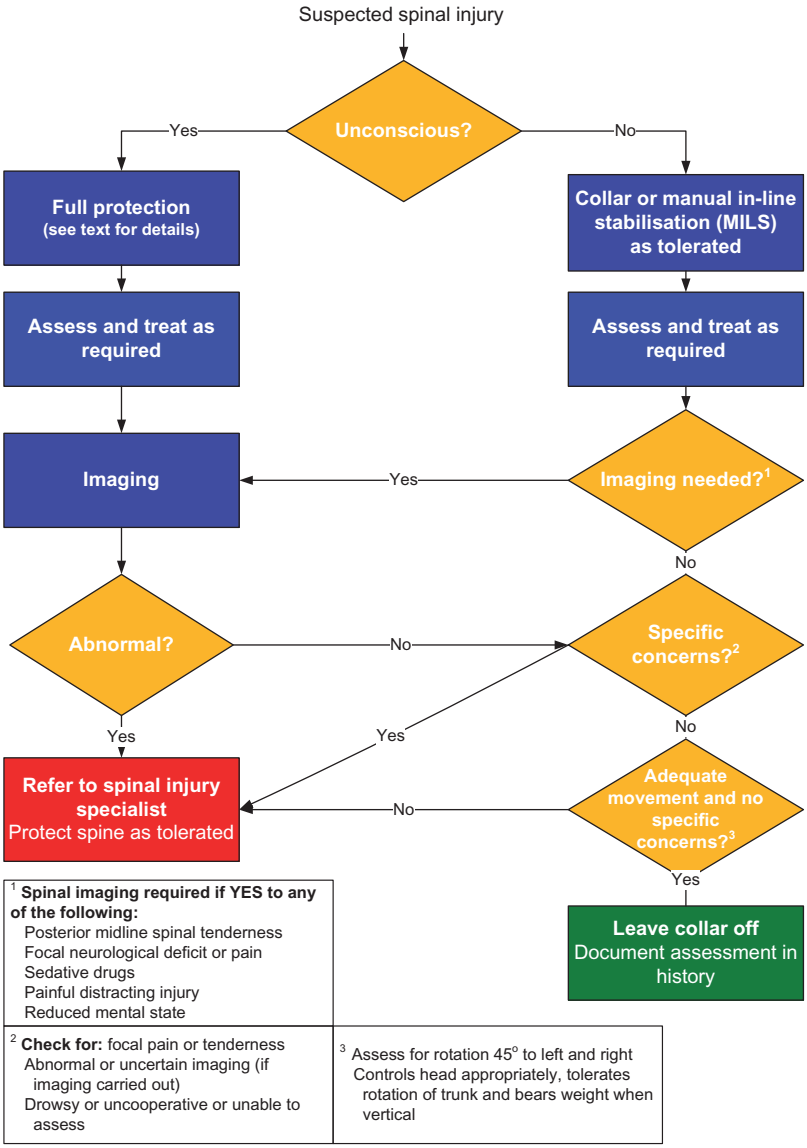

Figure 13.5 Algorithm for the management and clearance of cervical spine

If breath sounds are unequal then pneumothorax, haemopneumothorax, misplaced tracheal tube, blocked main bronchus or pulmonary collapse, diaphragmatic rupture, pulmonary contusion and aspiration of vomit or blood should be considered, and appropriate measures should be taken (see Chapter 14).

Circulation

All seriously injured children require vascular access to be established urgently. Two relatively large intravenous cannulae are mandatory. Peripheral veins are preferred, but in the event of failure, other options should be considered:

- Intraosseous cannulation of the tibia, femur or humerus.
- Direct cannulation of the external jugular vein.
- Indirect cannulation of the femoral vein using the Seldinger technique ('wire through needle' followed by 'catheter over wire').
- Cut-down onto the cephalic vein at the elbow or the long saphenous vein at the ankle.

Intraosseous infusion is warranted from the outset in urgent situations or later if other options have failed. It usually proves to be quicker and easier than the other more specialised techniques mentioned above. Vascular access techniques are discussed in detail in Chapter 21.

Central venous cannulation (other than by the femoral route) is hazardous in children and should not be attempted by the inexperienced clinician. Femoral cannulation is not without risk, given the proximity of the site to the femoral joint. The main use of an internal jugular or subclavian line is for monitoring central venous pressure.

If there are no immediate signs of shock, there is no need to give an *immediate* fluid bolus to an injured child. If there are signs of circulatory compromise, *uncontrolled* bleeding must be considered and confirmed or excluded quickly and pragmatically. Surgeons who can deal with any of the injuries that are suspected should be called in immediately (if they are not already part of the trauma team) and the operating theatre team alerted.

There is evidence that vigorous fluid administration is harmful in the presence of uncontrolled bleeding. The concern is that rapid normalisation of blood pressure may disrupt early clot formation and dilute the clotting factors, leading to greater blood loss. Fluid resuscitation therapy therefore depends on whether or not severe uncontrolled bleeding is suspected. For this reason it would be appropriate for children to be given fluid aliquots of 10 ml/kg (crystalloid) followed by rapid reassessment and subsequent 10 ml/kg as required (Figure 13.6).

If 40 ml/kg of crystalloid has been administered to a child who remains unstable, blood should be used for further fluid replacement.

> The surgical team must be involved in the child's management as soon as it is clear that 20 ml/kg has not stabilised the child.

In uncontrolled haemorrhage evidence has shown that the early transfusion of blood coupled with fresh frozen plasma and platelets has been beneficial. Cross-matching of blood takes time, and clinical urgency may dictate that type-specific or O-negative blood must be given. The times necessary to obtain blood are shown in Table 13.3.

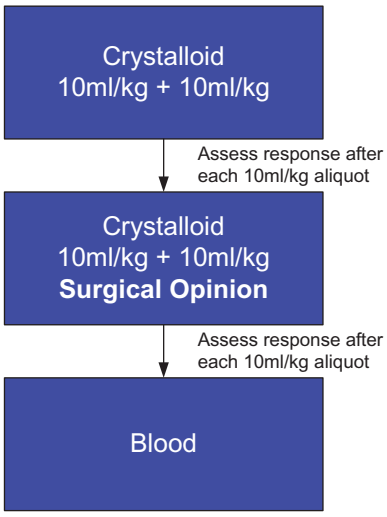

Figure 13.6 Fluids in hypovolaemic shock after trauma

Table 13.3 Cross-match times

Blood type	Cross-match	Time (min)
O-negative	Nil	0
Type-specific	ABO	10–15
Full cross-match	Full	45–60

Disability

If the primary survey reveals that the child has a decompensating head injury, *neurological* resuscitation is required. If the Glasgow Coma Scale score is less than 8 and there are pupil inequalities, immediate intervention is necessary. Lesser degrees of unconsciousness or the presence of focal signs also indicate the need for urgent action. Remember that the scale is modified in the smaller child.

Interventions to be considered include:

- Oxygenation (which will already have been addressed).
- Control of carbon dioxide tension (by controlling ventilation).
- Maintenance of blood pressure to support cerebral perfusion.
- Mannitol or hypertonic saline (if indicated) to lower the intracranial pressure.
- Anaesthesia/sedation/analgesia to reduce cerebral metabolism.
- Prompt treatment of any fits.

> As soon as a serious head injury is suspected, a CT scan should be ordered and the neurosurgical team (which will usually be off site) alerted.

This will help to reduce delay in reaching a decision on the need for neurosurgical intervention. A full Glasgow Coma Scale score is needed at this point.

Other procedures carried out during the resuscitation phase

Chest and pelvic radiographs are not required to identify the conditions (refer to list above) that should be found during the primary survey – these life-threatening conditions are *clinical* diagnoses – but they contribute to resuscitation and subsequent decision making. In unstable or high-risk children, it is appropriate to perform these radiographs during the resuscitation phase in the resuscitation room bay.

When venous access is achieved and blood is taken for cross-matching, samples for other investigations should be taken at the same time, including full blood count, clotting screen, amylase/trypsinogen, urea and electrolytes. Remember to measure the glucose, especially in adolescents (who are prone to both injury and hypoglycaemia after drinking alcohol) and in very small children.

A brief history will usually have been given by the ambulance staff together with details of the child's condition at the scene. This information is useful in allowing the clinician to consider what injuries the mechanism might have produced and to assess whether clinical deterioration is occurring. Monitoring of the respiratory rate, pulse rate, blood pressure and oxygen saturation are important adjuncts to the primary survey and resuscitation. A urinary catheter and a gastric tube may be inserted during this phase in severely injured children.

Urinary catheterisation

In a child, a urinary catheter should only be inserted if the child cannot pass urine spontaneously or if continuous accurate output measurement is required to achieve stabilisation after a serious physiological insult. The route (urethral or suprapubic) will depend on factors related

to signs of urethral, bladder, intra-abdominal or pelvic injury (such as blood at the external meatus, or bruising in the scrotum or perineum; see Chapter 15). If a boy requires urethral catheterisation, urethral damage must be excluded first. The smallest possible silastic catheter should be used in order to reduce the risk of subsequent urethral stricture formation. If any doubt exists then the decision to catheterise the child can be left to the responsible surgeon. Urine should be stick tested and sent for microscopy.

Nasogastric tube placement

Acute gastric dilatation is common in children and the stomach should be decompressed. If there is evidence or suspicion of basal skull fracture, the tube should not be passed by the nasal route. In the intubated patient, the oral route is a simple alternative.

Analgesia

Analgesia can usually be administered just after completing the primary survey and resuscitation. A brief examination of the conscious level and of the body part in pain helps to set the baseline for titrating the dose. Morphine is the standard drug in acute trauma care. A dose of 100–200 micrograms/kg (<1 year: 80 micrograms/kg) should be drawn up in a convenient concentration (e.g. 1 mg/ml in 0.9% saline) according to the size of the child, and administered in increments. Remember that morphine may take more than 10 minutes to provide maximal effect. Fentanyl given in increments of 0.5 micrograms/kg is a useful alternative (in the first instance) as it has a much quicker onset, though it is of shorter duration. Both of these opioids should be at least halved in dose if there is any alteration in the conscious level or any evidence of hypovolaemia. Titrated pain relief not only reduces distress, it limits catecholamine surges that may increase bleeding. It also makes it easier to assess the response to blood and fluid boluses. *There is no place for the administration of intramuscular analgesia in trauma*. Entonox (a 50:50 mix of O_2 and N_2O) may be considered, but is contraindicated if there is a possibility of pneumothorax or basal skull fracture.

13.4 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES

Having finished the primary survey and set in place appropriate resuscitative measures, *focused care* is the next phase of management. The central diagnostic process during this phase is the *secondary survey*, a systematic clinical examination to identify injuries. It is supplemented by observations, imaging and other investigations. Further information is gathered at this time, especially the history of the events leading up to the injury and the presence of any co-morbid factors.

History

The history should be sought from the child, ambulance personnel, relatives and witnesses of the accident. Some history may have already been relayed from the ambulance control prior to admission, though it will need to be confirmed as the initial information is often sketchy and incomplete. On arrival, ambulance staff should be able to provide a great deal of information, including details of the accident site and of the pre-hospital care given. Relatives should be able to give the child's past medical history, allergies and any regular medications. Pre-existing medical conditions such as haemophilia or osteogenesis imperfecta will affect how the child is treated. It is conventional to seek details of the time of the last meal, but it is never wise to assume that the stomach is empty, as gastric stasis is a frequent consequence of major trauma. The child may withhold relevant information, such as glue sniffing or drug abuse, especially in the presence of parents. Alcohol ingestion is usually obvious despite earnest denial.

The mechanism of injury is useful in predicting potential injuries and setting the level of concern. The information in Table 13.4 should be sought.

Table 13.4 Relevant history of injury mechanism

Road accident	Other
Car occupant/cyclist/pedestrian	Nature of accident
Position in vehicle	Objects involved
Restraints worn	Height of fall
Head protection	Landing surface
Thrown from vehicle	Environment
Speed of impact	Temperature
Damage to vehicle	Contamination
Other victim's injuries	

Secondary survey

The secondary survey is a simple but thorough search for key anatomical features of injury. It is helpful to think in terms of the following:

- Surface (head to toe, front and back).
- Orifice (mouth, nose, ears, orbits; rectum, genitals).
- Cavity (chest, abdomen, pelvic cavity, retroperitoneum).
- Extremity (upper limbs including shoulders, lower limbs including pelvic girdle).

In blunt trauma, the child is often brought to hospital on a spine board. It is generally convenient to perform the 'anterior' part of the secondary survey first. The head restraints (if in place) and collar are then taken off, while an assistant holds the neck in-line, to examine the head, neck, face, ears, nose and mouth. The collar can then be reapplied and, with the assistant still holding the neck in alignment, a log-roll is performed to examine the spine, scapula, renal angles, sacroiliac joints and other posterior structures. The spine board is removed at this time, if it has not been earlier.

Occasionally, a full secondary survey is delayed. If immediate life-saving interventions in the operating theatre are required, the secondary survey will have to be completed postoperatively.

Throughout this stage of management, the vital signs and neurological status should be continually reassessed, and any deterioration should lead to an immediate return to the primary survey.

Head

Clinical examination

- Inspect for bruising, haemorrhage, deformity and cerebrospinal fluid (CSF) leak from the nose or ears.
- Palpate for lacerations, bruising and skull depressions.
- Perform otoscopy (for haemotympanum) and ophthalmoscopy (for retinal haemorrhage).
- Perform a mini-neurological examination:
  - pupillary reflexes,
  - conscious level: according to the Glasgow Coma Scale (see Chapter 16),
  - motor function: reflexes, tone, power (noting symmetry), and
  - sensation.

Investigations (as indicated)

- CT scan of the head (see Chapter 16 for indications).
- Skull radiographs (now rarely indicated).

Face

Clinical examination

- Inspect for bruising, lacerations and deformity.
- Inspect the mouth inside and out.
- Palpate the bones for deformity and stability.
- Palpate the teeth to check if any are loose.

Investigations (as indicated)

- Facial radiographs.
- CT scan.

Neck

Clinical examination

> Extreme care should be taken not to move the cervical spine during the initial assessment, when the spine has not been cleared. While the blocks and straps (if in place) and hard collar are removed, an assistant should maintain in-line cervical stabilisation throughout.

- Inspect the front and back of the neck for bruising, lacerations and swelling.
- Palpate the cervical spine for tenderness, bruising, swelling and deformity.
- Palpate for surgical emphysema.

Investigations (as indicated)

Cervical spine imaging should be performed according to a locally agreed protocol; in the UK, this will be according to the National Institute of Health and Clinical Excellence (NICE) guidelines (see further details on p. 248).

- Lateral view (with the arms pulled down).
- Anteroposterior view.
- Odontoid (open mouth) view – omit if a CT scan of the head is separately required; a CT of the upper cervical spine should then be done instead.
- Oblique views (if the lower cervical spine is inadequately visualised on the lateral view – this is rarely needed in children).
- CT scan.
- MRI scan (especially if there are features of neurological deficit).

Flexion and extension views are controversial and should only be obtained in very specific circumstances.

Chest

Clinical examination

- Inspect for bruising, lacerations, deformity and movement.
- Inspect neck veins.
- Feel for tracheal deviation.
- Feel for tenderness, crepitus and paradoxical movement.
- Percuss.
- Listen for breath sounds and added sounds.
- Listen for heart sounds.

Investigations (as indicated)
- Electrocardiogram (ECG).
- Further chest radiographs.
- Special radiographs as indicated (e.g. arch aortogram).
- CT scan.

Abdomen
Clinical examination
- Observe for movement.
- Inspect for bruising, lacerations and swelling.
- Palpate for tenderness, rigidity and masses.
- Auscultate for bowel sounds.

Rectal examination should only be performed if the result is going to alter management of the child. In major blunt trauma, it is necessary (in males) to exclude evidence of urethral disruption. It may be necessary to assess anal tone in spinal injuries or to exclude rectal penetration by bony fragments in major pelvic fractures. Vaginal examinations should not be performed on children.

Investigations (as indicated)
- Ultrasound.
- CT scan (double contrast).
- Intravenous urogram.

Diagnostic peritoneal lavage has been replaced by ultrasound and CT scanning.

Pelvis
Clinical examination
- Inspect for bruising, lacerations and deformity.
- Inspect the perineum.
- Inspect the external urethral meatus for blood.
- Press over the anterior iliac crests for tenderness and check for abnormal mobility – do this once, to minimise the risk of dislodging clots and re-starting bleeding.

Investigations (as indicated)
- Ultrasound.
- Retrograde urethrography.

Spine
Clinical examination

Examination of the spine requires the child to be log-rolled (see Chapter 22).
- Observe for swelling and bruising.
- Palpate for tenderness, bruising, swelling and deformity.
- Assess motor and sensory function.

Investigations (as indicated)
- Radiographs.
- CT scans.
- MRI scans.

Extremities
Clinical examination
- Observe for bruising, swelling and deformity.
- Palpate for tenderness. Crepitus and abnormal movement may be found, but do not elicit deliberately as these are painful. The pattern and degree of tenderness alone will identify the need for X-rays.

- Assess peripheral circulation – pulses and capillary return.
- Assess peripheral sensation to touch. It is rarely useful to test pinprick in a frightened child.

Investigations (as indicated)
- Radiographs.
- Angiograms.

13.5 EMERGENCY TREATMENT

Emergency treatment represents the early response to key findings in the secondary survey and its adjunct investigations. While the interventions are less urgent than those in the resuscitation phase, they will still need to be carried out promptly to minimise the risk of deterioration or unnecessary morbidity. The emergency treatment plan will include treatments for any potentially life-threatening or limb-threatening injuries discovered during the secondary survey. If it does not put the child at undue risk, this plan may be extended to include definitive care of other (more minor) injuries discovered at the same time.

Emergency treatments are discussed in more detail in subsequent chapters.

13.6 REASSESSMENT

The initial emphasis was on crude physiological assessment (<C>ABCD) in the primary survey, followed by focusing on the anatomical evaluation of injuries in the secondary survey. From the time of the initial resuscitation, pulse rate, blood pressure, respiratory rate and oxygen saturation should be measured and charted frequently (every 5 minutes initially). Beyond these continuing observations, there is now a need to return to overall physiological control by considering the following systems in more detail, especially in a critically injured child:
- Respiration.
- Circulation.
- Nervous system.
- Metabolism.
- Host defence.

Respiration (A and B)

The airway should be rechecked. If intubated, is the endotracheal tube of an expected length at the teeth (for the size of the child)? Are the breath sounds symmetrical? Could the tube have migrated into a main-stem bronchus?

Arterial blood gas analysis provides essential information in the child with serious head, chest or multiple injuries (arterial oxygen and CO_2 tensions) or any child who has been intubated. Inserting an arterial line facilitates repeated measurements.

Pulse oximetry readings should be displayed continuously. End-tidal CO_2 monitoring is mandatory in the ventilated child. It shows that the breathing circuit is still connected and that the endotracheal tube has not become dislodged. The *end-tidal* CO_2 should not be regarded as a reliable indicator of *arterial* CO_2 tension, especially in a shocked child. Ventilation–perfusion mismatch causes it to underrepresent the arterial level. It can be regarded as a crude indicator of pulmonary perfusion.

Circulation (C)

This system comprises the three 'haems': *haemodynamics*, *haemoglobin* and *haemostasis*.

In a child with serious injuries, the pulse rate and rhythm should be monitored electrocardiographically. Non-invasive blood pressure readings are generally reliable, though in

serious head injuries and multiple injuries, it is better to monitor on a beat-to-beat basis using direct arterial measurements via an arterial line usually at the radius. This also allows estimation of the haemoglobin (or haematocrit) at hourly intervals to help detect ongoing bleeding and determine the requirement for further transfusion. Base-deficit (or lactate) measurements indicate the adequacy of tissue perfusion, though it is still important to reassess the child clinically. Other invasive techniques, such as central venous pressure monitoring, may be considered at this stage, but should only be undertaken by appropriately trained personnel.

In seriously injured children, the urinary output serves as an indicator of systemic perfusion and should be recorded hourly. It should be maintained at 1–2 ml/kg/h, or higher if there has been a major crush injury or electrical burn with a high risk of myoglobinuria. If it is low, hypovolaemia is the likely cause, though other causes should be considered. If it is high, it may reflect excessive fluid therapy, but remember that diabetes insipidus can occur within a few hours of a serious head injury. After major blood loss, fresh frozen plasma and platelets may be needed to correct coagulopathy following measurement of clotting times and platelet count. Remember that hypothermia affects clotting.

Nervous system (D)

Pupil size and reactivity and the Glasgow Coma Scale score should be checked and recorded every 15 minutes initially. Any deterioration should prompt the need to discuss the case with a neurosurgeon or consider a CT scan (or repeat one). Intracranial pressure (ICP) monitoring is an important means of identifying life-threatening rises in pressure. In conjunction with invasive blood pressure measurements, it provides a means of tracking cerebral perfusion pressure. ICP monitoring can be established in the operating theatre or the intensive care unit. Its use should be confined to hospitals with appropriately skilled personnel, but the importance of cerebral perfusion pressure should be understood by all those who deal with critical head injuries in children.

Metabolism (electrolytes, fluid balance, gut and hormones)

This system refers to biochemical processes and includes renal, hepatic, gastrointestinal and endocrine problems. Glucose control (DEFG: 'Don't ever forget glucose', especially in very young children and in adolescents who have taken alcohol) and urine output are key issues (see 'Circulation' above).

Host defence (injury, infection, immunity, intoxication)

Host defence represents the interaction between the body as a whole and external influences. As such, it encompasses injury (including injury from poor positioning and thermal injury), infection (including wound care), immunity (including the need for tetanus prophylaxis) and intoxication (including alcohol and drugs that may be present in the circulation).

Thermal injury is an important concern: hypothermia hinders blood clotting and predisposes to infection, while fever must be avoided in the severely head-injured child. Wound care, antibiotic prophylaxis for open fractures, and checking that tetanus immunisations are up to date (has the child been immunised at all?) are all considered at this stage, as is careful positioning to avoid problems such as pressure injury from a badly fitting collar.

The 'tertiary survey'

In addition to physiological system control, it is essential for transport escorts, intensive care staff or receiving unit medical staff, who may take over care at this stage, to re-examine the child and review the investigations (especially the imaging) from an anatomical viewpoint to seek out any missed injuries.

Any sudden deterioration in the child's condition should trigger an immediate reassessment of the airway, breathing, circulation and disability so resuscitation can once more be undertaken.

In the face of a serious deterioration, return to the primary survey.

13.7 CONTINUING STABILISATION

Continuing stabilisation and definitive care constitute the final part of the structured approach to trauma care. It goes hand in hand with the detailed physiological system control outlined above and is often carried out by teams other than those that initially received the patient. Patient stability will be assessed effectively using one of a variety of checklists commonly used to review patients receiving intensive care. An example is shown in the box below. Good note taking and appropriate, timely referral are essential if time is not to be lost. If definitive care is to be undertaken in a specialist centre then transfer may be necessary at this stage.

Respiratory	Endotracheal tube, humidification, ventilation parameters, chest X-ray, blood gases
Cardiovascular	Circulatory status, hepatic size, use of inotropes, ECG, chest X-ray
Neurological	Glasgow Coma Scale score, pupils, use of sedation, analgesia and paralysis, imaging neuro-protection for raised intracranial pressure
Gastroenterological	Nutrition, gastro-protection, ileus
Renal and fluids	Urine output, fluid balance, urea and creatinine, need for renal support
Hepatic	Liver function tests
Biochemistry	Electrolytes, blood sugar, calcium, magnesium
Haematology	Hb, clotting studies
Infection	Temperature, white cell count, review cultures/swabs, C-reactive protein, specific polymerase chain reaction, antibiotics
Skin/joints	Skin, mouth and eye care, rashes, passive movements
Drugs	Complete list of enteral and IV drugs, drug levels
Lines and tubes	Access for monitoring, blood sampling and IV drugs, security of catheters and drains
Parents and family	Communications, concerns, support

Note taking

The structured approach discussed in this chapter can provide a framework for the writing of notes. It is recommended that these should be set out as shown in Table 13.5.

Referral

Many teams may be involved in the definitive care of a seriously injured child. It is essential that referrals are made appropriately, clearly and early. Guidance about which children to refer to which teams is given in subsequent chapters.

Table 13.5 Template for note taking

History
- Mechanism of injury
- Past history

Primary survey and resuscitative interventions
- <C>
- A
- B
- C
- D

Secondary survey and emergency treatment of injuries
- Head
- Face
- Neck
- Chest
- Abdomen
- Pelvis
- Spine
- Extremities

Continuing stabilisation
- Respiration
- Circulation
- Nervous system
- Metabolism
- Host defence

Transfer

Injured children may require transfer either within the hospital or to another centre. In either case thorough preparation of equipment, patient and documentation is essential. Secondary transfer should not be undertaken until all life-threatening problems have been addressed, and the child is stable. Occasionally, transfer is the means of delivering life-saving care (e.g. an acute extradural haematoma) and a careful balance must be achieved between delaying such care and setting off with an inadequately stabilised child. Transport of children is discussed in more detail in Chapter 24.

13.8 SUMMARY

The structured approach to initial assessment and management allows the clinician to care for the seriously injured child in a logical, effective way.

Assessment of vital functions (airway, breathing, circulation and disability) is carried out first and resuscitation for any problems found is instituted immediately:
- Primary survey.
- Resuscitation.

A complete head-to-toe examination is then carried out, adjunct investigations are performed and emergency treatment is instituted:

- Secondary survey and the search for key features.
- Emergency treatment.

Finally, a detailed review is undertaken and definitive care is provided:

- Reassessment and physiological system control.
- Continuing stabilisation and definitive care.

CHAPTER 14

The child with chest injury

LEARNING OBJECTIVES

In this chapter, you will learn:

- Which chest injuries pose an immediate threat to life and those that are discovered later
- How to manage these injuries

14.1 INTRODUCTION

Establishing a secure airway is the first priority in the resuscitation of a child. The next most important consideration is the assessment of breathing. Respiratory problems may result from direct chest injury or may reflect the effect of other anatomical injuries on breathing (especially tachypnoea from shock and respiratory irregularity from head injury). General consequences of severe trauma, such as gastric dilatation or pulmonary aspiration after vomiting or regurgitation, may further compromise respiratory function.

Children have relatively elastic tissues. Substantial amounts of kinetic energy may be transferred through a child's chest wall to deep structures with little or no external sign of injury and without rib fractures. A lack of evident rib fractures on the chest radiograph does not exclude major thoracic visceral disruption; conversely, the presence of rib fractures indicates high-energy transfer. Children have relatively little respiratory reserve. Their high metabolic rate and small functional residual capacity allow them to desaturate more rapidly – when their oxygen supply is curtailed. Their horizontal ribs and underdeveloped musculature make them tolerate chest wall disruption badly. Flail chest, for example, is poorly tolerated.

The risk of iatrogenic chest problems must be appreciated. The child's relatively short trachea allows the endotracheal tube to become easily displaced into a main-stem bronchus or into the oesophagus. Mask ventilation can cause inadvertent gastric distension and overinflation of the lungs can result in a pneumothorax (especially after intubation, if the endotracheal tube has migrated beyond the carina). If a traumatic pneumothorax already exists, ventilation will cause it to increase in size and turn it into a tension pneumothorax. Thoracic injuries must be considered in all children who suffer major trauma. Some may be life threatening and require immediate resuscitative therapy during the primary survey and resuscitation. Others may be discovered during the secondary survey (and its associated investigations) and be dealt with by emergency treatment. Some situations will need prompt, specialist surgical intervention, but most chest injuries can be managed in the first hour using general advanced life support skills. Practical procedures are described in detail in Chapter 22. During subsequent detailed review, attention will be redirected to the chest to maintain respiratory control and to search for missed injuries.

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14.2 INJURIES POSING AN IMMEDIATE THREAT TO LIFE

The following conditions are life threatening. They should be identified during the primary survey and treated immediately. They do not need to be confirmed by adjunct investigations.

Tension pneumothorax

This is a relatively common life-threatening emergency that can be rapidly fatal if not treated promptly. Yet treatment is simple and effective.

Air accumulates under pressure in the pleural space. This pushes the mediastinum across the chest and kinks the great vessels, compromising venous return to the heart and reducing cardiac output. The diagnosis is a *clinical* one. A radiograph that shows a tension pneumothorax should never have been taken.

Signs

- The child will be hypoxic and may be shocked.
- Unless the child is deeply unconscious, there will be signs of respiratory distress.
- There will be decreased air entry and hyperresonance to percussion on the side of the pneumothorax.
- Distended neck veins may be apparent in thin children.
- The trachea deviates away from the side of the pneumothorax, although this is not always easy to identify clinically.

Resuscitation

- High-flow oxygen should be given through a reservoir mask.
- Immediate needle thoracocentesis should be performed to relieve the tension.
- A chest drain should be inserted urgently to prevent recurrence.

Air may be forced into the pneumothorax by positive pressure ventilation. If the child is ventilated, a simple pneumothorax is very likely to progress rapidly into a tension pneumothorax.

Massive haemothorax

A massive haemothorax will be identified during the B (*breathing*) stage of the primary survey, although it is even more of a circulatory problem than a respiratory one.

Blood accumulates in the pleural space. This may result from damage to blood vessels (arteries or veins from the pulmonary or systemic vessels) within the lung, the mediastinum or the chest wall (or from a combination). The hemithorax can contain a substantial proportion of a child's blood volume, causing haemorrhagic shock as well as local pressure effects.

Signs

- The child will show signs of shock and may be hypoxic despite added oxygen.
- There will be decreased chest movement, decreased air entry and dullness to percussion on the side of the haemothorax.

Resuscitation

- High-flow oxygen should be given through a reservoir mask.
- Intravenous access should be established and volume replacement commenced.
- A relatively large chest drain should be inserted urgently.

Open pneumothorax

In this situation there is a penetrating wound in the chest wall with associated pneumothorax. The wound may be obvious, but if it is on the child's back it will not be seen unless actively looked for. If the diameter of the defect is greater than about one-third of the diameter of the trachea, air will preferentially enter the pleural space via the defect rather than be drawn into

the lungs via the trachea when the child takes a breath. It is then referred to as a *sucking chest wound*.

Signs

- Air may be heard sucking and blowing through the wound.
- The other signs of pneumothorax will be present.
- There may be an associated haemothorax (i.e. a haemopneumothorax).

Resuscitation

- High-flow oxygen should be given through a reservoir mask.
- The conventional immediate treatment for a sucking wound is to cover it with an occlusive dressing that is taped down on three sides. This is intended to act as a flap valve, allowing air to escape from the untaped side during expiration. A more reliable solution is to use an Asherman chest seal, provided that the defect is not larger than the base of this device. The Asherman seal consists of an adhesive base, similar to that on a colostomy or ileostomy stoma bag, and a short, flexible pipe that protrudes outwards, acting as a one-way valve. However, recent studies have questioned the effectiveness of the Asherman seal and its adherence to traumatised wet skin. Alternatively, the wound may be occluded completely and a separate chest drain inserted.
- Whichever immediate treatment option is undertaken, a chest drain will be required as part of emergency treatment. It should not be inserted through the defect itself as this may spread contamination and restart bleeding.

Flail chest

If a number of adjacent ribs are fractured in two or more places, a segment of the chest wall may be free floating, moving inwards with inspiration and outwards with expiration (*paradoxical movement*). Such a *flail segment* is rare in children because of the elasticity of the child's chest wall. When it does occur, we expect major force to have been involved and serious underlying lung (and mediastinal) injury should be anticipated. If the reported mechanism does not involve significant force, suspect an erroneous history (remember non-accidental injury) or, more rarely, osteogenesis imperfecta.

Flail segments may not be noticed on initial examination for three separate reasons: firstly severe pain on breathing will cause the child to splint the chest wall (this may be unmasked by analgesia); secondly a child who has already been intubated will be receiving positive pressure ventilation, which moves the floating segment in unison with the rest of the chest wall; or thirdly the flail segment may be posterior and unnoticed if the back of the chest is not examined carefully. Rib fractures do not always show up well on the chest radiograph, so that imaging should not be relied upon in making the diagnosis.

Signs

- The child may be hypoxic despite added oxygen and in considerable pain.
- Paradoxical chest movement is characteristic, but may not be obvious as indicated above. A high index of suspicion should be retained.
- Other evidence of rib fractures (e.g. crepitus on palpation) may be seen.

Resuscitation

- High-flow oxygen should be given through a reservoir mask.
- Tracheal intubation and ventilation should be considered immediately if the child is compromised. If ventilation is necessary, it may need to be continued for up to 2 weeks before the flail segment becomes 'sticky' and stabilises. On the other hand, minor cases may do well simply with good pain relief and with oxygen by face mask. Nasal or facial continuous positive airway pressure (CPAP), combined with pain relief, may be effective in intermediate cases.

- Pain relief should be given using titrated intravenous opioids in the first instance. Local or regional neural blockade avoids the respiratory depressant effects of opioids and should be considered. However, intercostal blocks and epidural catheters are hazardous in the uncooperative patient and sedation may be needed to achieve safety – the risks and benefits of the decision must be carefully considered. Epidural analgesia in children should be carried out by an expert and only after injury to the spine has been ruled out formally.

Cardiac tamponade

Cardiac tamponade can occur after both penetrating and blunt injury, though it is much more common after penetrating trauma. The blood that accumulates in the fibrous pericardial sac reduces the volume available for cardiac filling during diastole. As more blood accumulates, the cardiac output is progressively reduced.

Signs

- The child will be in shock.
- The heart sounds may be muffled.
- The neck veins may be distended, though this will not be apparent if the child is also hypovolaemic.

Resuscitation

- High-flow oxygen should be given through a reservoir mask.
- Intravenous access should be established and volume replacement commenced. This may temporarily increase cardiac filling.
- Emergency needle pericardiocentesis should be performed. Removing even a small volume of fluid from within the pericardial sac can dramatically increase cardiac output.
- Emergency thoracotomy will generally be required. A cardiothoracic surgeon (or a paediatric or general surgeon, in centres without cardiothoracic surgery) should be involved as soon as the diagnosis is suspected.

14.3 SERIOUS INJURIES DISCOVERED LATER

These conditions will generally be discovered during the secondary survey and its associated investigations, but delayed presentation and masking by other injuries can occur, demanding continual vigilance into the detailed review phase and beyond.

Pulmonary contusion

Children have a high incidence of pulmonary contusion. Energy is readily transmitted to the lungs, as the ribs are elastic and do not easily dissipate energy by fracturing. If the ribs do fracture, the degree of force is such that pulmonary contusion is likely too. Pulmonary contusion usually results from blunt trauma, although the shock wave from a high-speed bullet can also cause it. At the microscopic level, pulmonary contusion manifests itself as oedema and interstitial and intra-alveolar haemorrhage.

Clinical features include hypoxia, dyspnoea and haemoptysis, but are not specific. Initially, there may be little to show on the chest radiograph, although an area of non-segmental opacification may be clearly visible from the outset. It is important to realise that the clinical features and radiological findings may progress over the next few hours. The appearance on the plain chest film is not specific and may be confused with aspiration, other causes of consolidation/collapse, and even with haemothorax (on a supine film). A computed tomography (CT) scan, when indicated for other reasons, helps to distinguish pulmonary contusion from other diagnoses, but it is not warranted for this purpose alone. There are simpler means of demonstrating a haemothorax, for example clinical examination and bedside ultrasound.

Treatment consists of the administration of high-flow oxygen and artificial ventilation if necessary. Uncomplicated contusions will largely resolve within the next 36 hours.

Physiotherapy plays an important role in reducing the risk of pulmonary collapse and secondary infection.

Tracheal and bronchial rupture

Tracheobronchial disruption has a high mortality and requires prompt referral to a specialist cardiothoracic surgeon. It presents as a pneumothorax or haemopneumothorax, typically with a persistent (and often vigorous) air leak after the insertion of a chest drain. Subcutaneous emphysema is frequently present.

Emergency care may involve the insertion of more than one chest drain (with suction applied). When intubation is required, the passage of the endotracheal tube may further disrupt a tracheal tear. An expert in airway care must be involved. When mechanical ventilation is needed, it is important to limit the pressure applied to the airway. This requires specialist ventilation techniques. Unless the leak is small enough to seal spontaneously, definitive surgical repair will be needed.

Disruption of great vessels

This is usually due to a high-speed motor vehicle crash and is generally fatal at the scene. A child with aortic rupture who survives to get to hospital has a tear that has tamponaded itself within an intact adventitial (outermost) layer. The commonest site of rupture is at the insertion of the ligamentum arteriosum (the residual ligament from the ductus arteriosus that connected the pulmonary and systemic circulations *in utero*), close to the origin of the left subclavian artery – this structure tethers the otherwise mobile aorta during severe deceleration.

The patient may be shocked and peripheral pulses may be poorly palpable. On the other hand, if the leak has (temporarily) sealed itself with little blood loss, interference with the baroreceptors in the aortic arch can lead to relative hypertension. Symptoms are generally non-specific. The diagnosis should be suspected if the mediastinum is widened or has an abnormal profile on chest radiograph. Remember that a supine anteroposterior film will increase the apparent width of the mediastinum and that the thymus is shown as a prominent mediastinal mass in small children. Sternal and spinal fractures can also cause apparent mediastinal widening.

Arch angiography remains the cornerstone of definitive diagnosis, though multislice CT promises to serve as an alternative. It is important to avoid surges in blood pressure that could precipitate rebleeding. Definitive repair requires cardiothoracic surgery.

Ruptured diaphragm

Diaphragmatic rupture is a rare blunt injury in children. It is generally thought to be more common on the left side, though some recent studies have questioned this. Penetrating trauma may also involve the diaphragm, usually in the form of knife stab wounds entering the chest or abdomen. Unless other structures are damaged at the same time, such knife wounds may be asymptomatic, only to present many years later as diaphragmatic hernias.

The child with a ruptured diaphragm may be hypoxic due to diaphragmatic dysfunction and to pulmonary compression from a herniated viscus. Shock may result from mediastinal distortion that affects venous return or result from haemorrhage from adjacent structures. The plain chest radiograph may show an apparently raised hemidiaphragm or evidence of abdominal contents within the chest, e.g. bowel shadowing or a nasogastric tube. Surgical referral should be made. Most ruptures can be repaired from the abdomen, without the need for thoracotomy.

14.4 OTHER INJURIES

Simple pneumothorax

Air is present in the pleural space with some degree of lung collapse, but it is not yet under pressure (*tension*). Signs of hypoxia with decreased chest wall movement, diminished breath

sounds and normal or increased resonance to percussion on the side of the pneumothorax may be found, but the signs may be subtle or barely perceptible compared with tension pneumothorax. The diagnosis is usually made on the plain chest radiograph as a lung edge with no lung markings beyond it. However, an anterior pneumothorax is often difficult to recognise. The increasing use of thoracic CT scanning in severe blunt trauma is picking up injuries that may have been missed on plain films.

As traumatic pneumothoraces do not usually resolve spontaneously, a chest drain should be inserted as a planned procedure, even if the child is relatively asymptomatic. If the patient needs to be ventilated, a chest drain must be inserted as a matter of urgency to avoid a simple pneumothorax developing into a tension pneumothorax.

14.5 PRACTICAL PROCEDURES

Needle thoracocentesis, chest drain insertion and pericardiocentesis are described in Chapter 22.

14.6 REFERRAL

A competent clinician, trained in advanced life support skills, can provide immediate management for most of the life-threatening injuries discovered during the primary survey. Emergency cardiothoracic surgical involvement will be needed if cardiac tamponade is diagnosed. Other serious injuries discovered during the secondary survey will need cardiothoracic referral. The major reasons for referral are shown in the box below.

Indications for cardiothoracic surgical referral

- Continuing massive air leak after chest drain insertion
- Continuing haemorrhage after chest drain insertion
- Cardiac tamponade
- Disruption of the great vessels

Patients who require ventilation as part of the treatment of their chest injury (such as those with significant pulmonary contusion) will need transfer to a paediatric intensive care unit. Critical care management will be needed for transfer and as part of continuing stabilisation in general. Appropriate medical and nursing referrals should be made.

14.7 CONTINUING STABILISATION

In serious chest injuries, the oxygen saturation and pulse rate must be continuously monitored through to the detailed review stage and beyond. The respiratory rate and blood pressure need to be checked frequently. Chest drains must be well secured. Arterial blood gas monitoring is invaluable in severe cases for confirming adequate oxygenation and adjusting carbon dioxide tensions (particularly if there is a concomitant head injury). Remember that many conditions worsen with time, especially pulmonary contusion. An arterial line will also allow haemoglobin, base deficit and lactate to be tracked.

Continual clinical review is required. Changes in the respiratory pattern and in the apparent degree of illness, in conjunction with trends in the monitoring data, will alert the vigilant clinician to new problems and missed injuries.

14.8 SUMMARY

- A clear airway must be established before attending to chest injuries.
- All children should receive high-concentration oxygen through a reservoir mask (if breathing spontaneously), through a mask and self-inflating bag with an oxygen reservoir (if receiving assisted ventilation) or via a ventilator (if intubated).
- Chest injuries are life threatening, but most can be managed successfully by any clinician capable of performing the following techniques:
  - needle thoracocentesis,
  - chest drain insertion,
  - intubation and ventilation,
  - fluid replacement, and
  - pericardiocentesis.
- Cardiothoracic surgical referral may be necessary once immediate management of life-threatening conditions has been carried out.
- Intensive care involvement will be needed for the continuing stabilisation of severe cases.

CHAPTER 15

The child with abdominal injury

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to assess the injured abdomen
- The options for definitive care

15.1 INTRODUCTION

Blunt trauma causes the majority of abdominal injuries in children. Most occur because of accidents on the roads, although a significant number happen during recreational activities. A high index of suspicion is necessary if some injuries are not to be missed.

The abdominal contents are very susceptible to injury in children for a number of reasons. The abdominal wall is thin and offers relatively little protection. The diaphragm is more horizontal than in adults, causing the liver and spleen to lie lower and more anteriorly. Furthermore, the ribs, being very elastic, offer less protection to these organs. Finally, the bladder is intra-abdominal, rather than pelvic, and is therefore more exposed when full. Respiratory compromise can complicate abdominal injury because diaphragmatic irritation or splinting may occur – reducing the use of the diaphragm during breathing.

15.2 HISTORY

A precise history of the mechanism of injury may help in diagnosis. Rapid deceleration, such as experienced during road accidents, causes abdominal compression. The solid organs (liver, spleen and kidneys) are at risk from such forces, and the duodenum may develop a large haematoma or may rupture at the duodenojejunal flexure. Direct blows, such as those caused by punching or impact with bicycle handlebars, readily injure underlying solid organs. Injury to the pancreas or duodenum is a particular feature of handlebar injury. Finally, straddling injuries associated with a significant perineal haematoma or urethral bleeding suggest urethral injury.

15.3 ASSESSMENT OF THE INJURED ABDOMEN

Initial assessment and management must be directed to the care of the airway, breathing and circulation as discussed in Chapter 13.

Examination

If shock is not amenable to fluid replacement during the primary survey and resuscitation, and no obvious site of haemorrhage exists, then intra-abdominal injury may be the cause of blood loss. The abdomen should be assessed urgently to establish whether early operative

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intervention is necessary. In other circumstances, the abdominal examination is carried out during the secondary survey.

The abdomen should be inspected for bruising, lacerations and penetrating wounds. Although major intra-abdominal injury can occur without obvious external signs, visible bruising increases the likelihood of significant injury. A high index of suspicion and frequent, repeated clinical assessment is appropriate in such cases. The external urethral meatus should be examined for blood.

Gentle palpation should be carried out. This will reveal areas of tenderness and rigidity. Care should be taken not to hurt the child because his or her continued cooperation is important during the repeated examinations that form an important part of management.

Rectal and vaginal examinations are rarely required in the injured child. Internal digital examination therefore should be limited to the surgeon who has overall responsibility for the child.

Aids to assessment

Both gastric and urinary bladder drainage may help the assessment by decompressing the abdomen.

Gastric drainage

Air swallowing during crying with consequent acute gastric dilatation is common in children. Early passage of a gastric tube of an appropriate size is essential. The tube should be aspirated regularly and left on free drainage at other times. A massively distended stomach can mimic intra-abdominal pathology needing laparotomy, and cause serious diaphragm splintage with consequent respiratory compromise.

Urinary catheterisation

Catheterisation of a child should only be performed if the child cannot pass urine spontaneously or if continuous accurate output measurement is required. The route (urethral or suprapubic) will depend on factors related to signs of urethral, bladder, intra-abdominal or pelvic injury (such as blood at the external meatus, or bruising in the scrotum or perineum). If a boy requires urethral catheterisation, urethral damage must be excluded first. The catheter should be silastic and as small as possible in order to reduce the risk of subsequent urethral stricture formation.

Investigations

Blood tests

Intravenous access will have already been secured during the primary survey and resuscitation, and at that time blood will have been drawn for baseline blood counts, urea and electrolytes and cross-matching. An amylase estimation should be requested and can usually be performed on the sample sent for urea and electrolytes. Arterial blood gases should be sent if indicated. Repeated monitoring of blood parameters may be appropriate in some patients.

Radiographs

Views of the chest and pelvis will have been obtained earlier. Neither a normal chest radiograph nor a normal pelvic radiograph excludes abdominal injury. Subsequent plain abdominal radiography may be helpful to look for gastric tube position, abdominal gas distribution and free gas in cases of suspected small bowel injury, where diagnosis is often delayed and notoriously difficult. Blood at the external urethral meatus may require investigation using retrograde urethrography.

Computed tomography

A single-contrast CT scan of the abdomen is the radiological investigation of choice in children. CT will confirm renal perfusion and identify solid organ injury. While free intraperitoneal air

is pathognomonic of a perforated viscus, the presence of significant free fluid in the absence of solid organ injury heightens suspicion of a bowel or, rarely, bladder injury.

Ultrasound

The use of FAST (focused abdominal with sonography for trauma) scanning has become routine in most emergency departments and can be helpful in early detection of free fluid and lacerations of the liver, spleen or kidneys. A normal ultrasound early on does not exclude injury, thus serial scans and watchful waiting can be helpful.

Diagnostic peritoneal lavage

The use of this investigation in children has been rendered virtually obsolete in the trauma setting by modern imaging modalities. The presence of intraperitoneal blood *per se* is not an indication for laparotomy. Once lavage fluid has been introduced, the peritoneum shows signs of irritation for up to 48 hours, and hence reduces the possibility of accurate repeated assessment. The technique is nevertheless described in Chapter 22 as peritoneal lavage may be used to re-warm the hypothermic 'drowned' patient, and access to the peritoneum may provide a temporary dialysis route in children with acute renal failure.

15.4 DEFINITIVE CARE

Non-operative management

Until the early 1980s, both adult and paediatric patients with haemoperitoneum would undergo laparotomy. Damage to the spleen or liver would result in splenectomy or partial hepatectomy, respectively. It has since been shown that the haemorrhage is often self-limiting, and many of these operations can therefore be avoided. As well as avoiding the morbidity associated with laparotomy, this approach also reduces the number of children at risk of overwhelming, potentially fatal sepsis following splenectomy.

For non-operative management to be undertaken the following are essential:

- Adequate observation and frequent monitoring.
- Precise fluid management.
- The immediate availability of a surgeon trained to operate on the paediatric abdomen (should this become necessary).

The need for clotting factors such as platelets, fresh frozen plasma or cryoprecipitate must be monitored. Vigorous and early management of coagulopathy is indicated in order to improve clotting and hence achieve haemostasis.

Indications for operative intervention

Children whose circulation is not stable after replacement of 40 ml/kg of fluid are probably bleeding into the thoracic or abdominal cavities. In the absence of clear thoracic bleeding, urgent laparotomy may be necessary. All children with penetrating abdominal injuries and those with definite signs of bowel perforation will require urgent laparotomy.

A non-functioning kidney, as demonstrated on contrast studies, may have suffered renal pedicle injury. Exploration will not salvage the kidney since the warm ischaemia time is only 45–60 minutes.

Indications for operative intervention following abdominal injury

- Laparotomy
- Refractory shock with evidence of solid organ injury on CT scan
- Penetrating injuries
- Signs of bowel perforation

It is essential that the surgeon performing these procedures is competent to deal with paediatric trauma and can perform any reconstructive surgery that may be required.

15.5 SUMMARY

- The assessment and management of airway, breathing and circulation must be carried out first. Abdominal assessment is only carried out at this stage if shock is refractory.
- Abdominal assessment consists of careful observation and gentle, repeated palpation. Gastric and urinary drainage aid this assessment.
- Abdominal CT scan is the investigation of choice.
- The majority of children with solid organ injury may be managed non-operatively. Urgent operative intervention is required when children with solid organ injury have persisting haemodynamic instability despite adequate blood replacement, or for penetrating abdominal injury, or signs of a perforated viscus.

CHAPTER 16

The child with trauma to the head

LEARNING OBJECTIVES
In this chapter, you will learn:
- The structured approach in the context of the child with a head injury

16.1 INTRODUCTION

Epidemiology

Head injury is the most common single cause of trauma death in children aged 1–15 years. It accounts for 27% of deaths from injury and many (but largely unstudied) cases of permanent brain injury, probably up to 2000 or even 3000 children per year in the UK alone. Head injury deaths in children most commonly result from road traffic accidents – pedestrians are the most vulnerable, followed by cyclists, and then passengers in vehicles. Falls are the second most common cause of fatal head injuries. In infancy, the most common cause is child abuse.

Pathophysiology

Primary brain injury is the damage incurred as a direct consequence of the impact. Neurones, axonal sheaths and blood vessels may be physically disrupted at the moment of impact, often with irreversible cell damage. *Secondary* brain injury represents further damage to central nervous system tissue by *secondary insults*, and adverse physiological events that can occur minutes, hours or days after the initial injury. Such insults include hypotension, hypoxia, intracranial hypertension and seizures. A key aim of head injury management is to prevent or minimise secondary brain injury.

Primary damage

- Injury to neural tissue:
  - focal cerebral contusions and lacerations (direct impact and contrecoup), and
  - diffuse axonal injury (shearing injury).
- Injury to intracranial blood vessels:
  - extradural haematoma (especially middle meningeal artery),
  - subdural haematoma (especially dural bridging veins),
  - intracerebral haematoma, and
  - subarachnoid haemorrhage.

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Injury to the cranium and to the dural sac may be associated with the above neural and vascular injuries. Open skull fractures, where there is a breach in the skull (vault or base) and in the dural membrane, allow brain tissue to come into contact with the external environment (directly or via the sinuses), with consequent risk of infection.

Secondary damage

This may result from either the direct secondary effects of cerebral injury or from the cerebral consequences of associated injuries and stress.

- Ischaemia from poor cerebral perfusion secondary to raised intracranial pressure:
  - expanding intracranial haematoma (exacerbated by coagulopathy), and
  - cerebral swelling/oedema.
- Ischaemia secondary to hypotension and anaemia:
  - haemorrhage with hypovolaemia or dilutional anaemia, and
  - other causes of hypotension (spinal cord injury, drug-induced vasodilatation or *later* sepsis).
- Hypoxia:
  - airway obstruction,
  - inadequate ventilation (loss of respiratory drive or mechanical disruption of chest wall or diaphragm), and
  - shunt from pulmonary contusion or *later* respiratory failure.
- Hypoglycaemia and hyperglycaemia.
- Fever.
- Convulsions.
- *Later* infection.

Hypothermia may have a protective effect on neural tissue, but at temperatures below 35°C it affects platelet function and may exacerbate bleeding.

Raised intracranial pressure

Once sutures have closed at 12–18 months of age, the child's cranial cavity behaves like an adult's with a fixed volume. If cerebral oedema worsens or if intracranial haematomas increase in size, the pressure within the cranium increases. Initial compensatory mechanisms include diminution in the volume of cerebrospinal fluid and venous blood within the cranial cavity. When these mechanisms fail, intracranial pressure rises, compromising cerebral perfusion:

Cerebral perfusion pressure = Mean arterial pressure – Mean intracranial pressure

Normal cerebral blood flow is 50 ml of blood per 100 g brain tissue per minute. A fall in cerebral perfusion pressure decreases cerebral blood flow. A flow below 20 ml/100 g brain tissue/min will produce ischaemia. This in turn increases cerebral oedema, causing a further rise in intracranial pressure. A cerebral blood flow of below 10 ml/100 g brain tissue/min leads to electrical dysfunction of the neurones and loss of intracellular homeostasis.

A generalised increase of intracranial pressure in the supratentorial compartment initially causes transtentorial (uncal) herniation, leading to transforaminal (central) herniation and death. In uncal herniation, the third nerve is nipped against the free border of the tentorium, causing ipsilateral pupillary dilatation secondary to loss of parasympathetic constrictor tone to the ciliary muscles. In central herniation, also known as *coning*, the cerebellar tonsils are forced through the foramen magnum.

In childhood, the most common cause of raised intracranial pressure following head injury is cerebral oedema. Children are especially prone to this problem. They may, of course, also have expanding extradural, subdural or intracerebral haematomas that require prompt surgical treatment.

Depending on the aetiology of the raised intracranial pressure, treatment is either aimed at preventing it rising further or removing its cause (by surgical evacuation of haematomas).

There are special considerations in infants with head injuries. Unfused sutures allow the cranial volume to increase initially. Large extradural or subdural bleeds may occur before neurological signs or symptoms develop. Such infants may show a significant fall in haemoglobin concentration. In addition, the infant's vascular scalp may bleed profusely, causing shock. In children aged over 1 year with shock associated with head injury, serious extracranial injury should be sought as the cause of the shock.

16.2 TRIAGE

Head injuries vary from the trivial to the fatal. Triage is necessary in order to give more seriously injured patients a higher priority. Factors indicating a potentially serious injury are shown in the box below.

Factors indicating a potentially serious injury

- History of substantial trauma such as involvement in a road traffic accident or a fall from a height
- A history of loss of consciousness
- Children who are not fully conscious and responsive
- Any child with obvious neurological signs/symptoms such as headache, convulsions or limb weakness
- Evidence of penetrating injury

16.3 PRIMARY SURVEY

The first priority is to assess and stabilise the airway, breathing and circulation as discussed in Chapter 13. Head injury may be associated with cervical spine injury, and neck immobilisation must be achieved as previously described.

Pupil size and reactivity should be examined and a rapid assessment of conscious level should be made. In the first place the AVPU classification may be used.

A	**A**lert
V	Responds to **V**oice
P	Responds only to **P**ain
U	**U**nresponsive to all stimuli

In a time-limited situation, it is not essential to work out the numerical Glasgow Coma Scale (GCS) score immediately, although the EMV (eye, motor, verbal) responses will have been noted. But it is important to note the response to voice or pain (if not responding to voice) in more detail using the Glasgow Coma Scale before proceeding with neurological resuscitation. The assessment serves as a baseline for continuing care and as a key indicator of the need to intervene immediately.

16.4 RESUSCITATION

Immediate control of the airway, breathing and circulation should be carried out in response to the primary survey findings, according to the general approach in Chapter 13. This support

will help to prevent secondary cerebral damage caused by hypoxia and shock arising from both the head injury and other coexistent injuries.

During the primary survey assessment of disability, evidence of decompensating head injury will have been recognised. In the severely injured child, extra information from blood gas sampling will be obtained during the resuscitation phase or ongoing monitoring. On the basis of simple clinical evaluation, supported when necessary by blood gas data, a set of indications for immediate intubation and ventilation in severe head injury have been recommended (in the UK, the National Institute for Health and Clinical Excellence (NICE) has produced evidence-based guidelines for treatment, imaging and referral).

Indications for immediate intubation and ventilation

- Coma – not obeying commands, not speaking, not eye opening (equivalent to a GCS score of <8)
- Loss of protective laryngeal reflexes
- Ventilatory insufficiency as judged by blood gases: hypoxaemia (PaO_2 < 9 kPa on air or <13 kPa with added oxygen) or hypercarbia ($PaCO_2$ > 6 kPa)
- Spontaneous hyperventilation (causing $PaCO_2$ < 3.5 kPa)
- Respiratory irregularity

16.5 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES

History

The history of the injury itself and the child's course since the injury should be established from bystanders and pre-hospital personnel. Other history should be obtained from parents or carers.

Examination

The head should be carefully observed and palpated for bruises and lacerations to the scalp and for evidence of a depressed skull fracture. Look for evidence of a basal skull fracture, such as blood or cerebrospinal fluid (CSF) from the nose or ear, haemotympanum, panda eyes or Battle's sign (bruising behind the ear over the mastoid process).

The conscious level should be reassessed using the modified Glasgow Coma Scale if the child is less than 4 years old, or the standard scale in older children. These scales are shown in Table 16.1. It should be noted that the coma scales reflect the degree of brain dysfunction *at the time of the examination*. Assessment should be repeated frequently – every few minutes if the level is changing. Communication with the child's caregivers is required to establish the child's best usual verbal response. A 'grimace' alternative to verbal responses should be used in pre-verbal or intubated patients (Table 16.2).

The pupils should be re-examined for size and reactivity. A dilated non-reactive pupil indicates third nerve dysfunction due to an ipsilateral intracranial haematoma until proven otherwise.

The fundi should be examined using an ophthalmoscope. Papilloedema will not be seen in acute raised intracranial pressure, but the presence of retinal haemorrhage may indicate non-accidental injury in a young infant.

Motor function should be assessed. This includes examination of extraocular muscle function and facial and limb movements. Limb tone, movement and reflexes should be assessed and any focal or lateralising signs noted.

Table 16.1 Glasgow Coma Scale and Children's Glasgow Coma Scale

Glasgow Coma Scale (4–15 years)		Children's Glasgow Coma Scale (<4 years)	
Response	**Score**	**Response**	**Score**
Eye opening		*Eye opening*	
Spontaneously	4	Spontaneously	4
To verbal stimuli	3	To verbal stimuli	3
To pain	2	To pain	2
No response to pain	1	No response to pain	1
Best motor response		*Best motor response*	
Obeys verbal command	6	Spontaneous or obeys verbal command	6
Localises to pain	5	Localises to pain or withdraws to touch	5
Withdraws from pain	4	Withdraws from pain	4
Abnormal flexion to pain (decorticate)	3	Abnormal flexion to pain (decorticate)	3
Abnormal extension to pain (decerebrate)	2	Abnormal extension to pain (decerebrate)	2
No response to pain	1	No response to pain	1
Best verbal response		*Best verbal response*	
Orientated and converses	5	Alert; babbles, coos words to usual ability	5
Disorientated and converses	4	Less than usual words, spontaneous irritable cry	4
Inappropriate words	3	Cries only to pain	3
Incomprehensible sounds	2	Moans to pain	2
No response to pain	1	No response to pain	1

Table 16.2 The best grimace response

Grimace response	Score
Spontaneous normal facial/oromotor activity	5
Less than usual spontaneous ability or only response to touch stimuli	4
Vigorous grimace to pain	3
Mild grimace to pain	2
No response to pain	1

Investigations

Blood tests

Blood for full blood count, clotting, glucose, urea and electrolytes should already have been taken during the immediate care phase. Blood for cross-matching should have been sent off at the same time. Arterial blood gases should be taken in head-injured patients to allow careful control of $PaCO_2$ and PaO_2, as well as to check pH and base deficit or lactate.

Imaging

Plain radiographs of the chest and pelvis may have been taken during the primary survey and resuscitation phase, depending on the mechanism of injury and the current state of the child. If not, reconsider the need for them at this stage. Remember that the chest film will need to be repeated if the child is subsequently intubated.

In severe head injury, a skull radiograph will usually be superfluous, as equivalent information (and more) will be contained within the computed tomography (CT) scan. In non-accidental injury and in some cases of penetrating head trauma, plain skull films still have a useful role. For other head injuries, this once-common investigation has been supplanted by CT scanning, on the basis of evidence in the literature. Indications for performing an emergency head CT scan are summarised below (UK NICE guidelines):

Indications for performing an emergency head CT scan

- Witnessed loss of consciousness lasting more than 5 minutes
- Amnesia (antegrade or retrograde) lasting more than 5 minutes
- Abnormal drowsiness
- 3 or more discrete episodes of vomiting
- Clinical suspicion of non-accidental injury
- Post-traumatic seizure but no history of epilepsy
- Age more than 1 year: GCS less than 14 on assessment in the Emergency Department
- Age less than 1 year: GCS less than 15 on assessment in the Emergency Department
- Suspicion of open or depressed skull injury or tense fontanelle
- Any sign of basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from ears or nose, Battle's sign)
- Focal neurological deficit
- Age less than 1 year: presence of bruise, swelling or laceration more than 5 cm on the head
- Dangerous mechanism of injury (high-speed road traffic collision either as pedestrian, cyclist or vehicle occupant, fall from more than 3 m, high-speed injury from a projectile or an object)

An area of concern especially in small children is the need, on occasion, to anaesthetise them for a CT scan, if they are non-cooperative or the scanner is slow. This must only be done where skills and facilities are available for the anaesthetic care of young children.

Remember that the cervical spine is at risk and that in the presence of altered consciousness such as from sedative drugs, neck pain or tenderness, or distracting pain from other injuries, spinal imaging will be required.

16.6 EMERGENCY TREATMENT

The initial aim of management of a child with a serious head injury is prevention of secondary brain damage. The key aims are to maintain oxygenation, ventilation and circulation, and to avoid rises in intracranial pressure (ICP).

These can best be achieved by paying attention to the <C>ABCs discussed earlier. If the airway is at risk, it should be secured. Children with a GCS score of 8 or less should be intubated and ventilated without delay using rapid-sequence induction of anaesthesia. Capnography must be used immediately after intubation to confirm endotracheal tube placement, to serve as a disconnection monitor, and to help maintain normocapnia or mild hypocapnia if there is evidence of raised ICP. Remember that the end-tidal CO_2 level may differ significantly from the arterial level, especially in the shocked child – *it is essential to check the arterial* P_{CO_2} *level with a blood gas sample*. While routine hyperventilation has not been shown to improve outcome, arterial P_{CO_2} levels of 4–4.5 kPa are considered to be appropriate in the presence of an acutely rising ICP. Lower levels may adversely affect cerebral perfusion in the areas of brain still responsive to changes in P_{CO_2}. Except in life-threatening, uncontrolled bleeding (see Chapter 13), hypotension should be treated vigorously to avoid hypoperfusion of the brain.

Analgesia

There have been concerns that opioid analgesic agents will lower the conscious level, cause respiratory depression and conceal pain in the abdomen and elsewhere. However, withholding analgesia may contribute to deterioration of the child's condition by leading to a rise in intracranial pressure. Failing to control pain will leave the child agitated and uncooperative, making any assessment of the pain more difficult, rather than easier.

Following initial assessment, sufficient analgesia should be administered by careful titration. It is important to appreciate that head-injured children are often more sensitive to opioids. If the child's conscious level is normal, despite other evidence of head injury, IV morphine in an initial standard dose of 100–200 micrograms/kg (<1 year: 80 micrograms/kg) (administered in increments) is appropriate. In obtunded children, particularly if the GCS score is 8 or less, intubation and ventilation will have a higher priority than analgesia alone. In intermediate cases, a useful rule of thumb is to expect that half the standard dose may be sufficient in the first instance.

Remember that opioids can be rapidly reversed with naloxone if necessary, although it is clearly better to avoid overadministration by cautious titration. Alternative opioids such as fentanyl that act more quickly when given intravenously or that can be given by an alternative route (e.g. mucosal) may be considered, as described in Chapter 13. Local anaesthetic techniques such as femoral nerve block may also be used to good effect, avoiding opioid side effects.

Management of specific problems

Deteriorating conscious level

If airway, breathing and circulation are satisfactory and hypoglycaemia has been excluded, then a deteriorating conscious level is assumed to be due to increased intracranial pressure, resulting from an intracranial haematoma or cerebral oedema. CT scan and urgent neurosurgical referral are indicated and the temporising manoeuvres shown in the box may be instituted.

Measures to increase cerebral perfusion temporarily

- Nurse in the 20° head-up position and head in midline to help venous drainage
- Ventilation to Pa_{CO_2} of 4.0–4.5 kPa*
- Infusion of intravenous mannitol 0.25–0.5 g/kg or hypertonic saline
- Combat hypotension if present with colloid infusion or inotropes if necessary

*Note this level is lower than normal because it is a temporary, short-term, urgent intervention.

Signs of uncal or central herniation

These signs (discussed in Chapter 11) should lead to immediate institution of the measures in the box above and emergency neurosurgical referral.

Convulsions

A focal seizure should be regarded as a focal neurological sign of considerable concern. A general convulsion, while also worrying, has less prognostic significance in children. Seizure activity raises intracranial pressure in both non-paralysed and paralysed patients, as well as causing an acidosis and increased cerebral metabolic demand. The lack of limb or facial movement makes it more difficult to recognise a seizure if the child has been paralysed, but fitting should still be suspected if there is a sharp increase in heart rate and blood pressure, with dilatation of the pupils.

Seizures due to head injury should be controlled promptly. Hypoglycaemia should be excluded, especially in small children and in adolescents who have been drinking alcohol. The initial drugs of choice are diazepam, midazolam or lorazepam (see Chapter 12). However, phenytoin should be used for prolonged or persistent convulsions as it is less sedative. The dose is 20 mg/kg intravenously over 20 minutes, with appropriate monitoring for rhythm irregularities and hypotension.

Neurosurgical referral

Agreed indications for neurosurgical referral are shown in the box below (NICE guidelines):

Indications for referral to a neurosurgeon

- Persisting coma (GCS <8) after initial resuscitation
- Unexplained confusion lasting for more than 4 hours
- Deteriorating conscious level (especially motor response changes)
- Focal neurological signs
- Seizure without full recovery
- Definite or suspected penetrating injury
- A cerebrospinal fluid leak

Other cases may be discussed to consider referral and to ensure optimal management, such as when there is evidence of a depressed or basal skull fracture or if the initial GCS is between 8 and 12. In general, the care of all children with new, surgically significant abnormalities on imaging should be discussed with a neurosurgeon.

16.7 DETAILED REVIEW AND CONTINUING STABILISATION

Review anatomical injuries and physiological system control.

It is easy to miss injuries in the face of an altered conscious level. A high index of suspicion is essential. Reconsider the mechanism of injury, review the physical and radiological findings, and make sure that the appropriate specialists have been involved.

In the severely head-injured child, physiological system control is of critical importance in preventing secondary insults. The airway and ventilation have been dealt with as part of emergency treatment. The position of the endotracheal tube should now be checked on a chest radiograph and the tube fixation adjusted and re-secured, if necessary. Attention to detail in adjusting the ventilator settings, according to repeated arterial blood gas sampling, is vital. An adequate blood pressure and circulating haemoglobin level are required for cerebral perfusion. Preventing rises in intracranial pressure will help achieve the same goal. Sedation and paralysis play an important role in tolerating the endotracheal tube and in suppressing rises in intracranial pressure, but must not be allowed to cause hypotension. Bleeding from other injuries

should already have been stopped and the blood volume restored. Normoglycaemia and a normal or slightly reduced temperature help to guarantee an optimal outcome.

Vigilance is needed to recognise any significant deterioration in the child's condition. Any of the following examples of neurological deterioration should prompt urgent reappraisal by the supervising medical team (UK NICE guidelines):

Examples of neurological deterioration prompting urgent reappraisal

- Development of agitated or abnormal behaviour
- A sustained (>30 minutes) drop of 1 point in the GCS (especially in the motor score)
- Any drop of 2 points in the GCS
- Severe/increasing headache/vomiting
- New neurological signs

16.8 TRANSFER TO DEFINITIVE CARE

Transport of critically ill children is increasingly the responsibility of the receiving hospital. In general, the quality of transfer is more important than absolute speed. Within the time constraints imposed by the particular diagnosis, adequate time should be spent preparing the child before setting off (see Chapter 24). Despite this emphasis, remember that some conditions (especially an expanding intracranial haematoma) depend on timely surgical intervention for a successful outcome, and a proactive approach is necessary to optimise outcome. In such circumstances, the delay in waiting for a retrieval team to arrive may be unacceptable, so that the responsibility for transfer may revert to the primary hospital.

16.9 SUMMARY

- The impact causes primary brain damage. Secondary damage occurs principally as a result of hypoxia and poor cerebral perfusion and cerebral bleeding, but can also result from hypoglycaemia, seizures and fever.
- The first priority is assessment and management of the airway, breathing and circulation.
- The response to voice or pain (if not responding to voice) and the pupillary responses are checked to identify decompensating head injury. Immediate measures are taken to improve cerebral perfusion, when indicated.
- A thorough secondary survey involves an assessment of external signs of head injury and a more detailed neurological examination. This includes a repeated assessment of conscious level and pupillary responses, together with examination of the fundi and motor function.
- A head CT scan should be performed if indicated.
- The aim of initial management is to prevent secondary damage. This is achieved by attention to airway, breathing and circulation, and by prompt neurosurgical referral and transfer for operative decompression or neurocritical care, when indicated.

CHAPTER 17

The child with injuries to the extremities or the spine

LEARNING OBJECTIVES

In this chapter, you will learn:

- How to use the structured approach in the assessment and management of the child with injuries to the extremities or the spine

17.1 EXTREMITY TRAUMA: INTRODUCTION

Skeletal injury accounts for 10–15% of all childhood injuries – of these 15% involve physeal disruptions. It is uncommon for extremity trauma to be life threatening in the multiply injured child. It is crucial to recognise and treat associated life-threatening injuries before assessing and managing the skeletal trauma. This can often be difficult in children as the pain associated with the extremity trauma can mask serious life-threatening injuries. Although rarely life threatening, fractures and associated extremity trauma must be managed well or else they can have devastating implications for subsequent rehabilitation. This chapter deals with problems from the perspective of multiple injury; the principles apply equally to individual injuries.

The differences between the mature and immature skeleton have a bearing on initial treatment and eventual outcome. Use of the principles usually applied to injuries of the mature skeleton will result in errors of both diagnosis and treatment. Children's bones are prone to a greater range of injury than those of adults. This is a reflection of the different mechanical properties of the immature skeleton, in particular the greater plasticity of bones and the presence of growth plates. These differences explain the occurrence of fractures unique to childhood. Greenstick and torus fractures occur because one or both cortices deform without fracturing. The growth plate is 2–5 times weaker than any other structure in the paediatric skeleton (including ligaments and tendons), so it is not surprising that it is commonly involved in fractures. The chance of fracture propagation is reduced, and comminuted fractures are relatively rare. It should be remembered that children's bones can absorb more force than adults and this may result in an underestimation of the degree of trauma to associated soft tissues.

17.2 ASSESSMENT OF EXTREMITY TRAUMA

Unless extremity injury is life threatening, evaluation is carried out during the secondary survey and treatment commenced during the definitive care phase. Single, closed extremity injuries may produce enough blood loss to cause hypovolaemic shock, but this is not usually life threatening. Multiple fractures can, however, cause severe shock. Pelvic fractures are relatively uncommon in children – the energy that would have fractured a pelvis in an adult may have been transmitted to vessels within the pelvis of a child, leading to disruption and haemorrhage.

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Closed fractures of the femur may cause loss of approximately 20% of the intravascular volume into the thigh, and blood loss from open fractures can be even more significant. This blood loss begins at the time of the injury, and it can be difficult to estimate the degree of pre-hospital loss.

17.3 PRIMARY SURVEY OF EXTREMITY TRAUMA AND RESUSCITATION

All multiply injured children should be approached in the structured way discussed in Chapter 13. Relevant history should be sought from relatives and pre-hospital staff. Extremity deformity and perfusion prior to arrival at hospital are especially important, and information concerning the method of injury is helpful.

Life-threatening injuries

These include the following:

- Crush injuries of the abdomen and pelvis.
- Traumatic amputation of an extremity.
- Massive, open, long-bone fractures.

They should be dealt with immediately and take precedence over any other extremity injury.

Crush injuries to the abdomen and pelvis

The pelvic bones of a child are much more cartilaginous and thus more flexible than those of an adult; therefore if fractures occur it will only be after significant impact. A child's pelvis tends to be narrower than that of an adult and thus does not offer the same protection to the internal structure and organs. The significance of a fracture in itself is not important but the subsequent damage caused to the associated organs and structures can be life threatening and must be treated accordingly. Pelvic disruption can lead to life-threatening blood loss. The child will present with hypovolaemic shock; this may remain resistant to treatment until either the pelvic disruption is stabilised or the injured vessels are occluded.

Initial treatment during the primary survey and resuscitation phase consists of splinting of the pelvis with a temporary sling, rapid fluid and blood infusions. The diagnosis may be obvious if disruption is severe or if fractures are open. More often this cause of resistant hypovolaemia is discovered when the pelvic radiograph is taken.

Emergency orthopaedic opinion should be sought, and urgent external fixation of the pelvis should be considered. In some hospitals, radiographic identification and therapeutic embolisation of bleeding vessels may be attempted.

Traumatic amputation

Traumatic amputation of an extremity may be partial or complete. Paradoxically, it is usually the former that presents the greatest initial threat to life. This is because completely transected vessels go into spasm, whereas partially transected vessels may not. Blood loss can be large and the pre-hospital care of these injuries is critical; an exact history of this should be sought.

Once in hospital the airway should be cleared and breathing assessed as previously discussed. Exsanguinating haemorrhage must be controlled. Two wide-bore cannulae should be inserted. If the child is in shock, but the bleeding points are well controlled, vigorous fluid therapy may be instituted. If the bleeding is still uncontrolled, fluid boluses should be commenced in 5–10 ml/kg aliquots until control is achieved (see Chapter 13). If local pressure and elevation are not sufficient, pressure may also be applied temporarily over the femoral and brachial artery or by use of a tourniquet. Bleeding continues through bones that are not compressed by the tourniquet and tissue viability is generally compromised. On the basis of experience from land-mine injuries, an elasticated compression bandage and dressing, if applied carefully, may help stem the haemorrhage and better preserve tissue viability. Emergency orthopaedic and plastic surgical opinions should be sought. If no active bleeding is taking place, the stump

should be dressed with a sterile dressing soaked in normal saline and the limb splinted and elevated.

Reimplantation techniques are available in specialist centres. The success rate is improving, particularly in children. Urgent referral and transfer are necessary – the amputated part will only remain viable for 8 hours at room temperature, or for 18 hours if cooled. The amputated part should be cleaned, wrapped in a moist sterile towel, placed in a sterile, sealed plastic bag and transported in an insulated box filled with crushed ice and water *in the same vehicle as the child*. Care should be taken to avoid direct contact between the ice and tissue.

If, after discussion with the specialist centre, it is decided that reimplantation is not appropriate, the amputated part should still be saved because it may be used for grafting of other injuries.

> The child must be stabilised before transfer.

Massive, open, long-bone fractures

The blood loss from any long-bone fractures may be significant; open fractures bleed more than closed ones because there is no tamponade effect from surrounding tissues. As a general rule an open fracture causes twice the blood loss of the corresponding closed fracture. Thus a single, open, femoral shaft fracture may result in 40% loss of circulating blood volume. This in itself is life threatening.

After airway and breathing have been assessed and treated, two relatively large-bore cannulae should be inserted and fluid boluses should be commenced according to the child's overall circulatory state (see Chapter 13). Exsanguinating haemorrhage should be controlled both by the application of pressure at the fracture site, and by correct splinting of the limb; in certain cases the use of tourniquets may be indicated.

Emergency orthopaedic opinion should be sought. Angiography may be necessary to examine whether any major vessel rupture has occurred, and if such an injury is considered likely then a vascular surgical opinion should be obtained immediately.

17.4 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES OF EXTREMITY TRAUMA

In a conscious child, inspection is usually the most productive part of the examination. Causing pain or eliciting crepitus in an injured extremity will only increase anxiety, ultimately making the child more difficult to manage.

The extremities should be inspected for discolouration, bruising, swelling, deformity, lacerations and evidence of open fractures.

Next, *gentle* palpation should be undertaken to establish any areas of tenderness. Limb temperature and capillary refill should be assessed, and pulses sought – a Doppler flow probe should be used if necessary.

Finally, the active range of motion should be assessed if the child is cooperative. If there is an obvious fracture or dislocation, or the child refuses to move a limb actively, passive movement should be avoided.

Limb-threatening injury

The viability of a limb may be threatened by vascular injury, compartment syndrome or by open fractures. These situations are discussed below.

Vascular injury

Assessment of the vascular status of the extremity is a vital step in evaluating an injury. Vascular damage may be caused by traction (resulting in intimal damage or complete disruption), or by penetrating injuries caused by either a missile or the end of a fractured bone. Brisk bleeding

from an open wound or a rapidly expanding mass is indicative of active bleeding. Complete tears are less likely to bleed for a prolonged period due to contraction of the vessel. It should be remembered that nerves usually pass in close proximity to vessels and are likely to have been damaged along with the vessel.

The presence of a pulse, either clinically or on Doppler examination, does not rule out a vascular injury. *A diminished pulse should not be attributed to spasm*.

The signs of vascular injury are shown in the box below.

Signs of vascular injury
- Abnormal pulses
- Impaired capillary return
- Decreased sensation
- Rapidly expanding haematoma
- Bruit

If these signs are present, urgent investigation and emergency treatment should be commenced. The fracture should be aligned and splints checked to ensure that they are not restrictive; if no improvement occurs a vascular surgeon should be consulted and angiography considered. Vascular damage may not always be immediately apparent so constant reassessment is essential.

Compartment syndrome

If the interstitial pressure within a fascial compartment rises above capillary pressure, then local muscle ischaemia occurs. If this is unrecognised, it eventually results in Volkmann's ischaemic contracture. Compartment syndrome usually develops over a period of hours and is most often associated with crush injuries. It may, however, occur following simple fractures and also as a result of misplaced intraosseous infusions. The classic signs are shown in the box below.

Classic signs of compartment syndrome
- Pain, accentuated by passively stretching the involved muscles
- Decreased sensation
- Swelling
- Pallor of limb
- Paralysis
- Pulselessness

Distal pulses only disappear when the intracompartmental pressure rises above arterial pressure; by this time irreversible changes have usually occurred in the muscle bed. Initial treatment consists of releasing constricting bandages and splints. If this is ineffective then urgent surgical fasciotomy should be performed.

Open fractures

Any wound within the vicinity of a fracture should be assumed to communicate with the fracture.

Open wounds are classified according to the degree of soft tissue damage, amount of contamination and the presence or absence of associated neurovascular damage. Initial treatment includes removal of gross contamination and covering the wound with a sterile dressing. No attempt should be made to ligate bleeding points because associated nerves may be damaged as this is done. Bleeding should be controlled by direct pressure. Broad-spectrum antibiotics should be given, and tetanus immunisation status checked. Further management is surgical –

debridement should be carried out within 6 hours by the orthopaedic surgeons under operating theatre conditions. It may be useful to take a photograph of the wound to reduce the number of times the dressing is removed.

Other injuries

Non-accidental injury

This must always be considered if the history is not consistent with the injury pattern. It is discussed in detail in Appendix C.

Fracture–dislocation

It is difficult to distinguish fractures and fracture–dislocations on clinical grounds. Radiology is often helpful, but in very young children, where ossification centres have not yet formed, an ultrasound examination or magnetic resonance imaging (MRI) scan may be necessary. In an older child (when some of the ossification centres are present), a comparative radiograph of the normal side may be helpful before more invasive investigations are considered. These investigations should be performed in the definitive care phase, unless there are vascular or neurological complications. If suspected, early orthopaedic assistance must be sought.

Dislocations

Dislocations, other than of the elbow and hip, are rare in children but, as for adults, may produce neurovascular injury that can result in permanent impairment. All dislocations should therefore be reduced as soon as possible.

Epiphyseal injuries

Fractures involving the epiphysis may be displaced or non-displaced. An orthopaedic surgeon should manage them.

17.5 EMERGENCY TREATMENT OF EXTREMITY TRAUMA

Life-threatening problems identified during the primary survey in the multiply injured patient are managed first. Only then should attention be turned to the extremity injury. The specific management of complications such as vascular injury, compartment syndrome, traumatic amputation and open wounds have been discussed earlier in this chapter.

Alignment

Severely angulated fractures should be aligned. Gentle traction should be applied to the limb to facilitate alignment, particularly when immobilising long-bone fractures. Splints should extend one joint above and below the fracture site. Perfusion of the extremity, including pulses, skin colour, temperature and neurological status, must be assessed before and after the fracture is aligned. Radiographs, including arteriograms, should not be obtained until the extremity is splinted.

When aligning a fracture, analgesia is usually necessary. Entonox or intravenous opiates should be used. Analgesia and further pain management is discussed in Appendix F. In femoral fractures, femoral nerve block is very effective – the technique is discussed in Chapter 22.

Immobilisation

Fractures (or suspected fractures) should be immobilised to control pain and prevent further injury. Splintage is a most effective way of controlling pain, and subsequent doses of analgesia may be reduced. If pain increases after immobilisation, then an ischaemic injury and/or compartment syndrome must be excluded. Emergency splinting techniques for various injured extremities are described below.

Upper limb

- *Hand.* This should be splinted in the position of function with the wrist slightly dorsiflexed and the fingers slightly flexed at all joints. This is best achieved by gently immobilising the hand over a large roll of gauze.

- *Forearm and wrist.* They should be splinted flat on padded pillows or a dorsal above-elbow plaster back slab should be applied.
- *Elbow.* The elbow should be immobilised in a flexed position to a maximum of 80° of flexion from full extension with a sling and an above-elbow plaster back slab.
- *Arm.* The arm should be immobilised by a sling, which can be augmented with splints for unstable fractures. Circumferential bandages should be avoided, as they may be the cause of constriction, particularly when swelling occurs.
- *Shoulder.* This is immobilised by a sling.

Lower limb

- *Femur.* Femoral fractures should be treated in traction splints or skin traction with a Thomas splint. Ipsilateral femoral and tibial fractures can be immobilised in the same splint. Excess traction may cause perineal injury and neurovascular problems, and should be avoided.
- *Tibia and ankle.* Tibial and ankle fractures should be aligned and immobilised in padded box splints or an above/below-knee back slab depending on the position of the fracture. Foot perfusion should be assessed before and after application of the splint.

17.6 EXTREMITY TRAUMA: SUMMARY

- Extremity trauma is rarely life threatening *per se*, unless exsanguinating haemorrhage ensues. Multiple fractures can cause significant blood loss.
- The first priority is assessment of the airway, breathing and circulation.
- Full assessment of the extremities takes place during the secondary survey. Limb-threatening injuries should be identified at this stage and further investigation and management begun. Other injuries should be treated by splintage.

17.7 SPINAL TRAUMA: INTRODUCTION

Spinal injuries are rare in children, which does not mean that they are unimportant. For any mechanism of injury capable of causing cervical spine damage (or in cases with uncertain history), the cervical spine is presumed to be at risk. A high index of suspicion, correct management and prompt referral are necessary in order to prevent exacerbation of underlying cord injury. Every severely injured child should be treated as though he or she has spinal injury until adequate examination and investigation exclude it.

Immobilisation

If the child is unconscious, uncooperative or has had a significant mechanism of injury that makes it possible to have a spinal injury, the head and neck should be stabilised initially by manual immobilisation. A hard collar should be fitted and applied to all cooperative patients.

Some situations are particularly difficult. An injured child may be uncooperative for many reasons including fear, pain or hypoxia. Manual immobilisation should be maintained and the contributing factors addressed. Too rigid immobilisation of the head in such cases may increase leverage on the neck as the child struggles. The infant or baby who is too small for a hard collar should have manual immobilisation supported throughout.

Once a child has been immobilised, a member of staff must remain with the child at all times for reassurance, and to ensure there is minimal movement and that the airway remains patent. Immobilisation of the cervical spine can be very frightening and disorientating to a child and thus must be carried out supportively and sensitively with careful explanation appropriate to the child's age and cognitive level throughout the procedure.

Children being transported between institutions may require additional immobilisation. This may involve head blocks, sand bags or a vacuum mattress, where possible axial loading must be avoided. Spinal boards should only be used in the short term. Children who arrive in hospital on a spinal board must be removed from it as quickly as possible.

Guidelines for clinically clearing a cervical spine

- No midline cervical tenderness on direct palpation
- No focal neurological deficit
- Normal alertness
- No intoxication
- No painful distracting injuries

If guidelines for clinically clearing the cervical spine are met, indicating a low risk of cervical spine injury, the patient should be asked to rotate their neck 45° to the left and right. If any of these manoeuvres cause midline posterior pain the neck should be immobilised again and the spine imaged. If there is no pain on movement, immobilisation is no longer required. See in full in Figure 17.1 below.

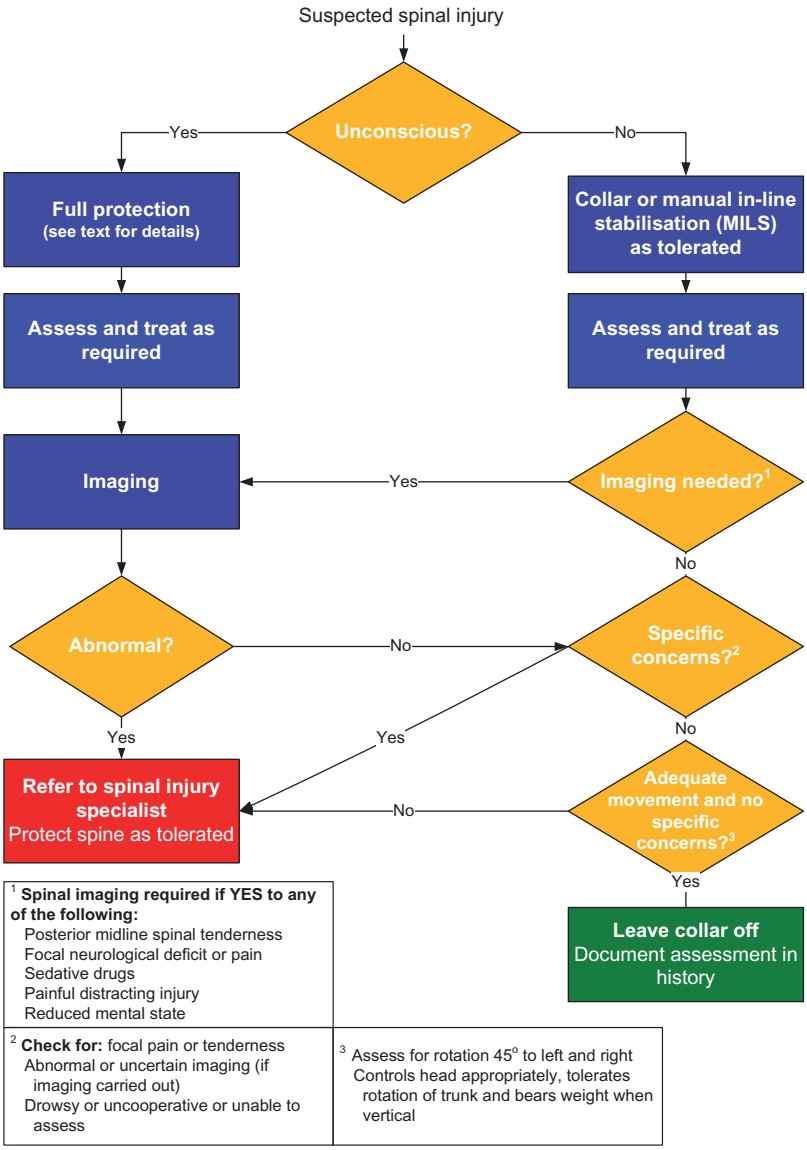

Figure 17.1 Algorithm for spinal imaging, referral and clearance. CT, computed tomography; GCS, Glasgow Coma Scale

17.8 INJURIES OF THE CERVICAL SPINE

Injuries to the cervical spine are rare in children; however they are associated with substantial levels of impact. The upper three vertebrae are usually involved – injury is more common in the lower segments of an adult. The low incidence (0–2% of all children's fractures and dislocations) of bony injury is explained by the mobility of the cervical spine in children, which dissipates applied forces over a greater number of segments.

Cervical spine imaging

Imaging must be taken in all children who cannot have their spine cleared clinically. Children with a Glasgow Coma Scale (GCS) score of 15 who need imaging and have no features suggestive of cord or nerve root injury can generally be imaged with *plain spinal radiographs* initially. These children should have a full cervical spine series including lateral, anteroposterior and odontoid peg views (the latter only in children over 5 years). Injury must be presumed until excluded radiologically and clinically. Spinal injury may be present even with a normal radiograph. The development of the cervical vertebrae is complex. There are numerous physeal lines (which can be confused with fractures), and a range of normal sites for ossification centres. Pseudosubluxation of C2 on C3 and of C3 on C4 occurs in approximately 9% of children; particularly those aged 1–7 years. Interpretation of cervical radiographs can therefore be difficult even for the most experienced.

Indirect evidence of trauma can be detected by assessing retropharyngeal swelling. At the inferior part of the body of C3, the pre-vertebral distance should be one-third the width of the body of C2. This distance varies during breathing and is increased in a crying child. Cervical spine X-rays are discussed in more detail in Chapter 23.

Some children will require further imaging and specialist consultation depending on their clinical and radiological features.

Proceed to a computed tomography (CT) scan if the plain views are abnormal or inadequate. Children with a GCS of <13 require a *CT scan of the entire cervical spine*, as well as a head CT scan. It is a matter of clinical judgement whether children with a GCS of 13–14 should have a CT scan of the upper or entire cervical spine, balancing the risk of missing an injury against unnecessary radiation to the thyroid and other tissues. Whether the rest of the spine needs CT scanning or plain views requires clinical judgement and depends on the overall clinical assessment.

After a high-energy mechanism of injury, if there is evidence of a serious trunk injury or cardiorespiratory instability, a *CT scan from the occiput to the pelvis* should be considered, irrespective of the conscious level. This will encompass the entire spine. Plain spinal films are not then required.

If there are features suggestive of cord or nerve root injury an *MRI scan* is indicated. The timing is a matter of clinical judgement by a spinal injury specialist.

Injury types

Atlantoaxial rotary subluxation is the most common injury to the cervical spine. The child presents with torticollis following trauma. Radiological demonstration of the injury is difficult, and CT or MRI may be necessary. Other injuries of C1 and C2 include odontoid epiphyseal separations and traumatic ligament disruption.

It should be noted that significant cervical cord injuries have been reported without any radiological evidence of trauma.

Immediate treatment

> Despite the rarity of fractures, a severely injured child's spine should be securely immobilised until spinal injury has been excluded. If in any doubt the child should continue to be immobilised and senior help sought.

Cervical spine immobilisation techniques are described in Chapter 22.

17.9 INJURIES OF THE THORACIC AND LUMBAR SPINE

Injuries to the thoracic and lumbar spine are rare in children. They are most common in the multiply injured child. In the second decade, 44% of reported injuries result from sporting and other recreational activity. Some spinal injuries may result from non-accidental injury.

When an injury does occur, it is not uncommon to find multiple levels of involvement because the force is dissipated over many segments in the child's mobile spine. This increased mobility may also lead to neurological involvement without significant skeletal injury.

The most common mechanism of injury is hyperflexion, and the most common radiographic finding is a wedge- or beak-shaped vertebra resulting from compression.

The most important clinical sign is a sensory level. Neurological assessment is difficult in children, and such a level may only become apparent after repeated examinations. Because of the difficulties of assessment, a child with multiple injuries should be assumed to have spinal injury, and should therefore remain immobilised. However, they should be log-rolled off the long spinal board onto a firm emergency department trolley. If injury is confirmed, further treatment is similar to that in adults. Unstable injuries may require open reduction and stabilisation with fusion.

If spinal cord damage has occurred the role of steroids remains controversial. Discuss the use with the local spinal injuries or trauma service. If it is to be used it should be commenced within 8 hours of the injury.

17.10 SPINAL CORD INJURY WITHOUT RADIOLOGICAL ABNORMALITY

Spinal cord injury without radiological abnormality (SCIWORA) is said to have occurred when the spinal cord has been injured without an obvious accompanying injury to the vertebral column. The cervical spine is affected more frequently than the thoracic spine. Because the upper segments of the cervical spine have the greatest mobility, the upper cervical cord is most susceptible to this injury.

Children who are seriously injured should have immobilisation of the spine maintained until such time as a full neurological assessment can be carried out, since normal X-rays do not exclude a cord injury. If there is any doubt, MRI scans should be obtained.

17.11 SPINAL IMAGING, REFERRAL AND CLEARANCE

The guidelines for clearing the spine are shown in Figure 17.1.

Spinal injury can often be excluded clinically without imaging or after normal imaging in alert, cooperative children, even if they have received morphine pain relief. If the initial imaging is normal but the child is too drowsy or uncooperative to make a safe assessment, the spine can still be cleared if an MRI scan has been indicated, performed and reported to be normal by a senior radiologist or spinal consultant.

In many centres, the spine may also be cleared by a normal high-quality CT scan with reconstruction views (on risk–benefit grounds) if it is predicted that the child will not be safely assessable within 24 hours. The small risk of missing a ligamentous injury is balanced against the risk of prolonged unnecessary immobilisation.

Otherwise, the spine remains uncleared and spinal precautions are maintained. If the child is to be left in a collar for more than 6 hours, the basic hard collar should be replaced by an appropriate two-piece collar (e.g. Aspen collar) that will be better tolerated.

In countries in which high-resolution scanners are not yet available, the spine may be cleared on the basis of plain imaging and clinical judgement by the attending specialist in spinal injury management.

17.12 SUMMARY: SPINAL TRAUMA

- Spinal injuries are rare in children but must be considered when associated with significant mechanism of injury.
- Assessment can be difficult and significant cord damage can occur without fractures.
- Spinal immobilisation must be applied until the assessment is complete and then can only be removed if the spine has been cleared.

CHAPTER 18

The burned or scalded child

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to use the structured approach to assess and manage the burned or scalded child

18.1 INTRODUCTION

Epidemiology

Each year some 37,000 burned or scalded children attend emergency departments. Of these, 4000–5000 require hospital admission. In England and Wales in 2007, eighteen children died from burns including 15 under the age of 5 years. Seventy per cent of those burnt are pre-school children, the most common age being between 1 and 2 years. Scalds occur mostly in the under 4-year-olds. Boys are more likely to suffer burns and serious scalds.

Most fatal burns occur in house fires and smoke inhalation is the usual cause of death. The number of deaths from burns has decreased because of a combination of factors. The move away from open fires, with safer fireguards, smoke alarms and more stringent low-flammability requirements for night clothes have all played a part. Non-fatal burns often involve clothing and are often associated with flammable liquids.

Scalds are usually caused by hot drinks, but bath water and cooking oil scalds are not uncommon. The improvement in survival following scalding (which followed improvements in treatment) has reached a plateau.

There is a strong link between burns to children and low socioeconomic status. Family stress, poor housing conditions and overcrowding are implicated in this.

Pathophysiology

Two main factors determine the severity of burns and scalds – these are the temperature and the duration of contact. The time taken for cellular destruction to occur decreases exponentially with temperature: at 44°C, contact would have to be maintained for 6 hours, at 54°C for 30 seconds, and at 70°C epidermal injury happens within a second. This relationship underlies the different patterns of injury seen with different types of burn. Scalds generally involve water at below boiling point and contact for less than 4 seconds. Scalds that occur with liquids at a higher temperature (such as hot fat or steam), or in children incapable of minimising the contact time (such as young infants and the handicapped), tend to result in more serious injuries. Flame burns involve high temperatures and consequently produce the most serious injuries of all.

It must be re-emphasised that the most common cause of death within the first hour following burn injuries is due to smoke inhalation. Smoke-filled rooms not only contain soot particles,

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hot gases and noxious substances but are also depleted of oxygen; inhalation of all or any of which can lead to cardiac arrest. Thus, as with other types of injury, attention to the airway and breathing is of prime importance.

18.2 PRIMARY SURVEY AND RESUSCITATION

When faced with a seriously burned child it is easy to focus on the immediate problems of the burn, and forget the possibility of other injuries. The approach to the burned child should be the structured one advocated in Chapter 13.

Airway and cervical spine

The airway may be compromised either because of inhalational injury and oral scalds or because of severe burns to the face. The latter are usually obvious, whereas the former two may only be indicated more subtly. The presence of inhalation injury is directly related to mortality – an observational study carried out in the USA found that there was a 15% higher mortality rate where inhalation injury was present. The indicators of inhalational injury are shown in the box below.

Indications of inhalational injury

- History of exposure to smoke in a confined space
- Deposits around the mouth and nose
- Carbonaceous sputum

Because oedema occurs following thermal injury, the airway can deteriorate rapidly. Thus even suspicion of airway compromise, or the discovery of injuries that might be expected to cause problems with the airway at a later stage, should lead to immediate consideration of tracheal intubation. This procedure increases in difficulty as oedema progresses; it is therefore important to perform it as soon as possible. All but the most experienced should seek expert help urgently, unless apnoea requires immediate intervention.

If there is any suspicion of cervical spine injury, or if the history is unobtainable, appropriate precautions should be taken until such injury is excluded.

Breathing

Once the airway has been secured, the adequacy of breathing should be assessed. Signs that should arouse suspicion of inadequacy include abnormal rate, abnormal chest movements and cyanosis (a late sign). Circumferential burns to the chest or abdomen (the latter in infants) may cause breathing difficulty by mechanically restricting chest movement.

All children who have suffered burns should be given high-flow oxygen. If there are signs of breathing problems, then intubation and ventilation should be commenced.

Circulation

In the first few hours following injury, signs of hypovolaemic shock are rarely attributable to burns. Therefore any such signs should raise the suspicion of bleeding from elsewhere, and the source should be actively sought. Intravenous access should be established with two cannulae during resuscitation, and fluids started. If possible, drips should be put up in unburnt areas, but burned skin, eschar, can be perforated if necessary. Remember that the intraosseous route can be used. Blood should be taken for haemoglobin, haematocrit, electrolytes and urea, blood glucose and cross-matching at this stage.

Disability

Reduced conscious level following burns may be due to hypoxia (remember smoke-filled rooms may contain little oxygen), head injury or hypovolaemia. It is essential that a quick assessment

is made during the primary survey as described in Chapter 13, because this provides a baseline for later observations.

Exposure

Exposure should be complete. Burned children lose heat especially rapidly, and should be kept in a warm environment and be covered with blankets when not being examined.

18.3 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES

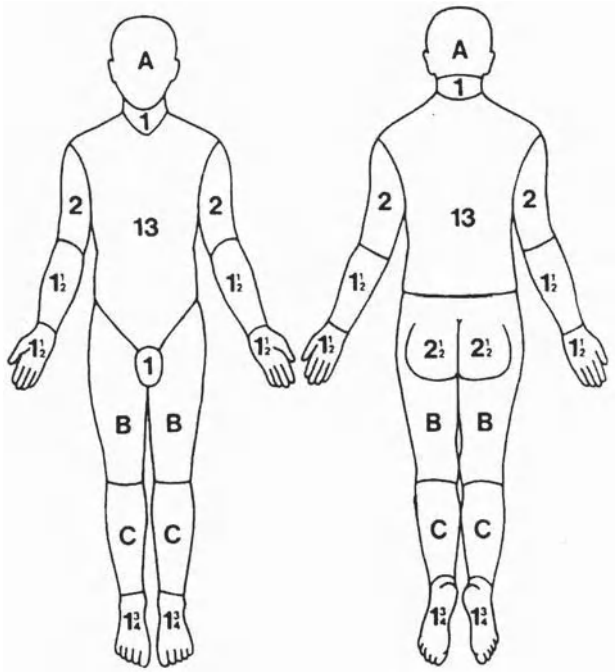
As well as being burned, children may suffer the effects of blast, may be injured by falling objects, or may fall while trying to escape from the fire. Thus other injuries are not uncommon and a thorough head to toe secondary survey must be carried out. This is described in Chapter 13. Any injuries discovered, including the burn, should be treated in order of priority.

Assessing the burn

The severity of a burn depends on its relative surface area and depth. Burns to particular areas require special attention.

Surface area

The surface area is usually estimated using burns charts. It is particularly important to use a paediatric chart when assessing burn size in children, because the relative surface areas of the head and limbs change with age. This variation is illustrated in Figure 18.1 and the table accompanying it.



Area indicated	Surface area at				
	0 year	1 year	5 years	10 years	15 years
A	9.5	8.5	6.5	5.5	4.5
B	2.75	3.25	4.0	4.5	4.5
C	2.5	2.5	2.75	3.0	3.25

Figure 18.1 Body surface area (percent). (Reproduced courtesy of Smith & Nephew Pharmaceuticals Ltd)

Another useful method of estimating relative surface area relies on the fact that the patient's palm and adducted fingers cover an area of approximately 1% of the body surface. This method can be used when charts are not immediately available, and is obviously already related to the child's size.

Note that the 'rule of 9s' cannot be applied to a child who is less than 14 years old.

Depth

Burns are classified as being superficial, partial thickness or full thickness. The first causes injury only to the epidermis, and clinically the skin appears red, with no blister formation. Partial-thickness burns cause some damage to the dermis; blistering is usually seen and the skin is pink or mottled. Deeper, full-thickness burns damage both the epidermis and dermis, and may cause injury to deeper structures as well. The skin looks white or charred, and is painless and leathery to touch.

Special areas

Burns to the face and mouth have already been dealt with above. Burns involving the hands or feet can cause severe functional loss if scarring occurs. Perineal burns are prone to infection and present particularly difficult management problems. Circumferential, full- or partial-thickness burns of the limbs or neck may require urgent incision to relieve distal ischaemia. Similarly circumferential burns to the torso may restrict ventilation and also require urgent incision. This procedure is called escharotomy and usually needs to be done before transfer to a burns centre.

18.4 EMERGENCY TREATMENT

Analgesia

Most burned children will be in severe pain, and this should be dealt with urgently. Some older children may manage to use Entonox, but most will not. Any child with burns that are anything other than minor should be given intravenous morphine at a dose of 100 micrograms/kg (<1 year: 80 micrograms/kg) as soon as possible. Further doses are often required but must be titrated against pain and sedation. There is no place for administration of intramuscular analgesia in burns because absorption is unreliable.

Fluid therapy

Two cannulae should already have been sited during the primary survey and resuscitation and therapy for shock (10+10 ml/kg) commenced if indicated. Children with burns of 10% or more will require intravenous fluids as part of their burns care. This fluid is in addition to their normal fluid requirement. The *additional* fluid (in ml) required per day to treat the burn can be estimated using the following formula:

Percentage burn × Weight (kg) × 4

Half of this should be given in the first 8 hours following the time of their burn. The fluid given is usually crystalloid. Remember that this is only an initial guide; subsequent therapy will be guided by urine output, which should be kept at 2 ml/kg/h or more. Urethral catheterisation should therefore be performed as soon as is practicable.

Wound care

Infection is a significant cause of mortality and morbidity in burns victims, and wound care should start as early as possible to reduce this risk. Furthermore, appropriate wound care will reduce the pain associated with air passing over burnt areas.

Burns should be covered with sterile towels, and unnecessary re-examination should be avoided. Blisters should be left intact. Although cold compresses and irrigation with cold

water may reduce pain, it should be remembered that burned children lose heat rapidly. These treatments should only be used for 10 minutes or less, and only in patients with superficial or partial-thickness burns totalling less than 10%. Children should *never* be transferred with cold soaks in place. Cling film is often used as a sterile dressing and can be applied *loosely* onto the burned area. No additional ointments or creams should be applied.

Management of carbon monoxide poisoning

During a fire, burning of organic compounds in a low-oxygen environment produces carbon monoxide. Inhalation by the victim induces the production of carboxyhaemoglobin, which has a 200-fold greater affinity for the oxygen molecule than haemoglobin. A high level will therefore cause cellular hypoxia as oxygen will not be given up to cells. Children who have been in house fires should have their blood carboxyhaemoglobin measured. (Note: most pulse oximeters show the oxygen saturation, regardless of haemoglobin concentration, i.e. normal Spo_2 does not exclude carbon monoxide poisoning).

Levels of 5–20% are treated with oxygen (which speeds up the removal of CO). Levels over 20% should prompt consideration of hyperbaric oxygen chamber treatment – discuss with the paediatric burns unit. In some environments the burning of plastics, wool and silk can produce cyanide. Assessment and treatment are complex. Be aware of the possibility of cyanide poisoning and consider it in a child from a house fire who is in a coma without apparent cause. In general, antidotes are used when blood levels of cyanide are greater than 3 mg/l. Discuss treatment with a poisons centre as other factors such as the concomitant presence of carboxyhaemoglobin are contraindications for some antidotes.

18.5 CONTINUING STABILISATION AND TRANSFER TO DEFINITIVE CARE

Definitive care requires transfer to a paediatric burns facility. Criteria for transfer are shown in the box below.

Criteria for transfer to a burns unit

- 10% partial- and/or full-thickness burns
- 5% full-thickness burns
- Burns to special areas: face, hands, feet or perineum
- Any circumferential burn
- Significant inhalational burn (excluding pure carbon monoxide poisoning)
- Chemical, radiation or high-voltage electrical burns

If in doubt discuss the child with the paediatric burns unit.

As with any injury in childhood, consider the possibility of child abuse. Note the timeliness of presentation, and assess whether the history given to account for the burn or scald fits in with the clinical appearance of the injury in size, shape, age and location. Consider whether the injury is consistent with the child's developmental attainments. If concerned or in doubt, consult with a child protection specialist.

18.6 SUMMARY

- Initial assessment and management of the burned child should be directed towards care of the airway, breathing and circulation. Intubation and ventilation should be performed early if indicated.

- Assessment of the area and depth of the burn should be undertaken during the secondary survey.
- Fluid replacement should be used initially to treat shock. Additional maintenance fluids will be needed for burns. Urine output should be used as an indicator of the efficacy of treatment.
- Specialist burns centres should be contacted, and transfer arranged if indicated.

CHAPTER 19

The child with an electrical injury or drowning

LEARNING OBJECTIVES
In this chapter, you will learn:
- How to use the structured approach to assess and manage the child with an electrical injury or drowning

19.1 ELECTRICAL INJURIES: INTRODUCTION

Epidemiology

Many minor electrical injuries do not require medical treatment and the instance of this sort of injury is unknown. Only a small percentage of electrical injuries requiring hospital attention occur in children. Electrical injuries usually occur in the home and involve relatively low currents and voltage. The mortality from electrical injuries from high power external sources such as electrified railways is high.

Other injuries may occur during the event: for example, the child may fall or be thrown from the source. As with all injuries, a systematic approach is required.

Pathophysiology

Alternating current (AC) produces cardiac arrest at lower voltages than does direct current (DC). Regardless of whether the electrocution is caused by AC or DC, the risk of cardiac arrest is related to the size of the current and the duration of exposure. The current is highest when the resistance is low and the voltage is high.

Current

A lightning strike is a massive direct current of very short duration which can depolarise the myocardium and cause an immediate asystole.

The typical *effects of an increase in current* are given in the following list:

- *Above 10 mA:* tetanic contractions of muscles may make it impossible for the child to let go of the electrical source.
- *50 mA:* tetanic contraction of the diaphragm and intercostal muscles leads to respiratory arrest, which continues until the current is disconnected. If hypoxia is prolonged, secondary cardiac arrest will occur.
- *Over 100 mA to 50 A:* primary cardiac arrest may be induced (defibrillators used in resuscitation deliver around 10 A).
- *50 A to several 100 A:* massive shocks cause prolonged respiratory and cardiac arrest and more severe burns.

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Resistance

The resistance of the tissues determines the path that the current will follow. Generally the current will follow the path of least resistance from the point of contact to earth. The relative resistance of the body tissues is, in increasing order: tissue fluid, blood, muscle, nerve, fat, skin, bone. Electrocution generates heat, which causes a variable degree of tissue damage. Nerves, blood vessels, the skin and muscles are damaged most. Swelling of damaged tissues, particularly muscle, can lead to a crush or compartment syndrome requiring fasciotomy. Water decreases the resistance of the skin and will increase the amount of current that flows through the body.

Voltage

High-voltage sources such as lightning or high-tension cables cause extremely high currents and severe tissue damage. However, very high voltages can cause severe superficial burns without damage to deeper structures (flash burns and arcing).

19.2 INITIAL TREATMENT OF ELECTRICAL INJURIES

The first priority is to disconnect the current. Be aware that high-voltage sources can discharge through several centimetres of air.

19.3 PRIMARY SURVEY OF ELECTRICAL INJURIES AND RESUSCITATION

The upper airway should be opened and secured, especially if this is compromised by facial or other injuries. The cervical spine should be immobilised especially in an unconscious child (see Chapter 13). Other injuries should be treated in an appropriate and structured manner.

The entry and exit point of the current should be sought in order to determine the sort of possible internal injuries that could have occurred.

19.4 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES OF ELECTRICAL INJURIES

Associated injuries are common in electrocution. Almost all possible injuries can occur as a result of falls or being thrown from the source. Burns are particularly common and are caused either by the current itself or by burning clothing. Tetanic contraction of muscles can cause fractures, luxations or muscle tearing.

Associated problems

Burns cause oedema and fluid loss. Myoglobinuria occurs after significant muscle damage. In this case it is important to maintain a urine production of more than 2 ml/kg/h with the judicious use of diuretics such as mannitol and appropriate fluid loading. Alkalisation of the urine with sodium bicarbonate increases the excretion of myoglobin.

Dysrhythmias can occur up to a considerable time after the electrocution, and continuous electrocardiogram (ECG) monitoring is essential. Children with significant internal injuries have a greater fluid requirement than one would suspect on the basis of the area of the burn.

19.5 STABILISATION OF ELECTRICAL INJURIES AND TRANSFER TO DEFINITIVE CARE

A significant electrical burn is an indication for transfer to a burns centre.

19.6 ELECTRICAL INJURIES: SUMMARY

- The first priority in electrical injuries is to switch off the current.
- The management of electrocution should be structured according to the ABCDE principle, including the appropriate treatment of a possible cervical spine injury.
- Almost all injuries can be associated with electrocution.
- The entry and exit wounds should be sought in order to form a picture of the possible internal injuries.
- Dysrhythmias can occur some considerable time following the electrocution.
- Myoglobinuria should be treated aggressively.

19.7 DROWNING: INTRODUCTION

The International Liaison Committee on Resuscitation (ILCOR) defines drowning as 'a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium'. The term 'near drowning' and 'wet' or 'dry' drowning are no longer official terms, mainly because they have been used differently worldwide, which has caused confusion.

Epidemiology

According to the annual World Health Organisation (WHO) report, some 450,000 die annually as a result of drowning worldwide. For children under the age of 15 years drowning is the leading cause for accidental death in the world. The age group from 0 to 4 years has a mortality rate of 18.9 per 100,000, the older age group up to 15 years has an incidence of 9.5 deaths per 100,000. Male victims are more likely to die from drowning than female victims. Infants die most commonly in bathtubs, older children die in private swimming pools, garden ponds and other inland waterways. It is estimated that up to 80% of drowning incidents are preventable. Prevention strategies like fencing of private pools and reinforcing the importance of adult supervision may reduce this number. Death from drowning is the third most common cause of accidental death in children in the UK (after road accidents and burns).

Pathophysiology

Bradycardia and apnoea occur shortly after submersion as a result of the diving reflex. As apnoea continues, hypoxia and acidosis causes tachycardia and a rise in blood pressure. Between 20 seconds and 5 minutes later a breakpoint is reached, and breathing occurs. Fluid is inhaled and on touching the glottis causes immediate laryngeal spasm. After a variable but short period of time, the laryngospasm subsides and fluid is aspirated into the lungs, resulting in alveolitis and pulmonary oedema. Hypoxia is by this time severe and the patient will have lost consciousness. Bradycardia and other dysrhythmias can also occur and may be fatal (ventricular fibrillation is rare). Hypoxia is thus the key pathological process which ultimately leads to death and needs to be corrected as quickly as possible.

Children who survive because of interruption of this chain of events not only require therapy for drowning, but also assessment and treatment of concomitant hypothermia, hypovolaemia and injury (particularly spinal). Major electrolyte abnormalities due to the amount of water swallowed seldom occur.

The type of water – freshwater or saltwater – does not predict the clinical course of drowning and should not influence treatment. However, immersion in severely contaminated water is associated with infections with unusual organisms, and aspiration of water contaminated with petroleum products can lead to severe acute respiratory distress syndrome (ARDS).

Submersion injuries are generally associated with hypothermia. The large body surface area to weight ratio in infants and children put them at particular risk. Hypothermia may have a protective effect against the neurological sequelae following hypoxia and ischaemia but is also associated with life-threatening dysrhythmias, coagulation disorders and susceptibility to infections.

The initial approach to drowning patients focuses on the correction of hypoxia, hypothermia and the treatment of associated injuries, which are common in older children and often overlooked. Cervical spine injury should always be suspected in drowning victims for whom the mechanism of injury is unclear, although these are rare (0.5% overall and much rarer in children under 5 years).

19.8 PRIMARY SURVEY OF DROWNING AND RESUSCITATION

The first priority is to move the victim from the water as quickly as possible without risk to the rescuer in order to allow CPR and ABC stabilisation without delay. Immobilisation of the neck should be instigated as soon as practicable until injury is excluded, although cervical spine injury is uncommon except after diving or traffic accidents. Rescue of the victim in a vertical position may lead to cardiovascular collapse due to venous pooling. However, horizontal rescue or cervical spine immobilisation in the water must not be allowed to delay the rescue. The initiation of early and effective basic life support (BLS) reduces the mortality drastically and is the most important factor for survival. Rescue breaths must be commenced as early as possible even in shallow water if this can be done without risk to the rescuer. Mouth-to-nose ventilation may be easier in this situation. BLS then proceeds according to the standard paediatric algorithm even in hypothermia. The presence of cardiac arrest can be difficult to diagnose as pulses are difficult to feel. If in doubt chest compressions should be given and continued. If an automatic external defibrillator (AED) is used it is vital to first dry the chest before applying the electrodes.

Following a submersion episode, the stomach is usually full of swallowed water. The risk of aspiration is therefore increased and the airway must be secured as soon as possible, usually by endotracheal intubation using a rapid sequence induction. Following this an oro- or nasogastric tube should be inserted. Ventilate the child to achieve an SpO_2 of 94–98% using additional oxygen and PEEP (positive end-expiratory pressure) as required. Respiratory deterioration can be delayed for 4–6 hours after submersion and even children who have initially apparently recovered should be observed for at least 8 hours. Chest X-ray changes may occur even later. Advanced life support proceeds according to the standard algorithm except for slight modifications in cases of hypothermia.

Hypothermia

A core temperature reading (rectal or oesophageal) should be obtained as soon as possible and further cooling prevented. The advantages of endotracheal intubation in hypothermia outweigh the small risk of precipitating malignant arrhythmias. Hypothermia is common following drowning, and adversely affects resuscitation attempts unless treated. Not only are arrhythmias more common but some, such as ventricular fibrillation, may be refractory at temperatures below 30°C when defibrillation should be limited to three shocks and inotropic or antiarrhythmic drugs should not be given. If unsuccessful the patient should be warmed to above 30°C as quickly as possible, when further defibrillation may be attempted. The dose interval for resuscitation drugs is doubled between 30°C and 35°C. Resuscitation should be continued until the core temperature is at least 32°C or cannot be raised despite active measures.

Rewarming strategies (see box on next page) depend on the core temperature and signs of circulation. External rewarming including a warm air system is usually sufficient if the core temperature is above 30°C. Active core rewarming should be added in patients with a core temperature of less than 30°C. Extracorporeal warming is the preferred method in circulatory arrest.

Rewarming

External rewarming

- Remove cold, wet clothing
- Supply warm blankets
- Warm air system
- Heating blanket
- Infrared radiant lamp

Core rewarming

- Warm intravenous fluids to 39°C to prevent further heat loss
- Warm ventilator gases to 42°C to prevent further heat loss
- Gastric or bladder lavage with normal (physiological) saline at 42°C
- Peritoneal lavage with potassium-free dialysate at 42°C, 20 ml/kg with a 15 minute cycle
- Pleural or pericardial lavage
- Endovascular warming
- Extracorporeal blood rewarming

The temperature is generally allowed to rise by 0.25–0.5°C per hour to reduce haemodynamic instability. Most hypothermic patients are hypovolaemic. During rewarming vasodilatation occurs, resulting in hypotension requiring large quantities of warmed intravenous fluids while avoiding overfilling and pulmonary oedema. Continuous haemodynamic monitoring is essential. Therapeutic hypothermia (32–34°C) for at least 24 hours has been shown to improve neurological outcome in some patients and may be of benefit in children who remain comatose.

19.9 SECONDARY SURVEY AND LOOKING FOR KEY FEATURES IN DROWNING

During the secondary survey, the child should be carefully examined from head to toe. Any injury may have occurred during the incident that preceded immersion including spinal injuries. Older children may have ingested alcohol and/or drugs.

Investigations

- Blood glucose.
- Blood gas analysis (preferably arterial) and blood lactate.
- Urea and electrolytes.
- Coagulation status
- Blood and sputum cultures.
- Chest X-ray.
- Electrocardiogram.
- Lateral cervical spine X-ray or computed tomography (CT) scan.

19.10 EMERGENCY TREATMENT IN DROWNING AND STABILISATION

The brain is the most vulnerable organ for asphyxia, and cerebral impairment occurs before cardiac problems in submersion. Except for early basic life support and possibly therapeutic hypothermia, there are few effective measures for reducing brain damage in drowning.

It is essential to monitor the vital functions closely, especially during the first couple of hours. An early suggestion of respiratory insufficiency, haemodynamic instability or hypothermia are indications for admission to the intensive care unit.

Prophylactic antibiotics have not been shown to be helpful but are often given after immersion in severely contaminated water. Fever is common during the first 24 hours but is not

necessarily a sign of infection, which usually becomes manifest later. Gram-negative organisms, especially *Pseudomonas aeruginosa*, are common and *Aspergillus* species have been reported. When an infection is suspected broad-spectrum intravenous antibiotic therapy (such as cefotaxime) should be started after repeating blood and sputum cultures.

Signs of raised intracranial pressure (ICP) may develop, as a result of a post-hypoxic injury, and this should be treated, although aggressive treatment to lower a raised ICP has not been shown to improve the prognosis. Other therapeutic measures, such as barbiturates, calcium channel blockers, surfactants, steroids and free-radical scavengers, have not been shown to be of benefit. However, keeping the patient normoglycaemic is important for the neurological outcome. Unless obvious, a careful search should be made for a precipitating cause of the drowning such as a channelopathy, particularly long QT-syndrome.

19.11 PROGNOSTIC INDICATORS IN DROWNING

The clinical course of drowning is determined by the duration of hypoxic–ischaemic injury and the adequacy of initial resuscitation. It is assumed that hypoxic brain damage is reduced when the brain cools before the heart stops. No single factor can predict good or poor outcome in drowning reliably; however, the following factors may give an indication of outcome.

Immersion time Most children who have been submerged for more than 10 minutes have a very small chance of intact neurological recovery or survival. Details of the incident are therefore vital.

Time to basic life support Starting basic life support at the scene greatly reduces mortality, whereas a delay of more than 10 minutes is associated with a poor prognosis.

Time to first respiratory effort If this occurs within 3 minutes after the start of basic cardiopulmonary support, the prognosis is good. If there has been no respiratory effort after 40 minutes of full cardiopulmonary resuscitation, there is little or no chance of survival unless the child's respiration has been depressed (e.g. by hypothermia, medication or alcohol).

Core temperature Pre-existing hypothermia and rapid cooling after submersion also seems to protect vital organs and can improve the prognosis. A core temperature of less than 33°C on arrival and a water temperature of less than 10°C have been associated with increased survival. This effect is more pronounced in small children because of their large surface area to weight ratio.

Persisting coma A persistent Glasgow Coma Scale (GCS) score of less than 5 indicates a bad prognosis.

Arterial blood pH If this remains below 7.1 despite treatment, the prognosis is poor.

Arterial blood P_{O_2} If this remains below 8.0 kPa (60 mmHg) despite treatment, the prognosis is poor.

Type of water Whether the water was salt or fresh has no bearing on the prognosis.

The duration of resuscitation efforts may not be a helpful prognostic factor. The decision to discontinue resuscitation attempts is particularly difficult in cases of drowning, and should be taken only after all the prognostic factors discussed above have been considered carefully. Resuscitation should only be discontinued out of hospital if there is clear evidence of futility such as massive trauma or rigor mortis.

19.12 OUTCOME OF DROWNING

Seventy percent of children survive drowning when basic life support is provided at the scene whereas only 40% survive without early basic life support, even with maximum therapy.

Of those who do survive, having required full cardiopulmonary resuscitation in hospital, around 70% will make a complete recovery and 25% will have a mild neurological deficit. The remainder will be severely disabled or remain in a persisting vegetative state.

19.13 DROWNING: SUMMARY

- Starting BLS as soon as possible is crucial to the outcome after drowning.
- Many associated injuries and underlying illnesses may be associated with submersion.
- If cervical injury cannot be excluded, stabilisation should be initiated as soon as practical but should not delay rescue and the start of BLS.
- Hypothermia should be actively sought and treated.
- Prolonged resuscitation may be necessary and the decision to stop resuscitation should be taken after all prognostic indicators have been considered.

PART 5

Practical application of APLS

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CHAPTER 20

Practical procedures: airway and breathing

In this chapter, the following procedures are explained:
- Oropharyngeal airway insertion for the:
  - Non-expert practitioner
  - Expert practitioner
- Nasopharyngeal airway insertion
- Orotracheal intubation including rapid sequence induction in the:
  - Infant/small child
  - Older child
- Laryngeal mask airway insertion
- Surgical airway procedures:
  - Needle cricothyroidotomy
  - Surgical cricothyroidotomy
- Ventilation without intubation:
  - Mouth-to-mask ventilation
  - Bag-and-mask ventilation
- Management of a blocked tracheostomy

20.1 OROPHARYNGEAL AIRWAY INSERTION

If gag reflex is present, it may be best to avoid the use of an oropharyngeal tube or other artificial airway, because it may cause choking, laryngospasm or vomiting.

Non-expert practitioner – all children

1 Select an appropriately sized Guedel airway (see Chapter 5).
2 Open the airway using a chin lift, taking care not to move the neck if trauma has occurred.
3 Use a laryngoscope blade or a tongue depressor to aid insertion of the airway 'the right way up'. This also provides an opportunity to examine the oropharynx for foreign material.
4 Recheck airway patency and look for improvement.
5 If necessary, consider a different size airway from the original estimate.
6 Provide oxygen; consider ventilation by pocket mask or bag-and-mask.

Expert practitioner – all children

1 Select an appropriately sized Guedel airway (see Chapter 5).
2 This can be inserted by either of the following:

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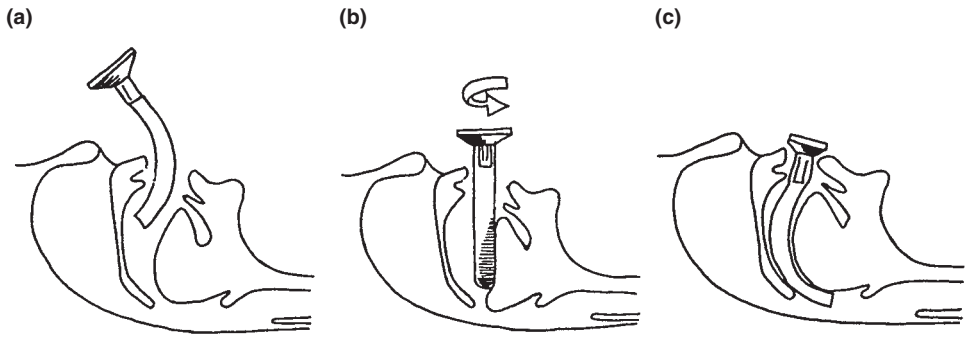


Figure 20.1 (a–c) Airway insertion in an older child

- Using the technique as described for the non-expert practitioner.
- Inserting the airway upside down until the tip has passed the soft palate and then rotating it 180° so that the natural curve of the Guedel airway follows the curve of the tongue and pharynx (Figure 20.1).

3 Recheck airway patency, looking for improvement following insertion.
4 Be prepared to change to a different size.
5 Provide oxygen and consider augmenting ventilation using either a pocket mask or a bag–valve–mask device.

20.2 NASOPHARYNGEAL AIRWAY INSERTION

Assess for any contraindications such as a base of skull fracture.

1 Select an appropriate size (length and diameter) of airway (see Chapter 5).
2 Lubricate the airway with a water-soluble lubricant and insert a large safety pin through the flange.
3 Insert the tip into the nostril and direct it posteriorly along the floor of the nose (rather than upwards).
4 Gently pass the airway past the turbinates with a slight rotating motion. As the tip advances into the pharynx, there should be a palpable 'give'.
5 Continue until the flange and safety pin rest on the nostril.
6 If there is difficulty inserting the airway, consider using the other nostril or a smaller size from the original estimate.
7 Recheck airway patency.
8 Finally, provide oxygen, considering ventilation by pocket mask or bag-and-mask.

20.3 OROTRACHEAL INTUBATION (INCLUDING RAPID SEQUENCE INDUCTION)

As resuscitation and stabilisation improve due to application of the advanced paediatric life support (APLS) principles, fewer children will require intubation when they are in extremis. Conversely, more will require intubation as a planned urgent rather than emergent procedure. This will often be outside the first hour during stabilisation or prior to transfer to definitive care.

Intubation may need to be carried out in the referring hospital prior to the arrival of a retrieval team and will require anaesthesia/sedation and muscle relaxation if the child is conscious. The technique used to secure the airway rapidly and safely in this situation is known as rapid sequence induction (RSI).

Rapid sequence induction

This has largely been the province of anaesthetists, for whom it is a core skill. However, it may also fall within the remit of paediatricians and emergency medicine specialists.

Crucially, the person carrying out the procedure should be a confident, competent and experienced intubator. They should have at least one and ideally two skilled assistants and all equipment available for intubation. In particular they should have the patient on a tilting trolley or bed and have available a powerful, high-volume sucker. They must have a plan of action for a failed intubation. It is also appropriate for those clinicians who will not have the skills to perform RSI to learn about the technique so that they can provide support for those who undertake it.

The steps in rapid sequence induction are as follow:

1. Prepare as above.
2. Pre-oxygenate with 100% oxygen for at least 3 minutes.
3. Induce anaesthesia using a sedative or anaesthetic induction agent (see 'Drugs' below).
4. Apply cricoid pressure via your skilled assistant. Cricoid pressure causes compression of the oesophagus against the vertebral body behind, thereby preventing passive regurgitation of gastric contents. It significantly enhances the safety of the intubation, but needs to be applied carefully and skilfully by an assistant specifically trained in the technique. It should be noted that in the trauma patient at risk of cervical spine injury, cricoid pressure should be applied with a two-handed technique, with the second hand *behind* the neck to provide additional stabilisation (in addition to the assistant who is stabilising the cervical spine by manual in-line immobilisation). The safety and value of cricoid pressure during emergency intubation is not clear. Therefore the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.
5. Administer a rapid-acting, short-lasting muscle relaxant for example suxamethonium.
6. Check that intubation is successful in the usual way.

Drugs

It must be stressed that *all* sedative and anaesthetic drugs may cause cardiovascular collapse in the ill, particularly hypovolaemic, child.

Drugs to support the failing circulation should be immediately to hand and the sedative/anaesthetic drugs should be given with extreme caution, often in very low doses.

Anaesthetic induction agents which may be used are listed in Table 20.1 (over page).

Technique of tracheal intubation: for immediate intubation in the apnoeic child or following RSI in the planned intubation

Infant or small child

1. Ensure that adequate ventilation and oxygenation by face mask are in progress. It is much less worrying for you and much safer for the child if adequate pre-oxygenation has been carried out. However, it should be realised that as one of the indications for intubation is failure to ensure adequate patency by any other means, this may not always be possible.
2. Prepare and check equipment (before inducing RSI if used under these circumstances).
3. Ensure manual immobilisation of the neck by an assistant if cervical spine injury is possible. Because of the relatively large occiput, it may be helpful to place a folded sheet or towel under the baby's back and neck to allow extension of the head.
4. The laryngoscope should be held in the left hand and inserted initially into the right-hand side of the mouth, thereby displacing the tongue to the left. Lift the epiglottis forward. The vocal cords should be sought in the midline directly underneath. It is easy to obscure the view by either looking too far to the left or too far to the right into either the piriform fossa or by inserting the blade too far past the larynx and down into the oesophagus. In circumstance where the laryngoscope blade has been inserted too far into the

Table 20.1 Anaesthetic induction agents for use in orotracheal intubation

Drug	Dose	Important notes
Ketamine	1–2 mg/kg	Potent analgesia, dissociative anaesthesia and less hypotensive. Causes intracranial pressure (ICP) to rise and should not be used in cases of head injury or potentially high ICP
Etomidate	100–400 micrograms/kg	Less hypotensive
Propofol	Dose in healthy child around 5 mg/kg, but 1–2 mg may suffice in a sick child	Most commonly used induction agent, therefore familiar. Very good intubating conditions, but much less cardiovascular system stability
Thiopental (thiopentone)	Dose in healthy child at least 5 mg/kg, but 1–2 mg may suffice in a sick child	Historically the agent of choice. Very smooth onset, but may induce marked cardiovascular impairment
Sedative drugs	Again, much lower doses may be needed in a sick child	Benzodiazepines: midazolam is the most commonly used drug in this group Opioid drugs: fentanyl and morphine are commonly used

oesophagus, if the blade is cautiously and slowly withdrawn the vocal cords may suddenly pop into view.

In an unconscious baby being intubated by a relatively inexperienced doctor, it is often easiest to place the laryngoscope blade well beyond the epiglottis. The laryngoscope blade is placed down the right side of the tongue into the proximal oesophagus. With a careful lifting movement, the tissues are gently tented up to 'seek the midline'. The blade is then slowly withdrawn until the vocal cords come into view. In some situations, it may be better to stay proximal to the epiglottis to minimise the risk of laryngospasm. This decision must be based on clinical judgement.

5 The tube should then be inserted through the cords, with due attention to the fact that in small children the trachea is very short. Whilst it is important not to insert the tube too far, thereby avoiding inadvertent bronchial intubation, it is much, much more dangerous to have a tube which is too short as this may be displaced any time by movement of the child's head.

6 Following intubation, placement of the tube should be confirmed by both inspecting the chest for equal bilateral movement and by auscultating the chest. It is also worth listening over the epigastrium for the *absence* of classic borborygmi following oesophageal intubation.

7 If intubation is not achieved within 30 seconds, discontinue the attempt, re-establish pre-oxygenation and try again.

8 The definitive test for successful placement is the presence of expired CO_2 in the exhaled air. This can be tested by either chemical colour change devices or, even better, by definitive end-tidal CO_2 measurement. This is recommended for secondary confirmation in neonates. A large randomised trial is underway.

9 Inflate the cuff if present, to provide an adequate seal. Note, however, that cuffed tubes should only be used in infants and small children by those who are trained and experienced in their use.
10 Once the tube is inserted and fixed into place, arrangements should be made to obtain a chest X-ray to confirm correct tube length. The end of the tube as seen on X-ray should be below the level of the vocal cords, but above the carina.

Older child

1 Ensure that adequate ventilation and oxygenation by face mask are in progress.
2 Prepare and check equipment (before inducing RSI if used under these circumstances).
3 Select an appropriate tube size, but prepare a range of sizes, including the size above and below the best estimate (see Chapter 5).
4 Ensure that manual immobilisation of the neck in cervical spine injury by an assistant is possible.
5 Hold the laryngoscope in the left hand and insert it into the right-hand side of the mouth, displacing the tongue to the left.
6 Visualise the epiglottis and place the tip of the laryngoscope anterior to it in the vallecula. The epiglottis is then pulled forwards by anterior pressure in the vallecula as demonstrated in Figure 20.2.
7 Gently but firmly lift the handle towards the ceiling on the far side of the room, while being careful not to lever on the teeth (Figure 20.3, over page).
8 Insert the endotracheal tube into the trachea, concentrating on how far the tip is being placed below the vocal cords. The tip should lie at least 2 cm below the vocal cords, depending on age. If the tube has a 'vocal cord level' marker, place this at the vocal cords. Be aware that flexion or extension of the neck may cause migration downwards or upwards, respectively.
9 Inflate the cuff if present, to provide an adequate seal.
10 Check the placement of the tube by inspecting the chest for movement and auscultating the chest (including the axillae) and epigastrium.
11 If endotracheal intubation is not achieved in 30 seconds, discontinue the attempt, ventilate and oxygenate by mask and try again.
12 Monitor expired carbon dioxide in the exhaled air by either colour change capnometry or end-tidal capnography.
13 Once the tube is in place, obtain a chest X-ray to confirm correct placement.

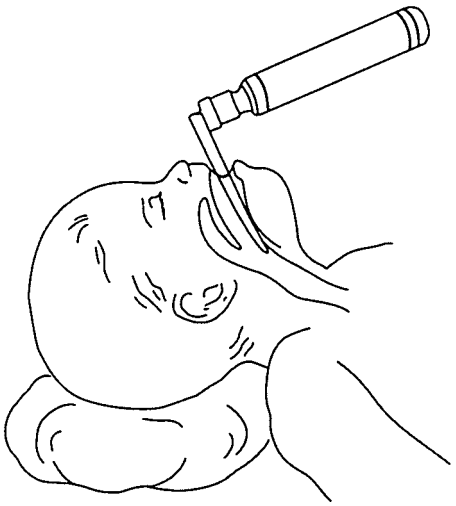


Figure 20.2 Technique 1: using the straight-bladed laryngoscope

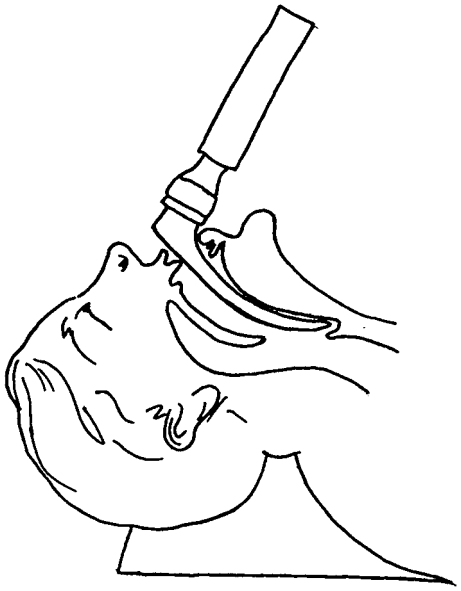


Figure 20.3 Technique 2: using the curved-bladed laryngoscope

Complications of endotracheal intubation

These include:

- Oesophageal intubation is the most dangerous complication of attempted intubation. It can cause severe hypoxia if not immediately recognised and is particularly dangerous when it occurs secondary to tube misplacement.
- Endobronchial intubation, resulting in lung collapse and/or risk of pneumothorax.
- Subglottic oedema or stenosis.
- Oesophageal perforation.
- Infection.

Exacerbation of an existing cervical spine injury leading to neurological deterioration should be preventable with a high index of suspicion and the use of trained assistant to provide immobilisation during airway manipulation.

20.4 INSERTION OF THE LARYNGEAL MASK AIRWAY

Procedure

1. Whenever possible, ventilate the patient with 100% oxygen using a bag–valve–mask device before inserting the LMA. During this time, check that the LMA and cuff are working with a syringe and have lubricant and suction to hand.
2. Deflate the cuff and lightly lubricate the back and sides of the mask.
3. Tilt the patient's head (if safe to do so), open the mouth fully, and insert the tip of the mask along the hard palate with the open side facing, but not touching the tongue (Figure 20.4a).
4. Insert the mask further, along the posterior pharyngeal wall, with your index finger initially providing support for the tube (Figure 20.4b). Eventually resistance is felt as the tip of the LMA lies at the upper end of the oesophagus (Figure 20.4c).
5. Fully inflate the cuff. The LMA will rise up slightly at this point.
6. Secure the LMA with adhesive tape and check its position during ventilation as for a tracheal tube. You will *not* usually obtain a completely gas-tight fit.
7. If insertion is not accomplished in less than 30 seconds, re-establish ventilation using a bag–valve–mask.

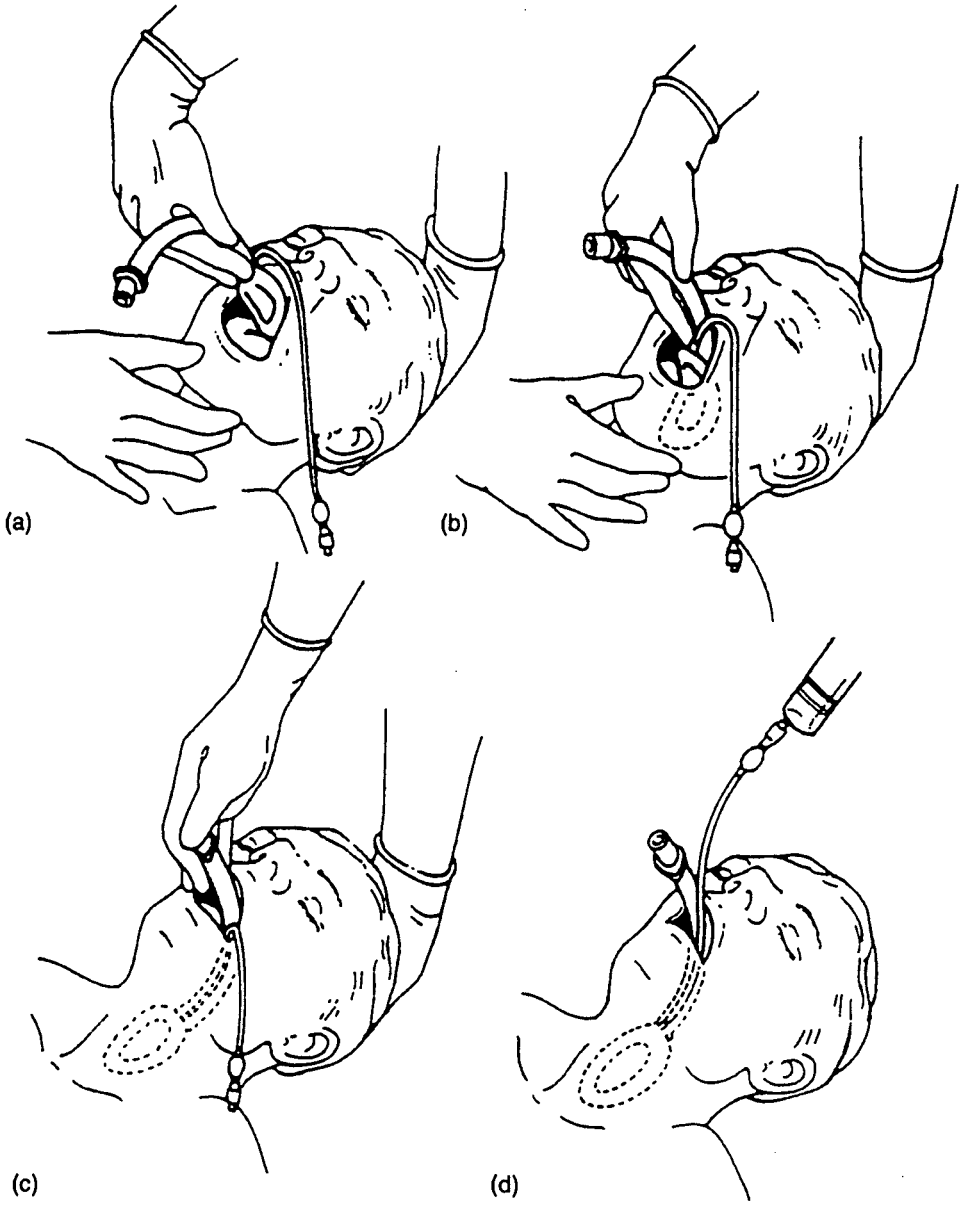


Figure 20.4 (a–d) Insertion of the laryngeal mask airway

Complications

- Incorrect placement is usually due to the tip of the cuff folding over during insertion. The LMA should be withdrawn and reinserted.
- Inability to ventilate the patient, because the epiglottis has been displaced over the larynx. Withdraw the LMA and reinsert ensuring that it closely follows the hard palate. This may be facilitated by the operator or an assistant lifting the jaw upwards. Occasionally rotation of the LMA may prevent its insertion. Check that the line along the tube is aligned with the patient's nasal septum; if not, reinsert.
- Coughing or laryngeal spasm is usually due to attempts to insert the LMA into a patient whose laryngeal reflexes are still present.
- Inadvertant displacement. This is very common with smaller LMAs, usually due to rotation once a circuit or self- inflating bag is attached.

20.5 SURGICAL AIRWAY

Cricothyroidotomy is a 'technique of failure'. It is indicated if a patent airway cannot be achieved by other means. It must be performed promptly and decisively when necessary. In children under the age of 12 years, needle cricothyroidotomy is preferred to surgical cricothyroidotomy. In the adolescent either technique can be used but the surgical technique allows better protection of the airway. The relevant anatomy is shown in Figure 20.5.

In a very small baby, or if a foreign body is below the cricoid ring, direct tracheal puncture using the same technique can be used.

Needle cricothyroidotomy

This technique is simple in concept, but far from easy in practice. In an emergency situation the child may be struggling and attempts to breathe or swallow may result in the larynx moving up and down:

1. Attach a cricothyroidotomy cannula-over-needle (or, if not available, an intravenous cannula and needle) of an appropriate size to a 5 ml syringe.
2. Place the patient in a supine position.
3. If there is no risk of cervical spine injury, extend the neck, perhaps with a sandbag under the shoulders.
4. Identify the cricothyroid membrane by palpitation between the thyroid and cricoid cartilages.
5. Prepare the neck with antiseptic swabs.
6. Place your left hand on the neck to identify and stabilise the cricothyroid membrane and to protect the lateral vascular structures from needle injury.
7. Insert the needle and cannula through the cricothyroid membrane at a 45° angle caudally, aspirating as the needle is advanced (Figure 20.6).
8. When air is aspirated, advance the cannula over the needle, being careful not to damage the posterior tracheal wall. Withdraw the needle.
9. Recheck that air can be aspirated from the cannula.
10. Attach the hub of the cannula to an oxygen flow meter via a Y-connector. Initially the oxygen flow rate (in litres) should be set at the child's age (in years).
11. Ventilate by occluding the open end of the Y-connector with a thumb for 1 second to direct gas into the lungs. If this does not cause the chest to rise the oxygen flow rate should be

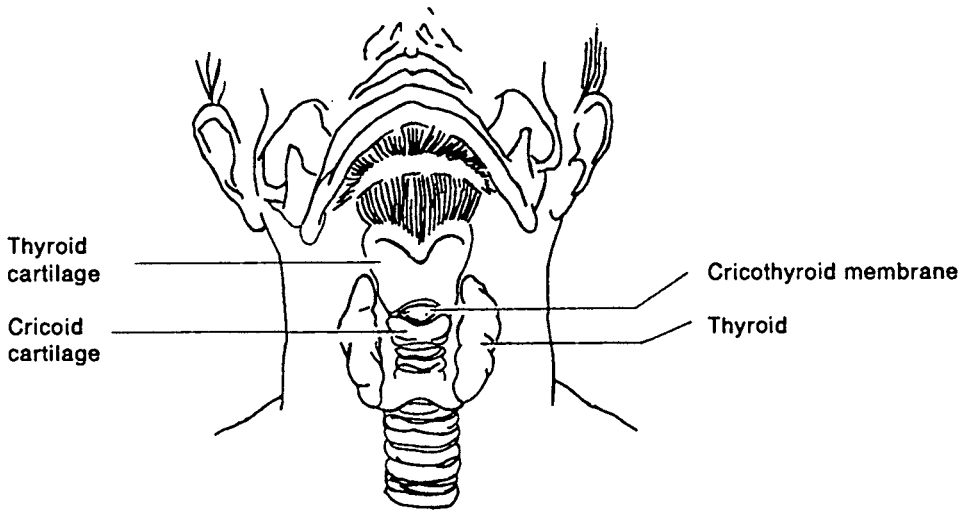


Figure 20.5 Surgical airway anatomy

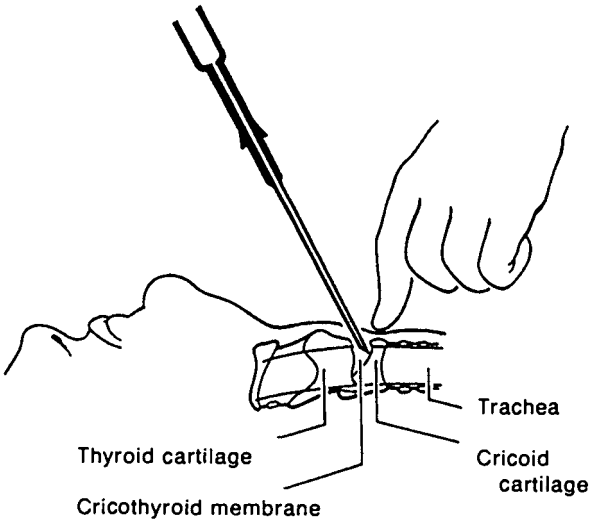


Figure 20.6 Needle cricothyroidotomy

increased by increments of 1 litre and the effect of 1 second's occlusion of the Y-connector reassessed.

12 Allow passive exhalation (via the upper airway) by taking the thumb off for 4 seconds.

13 Observe chest movement and auscultate breath sounds to confirm adequate ventilation.

14 Check the neck to exclude swelling from the injection of gas into the tissues rather than the trachea.

15 Secure the equipment to the patient's neck.

16 Having completed emergency airway management, arrange to proceed to a more definitive airway procedure, such as tracheostomy or intubation if more skilled help has arrived.

Important notes

There are two common misconceptions about transtracheal insufflation. The first is that it is possible to ventilate a patient via a needle cricothyroidotomy using a self-inflating bag. The maximum pressure from a bag is approximately 4.5 kPa (45 cmH_2O – the blow-off valve pressure) and this is insufficient to drive gas through a narrow cannula. In comparison, wall oxygen is provided at a pressure of 4 atmospheres (approximately 400 kPa or 4000 cmH_2O). The second misconception is that expiration can occur through the cannula, or through a separate cannula inserted through the cricothyroid membrane. This is not possible. The intratracheal pressure during expiration is usually less than 3 kPa (30 cmH_2O) – less than one-hundredth of the driving pressure in inspiration. Expiration must occur via the upper airway, even in situations of partial upper airway obstruction. Should upper airway obstruction be complete, it is necessary to reduce the gas flow to 1–2 l/min. This provides some oxygenation but little ventilation.

Nevertheless, insufflation buys a few minutes in which to attempt surgical airway.

Surgical cricothyroidotomy

This should only be considered in the older child (12 years or over):

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.

4 Prepare the skin and, if the patient is conscious, infiltrate with local anaesthetic.

5 Place your left hand on the neck to stabilise the cricothyroid 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° 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.
10 Ventilate the patient and check that this is effective.
11 Secure the tube to prevent dislodgement.

Complications of cricothyroidotomy

These include:
- Asphyxia.
- 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.
- Infection.

20.6 VENTILATION WITHOUT INTUBATION

Mouth-to-mask ventilation

1 Apply the mask to the face, using a jaw thrust grip, with the thumbs holding the mask. If using a shaped mask, it should be the right way up in children (Figure 20.7), or upside down in infants (Figure 20.8).
2 Ensure an adequate seal.
3 Blow into the mouth port, observing the resulting chest movement.
4 Ventilate at an initial 12–20 breaths/min, depending on the age of the child, if the child is apnoeic or is hypoventilating. If using the mask for cardiopulmonary resuscitation (CPR) then use two ventilations to 15 compressions.
5 Attach oxygen to the face mask if possible.

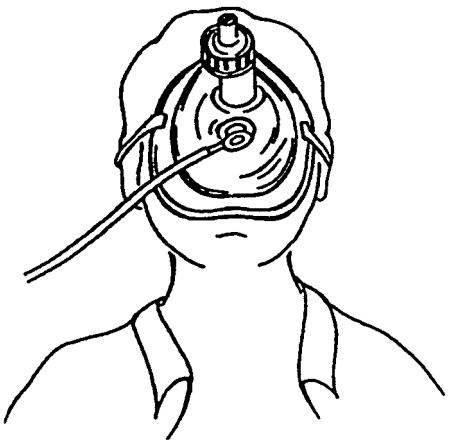


Figure 20.7 Mouth-to-mask in a child

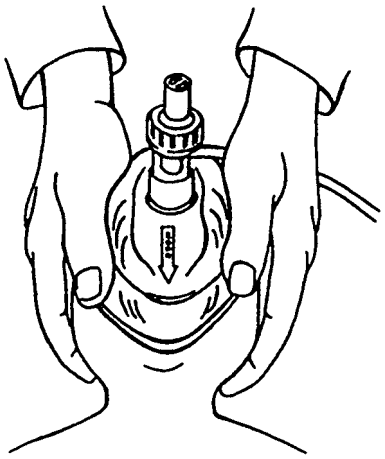


Figure 20.8 Mouth-to-mask in an infant

Bag-and-mask ventilation

1 Apply the mask to the face, using a jaw thrust grip. The fourth and fifth fingers should perform a jaw thrust and the other fingers hold the mask tightly in place (Figure 20.9).
2 Ensure an adequate seal.
3 Squeeze the bag, observing the resulting chest movement.
4 Ventilate at an initial 12–20 breaths/min, depending on the age of the child, if the child is apnoeic or is hypoventilating. If using the bag and mask for CPR then use two ventilations to 15 chest compressions.

If a two-person technique is used, one rescuer maintains the mask seal with both hands, while the second person squeezes the self-inflating bag.

This is to be recommended.

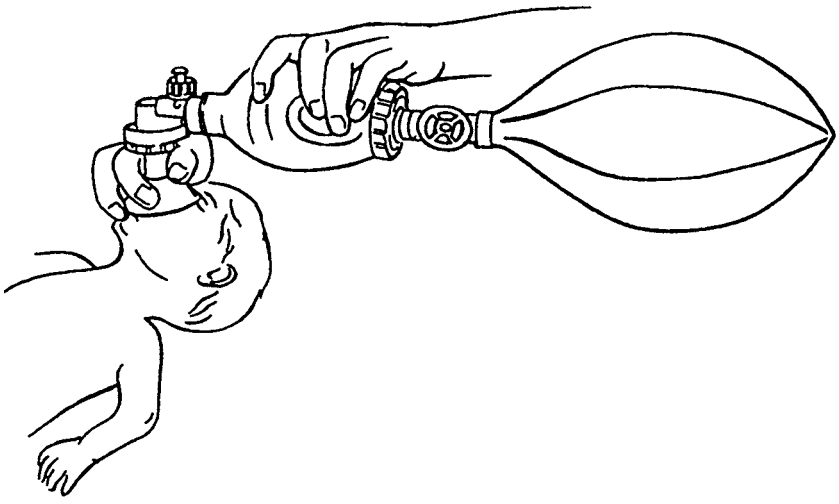


Figure 20.9 Bag-and-mask ventilation

20.7 MANAGEMENT OF A BLOCKED TRACHEOSTOMY

When you suspect a child with a tracheostomy tube is not breathing, the procedure to follow is:

1 Stimulate the child
2 Shout for help
3 Suction
4 Check for breathing
5 Tube change if not breathing
6 Check for breathing
7 Give five breaths if not breathing
8 Check signs of life/pulse
9 If no signs of circulation, start CPR with a ratio of 15 chest compressions to 2 ventilations
10 After 1 minute summon help if none available yet
Do not leave the child alone if his or her breathing returns to normal

Basic life support

1 Stimulate the child.
2 Shout for help.
3 Open and check the airway, and extend the neck with a head tilt. *This exposes the tracheostomy tube* (Figure 20.10). You may need to lift the chin also to expose the tracheostomy tube completely. If the tube is blocked attempt to clear it with a suction catheter *if you are unable to pass the suction catheter down the tracheostomy tube – then the tube must be changed immediately*.

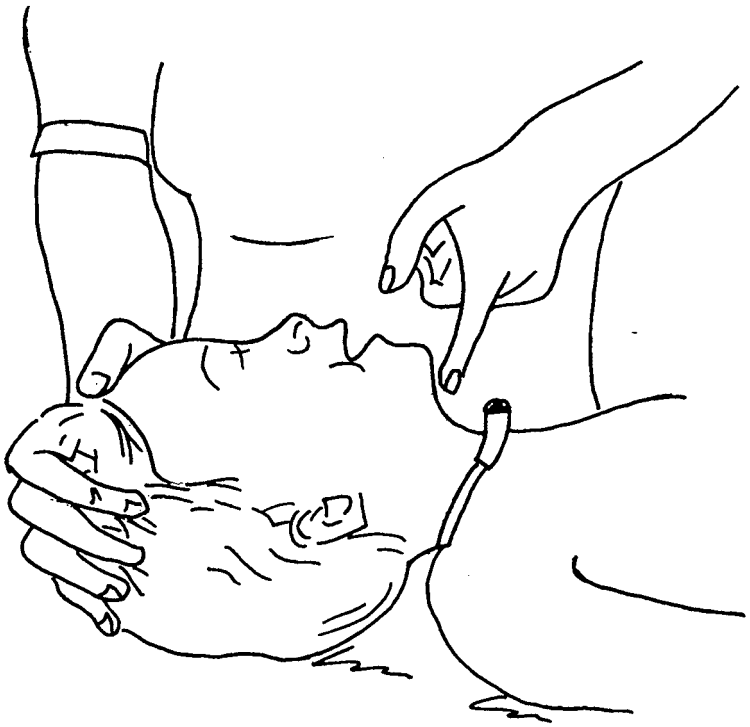


Figure 20.10 Chin lift

Figure 20.11 Check for breathing

If you are unable to insert the new tube:

- Try a smaller tube.
- If unable to insert this, thread a suction catheter through the tracheostomy tube. Insert the tip of the suction catheter into the stoma, then attempt to guide the tracheostomy tube along the catheter and into the stoma.
- If this is unsuccessful then attempt breathing via the suction catheter through the stoma (only if a large suction catheter) or attempt mouth-to-stoma/mouth-to-mouth resuscitation.

4 Check for breathing. Supporting the new tube, place the side of your face over the tracheostomy tube to listen and feel for any breaths, and at the same time look at the child's chest to observe any breathing movement (Figure 20.11). If the child is breathing satisfactorily, secure the tracheostomy tube, place him in a recovery position and continue to assess him. If the child is not breathing, or there are only infrequent gasps, you will have give rescue breaths.

5 Give five rescue breaths. Cover the child's tracheostomy tube with your mouth and exhale to make the chest rise. Remove your mouth from the child's tube to let the breath escape from their lungs. Repeat this five times at a rate of about one breath every 2 seconds.

You will know your breathing has been effective if you can see the child's chest rise and fall with each breath. After five breaths of mouth-to-tracheostomy rescue breathing, you must check to see whether or not the child has signs of life or a pulse.

6 If there are no signs of life/pulse or you are not sure, you will need to perform chest compressions as well as providing the child with rescue breathing.

7 Chest compressions should compress the lower half of the sternum. The rate for chest compressions is 100–120 per minute.

Using two fingers for an infant and one or two hands for a child, compress the lower half of the sternum 15 times by at least one-third of the diameter of the chest, and then give

two ventilations. If possible, support the trachestomy tube with the free hand, if one is available.

8 Give 1 minute of 15 chest compressions to two breaths and then call for help.

If the child recovers, place them in the recovery position, and continue to assess and support.

Emergency equipment for tracheostomy change

This should be checked daily:

- Usual size tracheostomy tube with tapes attached
- Smaller size tracheostomy tube with tapes attached
- Scissors
- Sterile sodium chloride 0.9% ampoules
- Five 2 ml syringes
- Tracheal dilators
- Spare tape
- Gauze swab
- KY jelly
- Gloves

CHAPTER 21

Practical procedures: circulation

In this chapter, the following procedures are explained:

- Vascular access:
  - Peripheral venous access:
    upper and lower extremity veins
    scalp veins
    external jugular vein
    venous cut-down
    umbilical vein
  - Central venous access:
    femoral vein
    internal jugular vein
    external jugular vein
    subclavian vein
  - Arterial cannulation
  - Intraosseous access
- Defibrillation

21.1 VASCULAR ACCESS

Access to the circulation is a crucial step in delivering advanced paediatric life support. Many access routes are possible: peripheral venous access, central venous access, arterial cannulation or intraosseous access. The one chosen will reflect both clinical need and the skills of the operator.

If fluids are to be given, infusion pumps or paediatric infusion sets must be used. This avoids inadvertent overtransfusion in small children.

Peripheral venous access

Upper and lower extremity veins

Veins on the dorsum of the hand, the elbow, the dorsum of the feet and the saphenous vein at the ankle can be used for cannulation. Standard percutaneous techniques should be employed if possible. Topical or injected local anaesthetic should be used whenever time allows.

Scalp veins

The frontal superficial, temporal posterior, auricular, supraorbital and posterior facial veins can be used.

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Equipment
- Skin-cleansing swabs.
- Butterfly needle.
- Syringe and 0.9% saline.
- Short piece of tubing or bandage.

Procedure
1. Restrain the child.
2. Shave the appropriate area of the scalp.
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 the site of puncture.
5. Fill the syringe with 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 into position.

External jugular vein

Equipment
- Skin-cleansing swabs.
- Appropriate cannula.
- Tape.

Procedure
1. Place child in a 15–30° head-down position (or with padding under the shoulders so that the head hangs lower than the shoulders).
2. Turn the head away from the site of puncture. Restrain the child as necessary in this position.
3. Clean the skin at 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 (Figure 21.1).
5. Have an assistant place his or her 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.
7. When free flow of blood is obtained, ensure no air bubbles are present in the tubing and then attach a giving set.
8. Tape the cannula securely in position.

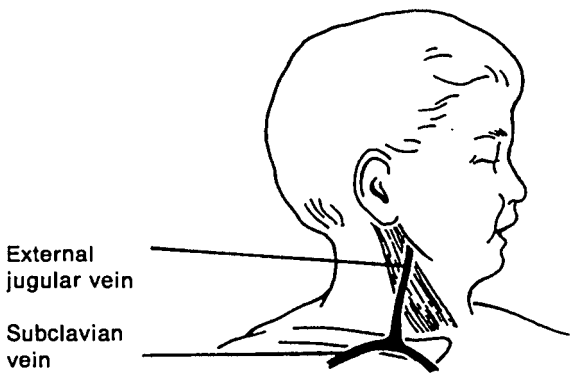


Figure 21.1 The course of the external jugular vein

Venous cut-down

If speed is essential, it may be more appropriate to use the intraosseous route for immediate access, and to cut down later for continued fluid and drug therapy.

Equipment

- Skin-cleansing swabs.
- Lidocaine 1% for local anaesthetic with a 2 ml syringe and a 25 gauge needle.
- Scalpel.
- Curved haemostats.
- Suture and ligature material.
- Cannula.

Procedure

1. Immobilise the appropriate limb.
2. Clean the skin.
3. Identify the surface landmarks for the relevant vein (Table 21.1).
4. If the child is responsive to pain, infiltrate the skin with 1% lidocaine.
5. Make an incision perpendicular to the course of the vein through the skin.
6. Using the curved haemostat tips, bluntly dissect the subcutaneous tissue.
7. Identify the vein and free 1–2 cm in length.
8. Pass a proximal and a distal ligature (Figure 21.2).
9. Tie off the distal end of the vein, keeping the ends of the tie long.
10. Make a small hole in the upper part of the exposed vein with a scalpel blade or fine-pointed scissors.

Table 21.1 Surface anatomy of the brachial and long saphenous veins

Child	Brachial	Saphenous (Figure 21.2)
Infant	One finger-breadth lateral to the medial epicondyle of the humerus	Half a finger-breadth superior and anterior to the medial malleolus
Small children	Two finger-breadths lateral to the medial epicondyle of the humerus	One finger-breadth superior and anterior to the medial malleolus
Older children	Three finger-breadths lateral to the medial epicondyle of the humerus	Two finger-breadths superior and anterior to the medial malleolus

Figure 21.2 Site of a long saphenous cut-down and its technique

11 While holding the distal tie to stabilise the vein, insert the cannula.
12 Secure this in place with the upper ligature. Do not tie this too tightly; doing so would cause occlusion.
13 Attach a syringe filled with 0.9% saline to the cannula and ensure that fluid flows freely up the vein. If free flow does not occur, then either the tip of the cannula is against a venous valve or the cannula may be wrongly placed in the adventitia surrounding the vein. Withdrawing the catheter will improve flow in the former case.
14 Once fluid flows freely, tie the proximal ligature around the catheter to help immobilise it.
15 Close the incision site with interrupted sutures.
16 Fix the catheter or cannula to the skin and cover with a sterile dressing.

Umbilical vein

Venous access via the umbilical vein is a rapid and simple technique. It is used during resuscitation at birth.

Equipment

- Skin-cleansing swabs.
- Umbilical tape.
- Scalpel.
- Syringe and 0.9% saline.
- Catheter.

Procedure

1 Loosely tie the umbilical tape around the cord.
2 Cut the cord with a scalpel, leaving a 1 cm strip distal to the tape.
3 If there is bleeding from the vein, gently tighten the tape to stop it.
4 Identify the umbilical vein. Three vessels will be seen in the stump: two will be small and contracted (the arteries sited inferiorly), and one at the head end will be dilated (the vein) (Figure 21.3).
5 Fill a French 5 gauge catheter with 0.9% saline.
6 Insert the catheter into the vein, and advance it approximately 5 cm.
7 Tighten the umbilical tape to secure the catheter. A purse-string suture may be used later to stitch the catheter in place.

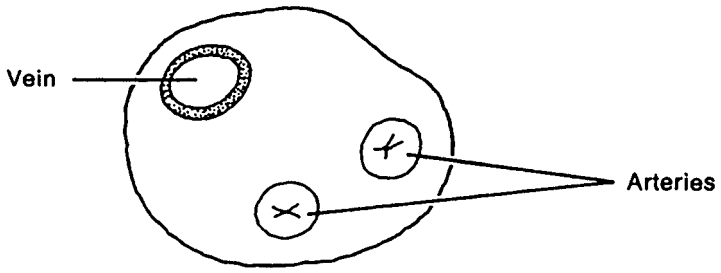


Figure 21.3 Umbilical cord cross-section

Central venous access

Central access can be obtained through the femoral, internal jugular, external jugular and (in older children) subclavian veins. The Seldinger technique is safe and effective. UK NICE guidelines recommend ultrasound guided insertion. The femoral vein is often used as it is relatively easy to cannulate away from the chest during cardiopulmonary resuscitation (CPR). Central venous access via the neck veins is not without dangers, and may be difficult in emergency situations. The course of the central veins of the neck is shown in Figure 21.4.

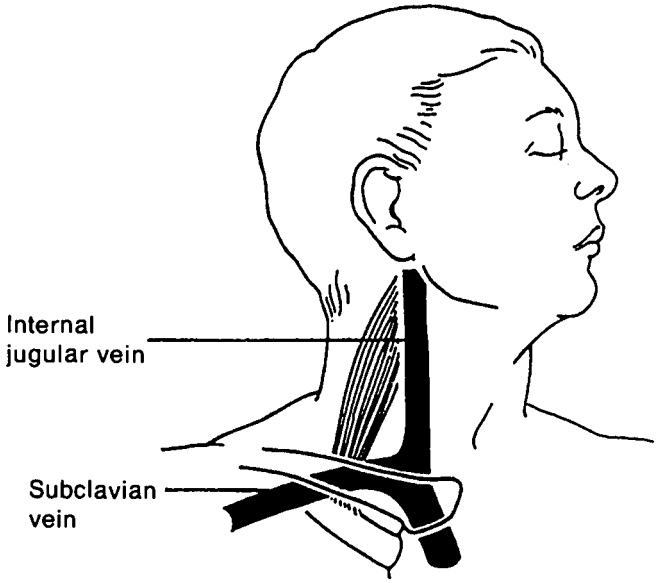


Figure 21.4 The course of the central veins of the neck

Femoral vein

Equipment

- Skin-cleansing swabs.
- Lidocaine 1% for local anaesthetic with a 2 ml syringe and a 23 gauge needle.
- Syringe and 0.9% saline.
- Seldinger cannulation set:
  - syringe,
  - needle,
  - Seldinger guide wire, and
  - cannula.
- Suture material.
- Prepared paediatric infusion set.
- Tape.

Procedure

1. Place the child supine with the groin exposed and leg slightly abducted at the hip. Restrain the child's leg and body as necessary.
2. Clean the skin at the appropriate side.
3. Under ultrasound guidance (if available) identify the puncture site. The femoral vein is found by palpating the femoral artery. The vein lies directly medial to the artery.
4. If the child is responsive to pain, infiltrate the area with 1% lidocaine.
5. Attach the needle to the syringe.
6. Keeping one finger on the artery to mark its position, introduce the needle at a 45° angle pointing towards the patient's head directly over the femoral vein. Keep the syringe in line with the child's leg. Advance the needle, pulling back on the plunger of the syringe all the time.
7. As soon as blood flows back into the syringe, take the syringe off the needle. Immediately occlude the end of the needle to prevent blood loss.
8. If the vein is not found, withdraw the needle to the skin, locate the artery again and advance as in point 6 above.
9. Insert the Seldinger wire into the needle, and into the vein.

10 Withdraw the needle along the wire, ensuring that the wire is not dislodged from the vein.
11 Place the catheter over the wire and advance it through the skin, into the vein.
12 Suture the catheter in place.
13 Withdraw the wire, immediately occluding the end of the cannula to prevent blood loss.
14 Attach the infusion set.
15 Tape the infusion set tubing in place.

Internal jugular vein

Equipment

- Skin-cleansing swabs.
- Lidocaine 1% for local anaesthetic with a 2 ml syringe and a 23 gauge needle.
- Syringe and 0.9% saline.
- Seldinger cannulation set:
  - syringe,
  - needle,
  - Seldinger guide wire, and
  - cannula.
- Suture material.
- Prepared paediatric infusion set.
- Tape.

Procedure

1 Place the child in a 15–30° head-down position.
2 Turn the head away from the side that is to be cannulated and restrain the child as necessary.
3 Clean the skin at the appropriate side of the neck.
4 Under ultrasound guidance (if available) identify the puncture site. This is found at the apex of the triangle formed by the two lower heads of the sternomastoid and the clavicle.
5 If the child is responsive to pain, infiltrate the area with 1% lidocaine.
6 Attach the needle to the syringe and puncture the skin at the appropriate place (see point 4 above).
7 Direct the needle downwards at 30° to the skin; advance the needle towards the nipple, pulling back on the plunger of the syringe all the time.
8 As soon as the blood flows back into the syringe, take the syringe off the needle. Immediately occlude the end of the needle to prevent air embolism.
9 If the vein is not found, withdraw the needle to the skin and advance it again some 5–10° laterally.
10 Insert the Seldinger wire into the needle, and into the vein.
11 Withdraw the needle along the wire, ensuring that the wire is not dislodged from the vein.
12 Place the catheter over the wire and advance it through the skin, into the vein.
13 Suture the catheter in place.
14 Withdraw the wire, immediately occluding the end of the cannula to prevent air embolism.
15 Attach the infusion set.
16 Tape the infusion set tubing in place.
17 Obtain a chest radiograph in order to see the position of the catheter and to exclude a pneumothorax.

External jugular vein

By using the Seldinger technique it is possible to obtain central venous access via the external jugular vein as described below. The anatomy is such that passage into the central veins can sometimes be more difficult compared with other approaches.

Equipment
- Skin-cleansing swabs.
- Lidocaine 1% for local anaesthetic with a 2 ml syringe and a 25 gauge needle.
- Syringe and 0.9% saline.
- Seldinger cannulation set:
  - syringe,
  - needle,
  - Seldinger guide wire (J wire), and
  - cannula.
- Suture material.
- Prepared paediatric infusion set.
- Tape.

Procedure
1. Place the child in a 15–30° head-down position (or with padding under the shoulders so that the head hangs lower than the shoulders).
2. Turn the head away from the site of puncture. Restrain the child as necessary in this position.
3. Clean the skin at 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 his or her 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. Attach the needle to the syringe and puncture the vein.
7. As soon as blood starts to flow freely, take off the syringe and occlude the end of the needle.
8. Insert a J wire into the needle and into the vein.
9. Advance the J wire. There may be some resistance as the wire reaches the valve at the proximal end of the vein. Gently advance and withdraw the wire until it passes this obstacle.
10. Gently advance the wire.
11. Withdraw the needle along the wire, ensuring that the wire is not dislodged from the vein.
12. Place the catheter over the wire and advance it through the skin, into the vein.
13. Suture the catheter in place.
14. Withdraw the wire, immediately occluding the end of the cannula to prevent air embolism.
15. Attach the infusion set.
16. Tape the infusion set tubing in place.
17. Obtain a chest radiograph in order to see the position of the catheter and to exclude a pneumothorax.

Subclavian vein

Equipment
- Skin-cleansing swabs.
- Lidocaine 1% for local anaesthetic with a 2 ml syringe and a 23 gauge needle.
- Syringe and 0.9% saline.
- Seldinger cannulation set:
  - syringe,
  - needle,
  - Seldinger guide wire, and
  - cannula.
- Suture material.
- Prepared paediatric infusion set.
- Tape.

Procedure

1. Place the child in a 15–30° head-down position.
2. Turn the head away from the site that is to be cannulated and restrain the child as necessary.
3. Clean the skin over the upper side of the chest to the clavicle.
4. Under ultrasound guidance (if available) identify the puncture site. This is 1 cm below the midpoint of the clavicle.
5. If the child is responsive to pain, infiltrate the area with 1% lidocaine.
6. Attach the needle to the syringe and puncture the skin at the appropriate place (see point 4 above).
7. Direct the needle under the clavicle, 'stepping down' off the bone.
8. Once under the clavicle, direct the needle towards the suprasternal notch. Advance the needle, pulling back on the plunger of the syringe all the time and staying as superficial as possible.
9. As soon as the blood flows back into the syringe, take the syringe off the needle. Immediately occlude the end of the needle to prevent air embolism.
10. If the vein is not found, slowly withdraw the needle, continuing to pull back on the plunger. If the vein has been crossed inadvertently, free flow will often be established during this manoeuvre.
11. If the vein is still not found repeat steps 7 to 10, aiming at a point a little higher in the sternal notch.
12. Insert the Seldinger wire into the needle, and into the vein.
13. Withdraw the needle along the wire, ensuring that the wire is not dislodged from the vein.
14. Place the catheter over the wire and advance it through the skin, into the vein.
15. Suture the catheter in place.
16. Withdraw the wire, immediately occluding the end of the cannula to prevent air embolism.
17. Attach the infusion set.
18. Tape the infusion set tubing in place.
19. Obtain a chest radiograph in order to see the position of the catheter and to exclude a pneumothorax.

Arterial cannulation

Arterial cannulation is used to monitor arterial blood pressure, guide dosage adjustments in shock and hypertensive crisis, obtain blood samples for respiratory and acid–base status, and calculate cerebral perfusion pressure.

It should not be performed in sites where there is skin infection or interruption, or absent collateral circulation, and care should be taken in severe haemorrhagic sites. In children, the preferred sites include the radial, posterior tibial, dorsalis pedis, ulnar and femoral arteries. The site should remain visible and not prone to contamination.

Radial artery cannulation

Equipment

- Skin-cleansing swabs.
- Lidocaine 1%.
- Heparinised syringe.
- Cannula:
  - pre-term: 24 gauge,
  - infant/pre-school: 22 gauge,
  - school-aged: 20–22 gauge, or
  - adolescent to adult: 18–20 gauge.
- T-connector or three-way tap with extension.

- Gauze, pad and tapes
- Transparent sterile dressing
- Flushed infusion set (saline 0.9% with heparin 0.5–1.0 U/ml) with pressure infusion bag or pump.
- Pressure transducer and monitor.

Procedure

1. Before using the radial artery check that the ulnar artery is present and patent. Occlude both arteries at the wrist and then release the pressure on the ulnar artery; circulation should return to the hand. (It will flush pink.) If this does not happen, do not proceed with a radial puncture on that side.
2. Keep the wrist hyperextended and restrained, and palpate the radial artery (usually located in the middle of the lateral third of the wrist).
3. Clean the skin, and infiltrate with local anaesthetic.
4. Insert the cannula over the artery at 45° to the skin and advance it slowly. When the artery is punctured, blood will be seen to pulsate into the syringe.
5. Advance the cannula over the needle and into the artery, and remove the needle whilst compressing the artery proximal to the position of the cannula tip.
6. Connect the T-connector or three-way tap with extension, ready flushed with 0.9% saline to test cannula patency.
7. Tape the cannula securely in place and cover with transparent dressing.
8. Connect the infusion set and calibrate the monitoring equipment.

Complications of cannulation

- Arteriospasm.
- Haematoma.
- Thrombosis.
- Bacterial colonisation and sepsis.

Intraosseous infusion

The technique of intraosseous infusion is not new. It was used in the 1930s as a quick method of gaining vascular access (the only alternative was to use a reusable, resharpened metal needle or to perform a venous cut-down). Because it is important to achieve vascular access quickly in many life-threatening situations, intraosseous infusion is again being recommended. Specially designed needles make this quick and easy. It is indicated if other attempts at venous access fail, or if they will take longer than 1.5 minutes to carry out. It is the recommended technique for circulatory access in cardiac arrest.

Equipment

- Alcohol swabs.
- An 18 gauge needle with trochar (at least 1.5 cm in length).
- A 5 ml syringe.
- A 20 ml syringe.
- Infusion fluid.

Procedure

1. Identify the infusion site. Fractured bones should be avoided, as should limbs with fractures proximal to possible infusion sites. The landmarks for the upper tibial and lower femoral sites are shown below, and the former approach is illustrated in Figure 21.5 (over page).
2. Clean the skin at the chosen site.
3. Insert the needle at 90° to the skin.
4. Continue to advance the needle until a ‘give’ is felt as the cortex is penetrated.

5 Attach the 5 ml syringe and aspirate – blood marrow may be used to check blood glucose and provide blood culture. Flush to confirm correct positioning.
6 Attach the filled 20 ml syringe and push in the infusion fluid in boluses.

Surface anatomy for intraosseous infusions

Tibial	Femoral
Anterior surface, 2–3 cm below the tibial tuberosity	Anterolateral surface, 3 cm above the lateral condyle

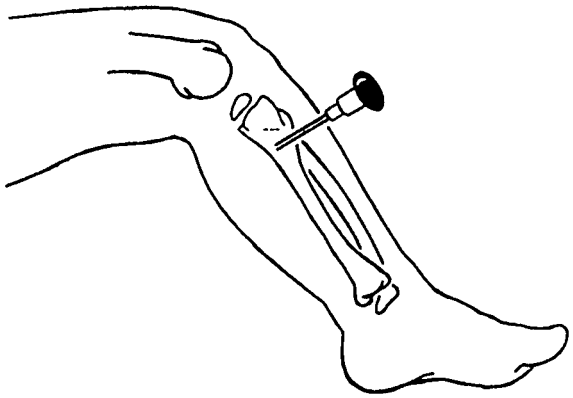


Figure 21.5 Tibial technique for intraosseous infusion

Other powered devices

The EZ-IO drill is a powered device that enables rapid insertion of an intraosseous needle. The same landmarks are used as for manual insertion and the procedure is less painful for the conscious victim due to its rapidity. The EZ-IO needles are in two sizes: under 40 kg and over 40 kg.

The procedure for insertion is as follows:

1 Universal precautions.
2 Clean site.
3 Choose appropriate size needle and attach to drill – it will fix magnetically.
4 Hold the drill and needle at 90° to the skin surface and push through the skin without drilling, until bone is felt.
5 Push the drill button and drill continuously and push until there is loss of resistance – there is a palpable give as the needle breaches the cortex.
6 Remove the drill and unscrew the trochar.
7 Aspirate the marrow if possible.
8 Attach the pre-prepared connection tube.
9 There is an optional device to secure the needle but this is not essential.
10 Proceed with required therapy.

It should be noted that rapid infusion of fluid may be painful for the conscious patient and if this proves to be the case 0.5 mg/kg of 2% lignocaine may be infused slowly to combat this.

Complications

- Compartment syndrome.
- Infection.
- Fracture.

21.2 DEFIBRILLATION

In order to achieve the optimum outcome, defibrillation must be performed quickly and efficiently. This requires the following:

- Correct paddle selection.
- Correct paddle placement.
- Good paddle contact.
- Correct energy selection.

Many defibrillators are available. Providers of advanced paediatric life support should make sure that they are familiar with those they may have to use.

Correct paddle selection

Most defibrillators are supplied with adult paddles attached (13 cm diameter, or equivalent area). Paddles of 4.5 cm diameter are suitable for use in infants, and ones of 8 cm diameter should be used for small children.

Correct paddle placement

The usual placement is anterolateral. One paddle is put over the apex in the midaxillary line and the other is placed just to the right of the sternum, immediately below the clavicle (Figure 21.6).

If the anteroposterior placement is used, one paddle is placed just to the left side of the lower part of the sternum and the other just below the tip of the left scapula (Figure 21.7).

Good paddle contact

Gel pads or electrode gel should always be used (if the latter, 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 levels are shown in Chapters 6 and 10.

Automated external defibrillators (AEDs) are now commonplace. The standard adult shock is used for children over 8 years. For children under 8 years attenuated paediatric paddles should be used with the AED.

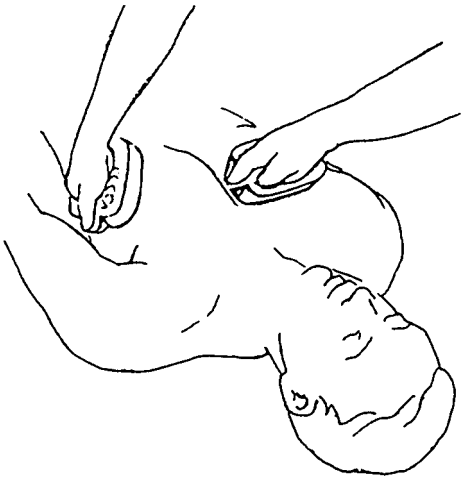


Figure 21.6 Standard anterolateral paddle placement

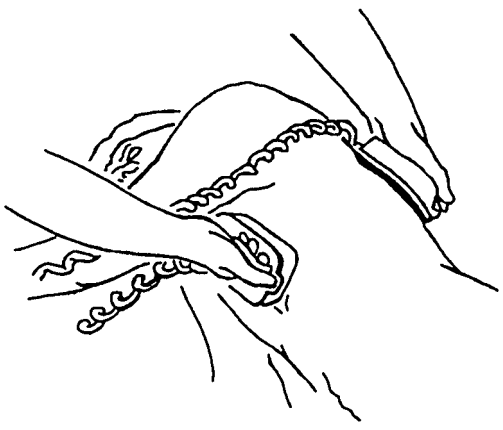


Figure 21.7 Anteroposterior paddle placement

For the infant of less than 1 year, 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. The order of decreasing preference for defibrillation in the under 1-year-olds is as follows:

1 Manual defibrillator.
2 AED with dose attenuator.
3 AED without dose attenuator.

Many AEDs can detect ventricular fibrillation/ventricular tachycardia (VF/VT) in children of all ages and differentiate 'shockable' from 'non-shockable' rhythms with a high degree of sensitivity and specificity.

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 the shock is delivered. The defibrillator should only be charged when the paddles are either in contact with the child or replaced properly in their storage positions.

Disconnect the oxygen supply to the patient.

Procedure: manual defibrillation

Basic life support should be interrupted for the shortest possible time (steps 7–9 below).

1 Apply gel pads or electrode gel.
2 Select the correct paddles.
3 Select the energy required.
4 Place the paddles onto the gel pads, and apply firm pressure.
5 Press the charge button.
6 Wait until the defibrillator is charged.
7 Shout 'Stand back!'
8 Check that all other rescuers are clear.
9 Deliver the shock.
10 Recommence CPR.

Procedure: hands-free defibrillation

Basic life support should be interrupted for the shortest possible time (steps 7–9 below).

1 Apply adhesive monitoring electrodes to the correct positions.
2 Continue compressions.
3 Interrupt briefly to confirm VF.
4 Select the energy required.
5 Press the charge button.
6 Wait until the defibrillator is charged.
7 Shout 'stand back!'
8 Check that all other rescuers are clear.
9 Deliver the shock.
10 Recommence CPR.

CHAPTER 22

Practical procedures: trauma

In this chapter, the following procedures are explained:
- Chest decompression:
  - Needle thoracocentesis
  - Chest drain placement
- Pericardiocentesis
- Femoral nerve block
- Focused assessment with sonography for trauma
- Diagnostic peritoneal lavage
- Cervical spine immobilisation:
  - Application of a cervical collar
  - Application of head blocks and straps
  - Log-rolling
- Helmet removal

22.1 NEEDLE THORACOCENTESIS

This procedure can be life saving and can be performed quickly with minimum equipment. It should be followed by chest drain placement.

Minimum equipment
- Alcohol swabs.
- Large over-the-needle intravenous cannula (16 gauge or larger).
- A 20 ml syringe.

Procedure
1 Identify the second intercostal space in the mid-clavicular line on the side of the pneumothorax.
2 Swab the chest wall with surgical preparation solution or an alcohol swab.
3 Attach the syringe to the cannula. Fluid in the cannula will assist in the identification of air bubbles.
4 Insert the cannula vertically into the chest wall, just above the rib below, aspirating all the time (Figure 22.1, over page).
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.

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

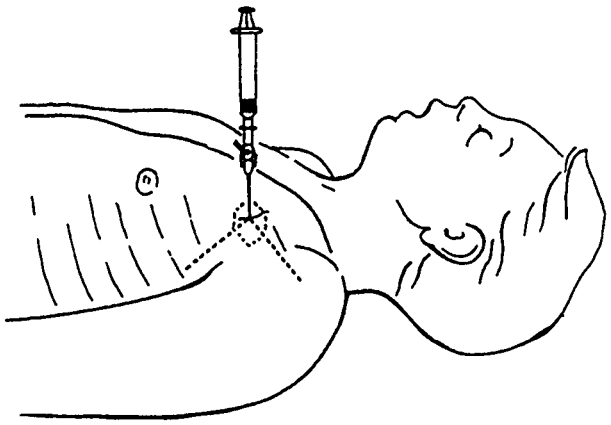


Figure 22.1 Needle thoracocentesis

If needle thoracocentesis is attempted, and the patient does not have a tension pneumothorax, the risk of causing a pneumothorax is 10–20%. Patients who have had this procedure must have a chest radiograph, and will require chest drainage if ventilated.

22.2 CHEST DRAIN PLACEMENT

Chest drain placement should be performed using the open technique described here. This minimises lung damage. In general, the largest size drain that will pass between the ribs should be used.

Minimum equipment

- Skin preparation and surgical drapes.
- Scalpel.
- Large clamps × 2.
- Suture.
- (Local anaesthetic.)
- Scissors.
- Chest drain tube.

Procedure

1. Decide on the insertion site (usually the fifth intercostal space in the mid-axillary line) on the side with the pneumothorax (Figure 22.2).
2. Swab the chest wall with surgical preparation or an alcohol swab.
3. Use local anaesthetic if necessary.
4. Make a 2–3 cm skin incision along the line of the intercostal space, just above the rib below.
5. Bluntly dissect through the subcutaneous tissues just over the top of the rib below, and puncture the parietal pleura with the tip of the clamp.
6. Put a gloved finger into the incision and clear the path into the pleura (Figure 22.3). This will not be possible in small children.
7. Advance the chest drain tube into the pleural space during expiration.
8. Ensure the tube is in the pleural space by listening for air movement, and by looking for fogging of the tube during expiration.
9. Connect the chest drain tube to an underwater seal.
10. Suture the drain in place, and secure with tape.
11. Obtain a chest radiograph.

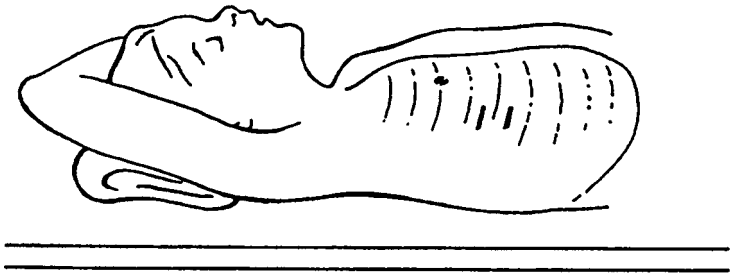


Figure 22.2 Chest drain insertion – landmarks

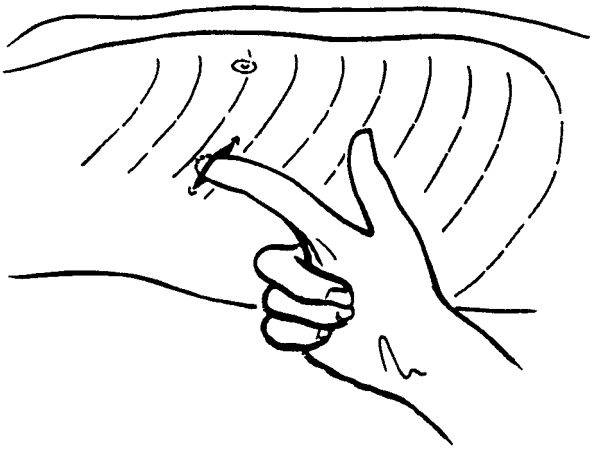


Figure 22.3 Chest drain insertion – clearing the path

22.3 PERICARDIOCENTESIS

The removal of a small amount of fluid from the pericardial sac can be life-saving. The procedure is not without risks and the electrocardiogram (ECG) should be closely monitored throughout. An acute injury pattern (ST segment changes or a widened QRS) indicates ventricular damage by the needle.

Minimum equipment

- Skin preparation and surgical drapes.
- ECG monitor.
- (Local anaesthetic.)
- A 20 ml syringe.
- Large over-the-needle cannula (16 or 18 gauge).

Procedure

1. Swab the xiphoid and subxiphoid areas with surgical preparation or an alcohol swab.
2. Use local anaesthetic if necessary.
3. Assess the patient for any significant mediastinal shift if possible.
4. Attach the syringe to the needle.
5. Puncture the skin 1–2 cm inferior to the left side of the xiphoid junction at a 45° angle (Figure 22.4, over page).
6. Advance the needle towards the tip of the left scapula, aspirating all the time (Figure 22.5, over page).
7. Watch the ECG monitor for signs of myocardial injury.
8. Once fluid is withdrawn, aspirate as much as possible (unless it is possible to withdraw limitless amounts of blood, in which case a ventricle has probably been entered).

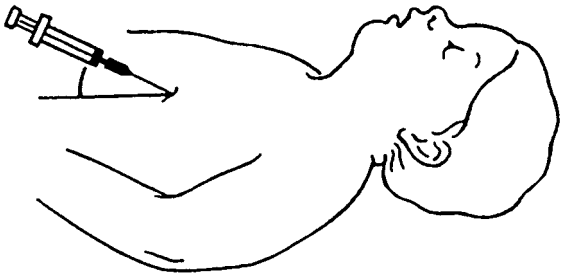


Figure 22.4 Needle pericardiocentesis – angle

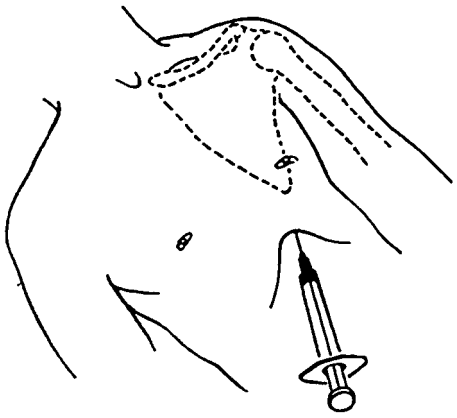


Figure 22.5 Needle pericardiocentesis – direction

9 If the procedure is successful, remove the needle, leaving the cannula in the pericardial sac. Secure in place and seal with a three-way tap. This allows later repeat aspirations should tamponade recur.

22.4 FEMORAL NERVE BLOCK

The femoral nerve supplies the femur with sensation, and a block is useful in cases of femoral fracture. The technique may also be of benefit when analgesic agents would interfere with the management or assessment of other injuries. A long-acting local anaesthetic agent should be used so that radiographs and splinting can be undertaken with minimal distress to the child.

Equipment

- Antiseptic swabs to clean.
- Lidocaine 1%.
- A 2 ml syringe and a 25 gauge needle.
- A syringe (5 or 10 ml) and a 21 gauge needle.
- Bupivacaine 0.25%: 0.8 ml/kg of 0.25% (maximum 2 mg/kg).

Procedure

1 Move the fractured limb gently so that the femur lies in abduction and the ipsilateral groin is exposed.
2 Swab the groin clean with antiseptic solution.
3 Identify the femoral artery and keep one finger on it. The femoral nerve lies immediately lateral to the artery.
4 Using the 2 ml syringe filled with lidocaine and a 25 gauge needle, infiltrate the skin just lateral to the artery. Aspirate the syringe frequently to ensure that the needle is not in a vessel.

5 Inject the bupivacaine around the nerve using the 21 gauge needle, taking care not to puncture the artery or vein.
6 Wait until anaesthesia occurs (bupivacaine may take up to 20 minutes to have its full effect).

22.5 FOCUSED ASSESSMENT WITH SONOGRAPHY FOR TRAUMA

Focused assessment with sonography for trauma (FAST) has become standard practice in most emergency departments as a diagnostic tool for assessing suspected haemoperitoneum; however, its use still remains contentious amongst certain professional groups. Studies have demonstrated that with adequate training and practice non-radiologists can become competent in carrying out FAST as accurately and reliably as professional sonographers. The basis of the FAST scan is to detect fluid in three dependent regions of the abdomen – the perihepatic space, perisplenic space and bladder/pelvis – a fourth view is often carried out assessing the pericardium looking for evidence of a cardiac tamponade.

Equipment

- Ultrasound gel.
- Ultrasound machine.
- 3–5 MHz transducer
- Darkened room.
- Trained operator.

Procedure

Any one wishing to carry out a FAST procedure must be adequately trained and practitioners in the UK should comply with the standards agreed by the College of Emergency Medicine.

1 The patient's details and examination site is recorded on the ultrasound machine along with the clinical details.
2 Gel is applied to the probe and the orientation of the probe is established.
3 Machine gain and depth are adjusted for optimum image quality.
4 For a pericardial view position the probe below the xiphiod process and rotate cephalad, pointing towards the left shoulder until a view of the heart and pericardium is obtained. The image should be saved and any pericardial fluid noted.
5 For a perihepatic view the probe should be placed on the anterior axillary line over the 10th intercostal space. The image should be obtained to reveal the liver and right kidney, observing the Rutherford–Morison pouch. The image should be saved and any free fluid noted.
6 For a perisplenic view the probe should be place along the posterior axillary line on the left side, using the intercostal space of the 10th and 11th rib. The image should reveal the spleen and left kidney.
7 For a pelvic view the transducer is placed above the pubic symphysis; most operators position the probe longitudinally and then rotate it transversely. The pelvis view is useful in visualising the pouch of Douglas in females, the rectovesical pouch in males, and the bladder. It is preferable to perform FAST before a urinary catheter is inserted as a full bladder provides an ideal acoustic window.

FAST is a 'rule in' investigation, and its limitations must be recognised. In children it should be regarded as a dynamic test, and repeated on a regular basis. FAST should not replace clinical findings or concerns of a seriously injured child, it does not replace the need for urgent surgical review or intervention or computerised tomography.

22.6 DIAGNOSTIC PERITONEAL LAVAGE

The technique described here is designed to maximise the sensitivity and specificity of diagnostic peritoneal lavage. Special care should be taken when performing diagnostic peritoneal lavage

in children, otherwise the unwary operator may be caught out by the relative thinness of the abdominal wall, the intra-abdominal position of the bladder and the high incidence of acute gastric dilatation. The use of this investigation in children has been rendered virtually obsolete in the trauma setting by modern imaging modalities. The presence of intraperitoneal blood *per se* is not an indication for laparotomy. The technique is nevertheless described as peritoneal lavage and may be used to re-warm the hypothermic 'drowned' patient, and access to the peritoneum may provide a temporary dialysis route in children with acute renal failure.

Equipment

- Antiseptic solution.
- Lidocaine 1% with adrenaline.
- Scalpel.
- Self-retaining retractors.
- Suture material.
- 500 ml sterile normal (physiological) saline (warmed).
- Sterile drapes.
- Syringe and needle.
- Artery forceps.
- Scissors.
- Peritoneal lavage catheter.
- Giving set.

Procedure

1. Ensure that the urinary bladder is catheterised and drained, and that a gastric tube has been passed to decompress the stomach.
2. Surgically prepare the abdomen with antiseptic solution and drapes.
3. Identify the site for incision – one-third of the way down from the umbilicus towards the pubis in the midline.
4. Anaesthetise the area to the peritoneum with 1% lidocaine and adrenaline.
5. Make a vertical incision through skin and subcutaneous tissue in the midline.
6. Incise the fascia.
7. Ensure haemostasis.
8. Apply two clips to the peritoneum and gently lift it away from underlying structures.
9. Using the scissors cut between the two clips – making a small hole in the peritoneum.
10. Insert the dialysis catheter through the hole, and gently advance it caudally into the pelvis.
11. Connect the dialysis catheter to a syringe and aspirate.
12. If blood is not aspirated, connect the catheter to the giving set and infuse 10 ml/kg of the warmed saline.
13. Use as indicated for dialysis or treating hypothermia (see Chapter 15).

22.7 CERVICAL SPINE IMMOBILISATION

All children with serious trauma must be treated as though they have a cervical spine injury. It is only when an adequate examination and history is taken or appropriate investigations have been performed that the decision to remove the cervical spine protection can be made. Specialist consultation may be needed prior to this decision. In-line cervical stabilisation should be continued until a hard collar has been applied. Children being transported between institutions may require additional immobilisation. This may involve head blocks, sand bags or a vacuum mattress.

Once the collar is in place, the neck is largely obscured. Before placing the collar look for the following signs quickly and without moving the neck:

- Distended veins.
- Tracheal deviation.

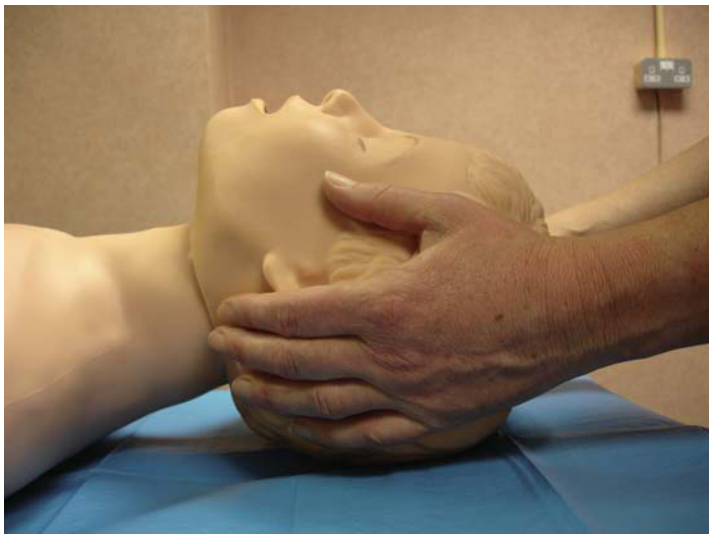


Figure 22.6 In-line cervical stabilisation

- Wounds.
- Laryngeal crepitus.
- Subcutaneous emphysema.

Application of a cervical collar

The key to successful, effective collar application lies in selecting the correct size.

Minimum equipment

- Measuring device.
- Range of paediatric hard collars.

Procedure

1. Ensure in-line cervical stabilisation is maintained throughout by a second person (Figure 22.6).
2. Using the manufacturer's method, select a correctly sized collar.
3. Fully unfold and assemble the collar.
4. Taking care not to cause movement, pass the flat part of the collar behind the neck.
5. Fold the shaped part of the collar round and place it under the child's chin.
6. Fold the flat part of the collar with its integral joining device (usually Velcro tape) around until it meets the shaped part.
7. Reassess the correct fit of the collar.
8. If the fit is wrong, slip the flat part of the collar out from behind the neck, taking care not to cause movement. Select the correct size and recommence the procedure.
9. If the fit is correct, secure the joining device.
10. Once the collar is fitted properly manual immobilisation may be discontinued.

Application of head blocks and tape

Equipment

- Two head blocks.
- Attachment system.

Procedure

1. Ensure in-line cervical stabilisation is maintained by a second person throughout.
2. Place a head block either side of the head.

3 Apply the forehead strap and attach it securely.
4 Apply the lower strap across the chin piece of the hard collar and attach it securely.
5 Apply tape across the chin piece of the hard collar and securely attach it to the long spinal board.

Exceptions
An injured child may be uncooperative for many reasons including fear, pain and hypoxia. Manual immobilisation should be maintained and the contributing factors addressed. Overzealous immobilisation of the head and neck may paradoxically increase the leverage on the neck as the child struggles. Children with traumatic torticollis should be manually immobilised in their current position.

Log-rolling

In order to minimise the chances of exacerbating unrecognised spinal cord injury, non-essential movements of the spine must be avoided until adequate examination and investigations have excluded it. If manoeuvres that might cause spinal movement are essential (e.g. during examination of the back in the course of the secondary survey), then log-rolling should be performed. The aim of log-rolling is to maintain the alignment of the spine during turning of the child. The basic requirements are an adequate number of carers and good control.

Procedure

1 Gather together enough staff to roll the child. In larger children four people will be required; three will be required in smaller children and infants (Figures 22.7 and 22.8).

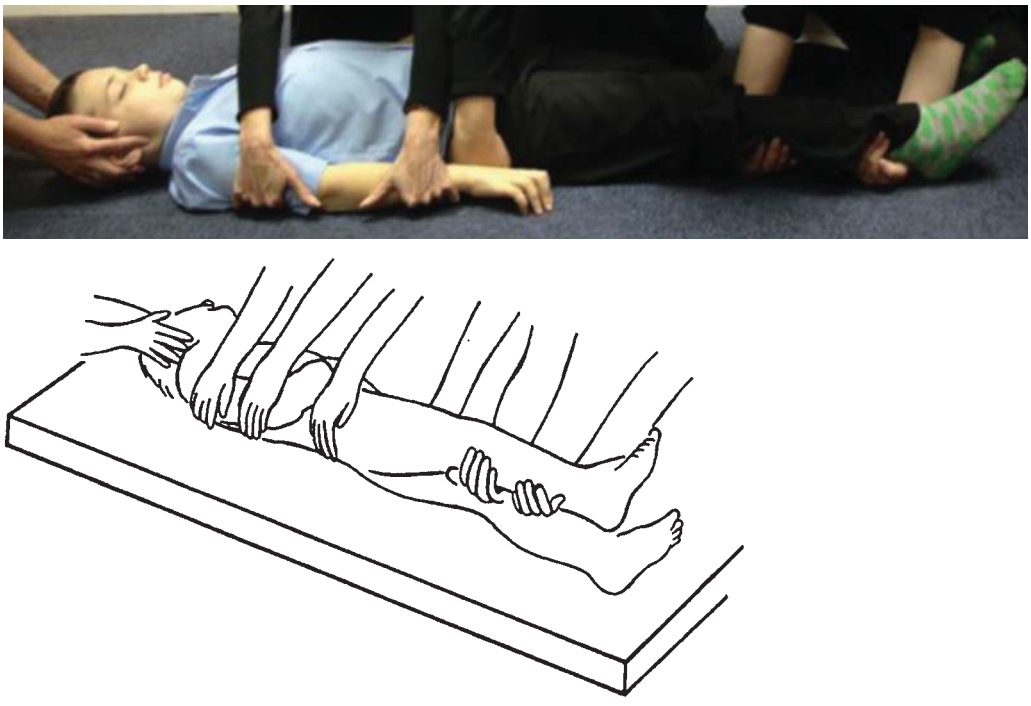


Figure 22.7 Log-rolling a child (four-person technique)

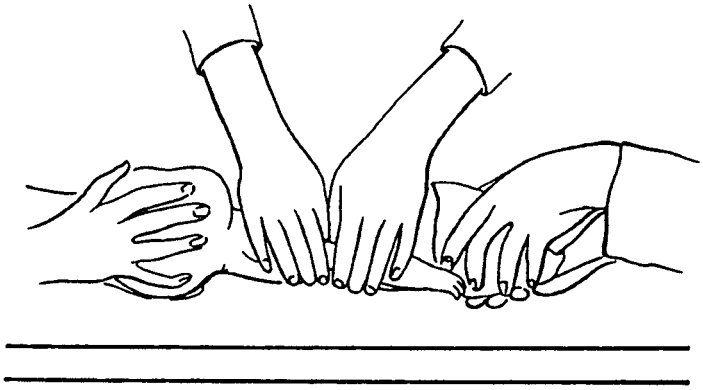


Figure 22.8 Log-rolling a small child or infant (three-person technique)

Table 22.1 Position of staff in log-rolling

	Position of staff for:	
Staff member no.	**Smaller child and infant**	**Larger child**
1	Head	Head
2	Chest	Chest
3	Legs and pelvis	Pelvis
4		Legs

2 Place the staff as shown in Table 22.1.
3 Ensure each member of staff knows what they are going to do, as shown in Table 22.2.
4 Carry out essential manoeuvres as quickly as possible.

22.8 HELMET REMOVAL

Cycle or motorcycle helmets must be removed without causing cervical spine movements. This requires a minimum of two staff.

Procedure

1 Obtain the history of the mechanism of injury.
2 Explain the procedure to the patient and parent(s).
3 Carry out a brief neurological examination.
4 Demonstrate the position of the hands on each side of the helmet with thumbs on the mandible and fingers on the occipital ridge. Keep this position whilst an assistant removes the chin straps.
5 Direct the assistant to take control of the in-line stabilisation by holding the occipital ridge with one hand and placing the thumb and forefinger of the other hand along the mandible.
6 Gently remove the helmet, spreading it laterally if necessary. Then resume in-line stabilisation with both thumbs on the mandible, and fingers of both hands on the occipital ridge.
7 Ensure removal of any jewellery from the neck.
8 Place the patient in a cervical collar. If the child is being transported between institutions you should also apply head blocks and straps and secure to the spinal board.
9 Carry out a brief neurological examination again.

Table 22.2 Tasks of individual members of staff

Staff member: position	Task
Head	Hold either side of the head (as for in-line cervical stabilisation) and maintain orientation of the head with the body in all planes during turning
	Control the log-roll by telling other staff when to roll and when to lay the child back onto the trolley
Chest	Reach over the child and carefully place both hands under the chest. When told to roll the child, support the weight of the chest and maintain stability. Watch the movement of the head at all times and roll the chest at the same rate
Pelvis and legs	*This only applies to smaller children and infants. If it is not possible to control the pelvis and legs at the same time, get additional help immediately*
	Place one hand either side of the pelvis over the iliac crests. Cradle the child's legs between the forearms. When told to roll the child, grip the pelvis and legs and move them together. Watch the movement of the head and chest at all times, and roll the pelvis and legs at the same rate
Pelvis	Place one hand over the pelvis on the iliac crest and the other under the top of the far leg. When told to roll the child, watch the movement of the head and chest at all times and roll the pelvis at the same rate
Legs	Support the weight of the far leg by placing both hands under it. When told to roll the child, watch the movement of the chest and pelvis and roll the leg at the same rate

CHAPTER 23

Interpreting trauma X-rays

23.1 INTRODUCTION

This chapter provides an overview of emergency imaging of the spine, chest and pelvis in children. It provides an introduction to interpretation for the clinician involved in managing paediatric trauma in the resuscitation room.

Radiological advice should be sought if there is any doubt that a film is normal. Discussing the film with an experienced emergency physician or trauma, orthopaedic or neurosurgeon may also help. An experienced emergency radiographer (technician) is a valuable asset to any department and, if they consider a film is abnormal, their comments should be carefully noted.

Radiography of a seriously injured child is technically challenging as access is often limited and films are often taken with a mobile machine. Equipment such as neck collars may obscure bony landmarks, and the position in which the child is lying may cause difficulty in radiographic interpretation due to rotation.

The radiology department is not a place to leave a sick or unstable patient without adequate clinical supervision. Plain films are taken by a radiographer, who will not be able to supervise an ill patient. Complex investigations including ultrasound scanning, computed tomography (CT) and contrast studies take time, during which the child may deteriorate significantly without appropriate treatment.

The commonest initial trauma films available to the emergency clinician include:

- Lateral cervical spine radiograph.
- Chest radiograph.
- Pelvic radiograph.

These three provide a basic screen for major injuries. They should only be taken after immediately life-threatening injuries have been identified and treated.

Viewing the film

Before reviewing any film, check the information shown in the box below.

- The name of the patient
- The date and time that the film was taken
- The orientation (side marker position)

The **ABCD** approach to radiographic interpretation is shown in the following box.

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Adequacy, **A**lignment and **A**pparatus
Bones
Cartilage and soft tissues
Disc spaces (in the spine), **D**iaphragm (in the chest)

23.2 CERVICAL SPINE

Cervical spine immobilisation should take place before any radiographs are performed. The standard film is a lateral radiograph, which may be supplemented by anteroposterior (AP) (lower cervical spine and odontoid peg views) radiographs when appropriate. If the child has an adequately fitted cervical collar for immobilisation, it is very difficult to get good quality AP views, including the odontoid peg. If sandbags rather than head blocks are used for immobilisation they may obscure bony landmarks.

Bony injury in itself is not the prime concern in spinal injury. The main risk is actual or potential injury to the cord. Any unstable fracture, if inadequately immobilised, may lead to progressive cord damage.

A lateral cervical spine film is often requested to 'clear' the cervical spine, but a normal film may be falsely reassuring. The plain film only shows the position of the bones at the time the film was taken, and gives no idea of the magnitude of flexion and extension forces applied to the spine at the time of injury. The cord may be injured even in a child without any apparent radiographic abnormality.

Unlike adult spines, most paediatric cervical spine injuries occur either through the discs and ligaments at the craniovertebral junction (C1, C2 and C3) or at C7/T1. The relatively large mass of the head, moving on a flexible neck with poorly supportive muscles, leads to injury in the higher cervical vertebrae.

Children develop three patterns of spinal injury:

1 Subluxation or dislocation without fracture.
2 Fracture with or without subluxation or dislocation.
3 Spinal cord injury without radiographic abnormality (SCIWORA).

The last of these, SCIWORA, is said to have occurred when radiographic films are totally normal in the presence of significant cord injury. If the film is normal in a conscious child with clinical symptoms (such as pain, loss of function or paraesthesia in a limb) then neck protection measures should be continued. In an unconscious child at high risk, a cord injury cannot be excluded until the patient is awake and has been assessed clinically, even in the presence of a normal cervical spine film. Adequate spinal precautions should be continued until the child is well enough to be assessed clinically, or magnetic resonance imaging (MRI) has been carried out.

The most common site of a 'missed' spinal injury is where a flexible part of the spine meets the fixed part. In the neck these are the cervicocranial junction and the cervicothoracic junction.

Adequacy

The whole spine should be viewed from the lower clivus down to the upper body of the T1 vertebra. If the C7/T1 junction is not seen initially then gentle traction should be applied by pulling the arms down, holding them above the elbow joint. If the child is conscious they should be asked to relax their shoulders as traction is applied. If the child is on a spinal board then this must be stabilised by an assistant.

Alignment

The four lines, three of which are shown in Figure 23.1, should be reviewed. These are:

1 The anterior vertebral line.
2 The posterior vertebral line (anterior wall of the spinal canal).

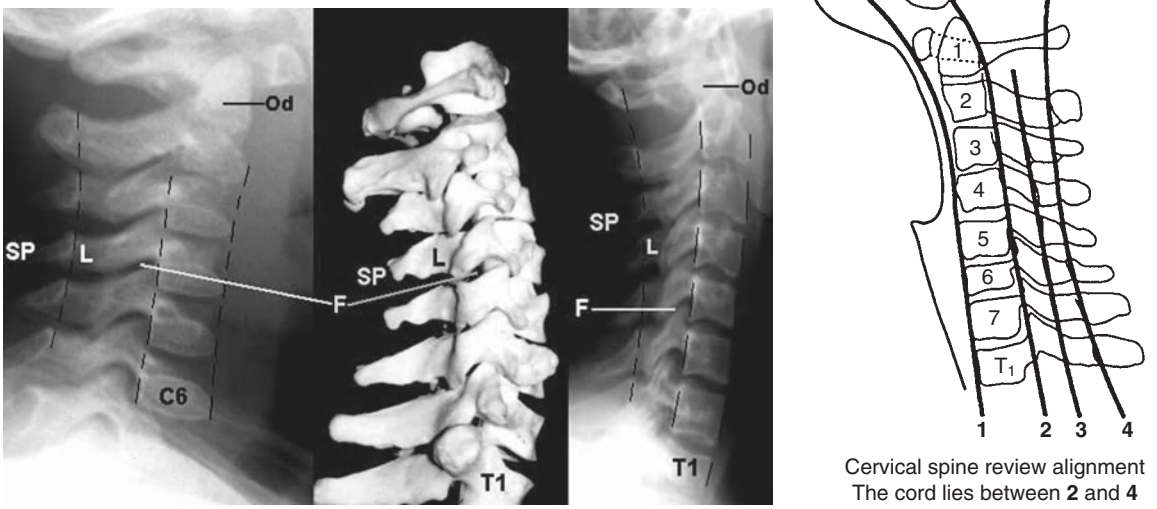


Figure 23.1 Lateral cervical spine showing the anatomy and three of the four review lines and disc spaces (facet joints and line not shown). F, facet joint; L, lamina; Od, odontoid; SP, spinous process spaces.

3 The facet line.
4 The spinolaminar line (posterior wall of the spinal canal).
The continuity of these lines should be maintained, no matter what the degree of flexion or extension seen on the neck film is. There should be no 'steps' or angulation.

The spinal cord lies in the canal between the posterior vertebral (2) and the spinolaminar (4) line. The former should line up with the clivus and the latter with the back of the foramen magnum.

Bones

The outline of each vertebra should be reviewed in turn. Fracture lines going through the cortex, vertebral bodies, laminae or spinous processes should be sought.

The spaces between the facet joints and the gaps between adjacent spinous processes should be similar (Figure 23.1).

The joint between the odontoid peg and the anterior arch of the atlas should be less than 3 mm in a child. This is illustrated in Figure 23.2.

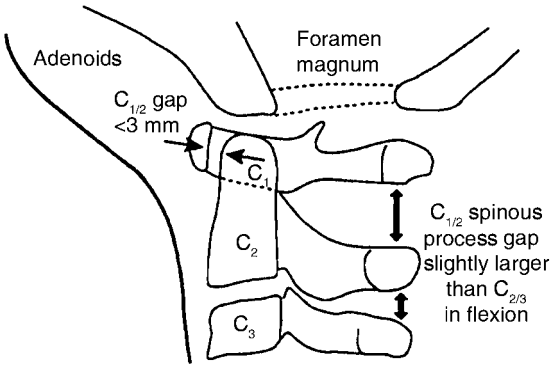
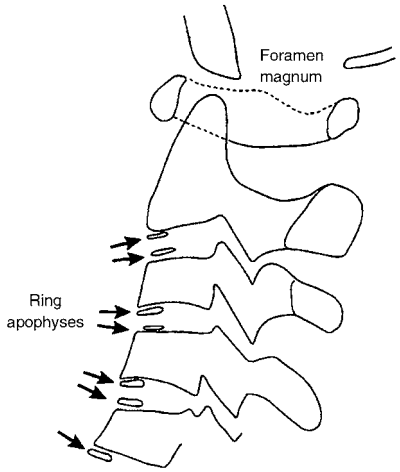


Figure 23.2 C1/C2 anatomy in an older child

The gap between the posterior arch of C1 and the spinous process of C2 may be slightly larger than the gaps at the other levels in flexion. The base of the odontoid peg may not be



They are parallel to the vertebral end plates
and with equal spacing from the vertebrae.

Figure 23.3 Ring apophyses in the adolescent spine

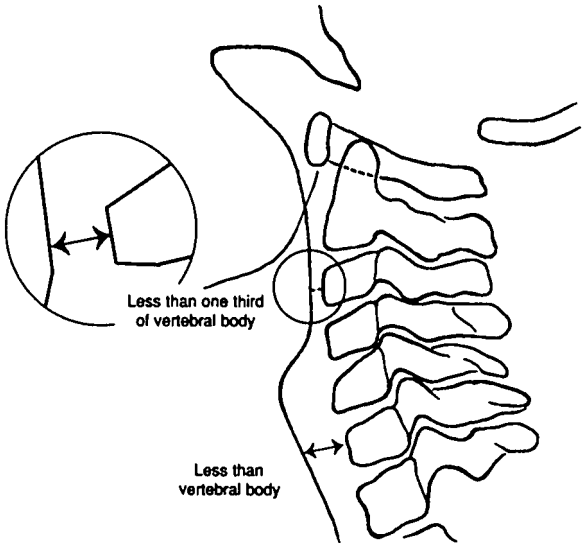


Figure 23.4 Lateral cervical spine – soft tissues (see also Figure 23.1)

completely fused onto the body of the axis (C2) in a small child, but the orientation of the odontoid peg should always be perpendicular to the body of C2.

In adolescence, ring apophyses are seen related to the vertebral bodies as shown in Figure 23.3. These are sometimes a site for fracture separation from the vertebral body. The appearances of the apophyses at each level should be compared with the vertebra above and below.

Cartilage and soft tissues

Abnormal widening of the prevertebral soft tissues may indicate a haematoma due to cervical spine injury. There may, however, be a significant spinal injury with normal soft tissues – thus the absence of soft tissue swelling does not exclude major bony or ligamentous injury. When a child is intubated, it is difficult to assess prevertebral soft tissue swelling. Small children have large adenoids, which are seen as well-demarcated soft tissue swelling at the base of the clivus. This is shown in Figure 23.4.

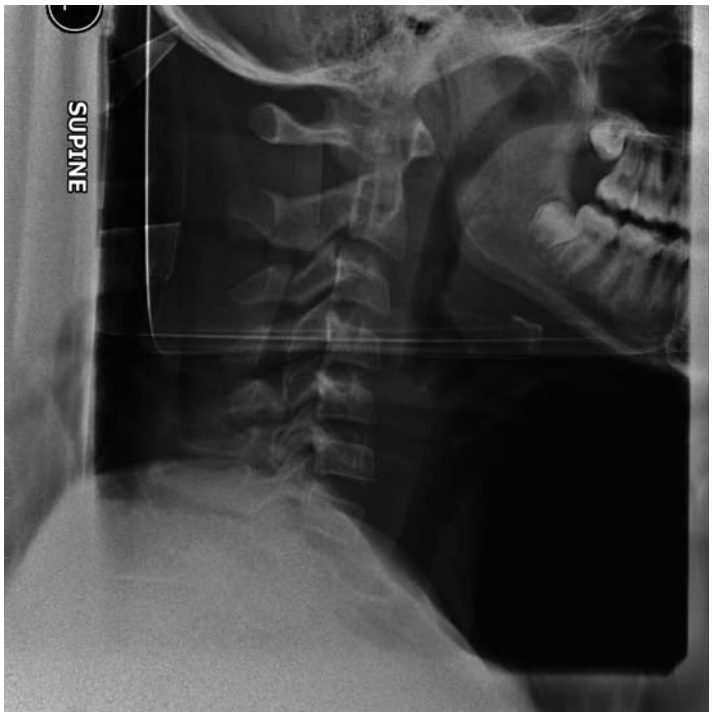


Figure 23.5 Lateral cervical spine: soft tissues (see also photograph in Figure 23.1)

Acceptable soft tissue thicknesses are:

- Above the larynx: less than one-third of the vertebral body width.
- Below the larynx: not more than one vertebral body width.

Below the level of the larynx the prevertebral soft tissues become progressively *narrower* towards the cervicothoracic junction (Figure 23.5). If the prevertebral soft tissues are wider at C7 than at the C5 level, then this suggests trauma at the C7/T1 level.

Any soft tissue swelling outside these limits should be regarded as abnormal and neck protection measures maintained until a further clinical and radiological opinion can be obtained. In small children the soft tissues may appear abnormally wide if the film is taken with the infant lying in flexion – if in doubt maintain the neck protection and ask for advice.

Discs

The height of the vertebral disc should be compared from C2/C3 to C7/T1. The discs should all be of similar height, as shown earlier in Figure 23.1.

Flexion and extension cervical spine films should never be performed in the acute trauma situation. Further imaging is obtained when the patient is stable, including CT to assess the bones or MRI for the spinal cord.

AP films of the cervical spine may be taken. The films should be reviewed using the same system as was used for the lateral cervical spine film:

Figure 23.6 (over page) shows five lines of alignment to assess. The spinal cord lies between lines 2 and 4.

23.3 CHEST RADIOGRAPH

Adequacy and alignment

Adequacy can be assessed by considering both penetration and the depth of inspiration. The film should be sufficiently penetrated to just visualise the disc spaces of the lower thoracic vertebrae through the heart shadow. At least five anterior rib ends should be seen above the diaphragm on the right side. An expiratory film may mimic consolidation.

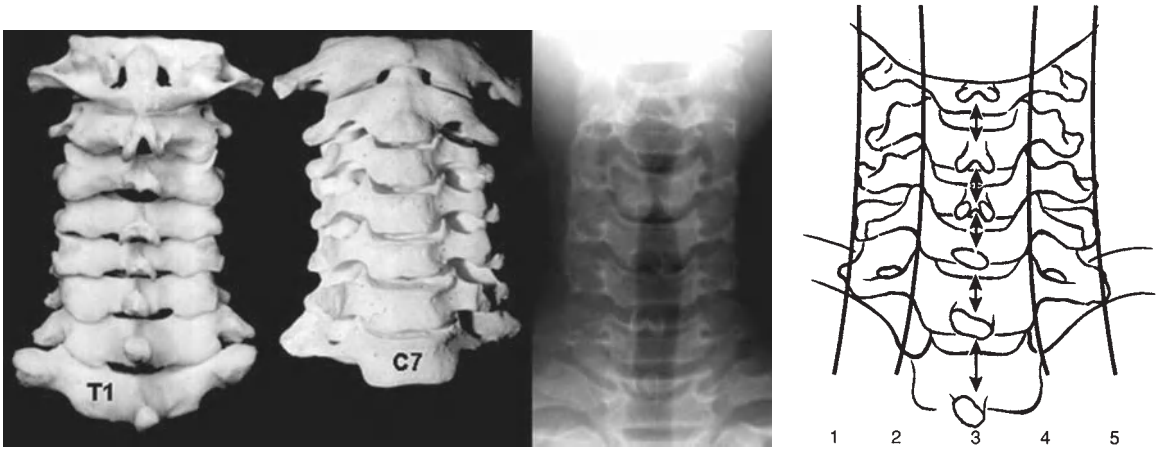


Figure 23.6 AP cervical spine showing the anatomy and five review lines, discs and spinous processes.

Alignment can be assessed by ensuring that the medial ends of both clavicles are equally spaced about the spinous processes of the upper thoracic vertebrae, as shown in Figure 23.7. Abnormal rotation may create apparent mediastinal shift. The trachea should be equally spaced between the clavicles.

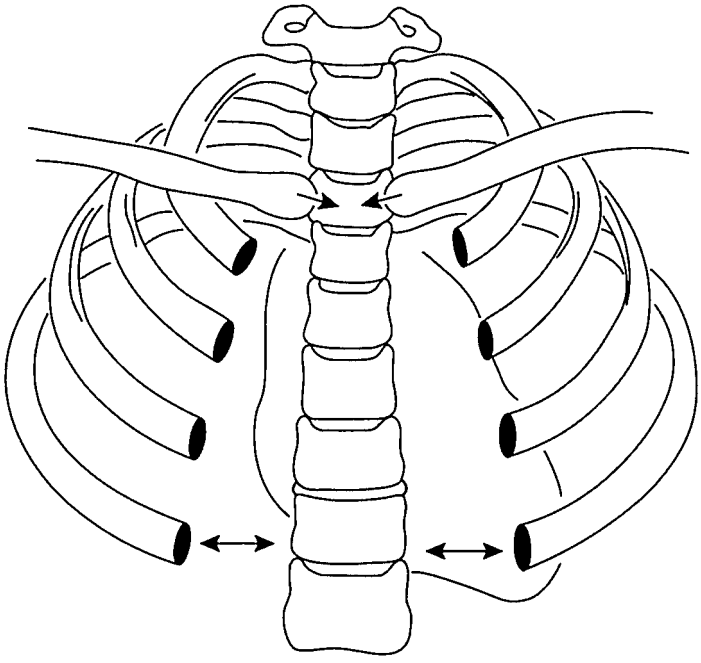


Figure 23.7 Assessing rotation – straight-chest film

Apparatus

Check the position of any apparatus, including:

- Tracheal tube.
- Central venous lines.
- Chest drains.

Misplacement of the endotracheal tube (ETT) should be evident clinically, but may be seen on a chest film if you look for it. Do this first when reviewing any chest X-ray on an intubated patient. Malposition of an ETT can result in reduced ventilation and hypoxia.

The ideal position for an ETT is below the clavicles and at least 1 cm above the carina. To find the carina, identify the slope of the right and left main bronchi – the carina is where the two lines meet in the midline.

Bones

The posterior, lateral and anterior aspects of each rib must be examined in detail. This can be done by tracing out the upper and lower borders of the ribs from the posterior costochondral joint to where they join the anterior costal cartilage at the mid-clavicular line. The internal trabecular pattern can then be assessed.

The ribs in children are soft and pliable and only break when subjected to considerable force. Even greater force is required to fracture the first rib or to break multiple ribs. Consequently, presence of these fractures should stimulate you to look for other sites of injury both inside and outside the chest.

Finish assessing the bones by inspecting the visible vertebrae, the clavicles, scapulae and proximal humeri.

Thoracic spine injuries may be overlooked on a chest radiograph. Abnormal flattening of the vertebral bodies, widening of the disc spaces or gaps between the spinous processes or pedicles may be seen. On AP views increased vertical or horizontal distances between the pedicles or spinous processes indicate an unstable fracture, as shown in Figure 23.8.

If there are rib fractures in the first three ribs, these may be associated with major spinal trauma and great vessel injury.

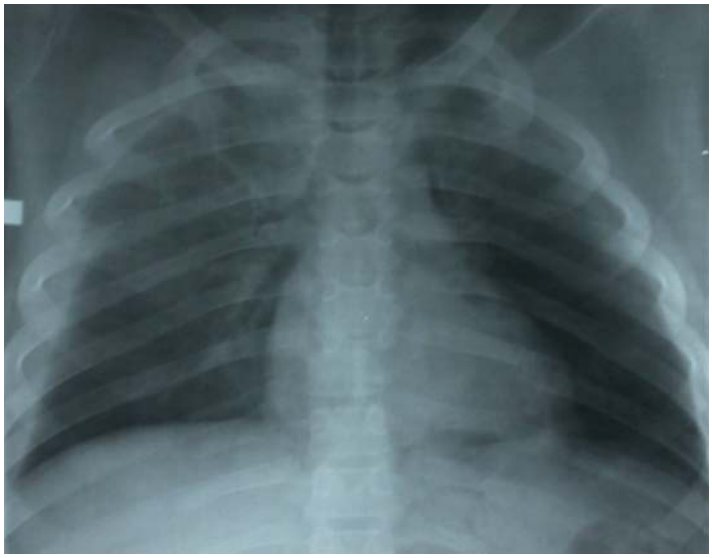


Figure 23.8 Vertical fracture of the thoracic spine

Cartilage and soft tissues

Lungs

In a well-centred film, the lungs should appear equally black on both sides. Compare the left and right lungs in the upper third, middle third and lower third of the chest.

Check that the lungs go all the way out to the rib cage – i.e. there is no pleural effusion or pneumothorax. A lung black on one side may be due to a pneumothorax or air trapping. A lung white on one side may be due to collapse, pulmonary haemorrhage, contusion or effusion (including haemothorax).

On the supine film, blood or fluid lies posteriorly, giving a generalised greyness to the lung, rather than the typical meniscus sign seen on the erect film. At the apex of each lung, an effusion displacing the lung down (apical cap) may indicate spinal injury or major vessel damage.

A suspected tension pneumothorax should be treated clinically in the emergency situation, without confirmatory X-ray. On a supine film, the air in a simple pneumothorax rises anteriorly and may only be evident from an abnormal blackness or 'sharpness' of the diaphragm or cardiac border. The standard appearances of a pneumothorax, where there is a sharp lung edge and the vessels fail to extend to the rib cage and the lung edges, may not occur in the supine film.

The heart

The cardiac outline should lie one-third to the right of midline and two-thirds to the left of midline. If the film is not rotated, then mediastinal shift is due to the heart being either pushed from one side or pulled from the other. For example, mediastinal shift to the left may be due either to a pneumothorax, air trapping or effusion on the right side, or to collapse of the left lung.

All trauma radiographs are taken in the supine position, often using portable X-ray machines. The tube is near to the patient and the heart is anterior with the film posterior. The heart in this situation appears abnormally magnified (widened) and the cardiothoracic ratio is difficult to assess on supine AP films.

The mediastinal cardiac outline should be clear on both sides. Any loss of definition suggests consolidation (de-aeration) of adjacent lungs. A 'globular' shape to the heart may suggest a pericardial effusion. Tamponade is managed clinically, not radiologically. A cardiac echo is useful in equivocal cases.

The upper mediastinum

In the teenager, the mediastinum should appear as narrow as in an adult. In children under the age of 18 months, the normal thymus may simulate superior mediastinal widening (above the level of the carina). A normal thymus may touch the right chest wall, left chest wall, left diaphragm or right diaphragm, making it very difficult to exclude mediastinal pathology. Fortunately, mediastinal widening due to aortic dissection or spinal trauma is very rare in small children.

In cases of doubt, where there is a normal clinical examination, an opinion from a radiologist should be sought. In the older child involved in trauma, mediastinal widening may mean aortic dissection, or major vessel or spinal injury. Ultrasound, CT or angiography may be required to resolve this when the child is stable.

Diaphragms

The cardiophrenic and costophrenic angles should be clear on both sides. The diaphragms should be clearly defined on both sides and the left diaphragm should be clearly visible behind the heart. Loss of definition of the left diaphragm behind the heart suggests left lower lobe collapse, an abnormal hump suggests diaphragmatic rupture and an elevated diaphragm suggests effusion, lung collapse or nerve palsy.

At the end of the systematic ABCD review of the X-ray, check again in the key areas shown in the box below.

- Behind the heart (left lower lobe consolidation or collapse)
- Apices for effusions, pneumothorax, rib fractures and collapse/consolidation
- Costophrenic and cardiophrenic angles – fluid or pneumothorax
- Horizontal fissure – fluid or elevation (upper lobe collapse)
- Trachea for foreign body (and ETT)

23.4 PELVIS

A single AP pelvic view is usually taken. As with other films this can be reviewed using the ABC approach.

Adequacy and alignment

Rotation of the pelvic film causes great difficulty in interpretation. In a non-rotated pelvic film the tip of the sacrum and spine will be aligned with the symphysis pubis.

The whole of the pelvis from the top of the iliac crests to the ischial tuberosities should be included, as should both hips, with the femoral necks shown to the level of the trochanters.

Bones

The pelvis is made up from the sacrum, innominate bones (iliac wings), ischium and pubic bones. These come together to form a Y-shaped cartilage in the floor of the acetabulum. In young children the joint between the ischium and the pubis (ischiopubic synchondrosis) is commonly seen and may simulate a fracture.

The pelvis is reviewed as a series of rings, including the pelvic brim, the two obturator rings and both acetabular fossae. These should appear smooth and symmetrical in a well-centred film. They are illustrated in Figure 23.9. Figure 23.10 (over page) shows a multiply fractured pelvis. The femoral necks must be checked for fracture and symmetry of the 'tear drop' gap.

Cartilage and soft tissues

Minor degrees of rotation, hip flexion or hip rotation will distort the fat plane and make assessment of soft tissue displacement difficult. Abnormal widening of the obturator fat pad may indicate a pelvic side wall haematoma.

The paediatric pelvis is held together by cartilage. Separation through the cartilage of the sacroiliac joint, the symphysis pubis or the 'Y' cartilage of the acetabular floor may occur without apparent bony injury. Comparison of both hips and sacroiliac joints on a well-centred film may show this. On a well-centred film the distance between the femoral head and the floor of the acetabular 'teardrop' should be symmetrical – it is abnormal in effusion or dislocation.

If further assessment is needed, a CT scan is performed after the patient is stabilised. Angiography may be needed for vessel injury, or cystourethrography to assess associated urethral or bladder damage.

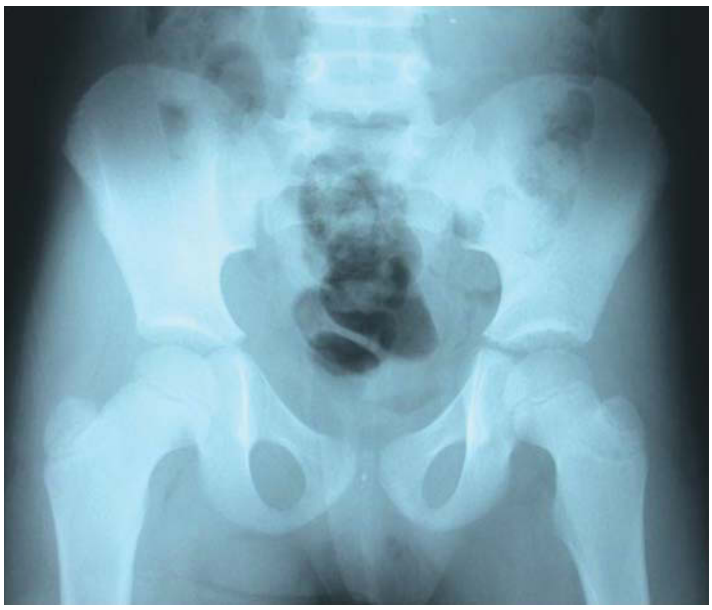


Figure 23.9 Normal, straight pelvis in a young child

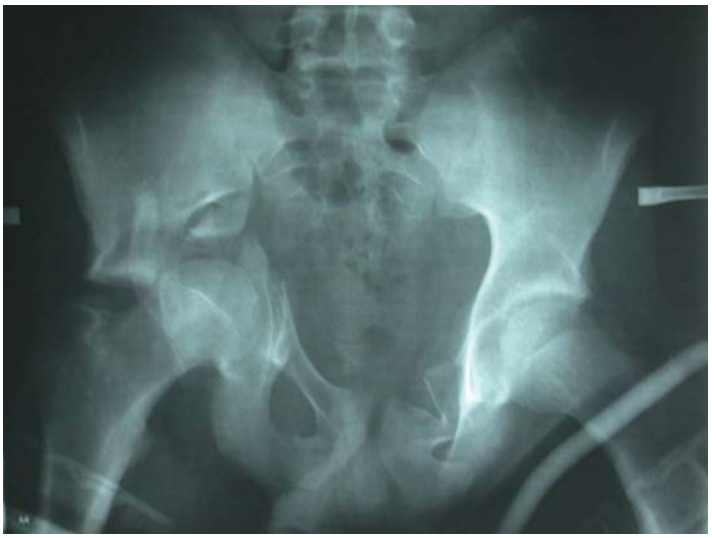


Figure 23.10 Multiple pelvic fractures

23.5 THE PLACE OF FURTHER IMAGING

Once the patient has been stabilised or resuscitated, further imaging of the brain and spine may be needed to exclude intracranial haemorrhage or spinal fracture. CT scanning is the first modality, but has a relatively high radiation dose.

NICE NHS guidance on head injuries

The National Institute for Health and Clinical Excellence (NICE) evidence is extrapolated from adult practice, as there are few studies to form a scientific basis for this guidance in the paediatric age group.

Plain X-rays of the cervical spine

- *Under 10 years*: AP views of the odontoid peg are difficult to obtain and are often obscured by overlying unerupted teeth. Request a lateral cervical spine and AP lower cervical spine.
- *Over 10 years*: request a lateral cervical spine, AP lower cervical spine and AP odontoid peg view.

CT of the cervical spine

If the plain films are unclear, or there is a high-risk mechanism of injury:

- *Under 10 years*: the recommendation is for CT of the upper cervical spine (from the occipital condyles and foramen magnum down to C3) – this covers the craniocervical junction. This is the most common site of fracture in this age group and it excludes the radiosensitive thyroid gland from the scan.
- *Over 10 years*: the recommendation is to image as adults – i.e. from the occipital condyles down to the C7/T1 junction.

Brain imaging

CT brain scanning

CT brain scanning is the prime modality for excluding acute intracranial haemorrhage. A child with clinical suspicion of intracranial bleeding requires a CT scan, not a skull film, as intracranial bleeding in children often occurs without a skull fracture. Before the child is sent into the radiology department for a scan, he or she must be resuscitated, stabilised and supervised at all times by a doctor or appropriately trained senior nurse.

If a child has an indication for a head CT and when the mechanism of injury might also have caused a neck injury, then the cervical spine is included at the same time (see CT of the cervical spine above).

MRI of the brain

This is not suitable for an acutely ill patient. Specific magnetic resonance-compatible anaesthetic equipment is needed and scanning takes much longer than CT. MRI is useful in assessing spinal cord injury, disc injury and soft tissue injury in the spine. It is a secondary imaging modality rather than a modality used in the acute phase.

Body imaging

There is often a temptation to scan the chest, abdomen and pelvis at the same time as the head. This extends the time a sick child spends in radiology and away from a high-dependency area. CT scanning is a high-radiation dose procedure, and the benefit from imaging multiple areas has to be carefully balanced against the time this takes and the radiation dose incurred. The decision to extend the scan to include the body must be based on clinical features and not requested simply 'whilst the child is being scanned'.

CHAPTER 24

Structured approach to stabilisation and transfer

24.1 STABILISATION OF THE CHILD

After successful resuscitation of the seriously ill or injured child, frequent clinical reassessment must be carried out to detect deterioration or improvement with therapy. All patients should have the following monitored:

- Oxygen saturation.
- CO_2 monitoring.
- Arterial pH and gases.
- Pulse rate and rhythm.
- Blood pressure.
- Urine output.
- Core temperature.
- Skin temperature.

Additionally, some patients will require:

- Invasive blood pressure monitoring.
- Central venous pressure monitoring.
- Intracranial pressure monitoring.

The facilities to measure some of these parameters may not be available until transfer to an intensive care setting.

The investigations shown in the box below should be considered following successful resuscitation or during subsequent stabilisation:

Post-resuscitation investigations

- Chest radiograph
- Arterial or central venous blood gasses
- Haemoglobin, haematocrit and platelets
- Group and save serum for cross-match
- Sodium, potassium, calcium, urea and creatinine
- Clotting screen
- Blood glucose
- Liver function tests
- Urinalysis, microscopy and culture
- Culture of blood and, if indicated, cerebrospinal fluid
- C-reactive protein or procalcitonin

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Children who have been resuscitated from cardiorespiratory arrest may die hours or days later from multiple organ failure. In addition, to the cellular and homeostatic abnormalities that occur during the preceding illness, and during the arrest itself, cellular damage continues after spontaneous circulation has been restored. This is called reperfusion injury and is caused by the following:

- Depletion of adenosine triphosphate (ATP).
- Entry of calcium into cells.
- Free fatty acid metabolism activation.
- Free-radical oxygen production.

Similarly, children resuscitated with serious illness or injury may suffer multisystem dysfunction as a result of hypoxia or ischaemia. Ongoing activation of inflammatory mediators, as occurs in serious sepsis, also contributes to multisystem organ failure. Post-resuscitation management aims to achieve and maintain homeostasis in order to optimise the chances of recovery. Management should be directed in a systematic way.

Airway and breathing

Seriously ill children often exhibit an impaired conscious level and a depressed gag reflex. Intubation should always be considered and will usually have occurred during resuscitation.

- The endotracheal tube should have a minimal leak, be appropriately secured and ventilation should be monitored by continuous capnography.
- Ventilation settings should be maintained to keep blood gases normal (PCO_2 4.5–6.0 kPa). Children with intracranial injuries should have their PCO_2 maintained as close to 4.5 kPa as possible. The rational for this is to minimise cerebral oedema and minimise any increase in intracranial pressure. If this is difficult because of airway or lung pathology, urgent advice should be obtained from a paediatric intensivist.
- In all non-cardiac cases, sufficient inspired oxygen should be given to maintain SpO_2 at between 94% and 98%. If anaemia or carbon monoxide poisoning is suspected inspired oxygen should be delivered at the highest possible concentration irrespective of the SpO_2 value. Specialist advice regarding target saturations should be sought from a paediatric intensivist or cardiologist for all children where a cardiac abnormality is either known or suspected.

Circulation

Following resuscitation, there will often be a poor cardiac output. This may be due to any combination of the following factors:

- An underlying cardiac abnormality.
- The effects on the myocardium of hypoxia, acidosis and toxins, preceding and during any arrest.
- Continuing acid–base or electrolyte disturbance.
- Hypovolaemia.

The following steps should be taken if there are signs of poor perfusion:

- Assess cardiac output clinically.
- Infuse crystalloid or colloid in aliquots of 10 ml/kg and reassess cardiac output clinically.
- Aim for a normal arterial pH (>7.3) and good oxygenation. This may require the use of inotropic drug support with or without further fluid boluses.
- Monitor ventilation and intervene with support when inadequate.
- Identify and start to correct hypoglycaemia and electrolyte abnormalities.

A central venous pressure (CVP) line will give useful information about the preload to the heart. This may assist in decisions about fluid infusion and inotropic support. A target value for the CVP should be discussed and established with an expert team.

The CVP is best used in assessing the response to a fluid challenge. In hypovolaemic patients, CVP alters little with a fluid bolus, but in euvolaemia or hypervolaemia it usually shows a sustained rise.

Drugs used to maintain perfusion following cardiac arrest or treatment of shock

There are no research data comparing one drug to another that show an advantage of any specific drug on outcome. In addition, the pharmacokinetics of these drugs vary from patient to patient and even from hour to hour in the same patient. Factors that influence, in an unmeasurable manner, the effects of these drugs include the child's age and maturity, the underlying disease process, metabolic state, acid–base balance, the patient's autonomic and endocrine response, and liver and renal function. Therefore, the recommended infusion doses are starting points, the infusions must be adjusted according to patient response.

Dopamine

Dopamine is an endogenous catecholamine with complex cardiovascular effects. At low infusion rates (1 ml/h = 5 micrograms/kg/min) dopamine may increase renal perfusion and has little effect on systemic haemodynamics. At infusion rates greater than 5 micrograms/kg/min, dopamine directly stimulates cardiac β-adrenergic receptors and releases noradrenaline from cardiac sympathetic nerves. Myocardial noradrenaline stores may be low in chronic congestive heart failure and in infants, so the drug may be less effective in these groups.

Dopamine is generally used in preference to dobutamine (see below) in the treatment of circulatory shock following resuscitation.

Dopamine infusions may produce tachycardia, vasoconstriction and ventricular ectopy. Infiltration of dopamine into tissues can produce local tissue necrosis. Dopamine and other catecholamines are partially inactivated in alkaline solutions and therefore should not be mixed with sodium bicarbonate.

Infusion concentration: 15 mg/kg in 50 ml of 5% dextrose or normal saline will give 5 micrograms/kg/min if run at 1 ml/h.

For a dose of 5–20 micrograms/kg/min, give 1–4 ml/h of the above dilution.

Dobutamine

Dobutamine increases the heart rate and myocardial contractility and has some vasodilating effect by decreasing peripheral vascular tone. It is potentially useful in the treatment of low cardiac output secondary to poor myocardial function. It is infused in a dose range of 5–20 micrograms/kg/min. Higher infusion rates often produce tachycardia or ventricular ectopy. Pharmacokinetics and clinical response vary widely, so the drug must be titrated according to individual patient response.

Infusion concentration: 15 mg/kg in 50 ml of 5% dextrose or normal saline will give 5 micrograms/kg/min if run at 1 ml/h.

For a dose of 5–20 micrograms/kg/min give 1–4 ml/h of the above dilution.

Adrenaline

An adrenaline infusion is a good first-line treatment for shock with poor systemic perfusion from any cause that is unresponsive to fluid resuscitation. Adrenaline may be preferable to dopamine or dobutamine in patients with severe hypotensive shock and in very young infants in whom other inotropes may be ineffectual. The infusion is started at 0.05–0.1 micrograms/kg/min and may be incrementally increased to 1 microgram/kg/min or higher depending on clinical response. Wherever possible adrenaline should be infused into a central intravenous or intraosseous line. Peripheral lines should only be used if there is no alternative because tissue infiltration can cause local ischaemia and ulceration.

Infusion concentration: 0.3 mg/kg in 50 ml of 5% dextrose or normal saline will give 0.1 micrograms/kg/min if run at a rate of 1 ml/h.

For a dose of 0.05–2.0 micrograms/kg/min, give 0.5–20 ml/h of the above dilution.

Kidneys

It is important both to maximise renal blood flow and to maintain renal tubular patency by maintaining urine flow. To achieve this, the following are necessary:

- Maintenance of an adequate blood pressure to drive renal perfusion.
- Maintenance of adequate filling and a good cardiac output using inotropes and fluids as required.
- Maintenance of good oxygenation.
- Diuretics (e.g. frusemide (furosemide) 1 mg/kg) may be utilised in children with a good cardiac output to maintain urine output at or above 1 ml/kg/h. However, great care must be exercised as excessive diuretics can cause further damage to the failing kidney. Wherever possible seek specialist advice before administration.
- Monitoring and normalisation of electrolytes (sodium, potassium, calcium, magnesium) and acid–base balance in blood should be undertaken as a supporting measure. Sodium bicarbonate should not usually be given to unintubated patients. Potassium should be given slowly and cautiously and only be given in small aliquots in oliguric or anuric patients.

Liver

Hepatic cellular damage can become manifest up to 24 hours following an arrest. Coagulation factors can become depleted, and bleeding may be worsened by concomitant, ischaemia-induced intravascular coagulopathy. The patient's clotting profile and platelets should be monitored and corrected, as indicated, with fresh frozen plasma, cryoprecipitate or platelets.

Brain

The aim of therapy is to protect the brain from further (secondary) damage. To achieve this, the cerebral blood flow must be maintained, normal cellular homeostasis must be achieved and cerebral metabolic needs must be reduced.

When intracranial pathology is present, cerebral autoregulation may not function correctly. In these circumstances adequate cerebral blood flow may be achieved if the cerebral perfusion pressure (mean arterial pressure – intracranial pressure) is kept above 50 mmHg. Maintenance of cellular homeostasis is helped by normalisation of the acid–base and electrolyte balances. Cerebral metabolic needs can be reduced by sedating and paralysing the child. Convulsions should prompt an investigation into their cause. They should be swiftly controlled and prophylactic medication, such as phenytoin, should be used if recurrent. Although a barbiturate coma reduces both cerebral metabolism and intracranial pressure, it has not been shown to improve neurological outcome.

Practical steps to minimise secondary brain injury are:

- Maintenance of good oxygenation.
- Protection of cerebral perfusion through:
  - maintenance of adequate blood pressure using inotropes and fluids,
  - intubation and maintenance of normal blood gases, and
  - nursing head-up at 20° and in midline.
- Using osmotic agents for acutely raised intracranial pressure such as:
  - mannitol 250–500 mg/kg IV over 15 minutes, or
  - hypertonic (2.7) 3% saline 3 ml/kg IV over 15 minutes.
- Control of blood glucose avoiding both hypoglycaemia and hyperglycaemia.
- Maintenance of good analgesia, sedation and paralysis (where indicated).
- Monitoring and normalisation of electrolytes and acid–base balance.
- Control of seizures.

Although there is some evidence that post-arrest hypothermia (core temperatures of 32–34°C) has beneficial effects on neurological recovery in adults, the evidence does not support the use of hypothermia in children outside the newborn period. Harm may occur, however, with raised core temperature, which increases metabolic demand by 10–13% for each degree centigrade increase in temperature above normal. Therefore, in the post-arrest patient with compromised cardiac output, hyperthermia should be treated with active cooling to achieve a normal core temperature. Shivering should be prevented since it will increase metabolic

demand. Sedation may be adequate to control shivering, but neuromuscular blockade is usually needed.

24.2 ASSESSMENT AFTER STABILISATION

After resuscitation and emergency treatment have been provided, consideration will need to be given to the best place to continue the child's care. This will usually involve a transfer to another unit, often another hospital. Critically ill children transferred by untrained personnel have been shown to be subjected to an excess of adverse events. Often a patient needs to be transported from the emergency department to another department within the same hospital. These transfers have also been associated with a high incidence of serious transport-related adverse events. The impact of these events on long-term outcome is unknown. Nevertheless, international practice has focused on minimising adverse events during transfer.

In the United Kingdom, the Paediatric Intensive Care Society has set a standard of practice for the transport of critically ill children. Where possible it is recommended that these are undertaken by specialised paediatric intensive care transfer teams. These teams can be contacted in the event of requests for transfer of a child to a paediatric intensive care unit (PICU) or a specialised facility such as a neurosurgical or burns unit. *Paediatric and Neonatal Safe Transfer and Retrieval: the Practical Approach* (ALSG, 2008) is a sister publication to this manual that supports a practical course of the same name. Although the interested reader should consider reviewing this text in detail, a summary of the principles described are detailed below.

24.3 PRINCIPLES OF SAFE TRANSFER AND RETRIEVAL

Transfers are undertaken to ensure that the child's care is of the highest possible standard at all times. To achieve this, the *right* child has to be taken at the *right* time, by the *right* people, to the *right* place, by the *right* form of transport, and receive the *right* care throughout. This requires a systematic approach that incorporates a high level of planning and preparation before the child is moved. One such approach is the ACCEPT method, which is described in detail in *Paediatric and Neonatal Safe Transfer and Retrieval: the Practical Approach* (ALSG, 2008).

The systematic approach to transfer of a child

A	**A**ssessment
C	**C**ontrol
C	**C**ommunication
E	**E**valuation
P	**P**reparation and packaging
T	**T**ransportation

Assessment

When commencing the transfer process a formal (re)assessment of the situation must be undertaken. Sometimes the clinicians undertaking the transportation may have been involved in the care given up to that point. Increasingly, however, the transport team will have been brought in specifically for that purpose and will have no prior knowledge of the child's clinical history. The process of assessment and reassessment continues throughout the time of the transfer, continually monitoring for changes in the child's condition and taking remedial action where appropriate (see below).

Control

Once the initial assessment is complete, the transport organiser needs to take control of the situation. This requires:

- Identification of the clinical team leader.
- Identification of the tasks to be carried out.
- Allocation of tasks to individuals or teams.

The lines of responsibility must be established promptly. In theory, ultimate responsibility is held jointly by the referring consultant clinician, the consultant clinician at the receiving centre and the transport personnel at different stages of the transport process. There should always be a clearly identified person with overall responsibility for organising the transport.

Communication

Moving ill children from one place to another requires cooperation and the involvement of many people. Key personnel need to be informed when transportation is being considered.

People who need to know about a transfer

Current (local) clinical team:

- Consultant in charge
- Clinicians at bedside
- Referring doctor/nurse
- Lead nurse

Transfer team

The transfer coordinator should disperse information to:

- Consultant in charge
- Clinician(s) undertaking transfer
- Ambulance providers

Receiving team

The transfer coordinator or receiving unit coordinator should disperse information to:

- Consultant accepting referral
- Other consultants who will need to be involved in care (PICU, surgical and anaesthetic teams)
- Receiving doctors
- Receiving nursing staff
- Child's relatives

Communication may take a long time to complete if one person does it all. It is therefore advisable to share the tasks between appropriate people, taking into account expertise and local policies. In all cases it is important that information is passed on clearly and unambiguously. This is particularly the case when talking to people over the telephone. It is useful to plan what to say before telephoning and to use the systematic summary shown below. It is also useful in complex conversations to summarise the situation and repeat what you need from the listener at the end.

The content of all discussions should be documented in the child's notes. The person in overall charge can then assimilate this information so that a proper evaluation of the child's requirements for transportation can be made.

Key elements in any communication
- Who you are
- What is needed (from the listener)
- What are the (relevant) child's details
- What the problem is
- What has been done to address the problem
- What happened
- What needs to be done next

Evaluation

The aim of evaluation is to confirm that transfer is appropriate for the child and, if so, what the clinical urgency is. Whilst evaluation is a dynamic process that starts from first contact with the child, it is usually only when the first phase of ACCEPT (that is, ACC) has been completed that enough information will have been gathered to fully evaluate the transport needs.

Is transport appropriate for this child?

Critically ill babies and children require transport because of the need for:
- Specialist treatment.
- Specialist investigations that are unavailable in the referring hospital.
- Specialist facilities that are unavailable in the referring hospital.

The risks involved in transport must be balanced against the risks of staying and the benefits of care that can be given only by the receiving unit.

What clinical urgency does this child have?

Once it has been established that transfer is needed, the urgency must be evaluated. The degree of urgency for transfer and the severity of illness may be used to rank the child's transfer needs. This decision will determine both the personnel required and the mode and speed of transport.

Preparation and packaging

Both preparation and packaging have the aim of ensuring that transport proceeds uneventfully, with no deterioration in the child's condition. The first stage (preparation) involves the completion of stabilisation and preparation of transfer team personnel and equipment. The second stage (packaging) involves the final measures that need to be taken to ensure the security and safety of the child, equipment and staff during the transportation itself.

Child preparation

To reduce complications during any journey, meticulous resuscitation and stabilisation should be carried out before transfer. This may involve carrying out procedures requested by the receiving hospital or unit. The standard airway, breathing and circulation (ABCDE) approach should be followed. The airway must be cleared and secured and appropriate respiratory support established. Venous access is essential and should preferably include a minimum of two easily accessible cannulae or a sutured multilumen central line. The child must have received adequate fluid resuscitation to ensure optimal tissue oxygenation. Hypovolaemic children tolerate the inertial forces of transportation very poorly. Children with a suspected spinal injury should be appropriately immobilised.

Occasionally, in time-critical situations such as an expanding intracranial lesion requiring neurosurgery, this process may not be fully completed before packing and transport. Decisions to transfer in these circumstances should be taken only by senior personnel.

Inadequate resuscitation or missed illnesses (and injuries) may result in instability during transfer and may adversely affect the child's outcome.

Equipment preparation

All equipment must be tested and have adequate power reserves. Supplies of drugs and fluids should be more than adequate for the whole of the intended journey. The essential items of equipment are shown in the box below.

Paediatric transport equipment

Airway

- Induction drugs for re-intubation
- Oropharyngeal airways: sizes 000, 00, 0, 1, 2, 3 and 4
- Tracheal tubes sizes 3.5–8.0 mm cuffed (in 0.5 mm steps) and 2.5–6.0 uncuffed
- Tracheal tube stylets
- Laryngeal masks sizes
- Laryngoscope handles × 2:
  - straight paediatric blades
  - curved blades
- Magill forceps
- Portable suction unit
- Yankauer suckers: paediatric and adult
- Soft suction catheters: sizes 6, 8, 10 and 12
- Humidity moisture exchange (HME) unit
- Needle cricothyroidotomy set

Breathing

- Oxygen masks with reservoir
- Self-inflating bags (with reservoir): sizes 240 ml (for pre-term infants only), 500 and 1600 ml
- Portable ventilator
- Face masks:
  - infant – circular 01, 1, 2
  - child – anatomical 2, 3
  - adult – anatomical 4, 5
- Catheter mount and connectors
- Ayre's T-piece

Circulation

- ECG monitor – defibrillator (with paediatric pads)
- Invasive and non-invasive (oscillometric) blood pressure monitor (with appropriate-sized cuffs)
- Pulse oximeter (with infant- and child-sized probes)
- End-tidal CO_2 monitor

Usually the four monitors above will be combined within one monitoring device, which will also include temperature and pressure channels

- Intravenous access requirements:
  - intravenous cannulae (as available) 18–25 gauge
  - intraosseous infusion needles 16–18 gauge
  - graduated burette
  - intravenous giving sets
  - syringes 1–50 ml
  - three-way taps, Luer-locking T-extensions, etc.
- Intravenous drip monitoring device/syringe pumps
- Central (or umbilical for newborns) and arterial line sets

Continued

Fluids
- 0.9% saline
- Hartmann's solution or Ringer's lactate
- 0.45% saline and 5% dextrose
- 10% dextrose
- Colloid

Drugs
- Adrenaline 1:10,000
- Adrenaline 1:1000
- Atropine 600 micrograms/ml or 1 mg/ml
- Sodium bicarbonate 4.2%
- Dopamine 40 mg/ml
- Lidocaine 1%
- Amiodarone
- Calcium chloride 10%
- Furosemide (frusemide) 20 mg/ml
- Mannitol 10% or 20%
- Antibiotics: penicillin, gentamicin, ampicillin, cefotaxime, cefuroxime
- Morphine, benzodiazepine and paralysing agent, made up as infusions

Miscellaneous
- Battery-operated suction device
- Nasogastric tubes: sizes 6, 8 and 10
- Chest drain set
- Stick test for glucose
- Sharps disposal box

Particular care should be taken with supplies of oxygen, inotropes, sedative drugs and batteries for portable electronic equipment. An example oxygen calculation is shown below:

Calculate the amount of oxygen required for the journey using the following:

$$\text{Number of cylinders} = \frac{2 \times \text{duration of journey} \times \text{flow [l/min]}}{\text{cylinder capacity [litres]}}$$

For example, if oxygen is provided at 10 l/min for a journey intended to take 120 minutes, this would need four size E cylinders, each containing 600 l. This allows for at least twice as much oxygen as the estimated journey time requires. Always take more than one cylinder in case of failure.

A member of the team should be allocated the task of ensuring that all of the child's documents, including case notes, investigations, radiographs, reports and a transfer form, accompany the child. The team should carry a mobile phone together with contact names and numbers to enable direct communication with both the receiving and base units. In addition, all personnel need appropriate clothing, food if the journey is long and enough money to enable them to get home independently if needed.

Personnel preparation

The number and nature of staff accompanying children during transport will depend on their transfer category. All staff must practise within their competences. Whatever the category of

the child, all personnel should be familiar with the relevant transfer procedures and the equipment that is to be used, as well as the details of the child's clinical condition. The team should be covered with accident insurance with adequate provision for personal injury or death sustained during the transfer.

Packaging

All lines and drains must be secured to the child, the child must be secured to the trolley and the trolley must be secured to the transport vehicle. This is especially important in neonatal transfers using a transport system that typically weighs over 100 kg. In an ambulance, all equipment fastenings should be CEN compliant. Chest drains should be secured and unclamped, with any underwater seal devices replaced by an appropriate flutter valve system. A special kit should be prepared to enable chest drain insertion or replacement *en route* if necessary. The child should be adequately covered to prevent heat loss. Care must be taken to ensure that coverings are arranged to permit ready access to the child, lines and drains during transfer.

Transportation

Mode of transport

The choice of transport needs to take into account several factors.

Factors affecting mode of transfer

- Nature of illness
- Urgency of transfer
- Mobilisation time
- Geographical factors
- Weather
- Traffic conditions
- Cost

Road ambulances are by far the most common means of transport used in the UK. They have a low overall cost and rapid mobilisation time, and are not generally affected by weather conditions. They also give rise to less physiological disturbance. Air transfer may be preferred for journeys of more than 80 km or 2 hours in duration, or if road access is difficult. The speed of the journey itself has to be balanced against organisational delays and also the need for inter-vehicle transfer at the start and end of the journey. Staff undertaking air transfers should have received specific training with regard to safety and flight physiology. They should not undertake such transfers without supervised experience.

Care during transport

Destabilisation may occur during transportation and may arise from the effects of the transport environment on the vulnerable physiology of the child. Careful preparation can minimise the deleterious effects of inertial forces, such as tipping, acceleration and deceleration, as well as changes in temperature and barometric pressure.

The standard of care and the level of monitoring carried out before transfer needs to be continued, as far as possible, during the transfer. Monitoring should include:

- Oxygen saturation.
- Electrocardiogram (ECG) and heart rate.
- Continuous intra-arterial pressure.
- End-tidal carbon dioxide in all intubated children and neonates.
- Core and ambulance temperature.

The child should be well covered and kept warm during the transfer.

Road speed decisions depend on clinical urgency. Although blue lights and sirens may be appropriate in order to get through heavy traffic, excessive speed is very rarely indicated. It is a risk to the child, the transfer team and the general public, and should be the exception rather than the rule.

With adequate preparation, the transportation phase is usually incident free. However, untoward events do occur. Should this be the case, the child needs to be reassessed using the ABC approach and appropriate corrective measures then instituted. If the transport team need to release their seatbelts, the ambulance must slow down immediately, and then stop at the first available safe place. If a major deterioration occurs, transfer to the nearest hospital for further stabilisation and support may be appropriate. The benefits of intervention should always be weighed against the risks of delaying arrival at the receiving hospital with its better facilities. Following any untoward events, communications with the receiving unit are important. This should follow the systematic summary described earlier.

Handover

At the end of the transfer direct contact with the receiving team must be established. A succinct, systematic summary of the child and transfer should be provided before transferring the child on to the local bed/cot. It must be accompanied by a written record of the child's history, vital signs, therapy and significant clinical events during transfer. All the other documents that have been taken with the child should also be handed over. Once verbal handover has been completed the child may be moved with monitoring and ventilator from the transport trolley to the receiving unit's cot or bed. The team can then retrieve all of their equipment and personnel and make their way back to their home unit.

24.4 SUMMARY

Meticulous attention to initial assessment and resuscitation together with appropriate emergency treatment will reduce the risk of transport-related morbidity and mortality. Where possible a specialised paediatric transport team should transfer critically ill and injured children to minimise the risk of adverse events. Irrespective of the origin of the transferring team a useful checklist is shown in the box on the facing page.

Checklist prior to transporting a child

1. Is the airway protected and is ventilation satisfactory? (Substantiated by blood gases, pH, pulse oximetry and capnography if possible)
2. Is the neck properly immobilised?
3. Is there sufficient oxygen available for the journey?
4. Is vascular access secure and will the pumps in use during transport work by battery?
5. Have adequate fluids been given prior to transport?
6. Is the child receiving adequate sedation, analgesia and, if used, paralysis?
7. Are fractured limbs appropriately splinted and immobilised?
8. Are appropriate monitors in use?
9. Will the child/baby be sufficiently warm during the journey – ambulance heater, head coverings for patient, etc?
10. Is documentation available? Include:
    - child's name
    - age and date of birth
    - known or estimated weight
    - clinical notes
    - observation charts, including neurological charts
    - time and route of all drugs given
    - fluid charts
    - ventilator records
    - results of investigations, including blood, urine, x-rays and scans
    - names and contacts of medical and nursing staff involved in referral, receipt and during transport
11. Is the necessary resuscitation equipment available?
12. Is appropriate treatment available for managing emergencies, e.g. rising intracranial pressure?
13. Has the case been discussed with the receiving team directly?
14. Has the receiving unit been advised of an estimated time of arrival?
15. Have plans been discussed with the parents, including issues of consent?

CHAPTER 25

Human factors

Error is inevitable, but harm is not
Sarah Corcoran, Associate Director of Clinical Effectiveness, Central Manchester Foundation Trust, 2009

25.1 INTRODUCTION

This manual is focused on the assessment and management of acute patient care. When clinicians utilise their knowledge and skills, they will often be caring for a deteriorating patient or one that has already collapsed. Usually the provision of patient care will be one of several concurrent responsibilities.

In an ideal world each patient would be managed by a dedicated, focused, mentally and physically fit and smoothly functioning team, that does not have competing commitments and is not interrupted with questions or requests not relevant to the patient or to the team function. In practice, it is clear we cannot easily and safely achieve this ideal. However, we can learn to work in a way that optimises the quality of the care we deliver.

There are a huge number of intrinsic and extrinsic factors that affect the performance of individuals and teams working in complex, high-pressure environments. Some 20 years ago the aviation industry started to recognise that knowledge of these factors, and how they impact on human performance, was critical to the maintenance of flight safety. Today, all airline staff undergo a rigorous *human factors* training programme that equips them with the tools and techniques to ensure that consideration of the safest option is at the centre of every decision. More recently there has been a developing movement within health care to embrace these concepts and principles in pursuit of the highest quality and safest health care provision. This has included recommendations from the Department of Health, most significantly that the importance of human factors training to safe care should be widely communicated ('Safer medical practice', CMO Report, 2000).

This chapter provides a brief overview of the human factors that can affect the performance of individuals and teams in health care environments. The reader is encouraged to consider these factors in their everyday practice. Those attending the courses associated with this manual may receive direct feedback on their non-technical skills performance.

25.2 HUMAN ERROR

Humans make mistakes. No amount of checks and procedures will mitigate this fact. Therefore it is vital that we aim to work in a way that minimises the occurrence of mistakes and ensures that when they do occur, we minimise the chance of them resulting in harm to patients or staff.

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25.3 ERROR CHAINS

Patient safety is generally not compromised through the occurrence of a single mistake. Behind any identified error (A), that leads to an untoward event (B), there is a sequence of factors arising from latent conditions or active failures that lead from error A to event B and without which event B would not have occurred. This is known as the error chain. James Reason (1997) showed this pictorially in what he called the 'Swiss cheese' model (Figure 25.1).

Each of the slices of cheese represents barriers which would, under ideal circumstances, prevent A leading to B. However, all checks and balances can fail at some stage. This is represented by the holes in the slices. For A to be followed by B, the holes need to line up through all the intervening slices. Simplistically viewed, the more checks that are put in place, the less likely an error is to occur. However, the increasing complexity of processes can be counterproductive as humans will avoid or modify one of more of the steps to make life easier.

Consider the following critical incident.

- *The wrong dose of a drug has been administered to a patient by a clinician. Why?* We know that the clinician should have checked the details of the prescription and confirmed the calculations to ensure that this all matched up with the formulation and strength of the medications to be administered. People do not deliberately make errors and therefore it is not unreasonable to conclude that the clinician thought they had checked and matched everything as described.
- *So why did the error occur?* Further information shows that two drugs had been replaced in one another's normal positions in the ward trolley. The packaging of both was very similar (Figure 25.2).

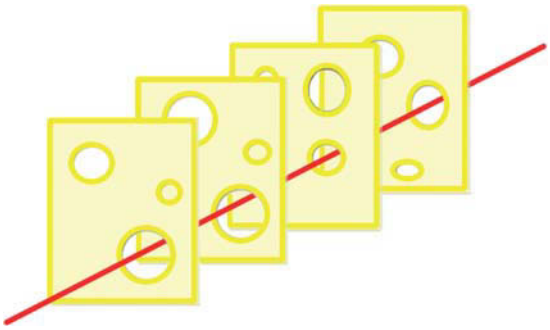

Figure 25.1 'Swiss cheese' model

Figure 25.2 Similar package design of two different medications

Ideally, the clinician should have read all of the relevant text on the packaging. However familiar tasks can lead to complaceny and subsequent inattention to detail. The clinician picked the medication from its usual place, thought they recognised the box and therefore did not actively review the name and concentration of the drug. Habit can blind us to what we are doing. Whilst incidents like this are well reported from the past, the problems of uniform packaging have now been recognised and highlighted through the National Patient Safety Agency (NPSA) 'Design for Medication Safety' initiative. Packages for different strength medications should now look completely different. This change in practice is a practical application of human factors theory to reduce the risk of error.

In the working environment we may be present at the right time to observe the breaching of a barrier that would normally prevent errors occurring. It is critical that we are vigilant for these breaches and draw the attention of our colleagues to them in order to prevent the progress and completion of an error chain. By convention, events or conditions that might be seen to be on the facilitative path to a critical incident are referred to as red flags. This approach is extremely useful. The more red flags that arise, the greater the risk of an adverse incident occurring and therefore the greater the need to alert the team to stop, review and if necessary resolve the situation.

25.4 COMMUNICATION

Problems with communication underpin a significant proportion of reported critical events. The fact that it is vital that clinicians communicate efficiently and unambiguously in their everyday clinical practice is undeniable. However, what steps do we take to ensure this happens?

When the speaker and listener do not share the same language, the communication issues are obvious and normal practice would be to look to using an interpreter to facilitate dialogue. Many recognise the limitations of discussions carried out through a third party. Even when all parties are using their native tongue, non-verbal signals carry a significant amount of information and meaning, in addition to the words themselves. With these facts in mind it is not difficult to understand why miscommunication is commonplace. This is particularly true in cross-cultural communications where both verbal and non-verbal elements can be completely misinterpreted by both parties.

Studies have shown that we understand around 61% of verbal communication and over 50% of written communication, with the remaining percentages being miscommunicated, misinterpreted or simply misunderstood (Beaty, 1995). The reason for this breakdown in communication is multifaceted and includes our use of non-verbal communication (outside the actual words we use), which has been shown to contribute up to 93% of what we understand (Barbour, 1976). Barbour's study identified that 38% of communication relates to how words are said (volume, pitch, rhythm, etc.) and 55% to body language (facial expressions, posture, etc.).

These observations are largely based on work from the 1960s exploring the nature of therapeutic counselling conversations. Emerging data show this to be an oversimplification of that which occurs in clinical communications. However, the message remains the same – words spoken or written by one, with an intent to convey a particular message, may be interpreted entirely differently by the listener or reader. This in itself provides a direct insight as to why in a busy clinical environment within which multiple tasks are being undertaken, and without direct visual connections between all team members, that miscommunication occurs so frequently. The process of communication can be described as three separate phases:

1 *The sender.* This is the process within which the originator of the message puts together the sentences in their mind (encoding), in what they perceive to be a meaningful and contextual manner.
2 *The channel.* This is the medium of communication chosen, verbal, non-verbal or written.

3 *The receiver.* This is the process within which the intended recipient makes sense of the information provided (decoding) and, due to multiple barriers, including euphemisms and localised terminology, is highly prone to distortion.

The resulting outcome in a noisy, highly pressured clinical arena is unsurprisingly one of poor information exchange. Multiple studies, including the document 'Why mothers die, the confidential inquiry into maternal deaths' (Lewis, 2007), have reached the same conclusion: poor and ineffective communication is central to clinical error.

The 'feedback loop' is an easily implemented technique that can be used to improve communication. It is a process within which the receiver repeats the message back to the sender to acknowledge and clarify that it has been correctly deciphered. It is quick and simple, easy to implement and shows an immediate benefit in busy clinical areas where requests and instructions are being passed on at breakneck speed.

In summary, the discussion above only begins to touch on the complexity of human communication. Beyond this there are many layers of subtlety in our interactions. To try and mitigate the risk of miscommunication, it is vital that both talkers and listeners actively engage in the process.

Teamwork

Teamwork is the cooperative effort of a team of people who have a shared goal and who have a leader with a defined role and the same goal. Good teamwork requires good relationships between team players, who are supportive of each other and can recognise problems amongst individuals within the team.

Body language and hierachy

All parties should always be aware of their non-verbal signals. Those that say 'I'm bored', 'I'm tired' or 'I don't value you' can all result in one person failing to pass on a key piece of information to another. The presence of a steep hierarchy can be particularly inhibitive as it embeds an attitude into the working culture where staff do not value one another's input. A culture where junior staff do not feel empowered to speak directly to senior staff, or senior staff are dismissive about concerns raised by junior staff, is inherently unsafe. If a clinical assistant walks into theatre and sees an expanding pool of blood under the operating table, they should feel able to raise their concerns. Whether or not it proves to be clinically important, their input should be positively acknowledged, as next time they might be the first to identify a critical issue.

Speaking up

Stage	Level of concern
P – Probe	*I think you need to know what is happening*
A – Alert	*I think something bad might happen*
C – Challenge	*I know something bad will happ*en
E – Emergency	*I will not let it happen*

A useful communication tool, widely used by staff in the airline industry, is shown in the box above. This structure can be used by any person concerned that they have information that might be important to others in the team. The levels *probe*, *alert*, *challenge*, *emergency* are used sequentially to express increasing concern. If a disaster appears to be imminent, it is entirely appropriate to use the 'challenge' or even 'emergency' stages without recourse to the initial stages. Although anyone can use this approach it becomes even more powerful when embedded in day-to-day working practice. In this circumstance, both the speaker and the

listener should recognise the level of the communication and react appropriately. In the aviation industry, any situation reaching the level of 'challenge' on the flight deck prompts an in-depth post-incidence review whether or not there was an adverse outcome.

These stages are described with examples below:

- *Probe* is used when a person notices something they think might be a problem. They verbalise the issue: 'Dr Adams, are you aware this child is cyanosed?'
- *Alert* is used when no one responds to the first prompt and the situation continues. The observer strengthens their statement and suggests a course of action: 'Dr Adams, I am concerned the child is deeply cyanosed; should we start BVM ventilation?'
- *Challenge* is used when the situation requires urgent attention and one of the key protagonists needs to be directly engaged. If possible, the speaker places themselves into the eye line of the person they wish to communicate with: 'Dr Adams you must listen to me now – this patient needs help with his ventilation'.
- *Emergency* is used when all else has failed and/or the observer perceives a critical event is about to occur. Where possible a physical signal or physical barrier should be employed together with clear verbalisation: 'Dr Adams, you are overlooking this child's respiratory state, please move out of the way as I am going to mask ventilate him'.

Organisations using this or similar systems sometimes make use of a code word or phrase to flag the level of concern. These may then be used to gain both the team's attention and a key individual's attention; for example 'Code Orange, Code Orange, please stop and listen'.

25.5 SITUATIONAL AWARENESS

People working in health care do not (or rarely) come to work to harm patients. Errors occur because the person undertaking a key trigger action does not see that their action will cause the patient to become harmed. We all operate in what we perceive to be the real world through our personal interpretation of its current status and our understanding of what will occur in response to our actions, or inaction, based on our experience and that perception. If we have insufficient or incomplete information, we can draw the wrong conclusions about what is going on. For example, we can make a misdiagnosis. If we then intervene on the basis of that flawed diagnosis it is not difficult to see that harm may ensue.

Good situational awareness is achieved when we have sufficient and correct information, have interpreted it correctly, and accurately project the outcome of an intervention into the future based on our current knowledge (Figure 25.3). It also includes the importance of sharing this perception with others in the team so that the mental model is shared by all.

Figure 25.3 Illustration of situational awareness

Consider the picture in Figure 25.3. What do you see? Around half of readers will see a young woman looking back/right and the other half will see an old woman looking forward/down (a few may see both). This is a simple example of how two people can look at the same thing yet make different interpretations.

The way we perceive a particular situation is affected by the information conveyed via our own sensorium, our past experience, our level of alertness, our current workload and the influence of intercurrent distractions. Simplistically, an experienced clinician might recognise a compromised patient more rapidly than a junior clinician.

In psychological terms our perception of the current situation is fitted into a model in our minds based on our knowledge and previous experiences. We use this model to plan our next actions and to anticipate the outcome of those actions. The projection into the future of what is likely to occur is then dictated by personal experience. A simple example of this might be a baby presenting with cyanosis and respiratory distress. The resuscitating clinician assessing a baby with congenital heart disease might conclude that the baby is suffering from a lower respiratory tract infection because of inexperience, or insufficient information secondary to tiredness or distractions.

The loss of situational awareness is often recognised whilst undertaking a specific psychomotor task; an example would be the team leader who had until this point maintained a position of objectivity and overall assessment of the situation at hand. However, when called to undertake a clinical skill, the ability to receive and process information is greatly diminished – clinical staff have reported a complete loss of situational awareness, in that they did not hear alarms or other team members alerting them to specific dangers whilst undertaking complex tasks. If it is the team leader undertaking the task and the team are also involved in busy practice, then it is not unusual for the entire team to miss critical elements of information that, if recognised, would have directly contributed to the correct diagnosis and care of the patient.

A common trap that people fall into is only seeing or registering the information that fits in with their current mental model. This is known as a *confirmation bias*. When this occurs, people favour information that confirms their preconceptions or hypotheses regardless of whether the information is true (Plous, 1993).

This may be observed within the health care setting during the process of a referral or handover. For example, a clinician receives a phone call requesting he or she to attend the ward to review an acutely deteriorating patient and is advised that the patient is a well known asthmatic. On their way to the ward, the clinician builds up a series of preconceived expectations of what they will find upon their arrival. They may even formulate a management plan based upon their expectations whilst travelling to the ward. Once this mindset is established it can be difficult to shift.

On arrival, the clinician examines the systems affected by the presumed diagnosis. They seek to confirm their expectation by focusing on auscultation of the chest at the expense of a thorough airway assessment. Upon hearing bilateral wheeze, their preconceived ideas are confirmed and the remainder of the assessment is completed without due attention and more as a rehearsed exercise than an open-minded exploration. They fail to notice that the patient also has a soft stridor and is hypotensive.

In an alternative scenario, the clinician knows they have several other patients they need to get to and does not undertake a thorough assessment. The junior staff in attendance point out the hypotension and signs of upper airway obstruction, but the clinician fails to recognise the significance of these signs. This occurs because psychologically they hold onto their preconceived diagnosis. In this situation they may dismiss these conflicting findings or may even manipulate the findings to fit their preconceived mental model.

In both cases, the eventual diagnosis of anaphylaxis becomes a late consideration, or a situation occurs that requires an objective newcomer to the team to point out the obvious.

It is vital all personnel understand the concept of situational awareness and continually question their own thought processes and those of others around them. It is also vital that the team shares their impressions of the current situation. There is good evidence that the

situational awareness of a well-functioning team is actually greater than the sum of its individual parts. This may in part be due to the elimination of bad data. Information or comments by colleagues that are outwith one's current mental picture should be treated as a trigger to consider whether anyone's situational awareness is lacking. A discussion of the disparity should uncover the true picture. Problems occur when individuals either ignore or rationalise the errant data to fit into their current picture of the world rather than treating them as a challenge.

25.6 FATIGUE

When we are tired, we find it more difficult to concentrate, our reactions are slower and our critical thought processes become imprecise. Our mood can be affected. This may be manifested by us becoming impatient, uninterested and irritable. It is not difficult to appreciate that these qualities are all likely to impact negatively on someone's ability to function both as an individual and as part of a team.

Clinicians work in a highly pressured environment. When an acute clinical crisis arises, there is an expectation that they should just keep going even when they know they are not functioning at 100% capacity. In a safe culture, staff should feel able to speak up and say 'I'm not fit to do this'. They should be able to do this with the knowledge that their colleagues will take this information on board and support them appropriately. Whilst it seems obvious that fatigued people are not working at the sharp end of health care delivery, this is not currently part of the mindset. All health professionals, throughout all clinical disciplines and management, need to alter their approach to translate an understanding of this into day to day practice. This is a culture change we all need to embrace.

It is important to note that even in normal working practice, circadian rhythms can produce both psychological and physiological disturbances that are disabling to some degree. Clinicians routinely work shifts, including nights, with a quick turnaround between patterns of rest and physiological stability. It is perhaps unsurprising to note that most of the recent major disasters attributed to human error (e.g. the *Exxon Valdez* oil spill, Three Mile Island, Bhophal chemical plant explosion and Chernobyl) occurred on the night shift, when alertness is at its lowest point.

It is suggested that the following factors may be exacerbated by circadian rhythm disturbance:

- Lack of concentration.
- Periods of inattention.
- Reduced vigilance.
- Reduction in alertness level.
- Slow actions and reactions.
- Alteration in short-term memory.
- Loss of critical analysis and advocacy.
- Interpretation errors.

Recognition of the impact of fatigue on our ability to perform effectively carries with it important, personal, responsibilities. People should look to arrive at work rested and prepared for the day ahead. If unforeseen events intervene that result in someone being unfit for work in any way, it is vital that each person takes responsibility for flagging it up to colleagues and managers. They, in turn, must take appropriate steps to ensure that anyone reporting such concerns is supported and, where necessary, allowed to step down from front line duties until fit. If this feels over-idealistic, consider the following – would you be comfortable boarding an aircraft when you knew the pilot had been up all night with his child who is critically unwell in hospital?

The discussions above are primarily focused on tiredness or fatigue due to lack of sleep. The expression of illness, use of medications, alcohol and personal stress can all manifest in a similar manner and should be sensitively examined and managed accordingly.

25.7 DECISION MAKING

On the surface, the practise of decision making is familiar to us all. However, to understand what factors can compromise this process it is important to understand the factors that will influence the reliability of any decision made. To make a good decision, a person needs to assess all aspects of a problem, identify the possible responses to the problem, consider the consequences of each of those responses and then weigh up the advantages and disadvantages in order to draw a conclusion. Having completed this, they then need to communicate their decision to their team.

Good situational awareness is a basic prerequisite of this process. To achieve this, the decision maker must ensure they have all the key information. It will be gathered through a combination of first-hand data and through two-way communication with their team. Decision makers should be on the alert for ambiguities or conflicting information. Any inconsistent facts should be treated as a potential marker for faulty situational awareness. They should never be brushed off as unimportant anomalies in the absence of evidence to support such a decision.

In many clinical situations there can be a significant pressure of time. Where this is not the case, then no decision-making process should be concluded until the team are satisfied they have all the information and have considered all the options. Where time is under pressure, a certain amount of pragmatism must be employed. There is plenty of evidence to confirm that practise and experience can mitigate some of the negative effects of abbreviating the decision-making process. Those making decisions under such circumstances need to remain consciously aware of the short-cuts they have taken. They should be ready to receive feedback from their team, particularly if any member of the team has significant concerns about the proposed course of action. Practical limitations accepted, there are few situations where a delay of a few minutes to share the rationale for the decision is not possible.

As discussed above, it is vital that team members feel able to raise their concerns and that the decision maker values and considers them appropriately. One only has to examine some high profile medical incidents, such as the Elaine Bromiley case (see below), to see that junior clinicians were trying to voice their (correct) concerns from a time when, if they had been heard, it may have altered the outcome.

> Elaine Bromiley died during a minor operation in 2005 after anaesthetists were unable to place an endotracheal (ET) tube. The doctors struggled for 20 minutes to insert the ET tube. They did not follow the standard 'Can't intubate, can't ventilate' protocol despite the appropriate kit being made available by theatre staff. During this episode Elaine Bromley suffered irreversible brain damage and subsequently died.

From time to time, there will be occasions where decision-makers disregard the opinions of others. Whatever their level of seniority, it is important that this is flagged with their line managers at the earliest opportunity. Don't wait for an incident to occur!

25.8 LEADERSHIP: PEOPLE, BEHAVIOURS

In social situations the variation in human personality is a stimulating source of intrigue and interest. To some extent this is the same when we come to work. In the professional environment, however, it is vital that people work well together and the mix of personalities can make or break the smooth functioning of the team.

Clinical medicine is often practised in a pressured, demanding environment. This can serve to exaggerate latent personality traits and bring out new ones. At the extremes, this can manifest as an aggressive or submissive affect. Dependant on the individual and the dynamics of the team, this can be highly detrimental to the process of communication, situational awareness and decision making.

A discussion of the ways to optimise team performance on the basis of personality is a book in itself. Suffice to say a degree of commonsense goes a long way. Wherever possible, the adoption of a facilitative role can serve to draw the best from all members of the team. In ideal circumstances, every opportunity should be taken to debrief teams after an episode of working together. This can be enhanced where there is opportunity for the team to practise and reflect on their interactions in a simulated environment.

25.9 SUMMARY

We all work in a complex clinical environment. Patient safety is everybody's responsibility and every clinician should seek to remain alert to red flags and promptly voice their concerns. To do this we need to recognise and respect both our own limitations and those of the people we work with. This safety surveillance can be facilitated by an understanding of the human factors that impact both negatively and positively upon our performance.

PART 6

Appendices

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APPENDIX A

Acid–base balance

A.1 INTRODUCTION

Under normal circumstances, blood pH is tightly controlled between 7.35 and 7.45. Although this sounds like very little variation, it has to be remembered that there is a logarithmic relationship between pH and hydrogen ion concentration ($[H^+]$). Thus a pH rise from 7.35 to 7.45 represents a fall in $[H^+]$ from 45 to 35 nmol/1. By the time the pH has fallen to 7.1, $[H^+]$ has doubled to 80 nmol/l.

A normal intracellular pH is required for the functioning of the many enzyme systems in the body. The body thus has mechanisms to maintain a normal pH, and many buffers exist to protect against the pH changes that occur as H^+ production increases in sepsis, injury, poor perfusion and catabolism, or if there is failure to excrete the normal acids produced.

Abnormalities of acid–base balance are important to understand for two major reasons. Firstly it is essential to identify and treat the cause of the acid–base disturbance, and secondly the acid–base abnormality may require correction. It is the intracellular pH that will affect the function of cells, and correction of the blood pH does not necessarily correct the intracellular pH. In the setting of poor tissue perfusion, administration of bicarbonate (which will increase blood pH) may actually aggravate intracellular acidosis. There is evidence that administration of bicarbonate in situations such as diabetic ketoacidosis may actually increase the risk of cerebral oedema and death.

The pH of blood affects the distribution of ions (such as potassium and calcium) throughout the body, and changes in pH may be associated with changes in ion concentration, which may have effects on body systems and thus require monitoring.

The acid–alkali balance equation

In the blood, bicarbonate reacts with hydrogen ions to produce CO_2 and water:

$$H^+ + HCO_3 \leftrightarrow H_2O + CO_2$$

The balance of this equation is reflected by the Henderson–Hasselbalch equation:

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$$pH = pKa + \log[HCO_3^-]/0.03PCO_2$$

which is used by blood gas analysers to calculate [HCO_3^-] once PCO_2 and pH have been measured.

The base deficit is calculated as the amount of base that would have to be added to the system to correct the pH to normal, while the base excess is the amount of base that would have to be removed (or balanced with H^+) in order to correct the pH. Unfortunately the base deficit is frequently expressed as a negative base excess, which may give rise to confusion unless great care is exercised. In practice, blood gas analysers calculate the base deficit from the difference between the calculated [HCO_3^-] and a predicted 'normal' of 24 mmol/l. As normal values of [HCO_3^-] are lower in young infants (18–20 mmol/l) this may be a source of confusion.

The acid–base balance is also affected by 'strong ions' in the body such as chloride. The term 'strong' simply refers to the fact that these ions have a strong tendency to exist in the ionic form. The presence of ions affects the amount of H^+ and HCO_3^- produced as electrochemical neutrality must be maintained in the body. An increase in the concentration of chloride ions will tend to produce acidosis, while low chloride ion concentrations will tend to induce metabolic alkalosis.

Short-term control of the blood pH is exerted via the respiratory system. Increased ventilation will drop the PCO_2 and thus increase the pH within minutes, while decreased ventilation will have the opposite effect. The respiratory centre is driven by the pH of the cerebrospinal fluid (CSF). Any drop in CSF pH will result in an increase in respiratory drive, with increased ventilation and loss of CO_2. Conversely an increase in CSF pH will tend to decrease the respiratory drive. Longer term control of the acid–base status of the body is exerted by the production of bicarbonate in the kidney, and the excretion of acid (over hours).

Figure A.1 shows the relationship between pH and bicarbonate concentration at different levels of CO_2. It can be seen that at a given bicarbonate concentration the pH falls as the CO_2 level rises. Also, it can be seen that at lower pH levels small falls in bicarbonate concentration produce dramatic reductions in pH. Similarly at low pH, administration of small amounts of bicarbonate may cause large shifts in pH. The nearer the pH gets to normal the larger the amount of bicarbonate required to produce any change.

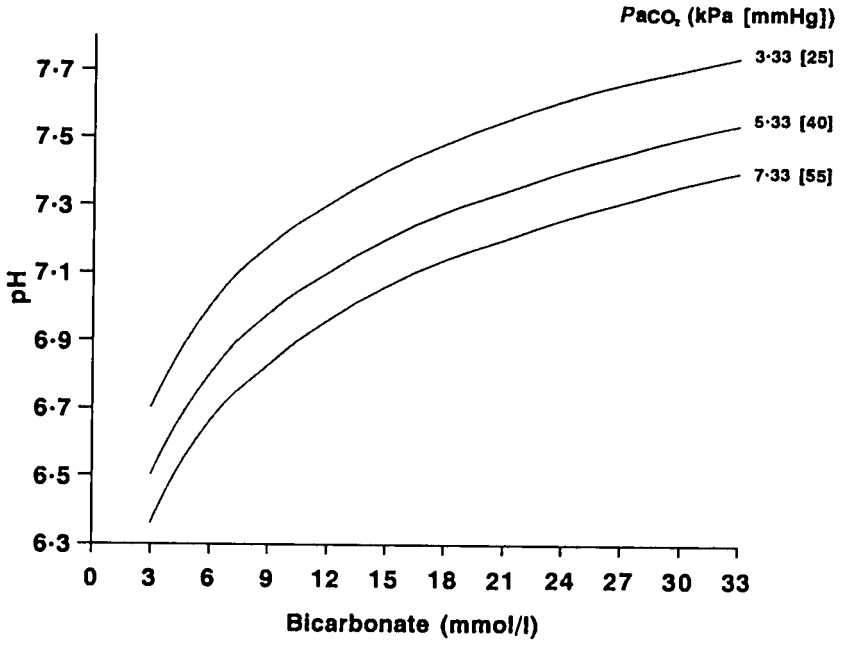

Figure A.1 The relationship between pH, bicarbonate and carbon dioxide

Abnormalities of acid–base balance

A drop in pH is referred to as acidosis, while an increase in pH above normal is alkalosis. Acidosis or alkalosis may be the result of respiratory or metabolic problems. Abnormalities of acid–base that are the result of respiratory dysfunction are generally referred to as respiratory acidosis or alkalosis, while acid–base abnormalities from all other systems are referred to as metabolic acidosis or alkalosis.

The body always attempts to correct abnormalities of pH and this may give rise to a 'corrected acidosis or alkalosis'.

It is possible to have concomitant metabolic acidosis and respiratory alkalosis (or vice versa). In this situation the pH will be normal, but the PCO_2, $[HCO_3^-]$ and base excess/deficit will be abnormal.

Acidosis (low pH)

Respiratory acidosis is characterised by a low pH with a high PCO_2, and results from retention of CO_2. In acute respiratory acidosis the $[HCO_3^-]$ will be normal, but in prolonged respiratory acidosis there is a tendency for the $[HCO_3^-]$ to gradually increase from renal compensation. It is important to note that the body will never overcompensate, so if the pH is normal or elevated in the face of an elevated PCO_2, it means that there is concomitant metabolic alkalosis.

In any patient with respiratory acidosis the questions to be asked are 'Is this a problem of respiratory drive, a problem of respiratory function, or a combination of both?' The presence of nasal flaring is strong evidence of adequate respiratory drive. Respiratory function may fail as a result of problems in the airways, lung, thorax, musculoskeletal system, brain or finally the neuromuscular system. Management must be directed at identifying the problem(s) and providing adequate respiratory support.

Metabolic acidosis (characterised by a low pH, low or normal PCO_2, and base deficit >2) is caused by the following:

- Abnormal production of acid:
  - lactic acid in patients with shock and poor tissue perfusion, and mitochondrial dysfunction,
  - ketoacidosis in patients with diabetic ketoacidosis, or
  - abnormal acids in children with inborn errors of metabolism, including lactic acidoses, organic acidurias and amino-acidopathies.
- Abnormal excretion of acid:
  - renal failure, or
  - distal renal tubular acidosis.
- Excessive tubular loss of bicarbonate:
  - proximal tubular acidosis, or
  - Fanconi's syndrome.
- Excessive levels of chloride: a feature of salt poisoning, or following resuscitation with large volumes of 0.9% sodium chloride.
- Toxin ingestion: salicylates.

The body will attempt to correct for the metabolic acidosis. A 'corrected metabolic acidosis' is characterised by a low pH with a low PCO_2.

In an acute life-threatening illness or injury, metabolic acidosis is most commonly due to a combination of lactic acidosis and acute renal failure. The primary management is thus to ensure that there is adequate resuscitation (ABC in particular) with correction of renal and other tissue perfusion. If the pH remains <7.15 in the face of adequate resuscitation, and it is felt that the acidosis is contributing to the clinical problems, a single dose of 1–2 mmol/kg (1–2 ml/kg of 8.4% $NaHCO_3$) may be given. This is based on the knowledge that this raises the $[HCO_3^-]$ by about 1.5–3 mmol/l, which should improve the pH by 0.1–0.2 units (see Figure A.1 on p. 274), whilst being unlikely to cause troublesome hypocalcaemia or hypokalaemia. If on recheck of the pH, after this dose has been given, significant correction has not occurred then it is due to the presence of a high acid load or high acid production rate. Expert assistance is required.

When correcting an acidosis, the serum calcium and potassium should be monitored carefully. Correction of acidosis reduces the ionised calcium and may produce symptomatic hypocalcaemia. Hypocalcaemia will have a negative inotropic effect on the heart. Although the serum potassium is often high in the face of acidosis, correction will cause it to fall because of intracellular movement, and supplementation may be required. Continued use of sodium bicarbonate will lead to hypernatraemia.

Where metabolic acidosis is inadequately explained, or more severe than expected, it is essential to collect specimens of blood and urine for later investigation of the causes of the metabolic acidosis. If this is not done, inborn errors of metabolism and toxin ingestion will be missed.

A 'compensated metabolic acidosis' is characterised by a low or normal pH with a low PCO_2, while a 'compensated respiratory acidosis' is characterised by a low or normal pH with a high PCO_2.

Alkalosis (high pH)

Respiratory alkalosis is characterised by a high pH with low PCO_2 and results from excessive blowing off of CO_2. In this situation it is important to identify what is causing the patient to overventilate. Pain and anxiety are the most common causes, but cerebral irritation (e.g. meningitis) may also cause overventilation. The CO_2 level can fall quite markedly, causing a significant rise in pH, which is often enough to produce hypocalcaemic tetany.

If a patient is overventilating from anxiety, the well-known remedy of breathing into a paper bag makes the patient rebreathe his or her own CO_2 and is very effective. Organic causes of overventilation must always be excluded before instituting rebreathing therapy.

Certain toxins such as salicylates may also be associated with respiratory alkalosis (although in children this is usually offset by a concomitant metabolic acidosis).

Metabolic alkalosis is characterised by a high pH in the face of a normal or high PCO_2. The base excess is >2.

Metabolic alkalosis is commonly associated with severe vomiting of gastric contents. Severe vomiting causes alkalosis in two ways. First, there is direct loss of acid from the stomach. Second, vomiting may induce severe hypovolaemia. This causes hyperaldosteronism in an attempt to promote salt and water retention, which, in turn, leads to increased renal potassium and hydrogen ion loss, with bicarbonate retention. This exacerbates the alkalosis. Volume expansion with normal saline promotes correction. Congenital hypertrophic pyloric stenosis is a good example of this pathophysiological process in action. It is important to exclude gastric outlet obstructions in small children with significant metabolic alkalosis. Renal causes of alkalosis, such as Bartter's syndrome, are rare, but diuretic agents such as a frusemide (furosemide) are relatively common causes of metabolic alkalosis.

In most of the cases of metabolic alkalosis noted above, hypochloraemia is a common feature. Administration of additional chloride may often be the most effective way of correcting the alkalosis. Very occasionally, agents such as acetazolamide may be used, but then only under specialist supervision.

A.2 ANALYSIS OF ARTERIAL BLOOD GAS

The proper interpretation of an arterial blood gas (ABG) sample requires clinical history, examination, knowledge of treatments given and other laboratory investigations. In emergencies, when many of the data are lacking, interpret the findings with caution.

What is the pH? (Normal: 7.36–7.45)

Is there an acidosis or alkalosis? If the pH is near normal it may be due to respiratory or metabolic compensation. This compensation is never complete, so if the pH is near normal it will

still always tell you if the child has a compensated acidosis (pH slightly lower than normal) or a compensated alkalosis (pH slightly higher than normal).

What is the $PaCO_2$? (Normal: 4.7–6.0 kPa or 35–45 mmHg)

This is a good indicator of ventilatory adequacy because it is inversely proportional to alveolar minute volume (respiratory rate × alveolar tidal volume). When the pH is known it can be used to determine if there are primary or compensatory ventilatory changes.

A higher than normal $PaCO_2$ indicates underventilation. In association with a low pH, this indicates respiratory acidosis.

A lower than normal $PaCO_2$ indicates overventilation. In association with a high pH this indicates respiratory alkalosis.

What is the base excess or deficit? (Normal: ±2)

A base excess indicates a metabolic alkalosis, while a base deficit indicates a metabolic acidosis. A base deficit is only treated if it is greater than 6 and the pH is low (and then only in specific circumstances).

Bicarbonate and base excess/deficit are *calculated* by blood gas analysers using the Henderson–Hasselbalch equation. These results must always be interpreted cautiously in clinical situations.

What is the PaO_2? (Normal: 10.6 kPa or >80 mmHg in air)

The partial pressure of oxygen in an arterial blood sample must be interpreted in the light of the inspired oxygen concentration and the pressure of its delivery.

An easy method for blood gas interpretation

Step 1

Assess the pH. Is it raised, >7.44 (alkalosis), or lowered, <7.36 (acidosis)? This is the overall status of the patient, regardless of compensatory mechanisms.

Step 2

	Acidosis	Alkalosis
Respiratory	CO_2↑	CO_2↓
Metabolic	Base excess ↓ or bicarbonate ↓	Base excess ↑ or bicarbonate ↑

Look at the CO_2 on the chart above.

- If the CO_2 provides a cause for the abnormal pH, i.e. low pH with high CO_2 (acidosis) and high pH with low CO_2 (alkalosis), then the overall picture is a *respiratory* acidosis or alkalosis.
- If the CO_2 does not provide a cause for the pH, it is compensating for a *metabolic* abnormality.

Step 3

Confirm your findings by looking at the base excess of bicarbonate.

- If the base excess provides a cause for the abnormal pH, i.e. low pH with negative base excess (acidosis) and high pH with positive base excess (alkalosis), the overall picture is a *metabolic* acidosis or alkalosis.
- If the base excess does not provide a cause for the pH, it is compensating for a *respiratory* abnormality.

Example

A patient with shock, showing sighing respirations, has the following measurements:

pH: 7.24
P_{CO_2}: 31 mmHg
[HCO_3^-]: 14 mmol/l
Base deficit: 8

- The pH is low, showing acidosis.
- The P_{CO_2} does not provide a cause for the abnormal pH (a low CO_2 indicates a respiratory alkalosis, not an acidosis); therefore it is compensating for a metabolic acidosis.
- The base deficit accounts for the abnormal pH (a base deficit indicates a metabolic acidosis).
- Therefore the patient has a metabolic acidosis, which is being partially compensated by a blowing off of CO_2.

Precautions when taking an arterial blood sample

The taking of arterial blood samples is described in Chapter 21. Certain errors must be avoided:

- Note the inspired oxygen concentration.
- Ensure an adequate sample and avoid bubbles. Air in the syringe will allow CO_2 and O_2 to diffuse in or out of the sample. Seal the syringe with a plastic cap for the same reasons.
- Avoid excess heparin (which may produce an artefactual acidosis). Fill the dead space of a 2 ml syringe and attached needle with 1:1000 heparin. A pre-heparinised syringe is preferable.
- Minimise metabolism in the sample. A delay in analysis allows O_2 consumption and CO_2 generation to continue in the syringe sample. If a delay of more than a few minutes is anticipated store the sample on ice.

APPENDIX B

Fluid and electrolyte management

B.1 INTRODUCTION

At birth approximately 80% of a child's body weight is water. This percentage falls gradually during childhood, reaching 60% water by adulthood. Total body water is distributed between the intracellular, interstitial and intravascular spaces moving from one compartment to another depending on various pressure and osmotic gradients. In illness and injury these fluid shifts may be rapid, with significant clinical consequences.

B.2 FLUID BALANCE

Normally fluid balance is tightly controlled by thirst, hormonal responses and renal function: the formulae in Table B.1 provide a guideline to appropriate fluid intake. These formulae are based on an assumption of 100 kcal/kg/day of caloric intake, 3 ml/kg/day of urine output and normal stool output.

For example:

- a 6 kg infant would require 600 ml/day,
- a 14 kg child would require 1000 + 200 = 1200 ml/day, and
- a 25 kg child would require 1000 + 500 + 100 = 1600 ml/day.

In critical illness or injury some or all of these mechanisms may be profoundly disrupted, and fluid therapy has to be tailored to the needs of the specific child. In the presence of acute respiratory or central nervous system (CNS) pathology, fluid requirements may be as low as 30 ml/kg/day, while in diarrhoea requirements may be as high as 300–400 ml/kg/day.

Fluid intake is required to replace fluid losses and to enable the excretion of various waste products through the urine. Insensible losses (via respiration and sweat) generally amount to between 10 and 30 ml/kg/day. The actual volume of insensible fluid loss is related to the caloric content of the feeds, the ambient temperature, humidity of inspired air, presence of pyrexia and quality of the skin. Insensible losses from a child on a ventilator in a cool environment with minimal caloric intake may be minimal. Usually between 0 and 10 ml/kg/day are lost in the stool (this will increase markedly in diarrhoea, where losses in excess of 300 ml/kg/day are not uncommon). Urinary losses are between 1 and 2 ml/kg/h (i.e. approximately 30 ml/kg/day).

Table B.1 Normal fluid requirements

Body weight	Fluid requirement per day (ml/kg)	Fluid requirement per hour (ml/kg)
First 10 kg	100	4
Second 10 kg	50	2
Subsequent kilograms	20	1

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

Dehydration and shock

Concepts

1 Dehydration does not cause death, shock does. Shock may occur with the loss of 20 ml/kg from the intravascular space, while clinical dehydration is only evident after total losses of >25 ml/kg
2 As a guide, the child with dehydration and no shock can be assumed to be 5% dehydrated; if shock is present, then 10% dehydration or greater has occurred
3 The treatment of shock requires rapid administration of intravascular volume of fluid that approximates in electrolyte content to plasma
4 The treatment of dehydration requires gradual replacement of fluids with an electrolyte content that relates to the electrolyte losses, or to the total body electrolyte content
5 Pathology from electrolyte changes is related to either extreme levels, or rapid rates of change
6 Administration of sodium bicarbonate is rarely indicated
7 Overhydration is potentially as dangerous as dehydration

The intravascular volume of an infant is approximately 80 ml/kg and of an older child 70 ml/kg. Rapid loss of 25% of this volume (i.e. 20 ml/kg) will cause shock unless that volume is replaced from the interstitial fluid at a similar rate. Clinical signs of dehydration (see box below) are only detectable when the patient is 2.5–5% dehydrated. Five per cent dehydration implies that the body has lost 5 g/100 g body weight, i.e. 50 ml/kg. Clearly, shock may occur in the absence of dehydration, dehydration may occur in the absence of shock, or both may occur together – all dependent on the rate of fluid loss and the rate of fluid shifts.

The priorities of management are to identify shock and treat it effectively and rapidly (see Chapter 9), identify dehydration and institute a treatment programme that will enable effective rehydration over 24–48 hours, identify the presence and aetiology of acid–base problems and correct these where necessary, and identify the presence and aetiology of electrolyte abnormalities and correct these gradually without precipitating complications.

One factor remains unknown at the initiation of therapy, namely the ongoing fluid losses that will occur during therapy. Thus any plan of fluid management represents a starting point, and this will have to be modified in the light of data from constant monitoring.

The critical clinical questions are therefore:

- Is the patient shocked?
- Is the patient dehydrated?
- Does the patient have a significant acid–base abnormality?
- Are there significant electrolyte problems?

Shock

Clinical signs of shock from fluid loss

Cardiovascular signs	Tachycardia usually associated with poor volume peripheral pulses
	Poor peripheral perfusion with prolonged capillary refill time and cool peripheries
	Low blood pressure as a pre-terminal sign
Consequences of poor perfusion	Alteration of mental status
	Development of metabolic acidosis with compensatory tachypnoea
	Poor urine output

The treatment of hypovolaemic shock secondary to fluid loss (after securing the airway and providing high-flow oxygen) is the rapid administration of crystalloid. The starting volume is 20 ml/kg, and this can be repeated if there is inadequate clinical response (with no evidence of intravascular overload). The fluids used should approximate in electrolyte concentrations to those of serum (options include 0.9% saline and Hartmann's solution, however the latter should be used with care in renal impairment). The presence of hyper- or hyponatraemia does not affect the choice of fluids during this phase of resuscitation.

Occasionally, shock may be precipitated by a cardiac dysrhythmia secondary to electrolyte abnormalities (most commonly potassium). In this situation rapid correction of the electrolyte anomaly may be essential; however, generally, electrolyte abnormalities should be corrected gradually.

Once shock has been adequately treated, attention can turn to management of hydration. Frequent reassessment remains necessary as the patient may well become shocked again if the underlying cause of the fluid shifts (e.g. gastroenteritis) is ongoing.

Dehydration

Clinical signs of dehydration

Drop in weight	This is the only objective measure of acute changes in hydration. Unfortunately the child's pre-sickness weight is usually not available, so this is not often useful in the initial evaluation of the child
Depressed fontanelle	Only useful if the fontanelle is still patent, and in the absence of disorders such as meningitis
Sunken eyes	Mothers may well report this sign but it is difficult to detect for clinical staff
Dry mouth	Mouth breathers tend to have dry mouths anyway, and the mouth will be wet if fluids have just been administered orally
Decreased skin turgor	This is difficult to interpret in malnourished children. It is particularly unreliable in fat children and in children with hypernatraemic dehydration
Decreased urine output	In renal disorders, the patient may be dehydrated in association with inappropriately high urine output (the same happens in diabetes). In the presence of gastroenteritis it is very difficult to establish urine output accurately, particularly in girls

Many clinical signs of dehydration are individually unreliable (see box above) and have poor interobserver reproducibility, but taken together they provide a reasonable estimate of total body fluid losses. Weight is the only clinically available objective measure of total body fluid changes, and enables an accurate assessment of fluid balance over time (unfortunately initial fluid therapy must usually be based on a clinical assessment of hydration because the pre-sickness weight is not often available).

The measured weight loss or percentage dehydration (5% dehydration = loss of 5 ml of fluid per 100 g body weight, or 50 ml/kg or 10 × percent dehydrated/kg) provides an estimate of the volume of fluid required to return to normal hydration. As a guide, the child with dehydration and no shock can be assumed to be 5% dehydrated; if shock is present, then 10% dehydration or greater has occurred.

Management of dehydration consists of the administration of calculated daily maintenance fluids in addition to calculated replacement fluids over a 24-hour period. The patient should thus achieve normal body weight over a 24-hour period; the period will be longer if electrolyte abnormalities are being corrected at the same time. Therapy should be monitored at 3–4-hourly intervals using weight as an objective measure, to ensure that the patient is gaining weight at an appropriate rate. If the calculated fluid administration rate is too slow or too fast, then the

Table B.2 Commonly available crystalloid fluids

Fluid	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^-(mmol/l)	Energy (kcal/l)	Other
Isotonic crystalloid fluids					
Saline 0.9%	150	0	150	0	0
Saline 0.45%, dextrose 2.5%	75	0	75	100	0
Saline 0.18%, dextrose 4%	30	0	30	160	0
Dextrose 5%	0	0	0	200	0
Hartmann's solution	131	5	111	0	Lactate
Hypertonic crystalloid solutions					
Saline 0.45%, dextrose 5%	75	0	75	200	0
Dextrose 10%	0	0	0	400	0
Saline 0.18%, dextrose 10%	30	0	30	400	0
Dextrose 20%	0	0	0	800	0

rate should be modified appropriately. See Table B.2 for the commonly available crystalloid fluids.

When there is excessive vomiting or there are signs of damaged bowel, fluid therapy should be given intravenously.

Example

A 6 kg child is clinically shocked and 10% dehydrated as a result of gastroenteritis.

Initial therapy

20 ml/kg for shock = 6 × 20 = 120 ml of 0.9% saline given as a rapid intravenous bolus.

Estimated fluid therapy over the next 24 hours

100 ml/kg for 10% dehydration = 100 × 6 = 600 ml
100 ml/kg for daily maintenance fluid = 100 × 6 = 600 ml
Rehydration + maintenance = 1200 ml
Therefore start with an infusion of 1200/24 = 50 ml/h

Application of fluid therapy

Reassess clinical status and weight at 4–6 hours, and if satisfactory continue. If the child is losing weight increase the fluid rate, and if the weight gain is excessive decrease the fluid rate. Start giving more of the maintenance fluid as oral feeds if the child is tolerating the fluids.

When the gut is functioning, oral rehydration using standard solutions is ideal (the World Health Organisation (WHO) formulation provides 75 mmol sodium, 20 mmol potassium, 65 mmol chloride, 10 mmol citrate and 75 mmol glucose per litre; the formulations generally used in the UK have lower sodium concentrations of 50–60 mmol/l). This fluid should be administered frequently in small volumes (cup and spoon works very well for this process). Generally, normal feeds should be administered in addition to the rehydration fluid, particularly if the infant is breast-fed.

Fluid overload and overhydration

In the same way that fluid losses may cause shock, dehydration or both, excessive fluid administration can cause intravascular fluid overload, overhydration or both.

In the patient with nephrotic syndrome, fluid has leaked out of the intravascular space and into the tissues because of a low serum albumin. Such children may be grossly overhydrated,

with diffuse severe oedema. However, many patients with nephrotic syndrome have a contracted intravascular space, and attempts to diurese these patients without first expanding the intravascular space with albumin may result in shock.

Clinical signs of overhydration

Oedema	Usually in dependent areas. In the infant or child who is either lying or sitting, this may not affect areas such as the buttocks or lower legs. Marked facial puffiness may be a feature.
Weight gain	Sudden increase in weight may be a marker of fluid overload

By contrast the patient with myocardial dysfunction may have an intravascular compartment that is grossly overfilled. The clinical signs of intravascular overload (see box below) may be present, and yet the patient (particularly if they have been on diuretics) may actually be total body fluid depleted and may appear dehydrated.

Children with other renal conditions may often have a combination of intravascular and total body fluid overload. They are then oedematous, but this is combined with features of intravascular fluid overload, and administering albumin would be dangerous.

Clinical signs of intravascular fluid overload

Raised jugular venous pressure	May be difficult to observe in a young child
Enlarged (and often tender) liver	Difficult to assess in a patient who already has a large liver. May also be difficult to assess if the patient has ascites
Cardiac gallop, usually together with cardiomegaly	May be difficult to assess in a patient with severe tachycardia, particularly if pulmonary oedema or other signs are present
Hypertension	An important clinical feature of excessive intravascular fluid, particularly in patients with renal problems
Pulmonary signs	Diffuse fine crepitations in the bases of the lungs, together with other clinical signs of fluid in the lungs

Therapy is dependent on an understanding of the pathology of the particular condition and the fluid shifts that have occurred.

The treatment of fluid overload is often complex and the non-specialist should seek help.

Electrolyte abnormalities

Table B.3 (over page) shows the normal electrolyte requirements.

Sodium

Both low and high sodium levels are potentially dangerous to the patient. Severe hypernatraemia may be associated with severe brain damage, because brain tissue shrinks as a result of intracellular dehydration and blood vessels may tear or clot up. Too rapid correction of hypernatraemia may lead to cerebral oedema and convulsions. Similarly, rapid correction of hyponatraemia may also be associated with demyelination and permanent brain injury.

Table B.3 Normal water, electrolyte and energy

Body weight	Water (ml/kg/day)	Sodium (mmol/kg/day)	Potassium (mmol/kg/day)	Energy (kcal/day)	Protein (g/day)
First 10 kg	100	2–4	1.5–2.5	110	3.00
Second 10 kg	50	1–2	0.5–1.5	75	1.5
Subsequent kilograms	20	0.5–1	0.2–0.7	30	0.75

The electrolyte losses during dehydration depend on the reason for dehydration. In gastroenteritis, sodium losses in diarrhoea range from approximately 50 meq/l (rotavirus) to approximately 80 meq/l (cholera and enteropathogenic *Escherichia coli*). In renal dysfunction sodium losses may be minimal (diabetes insipidus) or significant (renal tubular dysfunction).

Hypernatraemia Hypernatraemia in the dehydrated patient may be the end result of excessive loss of water (e.g. diabetes insipidus, diarrhoea), excessive intake of sodium (e.g. iatrogenic poisoning, non-accidental injury) or a combination of both (e.g. children with gastroenteritis given excessive sodium in rehydration fluid).

The electrolyte content of the replacement solution depends on the cause of the dehydration. In general 0.45% NaCl is a safe starting solution for intravenous rehydration. This is based largely on the electrolyte content of stool in diarrhoea. By contrast, patients with renal tubular dysfunction and natriuresis may require 0.9% saline to replace the renal losses of sodium. Nasogastric rehydration in moderate hypernatraemic dehydration is also a useful strategy and may be safer. Measurement of the sodium content of urine and stool may facilitate replacement therapy considerably.

The principles in the treatment of hypernatraemia are:

1 Treat shock first.
2 Calculate the maintenance fluid and estimate the fluid deficit carefully.
3 Aim to lower the serum sodium at a rate of no more than 0.5 mmol/h.
4 Check the calcium and glucose levels also.
5 Monitor the electrolytes frequently.
6 Clinically assess hydration and weigh frequently.

Hyponatraemia Hyponatraemia may be due to excessive water intake or retention, excessive sodium losses or a combination of both.

If the child is fitting from hyponatraemia, partial rapid correction of the serum sodium level will be necessary to stop the fitting. Administration of 4 ml/kg of 3% NaCl solution over 15 minutes will raise the serum sodium by approximately 3 mmol and will usually stop the seizures.

If hyponatraemia is due to excessive water intake or retention, and the patient is not symptomatic, the restriction of fluid intake to 50% of normal estimated requirements may be adequate therapy. If dehydrated and intravenous fluids are required then 0.9% NaCl is an appropriate fluid.

The principles in the treatment of hyponatraemia are:

1 Treat seizures with 3% NaCl.
2 Calculate the maintenance fluid and estimate the fluid deficit carefully
3 Aim to raise the serum sodium no more than 8 mmol/day.
4 Check the potassium, chloride, creatinine and glucose levels also.
5 Monitor the electrolytes frequently.
6 Clinically assess hydration and weigh frequently.

Potassium

Unlike sodium, potassium is mainly an intracellular ion and the small quantities measurable in the serum and extracellular fluid represent only a fraction of the total body potassium. However, the exact value of the serum potassium is important as cardiac arrhythmias can occur at values outside of the normal range. The intracellular potassium acts as a large buffer to maintain the serum value within its normal narrow range. Thus hypokalaemia is usually only manifest after significant total body depletion has occurred. Similarly, hyperkalaemia represents significant total body overload, beyond the ability of the kidney to compensate. The exception to both these statements is the situation in which the cell wall pumping mechanism is breached. A breakdown of the causes of hyper- and hypokalaemia is given in Table B.4.

Table B.4 Causes of hypo- and hyperkalaemia

Hypokalaemia	Hyperkalaemia
Diarrhoea	Renal failure
Alkalosis	Acidosis
Volume depletion	Adrenal insufficiency
Primary hyperaldosteronism	Cell lysis
Diuretic abuse	Excessive potassium intake
	In the critically ill neonate, inadequate cardiac output must always be excluded as a cause

Hypokalaemia Hypokalaemia is rarely an emergency and is usually the result of excessive potassium losses from acute diarrhoeal illnesses. As total body depletion will have occurred, large amounts are required to return the serum potassium to normal. The fastest way of giving this is with oral supplementation. In cases where this is unlikely to be tolerated, IV supplements are required. However, strong potassium solutions are highly irritant and can precipitate cardiac arrhythmias, thus the concentration of potassium in IV solutions ought not to exceed 40 mmol/l except when given centrally on intensive care units. Fortunately this is not usually a problem as renal conservation of potassium aids the restoration of normal serum levels.

Patients who are alkalotic, hyperglycaemic (but not diabetic) or are receiving insulin from exogenous sources will have high intracellular potassium stores. Thus hypokalaemia in these cases is the result of a redistribution of potassium rather than potassium deficiency, and treatment of the underlying causes is indicated.

Hyperaldosteronism is a cause of hypokalaemic alkalosis. Patients with this condition will have salt and water retention and will be hypertensive on presentation. Secondary hyperaldosteronism is the body's natural response to hypovolaemia and salt deficiency and is thus a common cause of hypokalaemic alkalosis. As there is primary salt and water deficiency the patient is not usually hypertensive. The most common causes are diarrhoeal illness and salt-losing conditions such as cystic fibrosis. Other causes include external loss of fluid from intestinal stomas or drains. Although potassium replacement is required in this condition the main thrust of therapy has to be with salt and water replacement to re-expand the circulation and cut down on aldosterone production.

Hyperkalaemia Hyperkalaemia is a dangerous condition. Although the normal range extends up to 5.5 mmol/l it is rare to get arrhythmias below 7.5 mmol/l. The most common cause of hyperkalaemia is renal failure – either acute or chronic. Hyperkalaemia can also result from

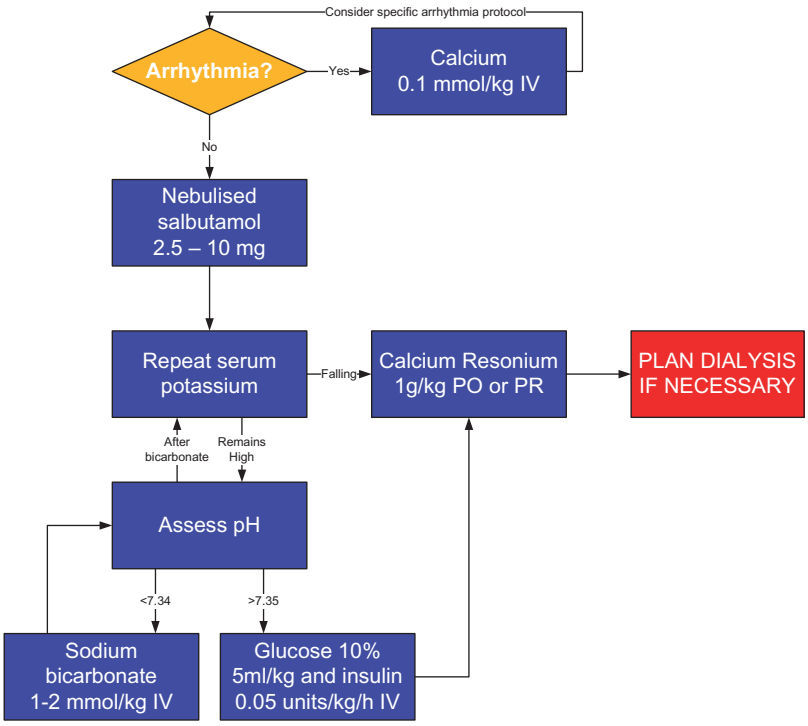

Figure B.1 Algorithm for the management of hyperkalaemia

potassium overload, loss of potassium from cells due to acidosis or cell lysis, hypoaldosteronism and hypoadrenalism.

The immediate treatment of hyperkalaemia is shown schematically in Figure B.1. If there is no immediate threat to the patient's life because of an arrhythmia then a logical sequence of investigation and treatment can be followed. Beta-2 stimulants, such as salbutamol, are the immediate treatment of choice. They act by stimulating the cell wall pumping mechanism and promoting cellular potassium uptake. They are best administered by a nebuliser. The dose to be given is shown in Table B.5. The serum potassium will fall by about 1 mmol/l with these dosages.

Sodium bicarbonate is also effective at rapidly promoting intracellular potassium uptake. The effect is much greater in the acidotic patient (in whom the hyperkalaemia is likely to be secondary to movement of potassium out of the cells). The dosage is the same as that used for treating acidosis, and 1–2 ml/kg of 8.4% $NaHCO_3$ is usually effective. It is mandatory to also check the serum calcium because hyperkalaemia can be accompanied by marked hypocalcaemia, particularly in patients with profound sepsis or renal failure. The use of bicarbonate in these situations can provoke a crisis by lowering the ionised calcium fraction, precipitating tetany, convulsions or hypotension and arrhythmias.

Table B.5 Salbutamol dose by age

Age (years)	Salbutamol dose (mg) nebulised
≤2.5	2.5
2.5–7.5	5
>7.5	10

Insulin and glucose are the classic treatment for hyperkalaemia. They are not, however, without risk, and the use of salbutamol has fortunately reduced the requirement for such therapy. It is very easy to precipitate hypoglycaemia if monitoring is not adequate. Large volumes of fluid are often used as a medium for the dextrose and, particularly in the patient with renal failure, hypervolaemia and dilutional hyponatraemia can then be a problem. Many patients are quite capable of significantly increasing endogenous insulin production in response to a glucose load, and this endogenous insulin is just as capable of promoting intracellular potassium uptake. It thus makes sense to start treatment with just an intravenous glucose load and then to add insulin as the blood sugar rises. The initial dosage of glucose ought to be 0.5 g/kg/h, i.e. 2.5 ml/kg/h of 20% glucose. Once the blood sugar is above 10 mmol/l, insulin can be added if the potassium level is not falling. The dosage of insulin is initially half that used in diabetic ketoacidosis, i.e. 0.05 units/kg/h. This can then be titrated according to the blood sugar.

The above treatments are the fastest means of securing a fall in the serum potassium, but all work through a redistribution of the potassium into cells. Thus the problem is merely delayed rather than treated in the patient with potassium overload. The only ways of removing potassium from the body are with dialysis or ion-exchange resins such as calcium resonium. If it is anticipated that the problem of hyperkalaemia is going to persist then the use of these treatments ought not to be delayed. Dialysis can only be started when the patient is in an appropriate environment, but will be the most effective and rapid means of lowering the potassium. Ion-exchange resins can be used at the outset. The dosage of calcium resonium is 1 g/kg as an initial dose either orally or rectally.

In an emergency situation where there is an arrhythmia (heart block or ventricular arrhythmia) the treatment of choice is intravenous calcium. This will stabilise the myocardium but will have no effect on the serum potassium. Thus the treatments discussed above will still be necessary. The dosage is 0.5 ml/kg of 10% Ca gluconate (i.e. 0.1 mmol/kg Ca); this dose can be repeated twice. With a very high potassium, more than one treatment can be used simultaneously.

Calcium

Some mention of disorders of calcium metabolism is relevant because both hyper- and hypocalcaemia can produce profound clinical pictures.

Hypocalcaemia Hypocalcaemia can be a part of any severe illness, particularly septicaemia. Other specific conditions that may give rise to hypocalcaemia are severe rickets, hypoparathyroidism, pancreatitis or rhabdomyolysis, and citrate infusion (in massive blood transfusions). Acute and chronic renal failure can also present with severe hypocalcaemia. In all cases hypocalcaemia can produce weakness, tetany, convulsions, hypotension and arrhythmias. Treatment is that of the underlying condition. In the emergency situation, however, intravenous calcium can be administered. As most of the above conditions are associated with a total body depletion of calcium and because the total body pool is so large, acute doses will often only have a transient effect on the serum calcium. Continuous infusions will also often be required, and must be given through a central venous line as calcium is irritant to peripheral veins. In renal failure, high serum phosphate levels may prevent the serum calcium from rising. The use of oral phosphate binders or dialysis or haemofiltration may be necessary in these circumstances.

Hypercalcaemia Hypercalcaemia usually presents as longstanding anorexia, malaise, weight loss, failure to thrive or vomiting. Causes include hyperparathyroidism, hypervitaminosis D or A, idiopathic hypercalcaemia of infancy, malignancy, thiazide diuretic abuse and skeletal disorders. Initial treatment is with volume expansion with normal saline. Following this, investigation and specific treatment are indicated.

B.3 DIABETIC KETOACIDOSIS

Diabetic ketoacidosis (DKA) is a condition in which a relative or absolute lack of insulin leads to an inability to metabolise glucose. This leads to hyperglycaemia and an osmotic diuresis.

Once urine output exceeds the ability of the patient to drink, dehydration occurs. In addition, without insulin, fat is used as a source of energy, leading to the production of large quantities of ketones and metabolic acidosis. There is initial compensation for the acidosis by hyperventilation and a respiratory alkalosis but, as the condition progresses, the combination of acidosis, hyperosmolality and dehydration leads to coma. DKA is often the first presentation of diabetes; it can also be a problem in known diabetics who have decompensated through illness, infection or non-adherence to their treatment regimes.

History

The history is usually of weight loss, abdominal pain, vomiting, polyuria and polydipsia, though symptoms may be much less specific in under 5-year-olds who also have an increased tendency to ketoacidosis.

Examination

Children are usually severely dehydrated with deep and rapid (Kussmaul) respiration. They have the smell of ketones on their breath. Salicylate poisoning and uraemia are differential diagnoses that should be excluded. Infection often precipitates decompensation in both new and known diabetics, and must be sought.

Management

Assess:

- **A**irway.
- **B**reathing.
- **C**irculation.

Give 100% oxygen and place on a cardiac monitor.

Take blood for:

- Bicarbonate/blood gases.
- Urea and electrolytes, creatinine, calcium and albumin.
- Glucose.
- Ketones.
- Haemoglobin and differential white cell count.

Take urine for:

- Culture.
- Sugar.

The principles of management of diabetic ketoacidosis are:

1. To reverse shock; fluid boluses should be given slowly in 10 ml/kg aliquots. It is rare to need more than 30 ml/kg. Resuscitation boluses are included as part of the total fluid needs (DKA only).
2. To rehydrate over 48 hours.
3. To replace insulin; the rate for insulin is under debate. Guidelines suggest 0.1 units/kg/h, but many paediatricians prefer 0.05 units/kg/h initially without a loading dose. Insulin should not be started until fluids have been running for 1 hour and therefore will probably commence after the child has been transferred.
4. To return the glucose level to that approaching normal.
5. To avoid hypokalaemia, hypoglycaemia and rapid changes in serum osmolarity.
6. To treat the underlying precipitating cause of the DKA.

The detailed management of DKA is complex. Advice should be sought from experienced local practitioners and published guidelines.

Complications

Major complications of diabetic ketoacidosis	
Cerebral oedema	Most important cause of death and poor neurological outcome. Attempt to avoid by slow normalisation of osmolarity with attention to glucose and sodium levels, and hydration over 36–48 hours
	Monitor for headache, recurrence of vomiting, Glasgow Coma Scale, inappropriate slowing of heart rate and rising blood pressure
	Treat with mannitol infusion (250–500 mg/kg over 20 minutes), or alternatively hypertonic saline may be used
	Hyperventilation has been associated with worse outcomes
Cardiac dysrhythmias	Usually secondary to electrolyte disturbances, particularly potassium
Pulmonary oedema	Careful fluid replacement may limit the occurrence of pulmonary oedema
Acute renal failure	Uncommon because of high osmotic urine flow

All of these complications require intensive monitoring on an intensive care unit.

APPENDIX C

Child abuse and neglect

C.1 INTRODUCTION

Child abuse is a universal occurrence. The medical aspects of this chapter are relevant in all settings, but the guidance for health professionals and legal details are specific to the countries indicated in the relevant parts of the text.

Health care workers will come into contact with:

- Children who have been abused by adults or by other children.
- Children who have abused other children.
- Adults who were abused as children.

Historical

The standard of care of children has varied over the centuries. Up to the nineteenth century, children were used in industry in a manner that today we would classify as abuse. In previous eras beating children as a means of discipline was accepted by most social groups. The extremes of physical abuse were described in 1962 by Kempe, an American paediatrician, as the 'battered baby syndrome' – multiple bruises, intracranial haemorrhages, fractures and internal injuries in children under the age of 1 year.

Since 1962 we have gradually recognised many more forms of abuse. Present classifications are as follows.

Classification of child abuse

Neglect

Neglect means the persistent or severe neglect of a child, or the failure to protect a child from exposure to any kind of danger, including cold or starvation, abandonment or extreme failure to carry out important aspects of care, resulting in the significant impairment of the child's health or development, including non-organic failure to thrive.

Physical injury

This is actual or probable physical injury to a child, or failure to prevent physical injury (or suffering) to a child, including deliberate poisoning, suffocation and illnesses or injuries, which are fabricated or induced.

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Sexual abuse

This is the involvement of dependent, developmentally immature children and adolescents in sexual activities that they do not truly comprehend, to which they are unable to give informed consent or which violate the social taboos of family roles. There are many types of sexual abuse:

- Touching, fondling or licking of genitals or breasts.
- Masturbation of a child by an adult or an adult by a child; or of an adult in the presence of a child.
- Body contact with the adult genitals including rubbing or simulated intercourse by the adult against or between thighs, buttocks or elsewhere.
- Heterosexual or homosexual intercourse with actual or attempted vaginal, anal or oral penetration.
- Exhibitionism (the display of genitals).
- Involvement in pornography, including photography and erotic talk.
- Involvement in prostitution, male or female.
- Other varieties of sexual exploitation, including internet child pornography and 'grooming' of a child.

Most of these abusive acts will leave no physical signs on the victim.

Emotional abuse

This is described as actual or probable severe adverse effect on the emotional and behavioural development of a child caused by persistent or severe emotional ill treatment or rejection. All abuse involves some emotional ill treatment. This category should be used where it is the main or sole form of abuse.

Grave concern

This is described in children whose situations do not currently fit the above categories, but where social and medical assessments indicate that they are at significant risk of abuse. These could include situations where another child in the household has been harmed or the household contains a known abuser, including situations where an adult is the subject of domestic abuse.

Organised abuse

This characteristically involves multiple perpetrators, involves multiple victims and is a form of organised crime. There are three subsections. The first is paedophile and/or pornographic rings. The second is cult-based ritualistic abuse in which the abuse has spiritual or social objectives. The third is pseudoritualistic abuse in which the degradation of children is the end rather than the means.

Presentations of physical abuse

- Head injuries – fractures, intracranial injury.
- Fractures of long bones:
  - single fracture with multiple bruises,
  - multiple fractures in different stages of healing, possibly with no bruises or soft tissue injury, or
  - metaphyseal or epiphyseal injuries, often multiple.
- Fractured ribs and spinal injuries.
- Internal damage, e.g. rupture of bowel.
- Burns and scalds – 'glove and stocking' appearance for scalds, implement imprints for contact burns.
- Cold injury – hypothermia, frostbite.
- Poisoning – drugs or household substances, suffocation.
- Cuts and bruises – imprints of hands, sticks, whips, belts, bites, etc. may be present.

Presentations of sexual abuse

- Disclosure by child.
- Disclosure by witness.
- Suspicion by third party because of the behaviour of the child, especially changes in behaviour. These include insecurity; fear of men; sleep disorders; mood changes, tantrums and aggression at home; anxiety, despair, withdrawal and secretiveness; poor peer relationships; lying, stealing or arson; school failure; eating disorders like anorexia and compulsive overeating; running away and truancy; suicide attempts, self-poisoning, self-mutilation and abuse of drugs, solvents and alcohol; unexplained acquisition of money; sexualised behaviour such as drawings with a sexual content; knowledge of adult sexual behaviour shown in speech, play or drawing; apparent sexual approaches; and promiscuity.
- Symptoms such as a sore bottom, vaginal discharge, bleeding perirectum or inflammed penis which the caregiver believes is due to sexual abuse.
- Symptoms as above and/or signs (e.g. unexplained perineal tear and/or bruising, torn hymen or perineal warts), but the doctor is the first person to suspect abuse.
- Sexually transmitted disease.
- Faecal soiling or relapse of enuresis.
- Child (usually adolescent girl) presents frequently with a variety of problems including recurrent abdominal pain, overdose of drugs and reluctance to go home.
- Pregnancy but the girl refuses to name the putative father or even indicate the category, e.g. boyfriend, casual acquaintance.

C.2 ASSESSMENT

The child who has disclosed abuse, or who is the subject of suspected abuse, will be overwhelmed by the number of professional people who are involved in the assessment of the situation. If the disclosure or suspicion arises in a nursery or school, then teachers and health visitors/school nurses will make preliminary enquiries and referrals. In all intrafamilial abuse, social workers will speak to the child and the family. They will be responsible for the safety of the child, for ongoing care of the family, and for any subsequent civil proceedings. All child abuse is criminal activity, so police officers will interview the alleged victim, the alleged offender and any other witnesses to the incidents. There should be good liaison between social workers and police officers so joint interviews can be done to minimise the number of times the child will have to relate the details of the incident(s). Whenever possible, these interviews are recorded on videotape to be used as evidence. Under the Criminal Justice Act 1991 (England and Wales), videotapes can be used as evidence in chief for children under the age of 14 years, provided that the child is available for cross-examination, and then a video link may be allowed by the judge. Under the Youth Justice and Criminal Evidence Act 1999, the age for child witnesses is now 17 years, but any vulnerable or intimidated adult is now also eligible for the same special measures.

Medical assessment will be carried out by a paediatrician with forensic training or jointly by a police surgeon and a paediatrician. If the child has severe psychological disturbance or psychiatric symptoms, then a psychologist and/or psychiatrist will also see the child and the family. The basic medical assessment should follow the pattern used for all other diagnostic problems. Consent is not mandatory for child protection concerns but it is good practice to involve the family wherever feasible.

Details of medical assessment

History

Full details of the history of the incident(s) should be obtained from the child and the caregivers. If social workers and police officers have previously talked to the child, then taking this history from them may be appropriate, especially for alleged sexual offences. Frequent repetition of the details can be very disturbing to the child and can jeopardise evidence. Remember

to remain objective and show professional sensitivity. Systemic enquiry is then done for the cardiovascular system, respiratory system, gastrointestinal tract (remember to ask about soiling, constipation, rectal pain and rectal bleeding), urogenital system (remember to ask about wetting, vaginal bleeding, vaginal discharge and, when appropriate, menarche, cycle, sanitary protection and previous sexual intercourse), central nervous system, musculoskeletal system, skin and behaviour.

Personal history must start with pregnancy, birth, the neonatal period and subsequent developmental milestones. Then details of immunisations, drug history (including alcohol and street drugs) and allergies are obtained. Information on the child's performance at nursery or school should include social factors.

Enquiries are made about previous illnesses and injuries, with dates of attendance at hospital or at the surgery of the family doctor. Past records should be obtained and relevant information should be extracted.

The traditional family history should include details of the natural parents, all cohabitees and any other people who regularly care for the child, e.g. relatives and childminders. Parental illness should be discussed, particularly psychiatric illness. The presence of domestic violence should be explored. Then the names, ages and medical histories of all siblings and half-siblings are obtained. Any miscarriages, stillbirths or deaths of siblings are discussed sensitively. Familial illnesses which are particularly important are inherited skin or blood disorders.

Examination

The general examination starts while the history is being taken. During that time the doctor observes the affect of the child, the relationships between the child, mother, father and others present and any behavioural problems. If the child is reluctant to be examined, then playing with toys or the doctor's stethoscope often breaks the ice. No child should be examined against his or her will as this constitutes an assault. Sometimes a child who refuses to be examined one day will come back quite cheerfully another day. Examination under anaesthesia is rarely required.

Each child is examined from head to toe rather than in systems. Height and weight are checked, as is head circumference in babies. Careful notes are made of all normal and abnormal findings, including any marks on clothing, e.g. tears or blood stains. Such clothing may be required by the police. All marks, contusions, abrasions and lacerations must be measured and related to anatomical landmarks and described as fully as possible. Drawings must be made. If an abnormality is found that has not been discussed previously in the history, then further questions are asked – most undisclosed events are recent minor childhood accidents or previous ones that have left scars. When the upper part of the body has been examined, the child is asked to put the clothes back on to that area before taking the clothes off the abdomen and legs. Finally, the genitalia and anal region are examined; this part of the examination should be recorded using a videocolposcope whenever possible, enabling future review of the videotape evidence and discussion with medical colleagues, avoiding unnecessary repeat examinations. This method minimises the embarrassment of the child.

Investigations

During the examination, specimens needed for forensic investigation will be taken by a police surgeon or a paediatrician with forensic experience. These are relevant when there has been contact within 7 days of the examination. Swabs for microbiological investigation will be taken if there is a vaginal discharge or if threadworms may be present. Investigations for sexually transmitted diseases are done ideally 7 days after the last alleged offence if oral, vaginal or anal intercourse has taken place.

If bruises are found, then organic disease may be present with or without abuse, so haematological investigations are needed. Venous blood is taken for full blood count, bleeding and clotting studies.

Radiograph interpretation

Occasionally old rib fractures may be seen on a chest radiograph. Posterior rib fractures in adjacent ribs are very suggestive of non-accidental injury due to abnormal squeezing or compression of the chest. Recent rib fractures, unless displaced, may be difficult to detect radiographically and may only be seen in the healing phase. Small children's ribs are relatively pliable compared to adults and will tend to bend rather than fracture with compressive forces. It is exceptionally unusual to fracture a child's ribs during cardiopulmonary resuscitation in a child with a normal skeleton. The presence of a rib fracture, recent or healed, is a significant finding. Metaphyseal fractures seen in the shoulders on a chest radiograph are significant.

Skull fractures may occur in small infants who fall from a significant height onto a hard floor, but are rarely seen when a child rolls off a sofa onto a carpeted floor. Femoral and humeral fractures occur infrequently in domestic accidents in infants. The history always needs to be correlated with the clinical and radiographical findings.

In suspected non-accidental injury the child should be protected from further assault and further assessment made. In physical abuse of children under the age of 2 years this involves a full skeletal survey. A skeletal survey can only be performed after adequate explanation to the child's carers, and does not normally need to be performed in the emergency situation. The components of a skeletal survey are shown in the box below.

Components of a skeletal survey

- Front and lateral skull films
- Lateral whole spine
- Chest radiograph
- Anteroposterior (AP) views of all the long bones
- AP views of lumbar spine, pelvis and hips
- Supplemented with lateral views of the metaphyses where there is any suspected abnormality or clinical symptoms
- Neurocranial imaging (e.g. computed tomography and/or magnetic resonance imaging) as appropriate to the child's symptoms

Diagnosis

Classic pointers to the diagnosis of inflicted injury are:

- There is delay in seeking medical help or medical help is not sought at all.
- The story of the 'accident' is vague, is lacking in detail and may vary with each telling and from person to person. Innocent accidents tend to have vivid accounts that ring true.
- The account of the accident is not compatible with the injury observed.
- The injury is not compatible with the child's level of development or of the level of development of another child alleged to have caused the injury.
- The parents' affect is abnormal. Normal parents are full of anxiety for the child who has been injured. Abusing parents tend to be more preoccupied with their own problems – for example, if they can return home as soon as possible.
- The parents' behaviour gives cause for concern. They may become hostile, rebut accusations that have not been made or leave before the consultant arrives.
- The child's appearance and his interaction with his parents are abnormal. He may look sad, withdrawn or frightened. There may be visible evidence of failure to thrive. Full-blown frozen watchfulness is a late stage and results from repetitive physical and emotional abuse over a period of time.
- The child may disclose abuse. Always make a point of talking to the child in a safe place in private if the child is old enough to be separated from the parents. Interviewing the child as an outpatient may fail to let the child open up as he is expecting to be returned home in the near future. He may disclose more in the safety of a foster home.

At the end of the medical assessment the diagnosis may be clear. More often the doctor has a differential diagnosis that includes abuse. Discussion then takes place among the social workers, health care workers and police officers, who have information about the family, to balance the probabilities of abuse having occurred. A child protection conference will be held as soon as possible. In the meantime it may be necessary to arrange for the child to be taken to a place of safety (see 'Emergency Protection Orders' below).

C.3 MANAGEMENT

All child protection work is based on cooperation between families, social workers, police officers, health care workers and educationalists. This multiagency approach is to ensure that all aspects of the care of the family are considered when decisions are being made. Certain decisions in management must be made by a professional, e.g. only a doctor can decide on the treatment required for fracture and only a police officer can decide the charge that is appropriate for the alleged offence. However, whenever possible, unilateral decisions are avoided in the best interests of the child and the family.

Because of the complexity of interaction between these agencies, the most common reason for child protection disasters has been failure of communication. Over the years, in the United Kingdom, there have been a number of high-profile cases that have been investigated and led to various reports. The most recent have been the Laming reports (2004 and 2009), which have made recommendations for health care professionals in the acute setting. The recommendations are designed to improve communication and to identify accountabilities. They include:

- Taking a history from the child where possible, with an interpreter when necessary.
- Having access to previous records and the Child Protection Register.
- Making comprehensive and contemporaneous records of all findings and communications.
- Seeking further opinion where needed and allowing discharge of the child only by a senior doctor and with a plan for future care.
- Having clear lines of responsibility and a single set of hospital records.
- Training and updates in child protection for those managing children.

Doctors may be concerned about sharing information with other professionals because of the ethical consideration of confidentiality. In the United Kingdom, the General Medical Council (2004) gives the following advice.

Disclosures where a patient may be a victim of neglect or abuse

If you believe a patient to be a victim of neglect or physical, sexual or emotional abuse and that the patient cannot give or withhold consent to disclosure, you must give information promptly to an appropriate responsible person or statutory agency, where you believe that the disclosure is in the patient's best interests. If, for any reason, you believe that disclosure of information is not in the best interests of an abused or neglected patient, you should discuss the issues with an experienced colleague. If you decide not to disclose information, you must be prepared to justify your decision.

When the diagnosis is one of child abuse then the decisions to be made on management are the following:

- Does the child need admission for treatment of the injuries?
- Will the child be safe if returned home?
- If the child needs protection from an abuser who is in his or her own home, how can this be done?
- What support/protection is needed for the rest of the family, including siblings?

If the alleged abuser is not in the same household as the child and the caregivers can protect the child, then he or she can return home. If the alleged abuser is in the same household as the child but is in custody, then the child will still be safe at home with another caregiver. When a person is charged and is allowed bail, one condition must be that he or she lives away

from the household of the child. If this is not done then alternative care will be needed for the child.

Whenever there is a disclosure or suspicion of abuse, the whole family needs support. Siblings may have been at risk of injury and so will need to be assessed. Spouses may be ambiguous in their loyalties to the child and to the alleged abuser. The child will need much support to withstand the stress of the investigation, especially if there are subsequent legal proceedings.

The details of management of these many facets are decided in the child protection conference. In this all the professional people meet with the family to share information and to produce a plan of care.

C.4 MEDICOLEGAL ASPECTS

Health care professionals must be familiar with the medicolegal aspects of their work. These may vary according to the jurisdiction where the clinician practices. In England and Wales the most important are the following:

- Emergency Protection Orders, Child Assessment Orders, Residence Orders, Police Protection Orders.
- Consent to examination.
- Writing of statements and reports for criminal and civil proceedings.
- Presentation of evidence.

Emergency Protection Order (EPO)

The Emergency Protection Order (Children Act 1989, sections 44 and 45) replaced the Place of Safety Order. It may be made for a maximum of 8 days, with a possible further extension of up to 7 days. An application for discharge of that order may be made. The court may only make the order if it is satisfied that there is reasonable cause to believe that the child is likely to suffer significant harm if either he is not removed to another place or his removal from a safe place (such as a hospital) is not prevented. Another clause is that, in the case of an application made by a social services department or the National Society for the Prevention of Cruelty to Children (NSPCC), the applicant 'has reasonable cause to suspect that a child is suffering or is likely to suffer significant harm' and enquiries which are being made with respect to the child 'are frustrated by access to the child being unreasonably refused' and the applicant believes that access is required as a matter of urgency.

Child Assessment Order (CAO)

The Child Assessment Order (Children Act 1989, section 43) addresses those situations where there is good cause to suspect that a child is suffering or is likely to suffer significant harm but is not at immediate risk, and the applicant believes that an assessment (medical, psychiatric or other) is required. If the parents are unwilling to cooperate, the social services department or the NSPCC can apply for a CAO. The CAO has a maximum duration of 7 days from the date on which it comes into effect. The court will direct the type and nature of the assessment that is to be carried out, and whether the child should be kept away from home for the purposes of the assessment. A child of reasonable understanding may refuse to have this assessment. Lawyers suggest that a child of reasonable understanding is a normal child of 10 years of age or more.

Residence Order

A Residence Order states with whom the child is to live. It has the effect of ending any care order and gives parental responsibility to the person with the order.

Police Protection Order

A constable has powers (Children Act 1989, section 46) to take a child 'into police protection' for up to 72 hours. This power can be used to prevent the removal of a child from hospital.

Consent to examination

Consent for all examinations that are for evidential purposes must be obtained from a person with parental responsibility. In England and Wales under the Children Act 1989 (section 3), parental responsibility is defined as 'all the rights, duties, powers, responsibilities and authority which by law a parent has in relation to the child and his property'. Those with parental responsibility are specified in the Children Act 1989 (section 2), and are summarised in the box.

Parental responsibility

- Parents married at the time of birth both have parental responsibility, which continues after separation or divorce
- An unmarried mother always has parental responsibility unless the child has been adopted
- An unmarried father has parental responsibility if he is named on the child's birth certificate and the birth is registered on or after 1 December 2003
- An unmarried father of a child born before this date or un-named on the birth certificate can acquire parental responsibility, by marrying the mother, by a parental responsibility agreement with the mother (stamped by the court) or by an order from the court
- A person in whose favour a Residence Order has been made has parental responsibility – this is for the duration of the Order
- An appointed guardian has parental responsibility
- The local authority has parental responsibility while a Care Order is in force
- A person who applied for an Emergency Protection Order has parental responsibility
- A person in whose favour an Adoption Order has been made has parental responsibility

When more than one person has parental responsibility, each of them can act alone and without the other in meeting that responsibility. Parents do not lose parental responsibility if a Care Order or an EPO is in force, but their responsibility may be limited by the local authority. Parents lose parental responsibility with an Adoption Order. Parental responsibility can be delegated to a person acting on their behalf, e.g. while they are on holiday.

To cover emergency situations, those caring for a child who do not have parental responsibility may do what is reasonable in all the circumstances for the purpose of safeguarding or promoting the child's welfare.

Consent from the child or young person is needed if that person is of sufficient understanding to make an informed decision. Lawyers suggest that in a normal child this would be at age 10 years. The Fraser (previously known as Gillick) ruling (1986) allows an individual under the age of 16 years to submit to examination and treatment without the parents being informed, provided that is the wish of the child or young person.

Court reports

When preparing a written report on a child for the court all health care professionals should keep in mind that the written report may be used in subsequent court appearances. Therefore, the report should be confined to the facts. Whenever possible, objective and measurable evidence of the child's health and development should be presented. Where subjective views must be given they should reflect balanced professional judgement. If the report is comprehensive and comprehensible, then the health care professional may not be called to give verbal evidence in person. Always keep a copy of your report.

Statements

The purpose of a statement is to provide the court with an informative and relevant account of the medical assessment of the child. The statement will give details of the persons involved, the observations and the findings. Information given by another person should not be included

unless this has been requested. In many areas, the prosecutors wish statements to record all information, although hearsay may be excluded before presentation to the court.

A statement is a professional document. It should be well written in clear, readily understandable language. Technical terms should be avoided or, if used, should be followed immediately by appropriate lay terms. Most statements will be for the prosecution and a printed statement form will be provided. The standard sequence of writing a statement is as shown in the box below.

Each page must be signed at the bottom, and the final page must be signed on the line below the completion of the writing. Any alterations must be initialled.

Always keep a copy of the statement.

Sequence for writing a statement

1. Full name with surname in capitals
2. Qualifications
3. Occupation
4. Name of person requesting the assessment
5. Date, time and place of the assessment
6. Name of person who was examined
7. Name of persons present
8. Details of the relevant history – if a general history was taken but produced nothing significant then make a general comment including the sight of the detailed notes
9. Details of examination – if it was a joint examination then specify who did each part
10. Investigations
11. Opinion on findings
12. The time at which the examination ended

Presentation of evidence

Dress in a professional manner. Arrive early in court. Take along all notes relevant to the case. Revise these on the day before the court proceedings. With permission from the magistrate or judge, you may refer to contemporaneous notes. However, revision helps to put the whole picture of the incident into the forefront of your mind so that you can find the appropriate notes more quickly.

When giving evidence stay calm even when challenged. Do not be persuaded to answer questions that are outside your knowledge or experience.

APPENDIX D

Prevention of injury in children

D.1 INTRODUCTION

In developed countries, injury is the leading cause of death in children aged between 1 and 14 years. Millions more children worldwide are injured in accidents that, although not causing death, cause pain, distress and permanent disability. The majority of these incidents are predictable and preventable.

Over the last few decades, injury prevention programmes in some countries have succeeded in halving childhood death rates from injury. This is a remarkable achievement and a good start to reducing childhood injury as far as possible. Injury prevention is a multifaceted, multidisciplinary process that provides many opportunities for clinicians, who are primarily involved in the management of acutely injured children, to play a major role.

D.2 EPIDEMIOLOGY

Circumstances and type of incident

A multitude of injury scenarios are possible, each of which involve the child interacting with their environment. The commonest injuries that cause death are those resulting from motor vehicle accidents, drownings, burns, falls from a height and poisonings. The commonest incidents overall, however, are falls leading to injuries such as bruises, abrasions and fractured limbs. Children in urban environments are at particular risk of motor vehicle accidents and playground falls whilst children in a rural environment are at risk of farm equipment injuries, unintentional chemical exposure and, in some countries, snakebites. Exposure to different circumstances also varies with age. Children under 5 years experience injuries at home. School-age children experience injuries at school, sport and play, and are especially at risk of death as pedestrians and cyclists. Adolescents may deliberately place themselves at risk of injury, especially where alcohol and drugs may impair their judgement.

Sex

Boys are more frequently injured than girls. The difference emerges at age 1–2 years of age. How much of this difference is innate and how much cultural is a subject for speculation. Girls may mature more rapidly in terms of perception and coordination.

Age

The type of injury sustained is closely related to the child's stage of development. Take falls as an example. A newborn baby can only fall if dropped, or if a parent falls holding the baby. An older baby can wriggle and roll off a changing table or a bed. A crawling baby can climb upstairs and fall back. A small child can climb and fall out of a window. An older child can climb a tree or fall in a playground.

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Social class

As with so many other health problems, injuries are linked to inequalities in environments. Children in social class V, derived from the occupation of the head of the household, are twice as likely to die from an injury as children in social class I and, for some injury types, such as burns, the chances are six times higher.

This does not mean that working class parents care less about their children than middle class parents, or that they do not know about risk. It may mean that there are other pressures such as overcrowding, lack of money or poor housing, and there is less ability for financial reasons to make safety-related changes.

Psychological factors

Injuries are more common in families where there is stress from mental illness, marital discord, moving home or a variety of similar factors.

D.3 INJURY PREVENTION

There are three levels of injury prevention. Primary injury prevention is any measure designed to reduce the incidence of injury. Examples of this are the use of speed limits, pool fences, fireguards and child-resistant medication closures. Secondary injury prevention is any measure designed to minimise injury even though an incident has occurred. Examples of this are the proper use of seat belts, bicycle helmets and other personal protective clothing. Tertiary injury prevention is any measure designed to limit the extent or consequences of an injury that has already occurred. Examples include the application of cold water to burns and scalds, or direct pressure on a laceration.

Whilst injury prevention can be addressed on an individual level it is most effective for the community as a whole when viewed as a public health issue. The most successful injury prevention campaigns have a number of common attributes. Firstly, they are carefully planned, with attention given to data collection and the identification of specific issues in the target population. Secondly, they attempt to permanently change behaviour by the use of education and enforcement. Finally, they include methods to monitor effectiveness, provide feedback and modify the campaign as necessary.

Clinicians involved in the acute management of injured children are in a unique position to be able to assist in injury prevention. Their daily work gives them first-hand knowledge of injured children and credibility with parents and government. Some of the ways that clinicians may be involved include the following.

Data collection and analysis

The provision of accurate and reliable data regarding incidence and circumstances of child injury occurring in a particular city or country underpins any injury prevention strategy. It identifies areas of high priority and enables the monitoring of effectiveness. In addition, it assists in the recognition of local and national factors that contribute to injury that may need to be specifically addressed. The power of the information and the ability to identify trends will be increased if the data are pooled into a national database that is accessible by many sources.

Education

Parents, community groups and politicians need information regarding childhood injuries and the methods likely to prevent them. Information can be delivered directly face to face in talks and interviews or by posters, books, pamphlets and the internet. The information must be relevant, accurate and presented at a level appropriate to the target group. Information based on local data presented by a credible person is most likely to be well received.

Publicising cases in the media can be an effective strategy to convey messages to parents, especially if the topic is newsworthy. Such publicity tends to be immediate and short lived but may be particularly useful in certain circumstances such as at the start of summer (e.g. snakebites, drowning).

The Injury Minimisation Programme for Schools (IMPS) provides an education pack with accident lessons drawn from the national curriculum in the UK and includes a hospital visit. Health professionals are actively encouraged to contact this group as its reach through schools in the UK has expanded and requires further support from interested health professionals.

Advocacy for legislation and design

Child injury prevention is all about changing behaviour. Whist education is the preferred way of encouraging people to do this, the introduction of legislation, regulations and enforceable standards have been an extremely effective adjunct. For example, legislation regarding pool fencing and regulations concerning the packaging of medications are two important legal measures specifically directed at child safety.

Whilst clinicians do not generally draft and enact legislation and regulations they can play an important role in convincing politicians that such measures are necessary and in ensuring that they are enforced. People must not only have the knowledge of what is safe, they must have the ability to select safe products and be protected against things that are inherently unsafe. Safe design of products designed for use by and around children is essential. Clinicians have a responsibility to notify authorities when they become aware of a dangerous toy that has injured a child.

Involvement with child safety organisations

Many countries have organisations dedicated to preventing childhood injuries. These organisations are often involved in all facets of injury prevention and provide ideal vehicles for individuals to work within. Be prepared to participate in working groups and campaigns. Health care professionals have special expertise and influence to offer. Local initiatives involving health professionals have had an impact, e.g. work in bicycle helmet use and playground safety has led to a better understanding of the effects of altering the environment and on implementing advances in design.

The collection of data about accidents can lead to a decrease in childhood injury; for example, by identifying accident black spots and collaborating with police and local council authorities, effective safety changes can be implemented. Similarly, types of frequently occurring domestic injuries can lead to targeted campaigns.

Children's accidents and injuries are the major public health problem for children in developed and developing countries today. All health care workers can learn more about them, and can be active in reducing their toll. Health care professionals can form powerful alliances with heads of schools, playgroup leaders, local media, police and local councils to launch injury-prevention schemes. Support for such initiatives can be given by charitable agencies such as the Child Accident Prevention Trust, the Gloucestershire Home Safety Check and the Royal Society for the Prevention of Accidents in the UK and Kidsafe in Australia.

Prevention measures

Primary prevention measures

- Parental knowledge regarding behaviour and supervision of young children
- Fencing around domestic swimming pools
- Child-resistant closures on medication containers
- Fireguards surrounding open fireplaces
- Motor vehicle speed limits around schools
- Automatic water temperature regulation in bathrooms
- Use of stair guards, window guards and toughened glass
- Installation of electrical safety switches
- Removal of unsafe toys from retail outlets

Continued

Secondary prevention measures
- Properly fitted child restraints in motor vehicles
- Wearing of bicycle helmets at all times
- Personal protective equipment such as mouth guards and wrist guards
- Installation of domestic smoke alarms, fire extinguishers and fire blankets

Tertiary prevention measures
- Cardiopulmonary resuscitation training
- Compressive bandage in snake bites
- Rapidly responding, well-trained ambulance service
- Excellent trauma care from retrieval to rehabilitation

APPENDIX E

When a child dies

E.1 INTRODUCTION

Even with the best preventative measures in place and the use of the most effective resuscitation methods, children will continue to die from serious illness and severe injury. When a death occurs, medical and nursing staff must be able to deal effectively with the child's family and the legal requirements of death as well as cope with their own emotional reactions. Sympathetic and sensitive support of the family at this time can do much to help the grief process and adjustment to the bereavement.

The principles in dealing with a family that has experienced a sudden child death are shown in the box below.

Principles in dealing with a family

- Display caring, kindness and compassion.
- Spend as much time as necessary with the family in an unhurried fashion.
- Offer information regarding the death as the family requires.
- Talk to colleagues later regarding your experience and feelings.

Unless a clear care plan indicating 'no resuscitation' has been negotiated in advance with the parents and recorded in the medical records, full resuscitation should be undertaken. Parental presence during resuscitation is increasingly common, and whether this occurs should be a decision made jointly by the child's parents and staff. Although being present at their child's resuscitation is always extremely traumatic for the parents, when it is over they are almost always left with the impression that everything possible was done to save their child. If parents are present during resuscitation, then a member of staff must be available exclusively for their support. If the presence of parents is impeding the progress of the resuscitation, they should sensitively be asked to leave.

E.2 DEALING WITH THE FAMILY

Breaking the news

Telling the parents that their child has died is a difficult task and is usually undertaken by a senior and experienced staff member. Before speaking to the parents ensure they are in a private, comfortable environment and that you know the name of the child.

A direct and sympathetic approach is best, avoiding euphemisms and clichés. If it is appropriate and you feel comfortable doing it you may show sympathy by holding the parent's hand or putting an arm around them. Usually the parents will turn away towards each other for a while but may wish to ask questions about the cause of death and what they should do now. The parents will often want to know what happened and what treatments were instituted. If you are asked about the cause of death answer as simply and honestly as you can, making it clear that some answers are not yet available.

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Caring for the parents

Provide the family with a private room in which they can be alone with their child for as long as they wish. Encourage the family to touch and hold the child. Offer to stay with the family; however if they wish to be left alone, assure them you will be nearby if they wish to speak with you. In cases where there have been child protection concerns it will be necessary for the parents to be accompanied by a professional when they are with the child.

Accept the family's distress as natural and support them in this by acknowledging their feelings. Be prepared for a variety of responses: there is no 'correct' way to grieve and each person will have a different reaction. Be sensitive to and respectful toward varying cultural norms and rituals surrounding death.

Facilitate contact with other family members and friends as required. Even very young children may be included in the grief process right from the start; assist the family in feeling comfortable with this.

Each institution will have its own bereavement support programme: ensure that you are familiar with local resources and that the family is offered ongoing support and medical advice.

E.3 POST-DEATH PROCEDURES

Every jurisdiction will have specific legal requirements that need to be adhered to. It is usually necessary for the coroner, the police or another statutory authority to be informed of the death. The requirements for a police or coronial investigation, an autopsy and an inquest will vary from case to case.

A customised checklist is invaluable for ensuring that procedures or information are not forgotten. The box opposite gives an outline of such a list; however, local hospital guidelines should be followed. In all cases of sudden unexpected death in the UK, there are local procedures for reporting to and investigation by a multi-agency team led by the Designated Doctor for Unexpected Deaths.

E.4 TAKE CARE OF THE STAFF

The sudden and unexpected death of a child is extremely distressing for all involved. Some staff members may be profoundly affected. Encourage staff members to talk about the event and their feelings in private soon afterwards with a colleague. Formal staff counselling should be available if needed.

Checklist for post-death procedure

The child

- Full and thorough examination
- Core temperature
- Wrap child in clean warm clothes for parents to see and hold (if consistent with forensic requirements)
- Samples or swabs if agreed as mandatory in local protocol

The parents

- Explain that the child (use name) has died
- Gently get as full a history as possible
- Ask if they would like a priest/religious leader present
- Ask if they want any close relative to be contacted
- Encourage the parents to see and hold the child
- Let them know if a post-mortem examination needs to be carried out and ensure that they understand all that they wish to know about the procedure and have given their written consent where appropriate
- Let them know that police and coroner are always informed of sudden unexpected deaths and will need to ask a few simple questions of the carers
- Ask what address the family will be going to on leaving the hospital
- Arrange transport from hospital to home and if alone make sure they are accompanied on the journey and not left alone at home
- Be gentle, unhurried, calm and careful
- Do not guess at the diagnosis

Obtain details of

- Child's and parents' names
- Child's date of birth
- Address at which death occurred
- Time of arrival in department
- Time last seen alive
- Usual address if different from above

Inform

- GP – advise of child's death and give the address to which parents will be going from hospital
- Health visitor
- Social worker
- Any relative as requested by the family
- Coroner – who will need to know the full name and address and date of birth of the child, time of arrival, place of death, brief recent history and any suspicious circumstances

APPENDIX F

Management of pain in children

F.1 INTRODUCTION

For many years children, especially the very young, have been undertreated for pain. Possible reasons for this include:

- A fear that the side effects of analgesics are more harmful in children.
- A reluctance to give injectable analgesia.
- An inability to measure pain in children.
- A failure to accept that children feel pain like adults.

Fortunately this situation is changing rapidly. More effective therapeutic options, a better understanding of pain and pain measurement, and an enhanced awareness of the need for analgesia have combined to lead to better treatment. Not only is this more humane but it has enhanced care as inadequate analgesia can be detrimental to the critically ill child. Bronchoconstriction and increases in pulmonary vascular resistance caused by pain can lead to hypoxia, whereas good pain control facilitates the assessment of the severity of illness.

F.2 RECOGNITION AND ASSESSMENT OF PAIN

There are three main ways in which we recognise that a child is in pain:

1. Firstly, listening to the child for statements that they are in pain.
2. Secondly, observing the child's behaviour and physiology for things such as crying, guarding of the injured part, facial grimacing, pallor, tachycardia and tachypnoea.
3. Thirdly, anticipating pain because of the event the child has experienced, e.g. fracture, burn or other significant trauma.

The purpose of pain assessment is to establish, as far as possible, the degree of pain experienced by the child so as to select the right level of pain relief. Additionally, reassessment using the same pain tool will indicate whether the pain management has been successful or whether further analgesia is required – the assess, treat and reassess cycle. The use of pain tools and protocols in the emergency setting has been shown to shorten the time to delivery of analgesia.

Pain assessment at triage in the emergency situation is unique and therefore a pain assessment tool, specifically designed for this situation, is desirable. The anxiety associated with a sudden and unexpected presentation in pain confounds the ability of an individual, especially a child, to make a satisfactory self-assessment of pain that can be used to guide analgesic requirements. Therefore an observational pain scale appears to be more appropriate in this setting. The Alder Hey Triage Pain Score is one such tool that has been developed specifically for this situation and is shown to have some validity as well as good levels of inter-rater reliability (Table F.1). It is an observation-based pain score, which is quick and easy to use.

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Table F.1 The Alder Hey Triage Pain Score: reference scoring chart

Response	Score 0	Score 1	Score 2
Cry/voice	No complaint/cry	Consolable	Inconsolable
	Normal conversation	Not talking negative	Complaining of pain
Facial expression	Normal	Short grimace <50% time	Long grimace >50% time
Posture	Normal	Touching/rubbing/sparing	Defensive/tense
Movement	Normal	Reduced or restless	Immobile or thrashing
Colour	Normal	Pale	Very pale/'green'

Guidance notes for the use of the score can be found at the end of this appendix.

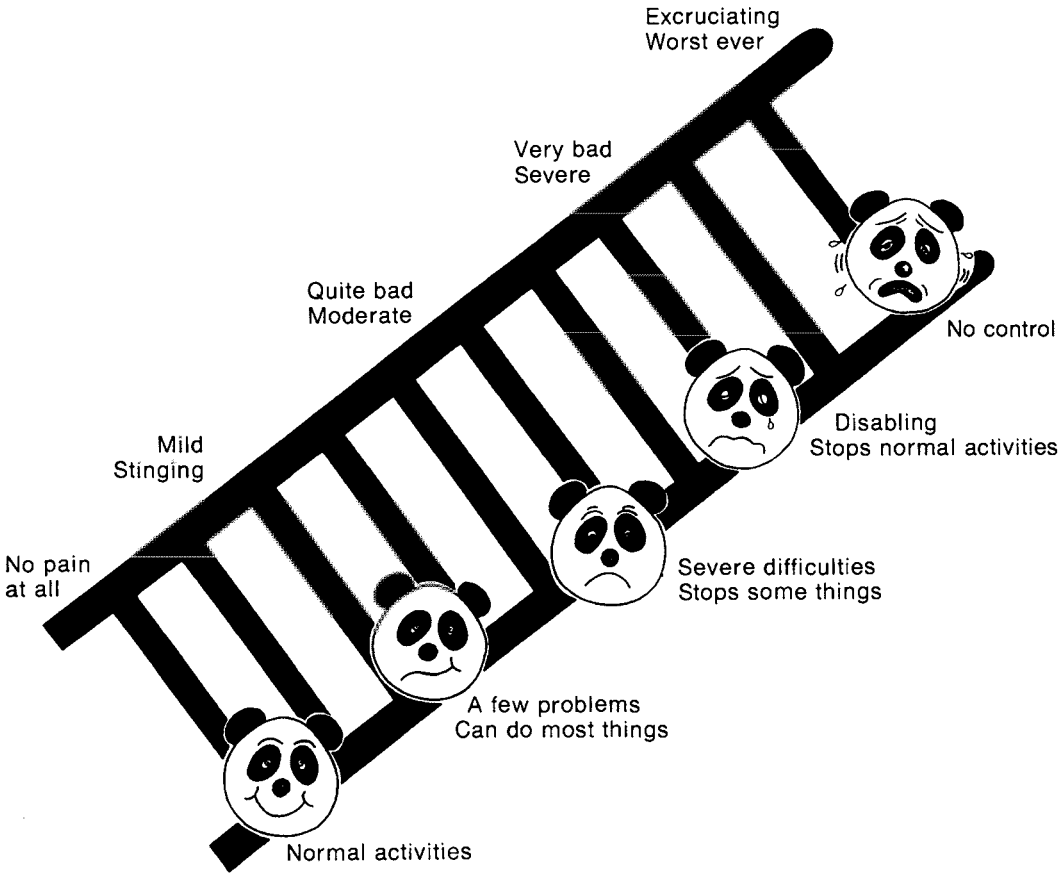

Figure F.1 Faces scale and pain ladder

Other commonly used pain scales are self-assessment tools, for example a faces scale or pain ladder (Figure F.1). Self-assessment tools, however, were primarily developed for use with children where there was the opportunity for explanation of the scale prior to the painful event, e.g. before surgery. This is clearly not the case in the emergency department.

All or any of these tools can be used to assess the pain experienced by the child and help, in the setting of the specific clinical situation, to guide the need for the level and route of analgesia. The tools can then be used again to assess the efficacy of the intervention and to guide further analgesia.

F.3 PAIN MANAGEMENT

Environment

The emergency department and the treatment room of the paediatric ward can be frightening places for children. Negative aspects of the environment should be removed or minimised. This includes an overly 'clinical' appearance and evidence of invasive instruments. An attractive, decorated environment with toys, mobiles and pictures should be substituted.

Preparation

Except in a life-threatening emergency or when dealing with an unconscious child, an explanation of the procedure to be undertaken and the pain relief planned should be given to the child and the parents. If time permits, they should contribute to the pain management plan by relating previous pain experiences and successful relief measures. If a play therapist is available they may be able to assist with the preparation and the procedure.

Physical treatments: supportive and distractive techniques

The presence of parents during an invasive procedure on their child is important. In one study almost all children between the ages of 9 and 12 years reported that 'the thing that helped most' was to have a parent present during a painful procedure. Parents need some guidance on how to help their child during the procedure beyond just being present. Studies suggest that talking to and touching the child during the procedure is both soothing and anxiety relieving. Other distractive strategies include:

- Looking at pop-up books or interactive toys.
- Listening through headphones to stories or music.
- Blowing bubbles.
- Video or interactive computer games.
- Moving images projected onto a nearby wall, e.g. fish swimming or birds flying.
- The presence of transitional objects (comforters), e.g. favourite blanket or soft toy.

Pharmacological treatment

Local anaesthetics: topical on intact skin

Ametop gel This contains tetracaine (amethocaine) base 4%.

- It is used under an occlusive dressing.
- Analgesia is achieved after 30–45 minutes.
- Anaesthesia remains for 4–6 hours after removal of the gel.
- Slight erythema, itching and oedema may occur at site.
- Not to be applied on broken skin, mucous membranes, eyes or ears.
- Can cause sensitisation on repeated exposure.
- Not recommended under 1 month of age

EMLA A mixture of lidocaine 2.5% and prilocaine 2.5% can be used in a similar fashion where sensitivity to Ametop occurs. EMLA however takes around 60 minutes to work effectively and tends to cause vasoconstriction rather than vasodilatation.

Local anaesthetics: infiltrated

Lidocaine (lignocaine) 1% lidocaine is used for rapid and intense sensory nerve block.

- The onset of action is significant within 2 minutes and is effective for up to 2 hours.
- It is often used with adrenaline to prolong the duration of sensory blockade and to limit toxicity by reducing absorption – adrenaline concentration 5 micrograms/ml. Adrenaline-containing local anaesthetic should not be used in areas served by an end artery, such as a digit.
- The maximum dose given locally is 3 mg/kg for plain solutions and 7 mg/kg for solutions that contain adrenaline.

Bupivacaine This local anaesthetic is used – at a concentration of 0.25% or 0.5% – when longer lasting local anaesthesia is required, such as in femoral nerve blocks. L-Bupivacaine used in the same dose is associated with less toxicity.

- The onset of anaesthesia is for up to 15 minutes but its effects last up to 8 hours.
- Maximum dosage is 2 mg/kg.

Local anaesthetics are manufactured to a pH of 5 (to improve shelf-life) and are painful for this reason. A buffered solution and the use of smaller needles will lessen the pain associated with infiltration, but local adrenaline cannot then be used because the bicarbonate buffer inactivates it.

Overdose or inadvertent injection of local anaesthetics into an artery or vein may result in cardiac arrhythmias and convulsions. Resuscitative facilities and skills must therefore be available wherever and whenever these drugs are injected.

Non-opioid analgesics

These drugs exhibit varying degrees of analgesic, antipyretic and anti-inflammatory activity.

Paracetamol Paracetamol is probably the most widely used analgesic in paediatric practice. It may be administered by the oral, rectal and intravenous routes. It is thought to work through inhibiting cyclo-oxygenase in the central nervous system but not in other tissues, so that it produces analgesia without any anti-inflammatory effect. It does not cause respiratory depression. It is very safe when administered at the recommended dose although overdosage in a large single dose or too frequent smaller doses may cause hepatotoxicity. Higher loading doses have been shown to improve pain control (see Table F.2).

Non-steroidal anti-inflammatory drugs (NSAIDs) These are anti-inflammatory and antipyretic drugs with moderate analgesic properties. They are less well tolerated than paracetamol, causing gastric irritation, platelet disorders, bronchospasm and renal impairment. They should therefore be avoided in children with a history of gastric ulceration, platelet abnormalities, asthma requiring regular medication and dehydration or renal problems. Their advantage is that they are especially useful for post-traumatic pain because of the additional anti-inflammatory effect. Ibuprofen is given by mouth, and if rectal administration is necessary then diclofenac can be used.

Opiate analgesics

Morphine Administered intravenously, morphine produces a rapid onset of excellent analgesia and remains the treatment of choice in many situations. It may be titrated to effect and reversed if necessary. Side effects include respiratory depression, nausea and vomiting. Cardiovascular effects include peripheral vasodilatation and venous pooling, but in single doses it has minimal haemodynamic effect in a supine patient with normal circulating volume. In hypovolaemic patients it will contribute to hypotension but this is not a contraindication to its use and merely an indication for cardiovascular monitoring and action as appropriate. Opioids produce a dose-dependent depression of ventilation primarily by reducing the sensitivity of brain-stem respiratory centres to hypercarbia and hypoxia. This means that a patient who has received a dose of an opioid requires observation and/or monitoring and should not be discharged home until it is clear that the effects of the opiate are significantly reduced. The nausea and vomiting produced in adults by morphine seems to be less common in children.

The intranasal route for the administration of opiates such as diamorphine and fentanyl has been shown to be a safe and effective route and is becoming increasingly popular for children. It also has the advantage of being quick and easy, avoiding the trauma of an intravenous cannula.

Codeine Oral codeine is often prescribed in combination with paracetamol for the treatment of moderate pain. It is less potent than morphine with less sedative and respiratory depressant effects.

Table F.2 Drugs used for pain relief

Analgesic	Pain severity	Single dose	Duration of effect	Common side effects	Comments
Morphine IV	Moderate to severe	*Over 1 year*: 100–200 mcg/kg *Under 1 year*: 80 mcg/kg	4 h	Respiratory depression Hypotension	Monitor respiration and pulse oximetry Electrocardiogram
Diamorphine intranasal spray	Moderate to severe	*Over 1 year*: intranasal single dose 0.1 mg/kg diluted to a volume to 0.2 ml		As above	As above Dilute with saline
Fentanyl intranasal	Moderate to severe	Initial dose 1.5 mcg/kg intranasal			Use an atomiser device for best effect
Morphine oral	Moderate	*Over 1 year*: 200–500 mcg/kg *Under 1 year*: 80 mcg/kg	4 h		Observe respiration
Codeine	Mild to moderate	Oral 1–1.5 mg/kg	4–6 h		Avoid in patients <1 year Do not give IV
Paracetamol	Mild	*Over 3 months*: single loading doses – 20 mg/kg orally or 40 mg/kg rectally; maintenance dose – 15 mg/kg orally or rectally	4–6 h	Further doses after 6 h	Avoid in liver impairment Total daily dose should not exceed 90 mg/kg
		Under 3 months: 20 mg/kg orally or 30 mg/kg rectally	8-hourly if >36 weeks' gestation		Total daily dose should not exceed 60 mg/kg
Ibuprofen	Mild to moderate	5 mg/kg	4–6 h		Caution where there is a history of hypersensitivity; not recommended for patients <10 kg
Diclofenac	Moderate	300 mcg to 1 mg/kg orally or rectally	8 h		Caution where there is a history of hypersensitivity; not for patients under 6 months of age
Midazolam	Not analgesic	0.5 mg/kg orally		Respiratory depression Hyperexcitability	Monitor SaO_2

Opiate antagonists

Naloxone Naloxone is a potent opioid antagonist. It antagonises the sedative, respiratory depressive and analgesic effects of opioids. It is rapidly metabolised and is given parenterally because of its rapid first-pass extraction through the liver following oral administration. Following IV administration naloxone reverses the effects of opiates virtually immediately. Its duration of action, however, is much shorter than the opiate agonist. Therefore, repeated doses or an infusion may be required if continued opiate antagonism is wanted.

Inhalational analgesia

Entonox Nitrous oxide is a colourless, odourless gas that provides analgesia in subanaesthetic concentrations. It is supplied in premixed cylinders at a 50% concentration with oxygen or at a concentration of up to 70% with oxygen via a blender. Delivery devices either act on a demand principle, i.e. the gas is only delivered when the patient inhales and applies a negative pressure, or via a free flowing circuit. The latter delivery system requires a scavenger circuit. Generally during nitrous oxide therapy the patient has to be awake and cooperative to be able to inhale the gas; this is an obvious safeguard with the technique.

- Because nitrous oxide is inhaled and has a low solubility in blood, its onset of effect is very rapid. It takes 2–3 minutes to achieve its peak effect. For the same reason, the drug wears off over several minutes, enabling patients to recover considerably quicker than if they received narcotics or sedatives. Laryngeal protective reflexes do not always remain intact.
- Nitrous oxide is therefore most suitable for procedures where short-lived intense analgesia is required, e.g. dressing changes, suturing, needle procedures such as venous cannulation, lumbar punctures and for pain relief during splinting or transport. It is also of benefit for immediate pain relief on presentation until definitive analgesia is effective.
- Using a free flow circuit, nitrous oxide can be used by children as young as 2 years of age although children will need to be 4 or 5 years of age before they can trigger the demand valve of a premixed cylinder.
- Nitrous oxide may cause nausea, vomiting, euphoria and disinhibition. Prolonged exposure to high concentrations can cause bone marrow depression and neuronal degeneration.
- Nitrous oxide is contraindicated in children with possible intracranial or intrathoracic air because gas diffusion into the confined space may increase pressure.

Sedative drugs

In addition to analgesics, psychotropic drugs may also be useful when undertaking lengthy or repeated procedures. Sedatives relieve anxiety and not pain and may reduce the child's ability to communicate discomfort and therefore should not be given in isolation. The problems associated with the use of sedatives are those of side effects (usually hyperexcitability) and the time required for the child to be awake enough to be allowed home if admission is not necessary.

Midazolam This is an amnesic and sedative drug. It can be given intravenously, intramuscularly,orally or intranasally (although this is unpleasant). It has an onset time of action of 15 minutes after an oral administration and recovery occurs after about an hour. It may cause respiratory depression, necessitating monitoring of respiratory function and pulse oximetry. A few children become hyperexcitable with this drug. Whilst its action can be reversed by flumazenil, intravenously this is rarely necessary and can precipitate seizures.

Ketamine Ketamine is a potent anaesthetic agent that has an established place in paediatric procedural pain relief in many emergency settings. It causes a dissociative anaesthesia as well as analgesia with maintenance of protective airway reflexes. Side effects include hypersalivation, tachycardia, hypertension and raised intracranial pressure. Laryngospasm is a rare complication that may be precipitated by instrumentation of the upper airway.

Ketamine should be considered as an anaesthetic agent and used with all the precautions generally associated with anaesthesia. Emergence phenomenon can be treated with midazolam if necessary but are much less common in paediatric than adult practice.

F.4 SPECIFIC CLINICAL SITUATIONS

Severe pain

Children in severe pain (e.g. major trauma, femoral fracture, significant burns, displaced or comminuted fractures, etc.) should receive IV morphine at an initial dose of 0.1–0.2 mg/kg infused over 2–3 minutes (Table F.1). A further dose can be given after 5–10 minutes if sufficient analgesia is not achieved. The patient should be monitored using pulse oximetry and electrocardiography.

Head injuries

There is often concern about giving morphine to a patient who has had a head injury and who could therefore potentially lose consciousness secondary to the head injury. If the patient is conscious and in pain then the presence of a potential deteriorating head injury is not a contraindication to giving morphine. First, an analgesic dose is not necessarily a significant sedative; second, if the child's conscious level does deteriorate, then the clinician's first action should be to assess airway, breathing and circulation, intervening where appropriate. If these are stable then a dose of naloxone will quickly ascertain whether the diminished conscious level is secondary to morphine or (as is much more likely) represents increasing intracranial pressure. There are significant benefits for the head-injured patient in receiving adequate pain relief as the physiological response to pain may increase intracranial pressure.

In the common situation of the patient who has an isolated femoral shaft fracture and a possible head injury, a femoral nerve block may be an effective alternative (see Chapter 22).

Emergency venepuncture and venous cannulation

At present the management of this problem is difficult as topical anaesthetics take up to an hour to be effective. Inhaled nitrous oxide given by one of the methods described earlier gives excellent results. Alternatives in an emergency include an ice cube inside the finger of a plastic glove placed over the vein to be cannulated or local anaesthetic infiltration (1% buffered lidocaine) using a very fine gauge (e.g. 29 gauge) needle.

F.5 EXPLANATORY NOTES OF THE ALDER HEY TRIAGE PAIN SCORE

Cry/voice

Score 0	Child is not crying and, although may be quiet, is vocalising appropriately with carer or taking notice of surroundings
Score 1	Child is crying but consolable/distractible or is excessively quiet and responding negatively to carer. On direct questioning says it is painful
Score 2	Child is inconsolable, crying and/or persistently complaining about pain

Facial expression

Score 0	Normal expression and affect
Score 1	Some transient expressions that suggest pain/distress are witnessed but less than 50% of time
Score 2	Persistent facial expressions suggesting pain/distress more than 50% of time

Grimace: open mouth, lips pulled back at corners, furrowed forehead and/or between eyebrows, eyes closed, wrinkled at corners.

Posture

This relates to the child's behaviour to the affected body area.

Score 0	Normal
Score 1	Exhibiting increased awareness of affected area, e.g. by touching, rubbing, pointing, sparing or limping

Score 2 Affected area is held tense and defended so that touching it is deterred; non-weight-bearing

Movement

This relates to how the child moves the whole body.

Score 0 Normal

Score 1 Movement is reduced or child is noted to be restless/uncomfortable

Score 2 Movement is abnormal, either very still/rigid or writhing in agony/shaking

Colour

Score 0 Normal

Score 1 Pale

Score 2 Very pale 'green', the colour that can sometimes be seen with nausea or fainting – extreme pallor

APPENDIX G

Triage

G.1 INTRODUCTION

Nurse triage is the process whereby each child presenting with potentially serious illness or injury is assigned a clinical priority. It is an essential clinical risk management step and is part of the process of recognition of the seriously ill or injured child that has been discussed earlier.

In the United Kingdom, Canada and Australia, five-part national triage scales have been agreed. The UK scale is shown in Table G.1. While the names of the triage categories and the target times assigned to each name vary from country to country, the underlying concept does not.

Table G.1 The UK triage scale

Number	Name	Colour	Maximum time (min)
1	Immediate	Red	0
2	Very urgent	Orange	10
3	Urgent	Yellow	60
4	Standard	Green	120
5	Non-urgent	Blue	240

G.2 TRIAGE DECISION MAKING

There are many models of decision making, each requiring three basic steps. These are: identification of a problem, determination of the alternatives and selection of the most appropriate alternative. The commonest triage method in the UK is that developed by the Manchester Triage Group. This method uses the following five steps:

1 Identifying the problem
2 Gathering and analysing information related to the solution
3 Evaluating all the alternatives and selecting one for implementation
4 Implementation of the selected alternative
5 Monitoring the implementation and evaluation of outcomes

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Identify the problem

This is done by taking a brief and focused history from the child, his or her parents and/or any pre-hospital care personnel. This phase is always necessary whatever the method used.

Gather and analyse information related to the solution

Once the presentation has been identified, discriminators can be sought at each level. Discriminators, as their name implies, are factors that discriminate between patients such that they allow them to be allocated to one of the five clinical priorities. They can be *general* or *specific*. The former apply to all patients irrespective of their presentation, whilst the latter tend to relate to key features of particular conditions. Thus severe pain is a general discriminator, but *cardiac pain* and *pleuritic pain* are specific discriminators. General discriminators would include life threat, pain, haemorrhage, conscious level and temperature.

Life threat

To an advanced paediatric life support (APLS) provider life threat is perhaps the most obvious general discriminator of all. Any cessation or threat to the vital (ABC) functions means that the patient is in the immediate group. Thus the presence of an insecure airway, inspiratory or expiratory stridor, absent or inadequate breathing, or shock are all significant.

Pain

From the child's and parent's perspectives pain is a major factor in determining priority. Pain assessment and management is dealt with elsewhere in this book and is not reiterated here. Children with severe pain should be allocated to the very urgent category while those with moderate pain should be allocated to the urgent category. Any child with any lesser degree of pain should be allocated to the standard category.

Haemorrhage

Haemorrhage is a feature of many presentations particularly those following trauma. If haemorrhage is exsanguinating, death will ensue rapidly unless bleeding is stopped. These children must be treated immediately. A haemorrhage that is not rapidly controlled by the application of sustained direct pressure, and which continues to bleed heavily or soak through large dressings quickly, should be treated very urgently.

Conscious level

All unresponsive children must be an immediate priority, and those who respond to voice or pain only are categorised as very urgent. Children with a history of unconsciousness should be allocated to the urgent category.

Temperature

Temperature is used as a general discriminator. It may be difficult to obtain an accurate measurement during the triage process; however modern rapid-reading tympanic membrane thermometers should make this aim attainable. A hot child (over 38.5°C) is always seen urgently; children who are cold (less than 32°C) are seen very urgently, as are hot infants.

Evaluate all alternatives and select one for implementation

Clinicians collect a huge amount of information about the children they deal with. The data are compared to internal frameworks that act as guides for assessment. The presentational flow diagrams developed by the Manchester Triage Group provide the organisational framework to order the thought process during triage.

Implement the selected alternative

As previously noted there are only five possible triage categories to select from and these have specific names and definitions (Table G.1). The urgency of the patient's condition determines their clinical priority. Once the priority is allocated the appropriate pathway of care begins.

Monitor the implementation and evaluate outcomes

Triage categories may change as the child deteriorates or gets better. It is important, therefore, that the process of triage (clinical prioritisation) is dynamic rather than static. To achieve this end all clinicians involved in the pathway of care should rapidly assess priority whenever they encounter the child. Furthermore any changes in priority must be noted and the appropriate actions taken.

G.3 SECONDARY TRIAGE

It may not be possible to carry out all the assessments necessary at the initial triage encounter – this is particularly so if the workload of the department is high. In such circumstances the necessary assessments should still be carried out, but as secondary procedures by a receiving nurse. The actual initial clinical priority cannot be set until the process is finished. More time-consuming assessments (such as blood glucose estimation and peak flow measurement) are often left to the secondary stage.

APPENDIX H

General approach to poisoning and envenomation

H.1 POISONING: INTRODUCTION

Suspected poisoning in children results in about 40,000 attendances at emergency departments each year in England and Wales. Around half of these children are admitted to hospital for treatment or observation. Precise data on hospital admissions for poisonings are affected by the fact that many emergency departments and paediatric wards have special areas where children who have taken a substance of low toxicity can be observed for a few hours without being formally admitted.

Deaths from ingested poisons are uncommon, and are due to drugs (especially tricyclic antidepressants), household products and, rarely, plants. As can be seen from Table H.1, more children die each year from inhalation of carbon monoxide and other gases in household fires than from accidental poisoning by drugs.

Incidence

There has been a steady decline in the number of childhood deaths from poisonings. The selective introduction of child-resistant containers (CRCs) in 1976, together with other measures, has reduced the number of poisonings and hospital attendances. In the case of salicylate poisoning the introduction of CRCs saw an 85% fall in hospital admissions from 1975 to 1978. It should be remembered, however, that 20% of children under the age of 5 years are capable of opening CRCs!

Table H.1 Deaths in children (ages 1–14 years) from poisons in England and Wales

Cause of death	1988	1998	2001	2008
From poisoning by drugs, medicaments and biological substances	16	18	2	11
From toxic effects of carbon monoxide	36	15	21	9
From toxic effect of other gases, fumes or vapours, including unspecified factors	56	24	7	12

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The decrease in deaths from the inhalation of toxic fumes may be related to the gradual effect of legislation in the UK on the banning of toxic substances in furnishing items. The continued substantial death rate from carbon monoxide poisoning is disappointing but may be related to the fact that although smoke alarms are more readily found in dwellings, they are often non-functional. The decline in mortality from drug poisoning may be due both to more effective treatment and possibly to the more widespread use of less toxic antidepressant drugs.

Accidental poisoning

This is usually a problem of the young child or toddler, with a mean age of presentation of 2.5 years. Accidental poisoning usually occurs when the child is unsupervised, and there is an increased incidence in poisoning following recent disruption in households, such as a new baby, moving house or where there is maternal depression.

Intentional overdose

Suicide or parasuicide attempts are usually made by young people in their teens; however, sometimes they may be as young as 8 or 9 years. These children or adolescents should undergo psychiatric and social assessment.

Drug abuse

Alcohol and solvent abuse are the commonest forms of drug abuse in children in the UK.

Iatrogenic drugs

The commonest offender is diphenoxylate with atropine (Lomotil). This combination is toxic to some children at therapeutic doses. The most frequently fatal drug is digoxin.

Deliberate poisoning

Rarely, symptoms are induced in children by adults via the administration of drugs. A history of poisoning will often not be given at presentation.

Most poisoning episodes in childhood and adolescence are of low lethality and little or no treatment is required. This appendix will not address the milder cases but will enable the student to develop an approach to the seriously ill poisoned child, with additional advice on the management of specific poisons.

H.2 PRIMARY ASSESSMENT IN POISONING

Airway

Assess airway patency by the 'look, listen and feel' method.

If the child can speak or cry in response to a stimulus, this indicates that the airway is patent, that breathing is occurring and that there is adequate circulation. If the child responds only with withdrawal to a painful stimulus or is unresponsive (AVPU score 'P' or 'U') his airway is at risk.

If there is no evidence of air movement then chin lift or jaw thrust manoeuvres should be carried out and the airway reassessed. If there continues to be no evidence of air movement then airway patency can be assessed by performing an opening manoeuvre and giving rescue breaths (see Chapter 4).

Breathing

Assess the adequacy of breathing.

- Effort of breathing:
  - Recession.
  - Respiratory rate: the rate may be increased in poisoning from amphetamines, ecstasy, salicylates, ethylene glycol and methanol.
- Efficacy of breathing:

- Breath sounds.
- Chest expansion/abdominal excursion.
- Monitor oxygen saturation with a pulse oximeter.

Circulation

Assess the adequacy of circulation.

- Cardiovascular status:
  - Heart rate: tachycardia is caused by amphetamines, ecstasy, β-agonists, phenothiazines, theophylline and tricyclic antidepressants (TCAs); bradycardia is caused by β-blockers, digoxin and organophosphates.
  - Pulse volume.
  - Capillary refill.
  - Blood pressure: hypotension is commonly seen in serious poisoning; hypertension is caused by ecstasy and monoamine oxidase inhibitors.
- Effects of circulatory inadequacy on other organs:
  - Acidotic sighing respirations: this may suggest metabolic acidosis from salicylates or ethylene glycol poisoning as a cause for the coma.
  - Pale, cyanosed or cold skin.

Monitor the heart rate/rhythm, blood pressure and core–toe temperature difference.

If heart rate is above 200 beats/min in an infant or above 150 in a child, or if the rhythm is abnormal, perform a standard electrocardiogram (ECG). QRS prolongation and ventricular tachycardia is seen in TCA poisoning.

Disability

Assess neurological function.

- A rapid measure of level of consciousness should be recorded using the AVPU scale. Depression of conscious level suggests poisoning with opiates, sedatives (such as benzodiazepines) antihistamines and hypoglycaemic agents.
- Pupillary size and reaction should be noted. Very small pupils suggest opiate or organophosphate poisoning; large pupils amphetamines, atropine and TCAs.
- Note the child's posture. Hypertonia is seen in amphetamine, ecstasy, theophylline and TCA poisoning.
- The presence of convulsive movements should be sought. Convulsions are associated with any drug that causes hypoglycaemia (ethanol) and with TCA poisoning.

Exposure

Take the child's core and toe temperatures. A fever suggests poisoning with ecstasy, cocaine or salicylates. Hypothermia suggests poisoning with barbiturates or ethanol.

H.3 RESUSCITATION IN POISONING

Airway

- A patent airway is the first requisite. If the airway is not patent it should be opened and maintained with an airway manoeuvre and the child ventilated by bag–valve–mask oxygenation. An airway adjunct can be used. The airway should then be secured by intubation by experienced senior help.
- If the child has an AVPU score of 'P' or 'U', his airway is at risk. It should be maintained by an airway manoeuvre or adjunct and senior help requested to secure it.

Breathing

- All children with respiratory abnormalities, shock or a decreased conscious level should receive high-flow oxygen through a face mask with a reservoir as soon as the airway has been demonstrated to be adequate.

- A number of agents taken in overdose (particularly narcotics) can produce respiratory depression. Oxygen should be given, but it is important to remember that these patients may have an increasing carbon dioxide level despite a normal oxygen saturation whilst breathing oxygen. Inadequate breathing should be supported using a bag–valve–mask device with oxygen or by intermittent positive pressure ventilation in the intubated patient.

Circulation

- A number of poisons can produce shock, by a number of different mechanisms. Hypovolaemia may be caused by gastrointestinal bleeding from iron poisoning or there may be vasodilatation from barbiturates. Shock should be treated with a fluid bolus, as usual. If possible, inotropes should be avoided in poisoning cases as the combination of toxic substance producing shock and an inotrope may be proarrhythmogenic.
- Cardiac dysrhythmias can be expected in TCA, digoxin, quinine and antiarrhythmic drug poisoning. Some antiarrhythmic treatments are contraindicated with certain poisons. See below for advice on TCA poisoning and contact a poisons centre urgently for other advice.

Gain intravenous or intraosseous access

- Take blood for a full blood count, urea and electrolytes, toxicology, paracetamol and salicylate levels (in patients who have taken an unknown drug), glucose stick test and laboratory test. Give 2 ml/kg of 10% glucose followed by maintenance glucose infusion to any hypoglycaemic patient.
- Give a 20 ml/kg rapid bolus of crystalloid to any patient with signs of shock.
- If a child has a tachyarrhythmia and is shocked, up to three synchronous electrical shocks at 1, 2 and 2 J should be given. If the arrhythmia is broad complex and the synchronous shocks are not activated by the defibrillator then attempt an asynchronous shock. A conscious child should be anaesthetised first if this can be done in a timely manner. DC shock may be dangerous in digoxin poisoning. Antiarrhythmics may be used on advice from a Poisons Centre.

Disability

- Treat convulsions with either diazepam, midazolam or lorazepam.
- Give a trial of naloxone in cases where depressed conscious level and small pupils suggest opiate poisoning.

In all cases of serious poisoning early consultation with a poisons centre is mandatory. Such centres have a wealth of expertise in the management of poisoning and will advise on the individual patient's needs.

Monitoring

- ECG.
- Blood pressure (use appropriate size of cuff).
- Pulse oximetry.
- Core temperature.
- Blood glucose.
- Urea and electrolytes.
- Blood gases (where indicated).

Lethality assessment

At the end of the primary assessment, it is important to assess the potential lethality of the overdose. This requires knowledge of the substance that has been taken, the time it was taken and the dosage. This information may be unattainable in the unwitnessed poisoning episode of a toddler or that of an unconscious or uncooperative adolescent. Some clues about the drug ingested may be available from physical signs noted during the primary assessment (Table H.2).

Table H.2 Diagnostic clues from the primary assessment

Signs	Drug
Tachypnoea	Aspirin, theophylline, carbon monoxide, cyanide
Bradypnoea	Ethanol, opiates, barbiturates, sedatives
Metabolic acidosis (sighing respirations)	Ethanol, carbon monoxide, ethylene glycol
Tachycardia	Antidepressants, sympathomimetics, amphetamines, cocaine
Bradycardia	Beta-blockers, digoxin, clonidine
Hypotension	Barbiturates, benzodiazepines, β-blockers, calcium channel blockers, opiates, iron, phenothiazines, phenytoin, tricyclic antidepressants
Hypertension	Amphetamines, cocaine, sympathomimetic agents
Small pupils	Opiates, organophosphate insecticides, phenothiazines
Large pupils	Amphetamines, atropine, cannabis, carbamazepine, cocaine, quinine, tricyclic antidepressants
Convulsions	Carbamazepine, lindane, organophosphate insecticides, phenothiazines, tricyclic antidepressants
Hypothermia	Barbiturates, ethanol, phenothiazines
Hyperthermia	Amphetamines, cocaine, ecstasy, phenothiazines, salicylates

Some investigation results can add clues to the diagnosis of an unknown poison.

- Metabolic acidosis can be found in poisoning from:
  - carbon monoxide,
  - ecstasy,
  - ethylene glycol,
  - iron,
  - methanol,
  - salicylates, and
  - tricyclic antidepressants.
- An enlarged anion gap $[(Na + K) - (HCO_3 - Cl)]$ of >18 can be found in poisoning from:
  - ethanol,
  - ethylene glycol,
  - iron,
  - methanol, and
  - salicylates.
- Hypokalaemia can be found in poisoning from:
  - β-agonists, and
  - theophylline.
- Hyperkalaemia can be found in poisoning from:
  - digoxin.

The risks of a particular overdose can be assessed once all the information has been gathered. Complex or life-threatening cases should be discussed with a poisons centre. The poisons centre will require the following information:

- Age and weight of the patient.
- The time since exposure.

- The substance.
- The amount taken together with any description or labeling.
- The patient's condition.

If the nature of the overdose is unknown then a high potential lethality should be assumed. Many childhood poisoning incidents have zero lethality and no treatment is required.

H.4 EMERGENCY TREATMENT IN POISONING

Drug elimination

Many children have taken a trivial overdose or an overdose of a non-poisonous substance. If the overdose episode is assessed as having a low lethality, then no treatment is required.

If the drug overdose is assessed as having a potentially high lethality or its exact nature is unknown, then measures to minimise blood concentrations of the drug should be undertaken. In general this means stopping further absorption. Occasionally measures to increase excretion can be employed and in some circumstances specific antidotes may be available. Seek advice from a poisons centre.

Activated charcoal

Activated charcoal has a surface area of 1000 m^2/g and is capable of binding a number of poisonous substances without being systemically absorbed. It is now widely used in cases of poisoning. However, there are some substances that it will not absorb. These include alcohol and iron. Repeated doses of activated charcoal are useful in some types of poisoning because they promote drug reabsorption from the circulation back into the bowel and interrupt enterohepatic cycling. These types include aspirin, barbiturates and theophylline.

It is often difficult to give charcoal to children as it is unpalatable. Flavouring may be necessary but can diminish the charcoal's activity. The charcoal can be given via a nasogastric or lavage tube after a gastric washout. The dose is at least 10 times the estimated dose of poison ingested. Children should usually be given 25–50 g.

Aspirated charcoal causes severe lung damage, so airway protection is especially important in the child who is not fully conscious.

Emesis

Emesis caused by ipecacuanha is now rarely used although for many years it was routinely given for the management of poisoning incidents in children. The dose schedule is 15 ml with water (10 ml in children of 6 months to 2 years), repeated once after 20 minutes if necessary. It must not be used in the child with a depressed conscious level. Evidence now suggests that unless emesis occurs within 1 hour of ingestion of the poison, little of the poison will be eliminated. Only about 30% is retrieved even within the hour.

Emesis should only be used for those poisons requiring removal that are not bound by charcoal, or in children who are at risk from developing symptoms from the poison they have taken, who present within 1 hour of ingestion and who will not take the charcoal.

Gastric lavage

Gastric lavage is rarely required as benefit rarely outweighs risk; advice should be sought from the National Poisons Information Service if a significant quantity of iron or lithium has been ingested within the previous hour.

There are a number of active elimination techniques such as haemoperfusion and plasmapheresis: their use is infrequent and should be guided by the advice of the poisons centre.

H.5 EMERGENCY TREATMENT OF SPECIFIC POISONS

Iron

The child with iron poisoning presents with shock, which may be due to gut haemorrhage. If over 20 mg/kg of elemental iron has been taken, toxicity is likely. Over 150 mg/kg may be fatal. Intubation, ventilation and circulatory support are necessary in the severely affected child.

Initial symptoms of toxicity are vomiting, diarrhoea and abdominal pain. These may lead on to drowsiness, fits and circulatory collapse.

Gastric lavage should be performed once the airway is secured and circulatory access has been gained. Charcoal is not helpful. Desferrioxamine can be left in the stomach, but the main treatment is to infuse desferrioxamine at a dose of 15 mg/kg/h. This treatment should be given immediately to children with serious symptoms such as shock, coma or fits and to all with a serum iron level (4 hours or more after ingestion) of 3 mg/l and gastrointestinal symptoms, leucocytosis or hyperglycaemia.

Radiography of the abdomen can help to show how much iron remains within. Whole-bowel irrigation with polyethylene glycol–electrolyte solutions may have a place in severe cases.

Tricyclic antidepressant poisoning

The toxic effects of these agents result from their inhibition of fast sodium channels in the brain and the myocardium, which action is known as 'quinidine-like'. With serious intoxication, the cardiac problems are due to intraventricular conduction delay. This results in QRS prolongation (a QRS of more than four little squares on the ECG paper is predictive of serious effects).

TCA poisoning causes anticholinergic effects (tachycardia, dilated pupils, convulsions) and cardiac effects (conduction delay, any arrhythmia). Convulsions should be treated as described in Chapter 12.

In addition, alkalinisation up to an arterial pH of at least 7.45, and preferably 7.5, has been shown to reduce the toxic effects on the heart. This can be achieved by hyperventilation (PCO_2 no lower than 3.33 kPa (25 mmHg)) and by infusing sodium bicarbonate (1–2 mmol/kg). Hypotension should be treated with volume expansion, and if an inotrope is necessary, noradrenaline is preferable to dopamine, dobutamine and adrenaline. Glucagon has an inotropic effect and can be used in this circumstance.

The use of antiarrhythmics should be guided from a poisons centre. Lidocaine and phenytoin may be helpful. Quinidine, procainamide and disopyramide are contraindicated.

Opiates (including methadone)

Following stabilisation of airway, breathing and circulation, the specific antidote is naloxone. An initial bolus dose of 10 micrograms/kg should be given. Naloxone has a short half-life, relapse often occurring after 20 minutes. Larger boluses, or an infusion of 5–20 micrograms/kg/h, may be required.

It is important to normalise CO_2 before the naloxone is given because adverse events such as ventricular arrhythmias, acute pulmonary oedema, asystole or seizures may otherwise occur. This is because the opioid system and the adrenergic system are interrelated. Opioid antagonists and hypercapnia stimulate sympathetic nervous system activity. Therefore if ventilation is not provided to normalise carbon dioxide prior to naloxone administration, the sudden rise in adrenaline concentration can cause arrhythmias.

Paracetamol

Significant paracetamol poisoning in childhood is almost always intentional; the accidental ingestion of paediatric paracetamol elixir preparations by the toddler very rarely achieves toxicity. Doses of less than 150 mg/kg will not cause toxicity except in a child with hepatic or renal disease. Current treatment of paracetamol poisoning includes oral charcoal and a paracetamol blood level to be taken at 4 hours or later. Figure H.1 shows a nomogram indicating the level of blood paracetamol at which acetylcysteine should be given intravenously. A total dose of 300 mg/kg is given over approximately 24 hours. Contact a poisons centre for individual details.

Salicylates

Aspirin slows stomach emptying, so gastric lavage can be undertaken up to 4 hours after ingestion. Repeated charcoal doses should be given for patients who have ingested sustained-release preparations. The salicylate level can be measured initially at 2 hours. However, repeated measurements are necessary and no reliance should be placed on a single salicylate level. The

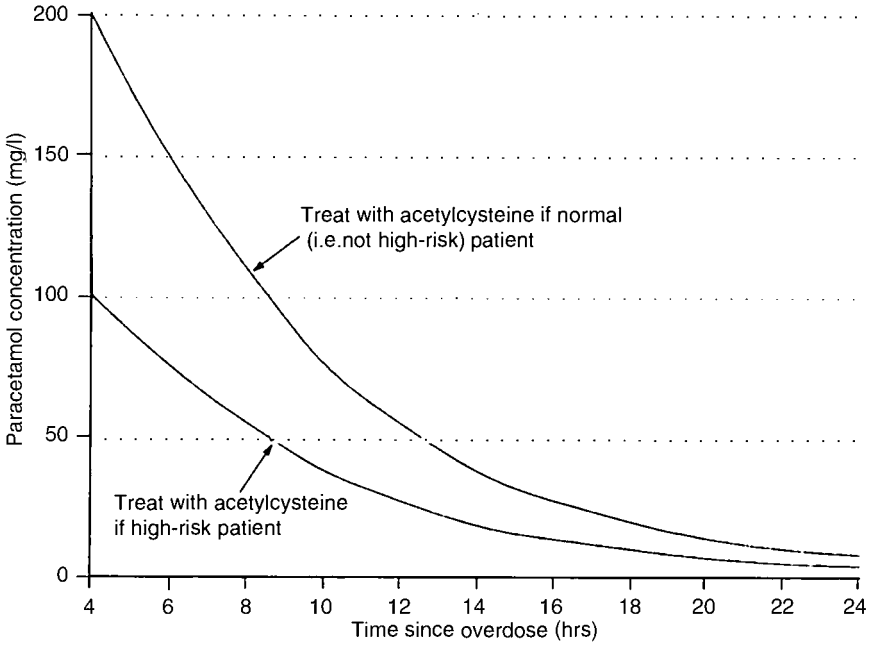

Figure H.1 Nomogram showing level of blood paracetamol; high risk = underweight or malnourished children, recent fasting or enzyme-inducing drugs

levels will usually rise significantly over the first 6 hours (longer if an enteric-coated preparation is used). Salicylate poisoning causes a respiratory alkalosis and metabolic acidosis. Arterial blood gas estimation is necessary for managing the patient. Alkalinisation of the patient improves the excretion of salicylate: 1 mmol/kg of sodium bicarbonate should be infused over 4 hours. Forced diuresis is no longer used.

Ethylene glycol

This sweet-tasting substance is available as an antifreeze and de-icer fluid for vehicles. It produces a clinical appearance of inebriation accompanied by metabolic acidosis and causes widespread cellular damage, especially to the kidneys. In unwitnessed ingestions the clue is in the metabolic acidosis with an inexplicable anion gap. Activated charcoal is ineffective. Ethanol is a competitive inhibitor of alcohol dehydrogenase and can block metabolism of the ethylene glycol to its poisonous metabolic byproducts. An oral loading dose of 2.5 ml/kg of 40% ethanol (the strength of most spirits) should be started. The aim is to have a blood ethanol concentration of 100 mg/dl. Fomepizole may also be used and haemodialysis may be necessary. Cofactors, thiamine and pyridoxine are also recommended.

Cocaine

Cocaine poisoning leads to the local accumulation of the neurotransmitters noradrenaline, dopamine, adrenaline and serotonin. Accumulation of noradrenaline and adrenaline leads to tachycardia, which increases myocardial oxygen demand while reducing the time for diastolic coronary perfusion. Vasoconstriction causing hypertension results from the accumulation of neurotransmitters at the peripheral β-adrenergic receptors, and peripheral hydroxytryptamine (5-HT) receptor stimulation causes coronary artery vasospasm. In addition, cocaine stimulates platelet aggregation. Together, these changes can produce what is effectively a coronary event in a child or an adolescent.

Acute coronary syndrome producing chest pain and varying types of cardiac rhythm disturbances is the most frequent complication of cocaine use, which leads to hospitalisation. Cocaine is also a sodium channel inhibitor, similar to a type I antiarrhythmic agent, and so can prolong the QRS duration and impair myocardial contractility. Through the combination of adrenergic

and sodium channel effects, cocaine use may cause various tachyarrhythmias including ventricular tachycardia and ventricular fibrillation. Treatment should be guided by a poisons centre.

Initial treatment of acute coronary syndrome consists of oxygen administration, continuous ECG monitoring, administration of a benzodiazepine (e.g. diazepam or lorazepam), aspirin and heparin. Hyperthermia should be treated with cooling. β-adrenergic blockers are contraindicated in the setting of cocaine intoxication.

Ventricular tachycardia should be treated with DC shock as antiarrhythmic drugs may cause further proarrhythmic effects.

Since cocaine is a sodium channel blocker, administration of sodium bicarbonate in a dose of 0.5–1 mmol/kg should be considered in the treatment of ventricular arrhythmias.

Ecstasy

Most ecstasy tablets contain 30–150 mg of 3,4-methylenedioxymethamphetamine (MDMA). This drug, which has a half-life of around 8 hours, most probably stimulates both the peripheral and the central α- and β-adrenergic receptors. Early deaths are usually due to cardiac dysrhythmias, while deaths after 24 hours occur from a neuroleptic malignant-like syndrome.

Mild adverse effects occur at low doses and include increased muscle tone, agitation, anxiety and tachycardia. Mild elevation of temperature may also occur. At higher doses, hypertonia with hyperreflexia, tachycardia, tachypnoea and visual disturbance can be seen. In the worst affected children, coma, convulsions and cardiac dysrhythmias can occur. Hyperpyrexia with increased muscle tone can lead to rhabdomyolysis, metabolic acidosis with acute renal failure and disseminated intravascular coagulation.

Activated charcoal should be given to conscious patients. Blood pressure and temperature must be monitored. Diazepam can be used to control anxiety – major tranquillisers should not be used because they exacerbate symptoms. If core temperature exceeds 39°C then active cooling should be commenced and the use of dantrolene sodium (2–3 mg/kg over 10–15 minutes) should be considered. Some children may require ventilation.

H.6 ENVENOMATION: INTRODUCTION

Envenomation may occur as a result of bites or stings from a wide variety of animals including snakes, bees, scorpions, spiders, jellyfish and fish. In many cases envenomation is unavoidable, but appropriate behaviour and clothing will limit exposure to envenomation.

Symptoms of envenomation may be the direct result of the venom, or allergic reactions to those toxins (e.g. to bee stings). In all cases the principles of management consist of:

- Standard resuscitation practice of managing airway, breathing and circulation.
- Limiting the uptake of venom into the circulation where possible.
- The administration of antitoxin where available and appropriate.
- Supportive care to the systems involved by the toxin.
- The management of pain.
- Treatment of sites of local injury.

Diagnosis of envenomation is often difficult, but essential to appropriate management (see Figures H.2 and H.3). The symptoms of stings are usually rapid in onset, and the diagnosis is usually more straightforward. Envenomation does not occur with all bites, even from venomous species, and it may be difficult to decide whether or not envenomation has occurred immediately after a bite. In this context it is appropriate to apply measures to reduce systemic uptake of venom from the site of the bite, and to delay the administration of antitoxin until a more definitive diagnosis of envenomation has been made. Occasionally, envenomation is part of the differential diagnosis of symptoms experienced by a patient who either was not aware of a sting or bite, or is too young to provide an appropriate history. Diagnosis of envenomation may be made by the development of specific symptoms, or by the use of diagnostic kits that identify the presence of specific venoms. Unfortunately, diagnostic systems are not generally available in many areas where stings or bites are common.

Although accurate identification of the cause of the envenomation may assist with specific therapy, attempts to capture the source of the bite or sting are more likely to result in extra victims than useful information.

H.7 RESUSCITATION AND SUPPORT IN ENVENOMATION

Airway

The airway may be threatened for a number of reasons, including depressed level of consciousness, bulbar palsy, paralysis and swelling of tissues around the airway. The airway must be assessed frequently. Clearance of secretions from the pharynx is the most common problem. Patients who require intubation for reasons other than a depressed level of consciousness require anaesthesia for intubation. It is extremely important to note that a totally paralysed patient may be fully conscious.

Breathing

Many venoms cause paralysis, and patients affected by these venoms require ventilatory support. The support must be provided prior to respiratory arrest. As patients may be paralysed but fully awake, anaesthesia for intubation is recommended. Severe muscle spasm or seizures may occur following some envenomation, and these patients will require ventilatory support. Also, secretions may contribute to respiratory embarrassment and ventilatory support may prevent the accumulation of secretions.

Circulation

Shock may occur for a variety of reasons including massive leakage of fluid into tissues damaged by cytotoxic venoms, cardiac arrhythmia and bleeding secondary to coagulopathy.

Adequate vascular access must be secured with fluid resuscitation appropriate to the clinical situation.

Some venoms are associated with the development of renal and/or electrolyte problems, and fluid and electrolyte therapy must be adapted to the specific venom.

Disability

Assess the patient's conscious level, remembering that failure to respond may be a consequence of paralysis and not of the level of consciousness. Look specifically to local neurological problems such as ophthalmoplegia and/or bulbar palsy.

Exposure

Full exposure may be required to identify the site of a bite, and in the case of stings it is also important to examine areas of the body covered by hair.

H.8 SPECIFIC ENVENOMATION ISSUES

Limiting the uptake of venom

Where possible, the rate of uptake of venom or toxin into the circulation should be limited. If the bite or sting has affected a limb, it may be possible to slow the rate of absorption of toxin from the bite or sting by the application of a crepe bandage to the limb, together with immobilisation of the limb. Neither the application of a pressure bandage nor immobilisation alone will significantly slow uptake of toxin into the circulation. Both measures should be applied. The bandage should be firmly applied (similar pressure to that required to strap a strained ankle), but should not interfere with the circulation to the limb. Splints should be applied to immobilise the limb. The pressure bandage should not be removed before the child is in a facility that can provide supportive care, and antivenin should be administered prior to the release of the bandage if envenomation has occurred.

There are few data about the efficacy of limb bandaging in the case of cytotoxic venoms. Theoretically, localisation of the venom may increase the local damage (this is controversial in

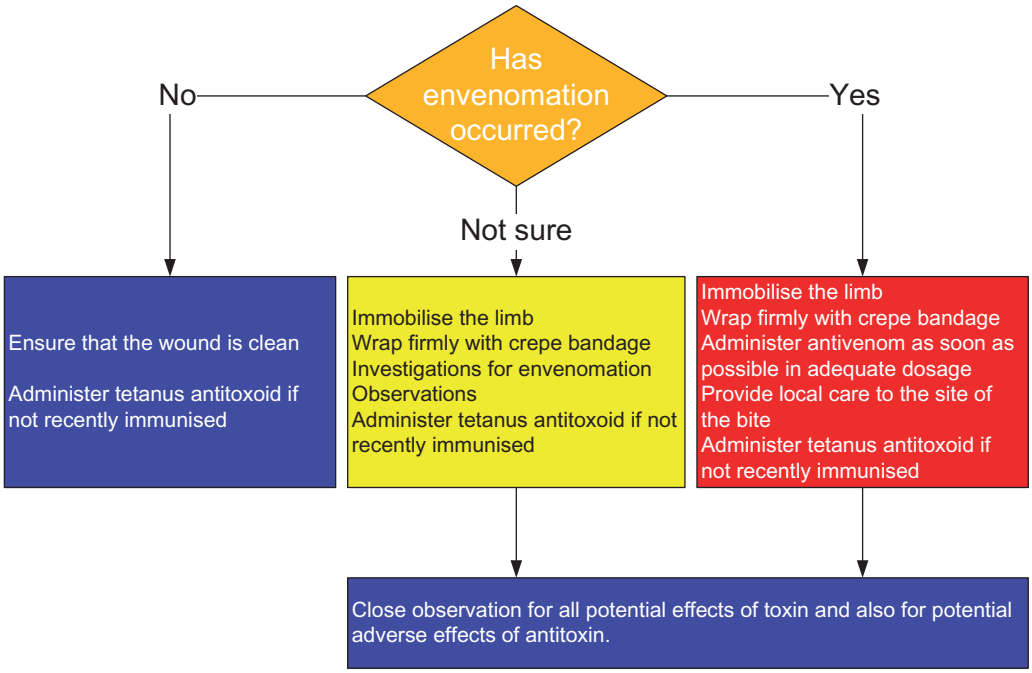

Figure H.2 Management of a potentially venomous bite

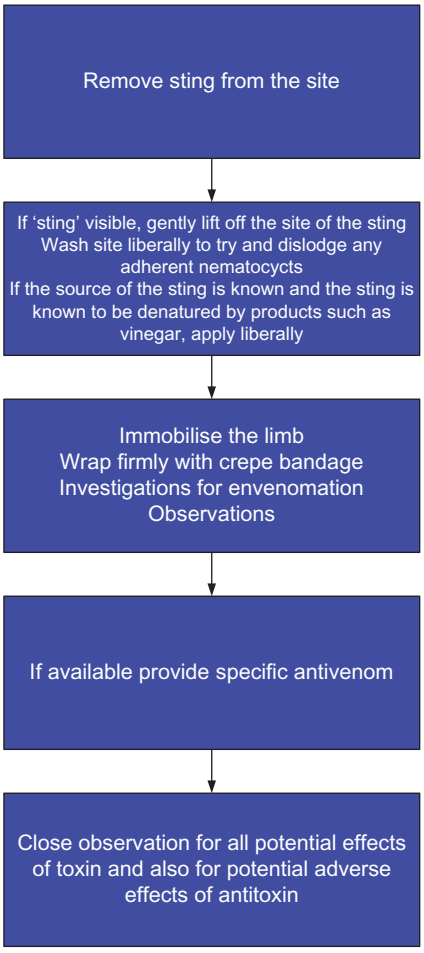

Figure H.3 Management of a potentially venomous sting

various articles). However, in children, uptake of predominantly cytotoxic venom into the circulation will cause systemic effects, and it is probably safer to immobilise the limb and apply a pressure bandage.

In the case of jellyfish stings there is a concern that application of pressure to the site of the sting may increase the release of toxin from stings on the skin. In this situation liberal washing of the site with vinegar and then gentle application of a vinegar-soaked bandage is appropriate.

Some marine toxins are heat sensitive, and application of warmth to the site of the injury may reduce symptoms considerably. Great care must be taken to avoid burning the patient.

If there is a specific antivenin or antiserum available, this should be administered as soon as possible. If there is doubt as to whether envenomation actually occurred, then there may be a place for observation and some delay in administration. Symptoms are usually evident within 1 hour of snakebite envenomation. In reality, antivenins are not usually available at the site of the incident, and by the time the victim has actually reached a medical facility it is usually clear whether envenomation has occurred. The dose of antiserum should not be based on the patient size, but on the amount required to neutralise the toxin; thus in general children will receive the full adult dose.

Although allergic reactions to antivenin are relatively common, they may be prevented or ameliorated by premedication with *subcutaneous* adrenaline 5–10 micrograms/kg. Additional protective agents such as a steroid (hydrocortisone) and an antihistamine may be indicated if the patient has a known allergic history.

Treatment of sites of local injury

If venom is sprayed into the eyes, it is important to wash out any venom as soon as possible, using clean fluids. There is no evidence that suction, electrotherapy or local incision provides any benefit to the patient. There is likewise no evidence that administration of prophylactic antibiotics is useful. Patients who suffer a snake bite and have not been immunised against tetanus within the last 5 years should receive antitetanus toxoid.

Envenomation by cytotoxic venoms may cause severe local swelling and pain. Limbs must be carefully observed for the development of compartment syndromes and if they develop early, fasciotomies must be performed. In these patients careful assessment of coagulation status with appropriate support is essential as severe bleeding may occur following fasciotomy.

Local and systemic infections may occur following bites. Although there is no place for prophylactic antibiotics, any necrotic tissue should be debrided, and infection treated vigorously with antibiotics based on bacterial cultures. Initial therapy prior to culture results should cover both Gram-negative and Gram-positive organisms.

Drugs

In addition to antivenin (where available) there are a number of problems that may require symptomatic therapy.

Analgesia

Pain may be an extremely important feature of envenomation, and adequate analgesia is critical. Severe pain is best treated with intravenous opiates titrated to effect. Local therapy with agents that neutralise toxins may be possible, e.g. application of vinegar in the case of jellyfish stings or application of warm water in some stonefish stings. In cases of severe limb-related pain, regional anaesthesia may be helpful, but must be used with care in view of potential bleeding problems.

Sedation

Bites or stings may be associated with extreme anxiety. Reassurance and supportive care is the basis of therapy, but sedation and anxiolysis may be helpful, particularly if patients require transportation.

Coagulation support

Antivenins do not correct coagulation defects, and coagulation problems must be treated using supportive administration of clotting factors.

Monitoring

If envenomation has occurred a wide range of side effects are possible depending on the particular source of envenomation, the dose of venom administered, the time to adequate therapy and the dose of antivenom administered. It is essential to monitor patients carefully for all the potential side effects of the supposed envenomation.

APPENDIX I

Resuscitation of the baby at birth

I.1 INTRODUCTION

The resuscitation of babies at birth is different from the resuscitation of all other age groups, and knowledge of the relevant physiology and pathophysiology is essential. However, the majority of babies will establish normal respiration and circulation without help. Ideally, someone trained in newborn resuscitation should be present at all deliveries. It is advisable that all those who attend deliveries should have been on courses such as the Newborn Life Support Course, organised by the Resuscitation Council (UK), European Resuscitation Council courses, or the Neonatal Resuscitation Programme, organised by the American Academy of Pediatrics. However, some babies are born in unexpected places such as A&E departments. For these situations it is important that clinicians have an understanding of the differences in resuscitating a baby at birth.

I.2 NORMAL PHYSIOLOGY

At birth the baby must change, often within a matter of moments, from an organism with fluid-filled lungs whose respiratory function is carried out by the placenta to a separate being whose air-filled lungs can successfully take over this function. Preparation for this begins during labour, when the fluid-producing cells within the lung cease secretion and begin reabsorption of that fluid. Delivery by caesarean section before the onset of labour may slow the clearance of pulmonary fluid from the lungs.

During vaginal delivery some lung fluid, perhaps 35 ml in a term baby, is expelled by passage through the birth canal. In a healthy baby the first spontaneous breaths may generate a negative pressure of between −30 and −90 cmH_2O, which aerates the lungs for the first time. This pressure is 10–15 times greater than that needed for later breathing but is necessary to overcome the viscosity of the fluid filling the airways, the surface tension of the fluid-filled lungs, and the elastic recoil and resistance of the chest wall, lungs and airways. These powerful chest movements cause fluid to be displaced from the airways into the lymphatics and circulation.

After delivery, a healthy term baby usually takes its first breath within 60–90 seconds of clamping or obstructing the umbilical cord. Separation of the placenta and clamping of the cord leads to the onset of hypoxia, which is initially a major stimulant to start respiration. Physical stimuli such as cold air or physical discomfort may also provoke respiratory efforts.

In a 3 kg baby up to 100 ml of fluid is cleared from the airways following the initial breaths, a process aided by full inflation and prolonged high pressure on expiration, i.e. crying. The effect of the first few breaths is to produce the baby's functional residual capacity. Neonatal circulatory adaptation commences with the detachment of the placenta, but lung inflation and alveolar distension releases mediators, which affect the pulmonary vasculature as well as increasing oxygenation.

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I.3 PATHOPHYSIOLOGY

Our knowledge of the pathophysiology of fetal asphyxia is based on pioneering animal work in the early 1960s. The results of these experiments, which followed the physiology of newborn animals during acute, total, prolonged asphyxia and subsequent resuscitation are summarised in Figure I.1.

When the placental oxygen supply is interrupted, the fetus attempts to breathe. Should these attempts fail to provide an alternative oxygen supply – as they will inevitably fail to do so *in utero* – the baby will lose consciousness. If hypoxia continues, the respiratory centre becomes unable, through lack of sufficient oxygen, to continue initiating breathing and the breathing stops, usually within 2–3 minutes (primary apnoea, Figure I.1).

Fetal bradycardia ensues blood pressure is maintained, primarily by peripheral vasoconstriction and diversion of blood away from non-vital organs, and also by an increased stroke volume. After a latent period of apnoea (primary), primitive spinal centres, no longer suppressed by neural signals from the respiratory centre, exert an effect by initiating primitive gasping breaths. These deep spontaneous gasps are easily distinguishable from normal breaths as they only occur 6–12 times per minute and involve all accessory muscles in a maximal inspiratory effort. After a while, if hypoxia continues, even this activity ceases (terminal apnoea). The time taken for such activity to cease is longer in the newly born baby than in later life, taking up to 20 minutes.

The circulation is almost always maintained until all respiratory activity ceases. This resilience is a feature of all newborn mammals at term, largely due to the reserves of glycogen in the heart. Resuscitation is therefore relatively easy if undertaken before all respiratory activity has stopped. Once the lungs are inflated, oxygen will be carried to the heart and then to the brain provided the circulation is still functional (Figure I.2). Recovery will then be rapid. *Most* infants who have not progressed to terminal apnoea will resuscitate themselves if their airway is patent. Once gasping ceases, however, the circulation starts to fail and these infants are likely to need more extensive resuscitation (Figure I.3).

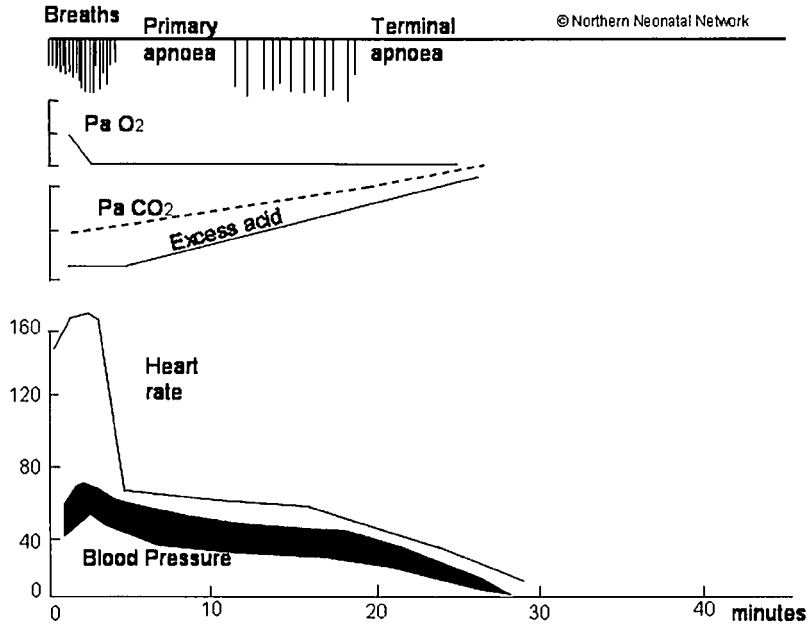

Figure I.1 Response of a mammalian fetus to total, sustained asphyxia started at time 0. (Reproduced with permission from the Northern Neonatal Network)

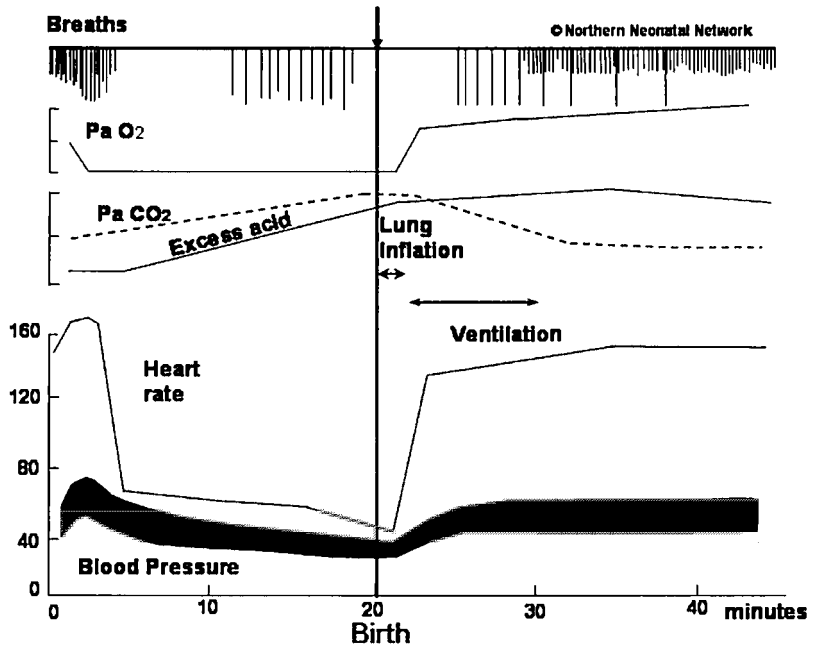

Figure I.2 Effects of lung inflation and a brief period of ventilation on a baby born in early terminal apnoea but before failure of the circulation. (Reproduced with permission from the Northern Neonatal Network)

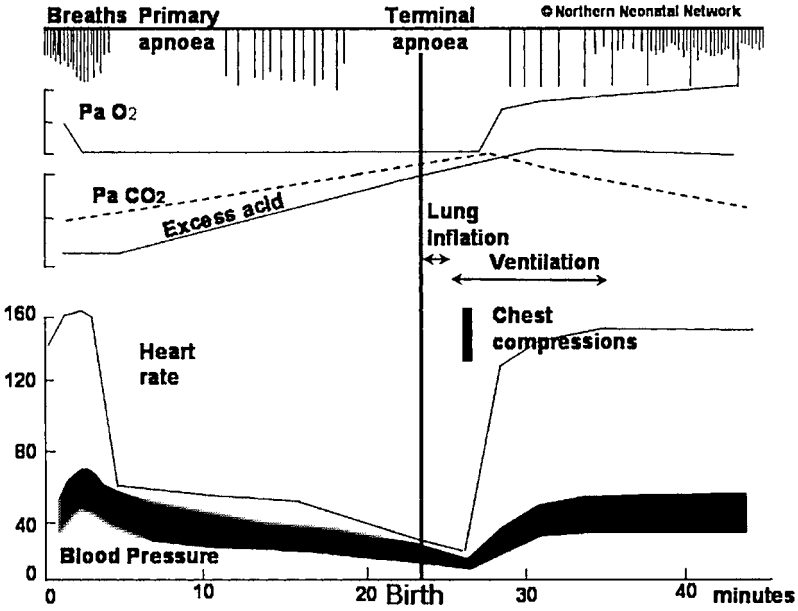

Figure I.3 Response of a baby born in terminal apnoea. In this case lung inflation is not sufficient because the circulation is already failing. However, lung inflation delivers air to the lungs and then a brief period of chest compressions delivers oxygenated blood to the heart which then responds. (Reproduced with permission from the Northern Neonatal Network)

I.4 EQUIPMENT

For many newborn babies, especially those born outside the delivery room, the need for resuscitation cannot be predicted. It is therefore useful to plan for such an eventuality. Equipment that may be required to resuscitate a newborn baby is listed in the box below. This will vary between departments; however, most babies can be resuscitated with a flat surface, warmth, knowledge and a way to deliver air or oxygen at a controlled pressure.

Equipment for newborn resuscitation

- A flat surface
- Radiant heat source and dry towels (or suitable plastic bags for preterm infants)
- Suction with catheters of at least 12 Fr gauge
- Face masks
- Bag–valve–mask or T-piece with pressure-limiting device
- Source of air and/or oxygen
- Oropharyngeal (Guedel) airways
- Laryngoscopes with straight blades, 0 and 1
- Nasogastric tubes
- Cord clamp
- Scissors
- Tracheal tubes sizes 2.5 to 4.0 mm
- Umbilical catheterisation equipment
- Adhesive tape
- Disposable gloves
- Plastic bag for preterm babies
- Saturation monitor/stethoscope

I.5 STRATEGY FOR ASSESSING AND RESUSCITATING A BABY AT BIRTH

Resuscitation is likely to be rapidly successful if begun before the baby has become so anoxic that all potential for respiratory activity has vanished. Babies in primary apnoea can usually resuscitate themselves if they have a clear airway. As you do not know whether a newborn, apnoeic baby is in primary or secondary apnoea you must develop a graded approach that will work in either situation. Always start by drying and covering the baby to prevent it from getting cold and then proceed as far as is necessary down the following list:

- Call for help.
- Start the clock or note the time.
- Dry and cover the baby.
- Assess the situation.

- Airway.
- Breathing.
- Chest compressions.
- (Drugs.)

Call for help

Ask for help if you expect or encounter any difficulty or if the delivery is outside the labour suite.

Start the clock

If available, or note the time of birth.

At birth

- There is no need to rush to clamp the cord, particularly if the baby appears well. Unless the baby is clearly in need of immediate resuscitation, wait for at least 1 minute from the complete delivery of the baby before clamping the cord. Keep the baby warm during this time.
- Dry the baby quickly and effectively. Remove the wet towel and wrap in a fresh, dry, warm towel. (For very small or significantly preterm babies it is better to place the wet baby in a food-grade plastic bag – and later under a radiant heater.)
- During this period it is possible to assess the baby and decide whether any intervention is going to be needed.
- Then clamp and cut the cord.

If the baby is thought to be in need of assistance then this becomes the priority. This may mean that the cord needs to be clamped in order to deliver that assistance.

Keep the baby warm

Dry the baby off immediately and then wrap in a dry towel. A cold baby has increased oxygen consumption and cold babies are more likely to become hypoglycaemic and acidotic. They also have an increased mortality. If this is not addressed at the beginning of resuscitation it is often forgotten. Most of the heat loss is caused by the baby being wet and in a draught – hence the

need to dry the baby and then to wrap the baby in a dry towel. Babies also have a large surface area to weight ratio, thus heat can be lost very quickly. Ideally, delivery should take place in a warm room, and an overhead heater should be switched on. However, drying effectively and wrapping the baby in a warm dry towel is the most important factor in avoiding hypothermia. A naked wet baby can still become hypothermic despite a warm room and a radiant heater, especially if there is a draught. Make sure that the head is covered as it represents a significant part of the baby's surface area (see 'Pre-term babies' below).

Assessment of the newborn baby

Whilst keeping the baby warm make an initial assessment by assessing:

- Respiration (rate and quality) — *Airway and breathing*
- Heart rate (fast, slow, absent)
- Colour (pink, blue, pale)
- Tone (unconscious, apnoeic babies are floppy)

Circulation

Unlike resuscitation at other ages, it is important to assess fully in order that one can judge the success of interventions. This is most true of heart rate and breathing, which guide further resuscitative efforts. However, a baby who is white and shut down peripherally is more likely to be acidotic and a baby who is atonic is likely to be unconscious. Subsequent assessments should focus on breathing and heart rate.

Respiration

Most babies will establish spontaneous regular breathing sufficient to maintain the heart rate above 100 beats/min and to improve the skin colour within 3 minutes of birth. If apnoea or gasping persists after drying, intervention is required.

Heart rate

In the first couple of minutes, auscultating at the cardiac apex is the best method to assess the heart rate. Palpating peripheral pulses is not practical and cannot be recommended. Palpation of the umbilical pulse can only be relied upon if the rate is ≥100 beats/min. A rate less than this should be checked by auscultation if possible or by a saturation monitor using Masimo (or similar) technology if available. A saturation monitor applied to the right hand or wrist can give an accurate reading of heart rate and saturations within 90 seconds of application (see later). An initial assessment of heart rate is vital because an increase in the heart rate will be the first sign of success during resuscitation.

Colour

Attempting to judge oxygenation by assessing skin colour is unreliable but it is still worth noting the baby's colour at birth as well as whether, when and how it changes. Very pale babies who remain pale after resuscitation may be hypovolaemic as well as acidotic.

If the baby has good tone, a good heart rate and is making good respiratory effort then further help is unlikely to be needed.

Using a saturation monitor will allow rapid assessment of heart rate and saturation within about 90 seconds of application. Oxygen saturation levels in healthy babies in the first few minutes of life may be considerably lower than at other times.

Acceptable pre-ductal SpO_2	
2 min	60%
3 min	70%
4 min	80%
5 min	85%
10 min	90%

This assessment will categorise the baby into one of the three following groups:

1 *Regular respirations, heart rate fast (more than 100 beats/min) and pink, good tone.* These are healthy babies and they should be kept warm and given to their mothers. The baby will remain warm through skin-to-skin contact with the mother under a cover and may be put to the breast at this stage.
2 *Irregular or inadequate respirations, heart rate slow (less than 100 beats/min) and blue, normal or reduced tone.* If gentle stimulation (such as drying) does not induce effective breathing, the airway should be opened and, if necessary, cleared. If the baby responds then no further resuscitation is needed. If there is no response, progress to lung inflation.
3 *Apnoeic, heart rate slow (less than 100 beats/min) or absent and blue or pale, floppy.* Whether an apnoeic baby is in primary or terminal apnoea (see Figure I.1) the initial management is the same. Open the airway and then inflate the lungs. A reassessment of any heart rate response then directs further resuscitation. Reassess the heart rate and respiration at regular intervals throughout.

Apnoea, low or absent heart rate, pallor and floppiness together suggest terminal apnoea. However, initial management of such babies is unchanged but resuscitation may be prolonged.

After assessment, resuscitation follows:

- Airway.
- Breathing.
- Circulation.
- With the use of drugs in a few selected cases.

Resuscitation of the newborn baby

Airway

The baby should be positioned with the head in the neutral position (Figure I.4; see also Chapter 4). The newborn baby's head has a large, often moulded, occiput which tends to cause the neck to flex when the baby is supine on a flat surface. However, overextension may also collapse the newborn baby's pharyngeal airway, leading to obstruction. A 2 cm folded towel placed under the neck and shoulders may help to maintain the airway in a neutral position and a jaw thrust may be needed to bring the tongue forward and open the airway, especially if the baby is floppy (Figure I.5). Visible secretions may be removed by gentle suction with a paediatric Yankauer or 12–14 gauge suction catheter, although these rarely cause airway obstruction. Blind deep pharyngeal suction should not be peformed as it may cause vagally induced bradycardia and laryngospasm. Suction, if it is used, should not exceed −100 mmHg (9.8 kPa). The presence of thick meconium (see below) in a non-vigorous baby is the only indication for considering visualisation of the pharynx and immediate suction.

Figure I.4 Chin lift in infants

Figure I.5 Jaw thrust

Meconium aspiration Meconium-stained liquor (light green tinge) is relatively common and occurs in up to 10% of births. Happily, meconium aspiration is a rare event. Meconium aspiration usually happens in term infants *in utero* before delivery. A large randomised trial has shown no advantage to suctioning the airway whilst the head is on the perineum and that this may delay resuscitation. This practice is, therefore, no longer recommended. If the baby is vigorous, a randomised trial has shown that suctioning at any time offers no advantage and no specific action (other than drying and wrapping the baby) is needed.

If the baby has absent or inadequate respirations, a heart rate <100 beats/min or hypotonia, inspect the oropharynx with a laryngoscope and aspirate any particulate meconium seen using a wide-bore catheter. If intubation is possible and the baby is still unresponsive, aspirate the trachea, using the tracheal tube as a suction catheter. However, if intubation cannot be achieved immediately, clear the oropharynx and start mask inflation. If, while attempting to clear the airway, the heart rate falls to less than 60 beats/min then stop airway clearance, give aeration breaths and start ventilating the baby.

Breathing (aeration breaths and ventilation)

The first five breaths in term babies should be 'inflation' breaths in order to replace lung fluid in the alveoli with air. These should be 2–3 second sustained breaths using a continuous gas supply, a pressure-limiting device and a mask. Use a transparent, circular, soft mask big enough to cover the nose and mouth of the baby. If no such system is available then a 500 ml self-inflating bag and a blow-off valve set at 30–40 cmH_2O can be used (Figure I.6). This is especially useful if compressed air or oxygen is not available.

Figure I.6 Bag-and-mask ventilation

The chest may not move during the first 1–3 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/min. Therefore, reassess the heart rate after delivery of the first five breaths. It is safe to assume the chest has been inflated successfully if the heart rate responds.

Once the chest is inflated and the heart rate has increased or the chest has been seen to move, then ventilation should be continued at a rate of 30–40 per minute. Continue ventilatory support until regular breathing is established. Where possible, start resuscitation of the baby at birth with air. There is now good evidence for this in term babies and oxygen toxicity is a real concern with premature babies. Use of supplemental oxygen should be guided by pulse oximetry, with reasonable levels listed in the box above (page 334) and on the algorithm (page 342).

If the heart rate has not responded, then check for chest movement rather than auscultation as in fluid-filled lungs breath sounds may be heard without lung inflation. Go back and check airway opening manoeuvres and repeat the inflation breaths.

Circulation

If the heart rate remains slow or absent, despite adequate ventilation for 30 seconds, as shown by chest movement, then chest compressions should be started. These chest compressions should help to move oxygenated blood from the lungs to the heart and coronary arteries. The blood you move can only be oxygenated if the lungs have air in them. Cardiac compromise is always the result of respiratory failure and can only be effectively treated if effective ventilation is occurring.

The most efficient way of delivering chest compressions in the neonate is to encircle the chest with both hands, so that the fingers lie behind the baby and the thumbs are apposed on the sternum just below the inter-nipple line (Figure I.7). Compress the chest briskly, *by one-third of its depth*. In newborn babies, current advice is to perform three compressions for each ventilation breath (3:1 ratio).

The purpose of chest compression is to move oxygenated blood or drugs to the coronary arteries in order to initiate cardiac recovery. Thus there is no point in starting chest compression before effective lung inflation has been established. Similarly, compressions are ineffective unless interposed by ventilation breaths of good quality. Therefore, the emphasis must be upon *good-quality breaths*, followed by effective compressions. Simultaneous delivery of compressions and breaths should be avoided, as the former will reduce the effectiveness of the breaths. It is usually only necessary to continue chest compressions for about 20–30 seconds before the heart responds with an increase in heart rate.

Once the heart rate is above 60 beats/min and rising, chest compression can be discontinued. Maintain ventilations until effective breathing or mechanical ventilation is established.

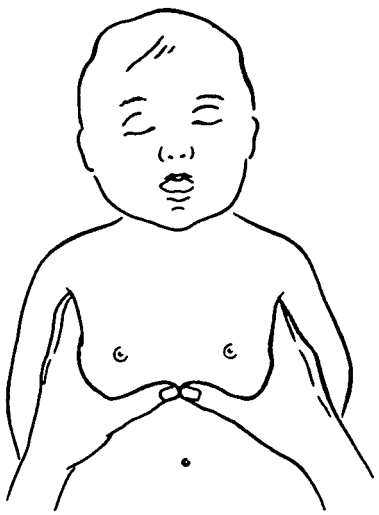

Figure I.7 Infant chest compression: hand-encircling technique

Drugs

If after adequate lung inflation and cardiac compressions the heart rate has not responded, drug therapy should be considered. However, the most common reason for failure of the heart rate to respond is failure to achieve lung inflation, and there is *no point* in giving drugs unless the airway is open and the lungs have been inflated. Airway and breathing must be reassessed as adequate before proceeding to drug therapy. Venous access will be required via an umbilical venous line, because ideally drugs should be given centrally. The outcome is poor if drugs are required for resuscitation.

Adrenaline The α-adrenergic effect of adrenaline increases coronary artery perfusion during resuscitation, enhancing oxygen delivery to the heart. In the presence of profound unresponsive bradycardia or circulatory standstill, 10 micrograms/kg (0.1 ml/kg 1:10,000) adrenaline may be given intravenously. Further doses of 10–30 micrograms/kg (0.1–0.3 ml 1:10,000) may be tried at 3–5-minute intervals if there is no response. The tracheal route cannot be recommended, as there are insufficient data. However, if it is used tracheally, animal evidence suggests that doses of 30 micrograms/kg will be ineffective.

Bicarbonate Any baby who is in terminal apnoea will have a significant metabolic acidosis. Acidosis depresses cardiac function. Bicarbonate 1–2 mmol/kg (2–4 ml/kg of 4.2% solution) may be used to raise the pH and enhance the effects of oxygen and epinephrine. Bicarbonate use remains controversial and it should only be used in the absence of discernible cardiac output despite all resuscitative efforts or in profound and unresponsive bradycardia.

Glucose Hypoglycaemia is a potential problem for all stressed or asphyxiated babies. It is treated using a slow bolus of 2.5 ml/kg of 10% glucose intravenously, and then providing a secure intravenous dextrose infusion at a rate of 100 ml/kg/day of 10% glucose. BM stix are not reliable in neonates when reading less than 5 mmol/l.

Fluid Very occasionally hypovolaemia may be present because of known or suspected blood loss (antepartum haemorrhage, placenta or vasa praevia, unclamped cord) or it may be secondary to loss of vascular tone following asphyxia. Volume expansion, initially with 10 ml/kg, may be appropriate. Normal saline can be used; alternatively Gelofusine has been used safely and if blood loss is acute and severe, non-cross-matched O-negative blood should be given immediately. Albumin cannot be recommended. However, most newborn or neonatal resuscitations do not require fluid unless there has been known blood loss or septicaemic shock.

Naloxone This is not a drug of resuscitation. Occasionally, a baby *who has been effectively resuscitated*, is pink, with a heart rate of over 100 beats/min, may not breathe spontaneously because of the possible effects of maternal opiates. If respiratory depressant effects are suspected the baby should be given naloxone intramuscularly (200 micrograms in a full-term baby). Smaller doses of 10 micrograms/kg will also reverse the sedation but the effect will only last a short time (20 minutes IV or a few hours IM). Intravenous naloxone has a half-life shorter than opiates, and there is no evidence to recommend intratracheal administration.

I.6 RESPONSE TO RESUSCITATION

The first indication of success will be an increase in heart rate. Recovery of respiratory drive may be delayed. Babies in terminal apnoea will tend to gasp first as they recover before starting normal respirations (see Figure I.3). Those who were in primary apnoea are likely to start with normal breaths, which may commence at any stage of resuscitation.

Tracheal intubation

Most babies can be resuscitated using a mask system. Swedish data suggest that if this is applied adequately, only 1 in 500 babies may actually need intubation. However, tracheal intubation only remains the gold standard in airway management if it is performed perfectly. It is especially

useful in prolonged resuscitations, pre-term babies and meconium aspiration. It should be considered if mask ventilation has failed, although the most common reason for this is poor positioning of the head with consequent failure to open the airway.

The technique of intubation is the same as for infants and is described in Chapter 20. A normal full-term newborn usually needs a 3.5 mm tracheal tube, but 4.0, 3.0 and 2.5 mm tubes should also be available.

Tracheal tube placement must be assessed visually during intubation and in most cases will be confirmed by a rapid response in heart rate on ventilating via the endotracheal tube. If in doubt exhaled CO_2 detection will identify most correctly sited tubes in the presence of any cardiac output. Detection of exhaled CO_2 should be used to confirm tracheal tube placement.

I.7 SPECIAL CASES

Pre-term babies

Unexpected deliveries outside delivery suites are more likely to be premature. Premature babies are more likely to get cold (higher surface area to mass ratio), and more likely to become hypoglycaemic (fewer glycogen stores). There are now several trials that support the use of plastic bags placed over babies of <29 weeks' gestation or <1000 g before drying in order to keep warm. The babies should then be placed under a radiant heater (see box below). The effectiveness of this technique without a radiant heater has not been tested in a trial.

Guidelines for the use of plastic bags for pre-term babies (<29 weeks) at birth

1. Pre-term babies born below 29 completed weeks' gestation may be placed in plastic bags or wrap for temperature stability during resuscitation. They should remain in the bag until they are on the neonatal intensive care unit (NICU) and the humidity within their incubator is at the desired level. It is a way of preventing evaporative heat loss and cannot replace incubators, etc. Neither should it replace all efforts to maintain a high ambient temperature around babies born outside delivery suites.
2. At birth the baby should not be dried, but should be slipped straight into the prepared plastic bag or wrapping. There is no need to wrap in a towel so long as this is done immediately after birth. This gives immediate humidity. The plastic bag only prevents evaporative heat loss – once in the bag the baby should be placed under a radiant heater.
3. Suitable plastic bags are food-grade bags designed for microwaving and roasting. They should be large. The bag is prepared with a V cut in the closed end. Purpose-made bags and wraps are also available.
4. The bag should cover the baby from the shoulders to the feet, with the head protruding through the V cut. This is most easily performed if the hand is placed through the V, the head placed in the hand, and the bag drawn back down over the baby.
5. The head will stick out of the V cut and should be dried as usual and resuscitation commenced as per standard guidelines. A hat should be placed over the head if practical to further reduce heat loss.
6. Standard resuscitation can be carried out without any limitations of access, but if the umbilicus is required for any access then a hole can be made above the area and the desired intervention done.
7. The bag should not be removed unless deemed necessary by the registrar or consultant.
8. After transfer to a neonatal unit and stabilising ventilation, if required, the baby's temperature should be recorded. The bag is only removed when the incubator humidity is satisfactory, and further care provided as per nursing protocols.

This is a potentially useful technique for keeping larger babies warm when born unexpectedly outside the delivery suite or in the community. However, it should be augmented by also wrapping with warm towels and ensuring a warm environment.

The more premature a baby, the less likely it is to establish adequate respirations. Pre-term babies of less than 32 weeks' gestation are likely to be deficient in surfactant especially after unexpected or precipitate delivery. The surfactant, secreted by pneumocytes in the alveolar epithelium, reduces alveolar surface tension and prevents alveolar collapse on expiration. Small amounts of surfactant can be demonstrated from about 20 weeks' gestation, but a surge in production occurs at 30–34 weeks. Surfactant is released at birth due to aeration and distension of the alveoli. The half-life of the surfactant is approximately 12 hours. Production is reduced by hypothermia (<35°C), hypoxia and acidosis (pH <7.25). In babies born before 32 weeks, one must anticipate a lack of surfactant. The effort of respiration will be increased, although the musculature will be less developed. They may require help to establish prompt aeration and ventilation, and may subsequently require exogenous surfactant therapy.

The lungs of pre-term babies are more fragile than those of term babies and thus are much more susceptible to damage from overdistension. Therefore, it is appropriate to start with a lower inflation pressure of 2.0–2.5 kPa (20–25 cmH_2O) but do not be afraid to increase this to 30 cm H_2O if there is no heart rate response.

It should be noted that very obvious chest wall movement in premature babies of less than 28 weeks' gestation may indicate excessive and potentially damaging tidal volumes.

Premature babies are more susceptible to the toxic effects of hyperoxia. Using a pulse oximeter to monitor both heart rate and oxygen saturation in these babies from birth makes stabilisation much easier. Exposing babies at birth to high concentrations of oxygen can have significant adverse longer term effects. Ranges of pre-ductal oxygen saturation found in the first few minutes of life in well pre-term infants are increasingly being reported, however normal values in well babies born before 32 weeks' gestation are based on small numbers. Therefore, at present, additional oxygen should not be given if the oxygen saturation from the right arm or wrist is above the values below:

Time from birth	Acceptable (25th centile) pre-ductal saturation (%) under 32 weeks' gestation
2 min	60
3 min	70
4 min	80
5 min	85
10 min	90

Saturation monitoring

Pulse oximetry gives a quick and relatively accurate display of both heart rate and oxygen saturation that can be easily seen by all involved in the resuscitation. This is particularly useful when stabilising significantly pre-term babies or when tempted to give additional oxygen to any baby. Once the oximeter is switched on, a reading can be obtained a few seconds faster if the probe is first attached to the right hand or wrist of the baby and only then connected to the machine. Once the heart rate is displayed it is likely that this will be more accurate than other commonly used methods of assessing heart rate.

Actions in the event of poor initial response to resuscitation

1 Check airway and breathing.

2 Check for a technical fault:

- Is mask ventilation effective? Observe chest movement.
- Is the tracheal tube in the trachea? Auscultate both axillae, listen at the mouth for a large leak, and observe movement. Use an exhaled CO_2 detector to ensure tracheal tube position.

- Is the tracheal tube in the right bronchus? Auscultate both axillae and observe movement.
- Is the tracheal tube blocked? If there is doubt about the position or patency of the tracheal tube re-place it. Use an exhaled CO_2 detector.
- Is a longer inflation time required?
- If starting in air then increase the oxygen concentration. This is least likely to be a cause, although if monitoring saturations it could be a cause for slow increase.

3 Does the baby have a pneumothorax? This occurs spontaneously in up to 1% of newborns, but those needing action in the delivery unit are exceptionally rare. Auscultate the chest for asymmetry of breath sounds. A cold light source can be used to transilluminate the chest – a pneumothorax may show as a hyper-illuminating area. If a tension pneumothorax is thought to be present clinically, a 21-gauge butterfly needle should be inserted through the second intercostal space in the mid-clavicular line. Alternatively, a 22-gauge cannula connected to a three-way tap may be used. Remember that you may well cause a pneumothorax during this procedure (see Chapter 22).

4 Does the baby remain cyanosed despite breathing with a good heart rate? There may be a congenital heart malformation, which may be duct dependent (see Chapter 9), or a persistent pulmonary hypertension.

5 If, after resuscitation, the baby is pink and has a good heart rate but is not breathing effectively, it may be suffering the effects of maternal opiates. Naloxone 200 micrograms IM may be considered, and this should outlast the opiate effect.

6 Is there severe anaemia or hypovolaemia? In case of large blood loss, 20 ml/kg O-negative blood or a volume expander should be given.

Birth outside the delivery room

Whenever a baby is born unexpectedly, the greatest difficulty often lies in keeping it warm. Drying and wrapping, turning up the heating and closing windows and doors are all important in maintaining temperature. Special care must be taken to clamp and cut the cord to prevent blood loss.

Hospitals with accident and emergency departments should have guidelines for resuscitation at birth, summoning help and post-resuscitation transfer of babies born within the department.

Babies born unexpectedly outside hospital will be at greater risk of being pre-term and of getting cold. However, the principles of resuscitation are identical to the hospital setting. Transport will need to be discussed according to local guidelines.

Discontinuation of resuscitation

The outcome for a baby with no detectable cardiac output for more than 10 minutes is likely to be very poor. Stopping resuscitation early, or not starting resuscitation at all, may be appropriate in situations of extreme prematurity (<23 weeks), birth weight of <400 g, or in the presence of lethal abnormalities such as anencephaly or confirmed trisomy 13 or 18. Resuscitation is nearly always indicated in conditions with a high survival rate and acceptable morbidity. Such decisions should be taken by a senior member of the team, ideally a consultant in consultation with the parents and other team members.

Communication with the parents

It is important that the team caring for the newborn baby informs the parents of the progress whenever possible. This is likely to be most difficult in unexpected deliveries so prior planning to cover the eventuality may be helpful. Decisions at the end of life must involve the parents whenever possible. All communication should be documented after the event.

I.8 SUMMARY

The approach to newborn resuscitation is summarised in the algorithm in Figure I.8.

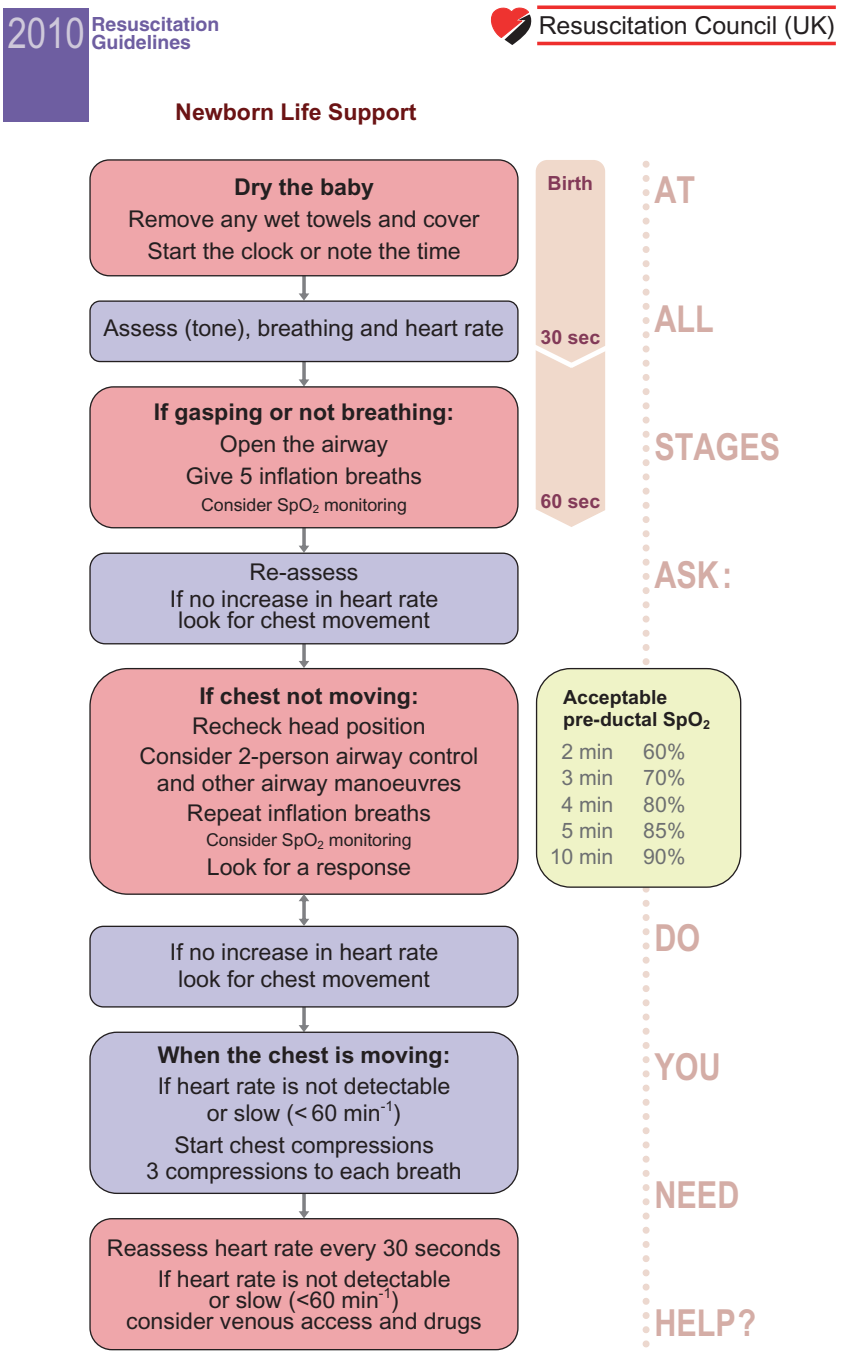

Figure I.8 Newborn resuscitation algorithm. HR, heart rate. (Reproduced with kind permission from the Resuscitation Council (UK))

APPENDIX J

Formulary

The formulary is intended as a reference to be used in conjunction with this book. To this end the drugs mentioned elsewhere are set out alphabetically below, along with their routes of administration, dosage and some notes on their use.

General guidance on the use of the formulary

- When dosage is calculated on a basis of per kilogram and a maximum dose is not stated, then the dose given should not exceed that for a 40 kg child.
- The exact dose calculated on a basis of per kilogram may be difficult to administer because of the make-up of the formulations available. If this is the case the dose may be rounded up or down to a more manageable figure.
- Doses in the formulary are sometimes written as µg or ng. When prescribing such doses these terms should be written in full (micrograms (mcg) or nanograms, respectively) in order to avoid confusion.
- Although every effort has been made to ensure accuracy, the writers, editors, publishers and printers cannot accept liability for errors or omissions.
- More detailed information about individual drugs is available from the manufacturers, from the *British National Formulary for Children*, from hospital drug information centres and from the pharmacy departments of children's hospitals.

Abbreviations

The following abbreviations to indicate administration route are used:

via ETT	via endotracheal tube
IO	intraosseous
IM	intramuscular
IV	intravenous
SC	subcutaneous

The final responsibility for delivery of the correct dose remains that of the physician prescribing and administering the drug.

Advanced Paediatric Life Support, Fifth Edition. Edited by Martin Samuels, Sue Wieteska.

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
		Under 12 years			
ACETYLCYSTEINE		**<20 kg**	**>20 kg**	**12–18 years**	
Treatment of paracetamol overdose	IV infusion over 15 minutes	150 mg/kg in 3 ml/kg	150 mg/kg in 100 ml	150 mg/kg in 200 ml	Single dose
	Then	Then	Then	Then	
	IV infusion over 4 hours	50 mg/kg in 7 ml/kg	50 mg/kg in 250 ml	50 mg/kg in 500 ml	Single dose
	Then	Then	Then	Then	
	IV infusion over 16 hours	100 mg/kg in 14 ml/kg	100 mg/kg in 500 ml	100 mg/kg in 1 litre	Single dose
		Notes: May be used up to 24 hours after paracetamol overdose After 24 hours seek guidance from a National Poisons Information Centre Infuse in glucose 5%, if this is unsuitable use normal saline 0.9% Use with caution if patient is taking liver enzyme-inducing drugs, e.g. phenytoin Contraindicated if previous hypersensitivity to any of the ingredients. Use with caution in patients with asthma or a history of bronchospasm Monitor serum potassium			

ACICLOVIR		Up to 2 years	2–12 years	12–18 years	
Herpes simplex virus treatment	IV infusion	*<3 months*: 10 mg/kg *3–24 months*: 250 mg/m^2	250 mg/m^2	5 mg/kg	3 times daily
		Notes: Normal immune status			
		<3 months: 10 mg/kg *3–24 months*: 500 mg/m^2	500 mg/m^2	10 mg/kg	3 times daily
		Notes: Immunocompromised or encephalitis In both cases: adjust dose if renal impairment. Not given by IV bolus, infuse over 1 hour			

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
ACTIVATED CHARCOAL		**1 month to 2 years**	**2–12 years**	**12–18 years**	
Absorption of poisons	Oral	Dose by weight: 1 g/kg			Single dose
		Dose by age:			Single dose
		–	25–50 g	50 g	
		Notes: Single dose treatment Because of the risk of aspiration, charcoal should never be given to a child with an absent gag reflex or impaired consciousness unless the airway is first protected by an endotracheal tube			

ADENOSINE		**Birth to 12 years**	**12–18 years**	
Antiarrhythmic to terminate SVT and to elucidate mechanism of tachycardia	IV bolus	100 mcg/kg	3 mg	Single dose
		Increase after 2 minutes if necessary to 200 mcg/kg and then 300 mcg/kg	Increase after 2 minutes if necessary to 6 mg	
		Maximum dose: 300 mcg/kg <1 month 500 mcg/kg >1 month	Increase after further 2 minutes if necessary to 12 mg	
		Notes: Initial dose as shown Subsequent doses if required as shown Increments should not be given if high level AV block develops at any particular dose *Contraindications or warnings*: second or third degree heart block, AV block, sick sinus syndrome and asthma. Beware of torsades de pointes in a child with prolonged QT interval		

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
ADRENALINE (EPINEPHRINE)		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Cardiopulmonary resuscitation (CPR)	IV/ETT	10 mcg/kg (0.1 ml/kg of 1:10,000)	–	–	–	Initial dose
		10–30 mcg/kg (0.1–0.3 ml/kg of 1:10,000)	–	–	–	Subsequent doses
		Notes: Endotracheal route is accepted but has unproven effectiveness in resuscitation at birth				
CPR	IV rapid bolus/IO	–	10 mcg/kg (0.1 ml/kg of 1:10,000)		1 mg (10 ml of 1:10,000)	Initial and usual subsequent dose
		Notes: If given by IO route flush with NaCl 0.9%				
		–	100 mcg/kg (0.1 ml/kg of 1:1000 or 1 ml/kg of 1:10,000)		5 mg (5 ml of 1:1000)	
		Notes: Maximum dose is 5 ml of 1:1000. Subsequent doses in exceptional circumstances, e.g. arterial monitoring, septicaemia, anaphylaxis				
	ETT	–	100 mcg/kg (0.1 ml/kg of 1:1000 or 1 ml/kg of 1:10,000)		5 mg (5 ml of 1:1000)	
Acute anaphylaxis	Deep IM	10 mcg/kg (0.01 ml/kg of 1:1000) or <6 years: 150 mcg 6–12 years: 300 mcg >12 years: 500 mcg			0.5–1 mg (0.5–1 ml of 1:1000)	Single dose
		Notes: Repeat at 5-minute intervals if necessary according to clinical response				
Low cardiac output	IV infusion	10 nanograms to 1 mcg/kg/min				Continuous
		Notes: Start at lower doses				
Croup	Nebulised	400 mcg/kg (0.4 ml/kg of 1:1000)			5 mg (5 ml of 1:1000)	Single dose
		Notes: Produces a transient improvement, rarely alters the long-term course of the illness. Observe closely with ECG and oxygen saturation monitoring				
Envenomation	Subcutaneous	5–10 mcg/kg				Single dose

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
ALPROSTADIL		**Birth to 1 month**	
Duct-dependent congenital heart defects in neonates	IV infusion	Start at 5 nanograms/kg/min increasing in increments of 5 nanograms/kg/min to 20 nanograms/kg/min	Single dose
	Then	Then	
	IV infusion	Decrease to lowest effective dose	
		Note: Intensive support required: maximum doses of 100 nanograms/kg/min have been used	
AMINOPHYLLINE		**1 month to 12 years** / **12–18 years**	
Bronchodilator in asthma or anaphylaxis	IV infusion over 20–30 minutes	5 mg/kg (max. 500 mg)	Single loading dose over 20–30 minutes
		Notes: Loading dose if no theophylline or aminophylline has been given in the last 24 hours	
	IV infusion	1 mg/kg/h / 500 mcg/kg/h	Continuous
		Notes: Maintenance dose. Infusion can usually be stopped and not tapered down. Monitor for arrhythmias, vomiting and seizures	
AMIODARONE		**Birth to 18 years**	
Stable arrhythmias	IV loading dose	5 mg/kg	Single dose
	IV infusion	300 mcg/kg/h (max. 1.5 mg/kg/h; do not exceed 1.2 g in 24 hours)	Continuous
		Notes: Slow IV injection over 20 minutes (neonates 30 min) Loading doses up to 15 mg/kg have been reported Give via a central line if possible. Adjust rate according to clinical response. Dilute to a concentration of not less than 600 mcg/ml with glucose 5%	
In cardiopulmonary resuscitation: shock-resistant VF and pulseless VT	Rapid IV bolus	5 mg/kg (max. 300 mg) over 3 min	Single dose
		Notes: The injection is compatible with glucose 5% only May be used in concentrations of 15 mg in 1 ml of 5% glucose	

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
ATROPINE SULPHATE		**Birth to 1 month**	**1 month to 12 years**	**12–18 years**	
Pre-intubation dose or to be given in bradycardia if induced by vagal stimulation	IV bolus over 1 minute	15 mcg/kg	20 mcg/kg (min. 100 mcg, max. 600 mcg)	300 mcg to 1 mg	Single dose
		Notes: May be repeated			
BUDESONIDE		**1 month to 18 years**			
Croup	Nebuliser	2 mg (may be repeated after 12 hours)			Single dose
BUPIVACAINE HYDROCHLORIDE		**Birth to 12 years**		**12–18 years**	
Infiltration and regional anaesthesia including peripheral nerve block	Local infiltration	Up to 0.8 ml/kg of 0.25% (up to 2 mg/kg)		Up to 60 ml 0.25% (up to 150 mg)	Single dose
		Notes: Do not administer more than every 8 hours			
CALCIUM GLUCONATE		**Birth to 18 years**			
Cardiopulmonary resuscitation only when there is electrolyte disturbance and in septicaemia where there is hypocalcaemia	IV bolus	0.3 ml/kg of 10% solution			Single dose
		Notes: Maximum dose 20 ml (4.5 mmol). Tissue damage if there is extravasation			
CALCIUM RESONIUM		**Birth to 18 years**			
Hyperkalaemia associated with anuria or severe oliguria and in dialysis patients	Oral	0.5–1 g/kg			May be repeated after 6–12 hours
		Notes: Contraindicated in obstructive bowel disease			

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
CEFOTAXIME		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Severe neonatal infections, meningitis	IV	*<7 days*: 50 mg/kg	–	–	–	Twice daily
		7–21 days: 50 mg/kg	–	–	–	3 times daily
		>21 days: 50 mg/kg	–	–	–	3–4 times daily
Meningitis, respiratory tract, urinary tract and soft tissue infections, epiglottitis	IV	–	50 mg/kg		1–3 g	Twice daily
		Notes: The frequency should be increased to 4 times daily in meningitis and other severe infections. In these cases a loading dose of 80 mg/kg can be given				

CEFTRIAXONE		Birth to 1 month	1 month to 18 years	
Severe neonatal infections, meningitis	IV	20–50 mg/kg	–	Once daily
		Notes: Do not exceed 50 mg/kg. Infuse doses of 50 mg/kg over at least 60 minutes Avoid in premature, acidotic or hyperbilirubinaemic neonates		
Meningitis, respiratory tract, urinary tract and soft tissue infections, epiglottitis	IV or IM	–	20–50 mg/kg	Once daily
		Notes: Maximum single dose 4 g		
	IV	–	80 mg/kg	Once daily
		Notes: In severe infections and meningitis, infuse over at least 30 minutes		

CETIRIZINE HYDROCHLORIDE		Birth to 1 year	1–2 years	2–6 years	6–18 years	
Symptoms of allergy in association with anaphylaxis	Oral	–	250 mcg/kg	2.5 mg	5 mg	Twice daily
		Notes: Can substitute twice daily dose with: *2–6-year-olds*: 5 mg once daily *6–18-year-olds*: 10 mg once daily Renal impairment: use half normal dose if estimated glomerular filtration rate is <30 ml/min/1.73 m^2				

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
CHLORPHENAMINE (CHLORPHENIRAMINE)		**1 month to 2 years**	**2–12 years**	**12–18 years**	
Antihistamine with sedative and antimuscarinic effects	IV, IM or SC	*<1 year* (dose by weight): 250 mcg/kg *>1 year* (dose by age): 2.5–5 mg	*2–5 years*: 2.5–5 mg *6–12 years*: 5–10 mg	10–20 mg	Single dose
		Notes: Can be repeated up to 4 times in 24 hours if necessary Note adult maximum *daily dose* is 40 mg In anaphylaxis, administer IV, as SC or IM rarely acts quicker than oral dosing			
CODEINE PHOSPHATE		**Birth to 18 years**			
Mild to moderate pain	Oral/ IM	0.5–1 mg/kg Notes: Max. 240 mg daily Contraindicated in paralytic ileus *Warning*: avoid in acute respiratory depression *Caution*: renal impairment. Hepatic impairment may precipitate coma			Repeat after 4–6 hours
DANTROLENE		**1 month to 18 years**			
Malignant hyperthermia	IV bolus	2–3 mg/kg initially Notes: Repeat with 1 mg/kg as required at 5–10-minute intervals to a maximum cumulative dose of 10 mg/kg			Single dose
DESFERRIOXAMINE MESILATE		**1 month to 18 years**			
Acute iron poisoning	IV infusion	Initially 15 mg/kg/h Reducing after 4–6 hours as indicated Notes: If shocked, hypotensive or seriously ill, administer IV Decreased rate of administration after 4–6 hours to ensure that total maximum dose does not exceed 80 mg/kg/day Continue until serum iron is less than total iron-binding capacity Use with caution in patients with renal impairment			Continuous

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
DEXAMETHASONE		**1 month to 18 years**	
Croup	Oral	150 mcg/kg	Twice daily
		Notes: No definitive standard dose has been agreed in the UK. Suggested maximum single dose of 12 mg	
Short course to relieve symptoms of brain tumour	IV or oral	500 mcg/kg	Twice daily
		Notes: Can also be used to reduce oedema around tumours compressing nerves	

DIAMORPHINE		Birth to 1 month	1 month to 18 years	
Control of severe pain	Intranasal	Not recommended	0.1 mg/kg	Single dose
		Notes: Dilute with saline to volume of 0.2 ml Monitor closely for at least 30 minutes and repeat if needed; may repeat 6 hourly Avoid in acute respiratory depression Naloxone is an antidote Use with caution in head injury		

DIAZEPAM		Birth to 18 years	
Treatment of status epilepticus	Rectal	0.5 mg/kg	Single dose
		Notes: If needed, repeat after 5 minutes Parenteral and rectal use can depress respiration Caution with other central nervous system depressants	
In place of lorazepam where this is not available	IV or IO	0.25 mg/kg	

DICLOFENAC		1 month to 2 years	2–18 years	
Non-steroidal anti-inflammatory drug (NSAID)	Oral or rectal	*<6 months*: not recommended *>6 months*: 300 mcg to 1 mg/kg	300 mcg to 1 mg/kg	3 times daily
		Notes: Up to a maximum of 150 mg per day Caution where there is a history of hypersensitivity and in dehydration (risk of renal failure)		

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
DOBUTAMINE		**Birth to 18 years**	
Provides inotropic support in the treatment of low output cardiac failure, e.g. in septicaemia	IV infusion	5–20 mcg/kg/min Notes: Dose can be increased up to a maximum of 40 mcg/kg/min in older children, if necessary (20 mcg/kg/min in newborn infants) but side effects are more likely at this higher dose	Continuous

DOPAMINE HYDROCHLORIDE		Birth to 1 month	1 month to 18 years	
Treatment of low cardiac output states	IV infusion	Start at 3 mcg/kg/min, increasing as clinically indicated to a max. of 20 mcg/kg/min	5–20 mcg/kg/min	Continuous
		Notes: Direct inotropic effect but vasoconstriction may occur at higher doses		

ERYTHROMYCIN		Birth to 1 month	1 month to 2 years	2–12 years	12–18 years	
Upper and lower respiratory tract infections	Oral or IV	12.5 mg/kg	–	–	–	4 times daily
	Oral	–	125 mg	*2–8 years*: 250 mg *9–12 years*: 500 mg	500 mg	4 times daily
		Notes: Doses can be doubled in severe infections Maximum single dose 1 g				

FLECAINIDE ACETATE		Birth to 18 years	
Treatment of resistant re-entry SVT, ventricular ectopics or ventricular tachycardia	IV bolus	2 mg/kg Notes: Give over at least 10 minutes with ECG monitoring Avoid in patients with pre-existing heart block Maximum dose 150 mg	Single dose

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
FLUCLOXACILLIN		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Treatment of infections due to Gram-positive organisms (antistaphylococcal)	Oral or IV	*<7 days*: 25–50 mg/kg	–	–	–	Twice daily
		7–21 days: 25–50 mg/kg	–	–	–	3 times daily
		>21 days: 25–50 mg/kg	–	–	–	4 times daily
		Notes: Dose may be increased to 100 mg/kg per dose IV in severe infection (meningitis, cerebral abscess, staphylococcal osteitis) Oral route only recommended for minor infection				
	IV or IM bolus	–	12.5–25 mg/kg			4 times daily
		Notes: Maximum single dose 1 g Dose may be doubled in severe infection, maximum single dose 2 g				
	Oral	–	*<1 year*: 62.5 mg	*<5 years*: 125 mg	250 mg	4 times daily
			>1 year: 125 mg	*>5 years*: 250 mg		
		Notes: Doses may be doubled in severe infection				

FLUMAZENIL		Birth to 1 month	1 month to 2 years	2–12 years	12–18 years	
Reversal of acute benzodiazepine overdosage	IV bolus over 15 seconds	10 mcg/kg	10 mcg/kg (max. dose 200 mcg)		200 mcg	Single dose
		Notes: Initial dose as shown If the desired effect is not achieved – repeat at 1-minute intervals to a max. total dose of 40 mcg/kg (2 mg max. dose in 12–18-year-olds)				
	IV infusion	2–10 mcg/kg/h (max. dose 400 mcg/h)			100–400 mcg/h	Continuous
		Notes: This should be individually adjusted to achieve the desired level of arousal There is limited experience of the use of flumazenil in children				

INDICATION	ROUTE	AGE/WEIGHT		FREQUENCY
FRUSEMIDE (FUROSEMIDE)		**Birth to 12 years**	**12–18 years**	
To induce diuresis in cardiac or renal failure or fluid overload; hypertension	IV bolus	500 mcg to 1 mg/kg	20–40 mg	Single dose
		Notes: Single doses up to 4 mg/kg have been used. Dose can be repeated every 8 hours		

GLUCAGON		Birth to 1 month	1 month to 2 years	2–18 years	
Severe insulin-induced hypoglycaemia in the treatment of diabetes	IM, SC	Not recommended	500 mcg	500 mcg to 1 mg (*<25 kg*: 500 mcg *>25 kg*: 1 mg)	Single dose
		Notes: Should be effective within 10 minutes Only use when IV glucose is difficult or impossible to administer. If not, give IV glucose 5–10% instead			

HYDROCORTISONE		Birth to 1 month	1 month to 12 years	12–18 years	
Anaphylaxis and emergency treatment of severe acute asthma	IV bolus, IM or IO	2.5 mg/kg	4 mg/kg (max. dose 100 mg)	100–300 mg	Single dose
		Then: 2 mg/kg	Then: 2–4 mg/kg	Then: 100–300 mg	4 times daily
		Notes: Maintenance dose may be repeated if necessary every 6 hours. May be given by IO route if IV is not possible			

IBUPROFEN		1 month to 2 years	2–12 years	12–18 years	
Pyrexia, mild to moderate pain	Oral dose by weight	5 mg/kg		–	3–4 times daily
	Oral dose by age	*1–2 years*: 50 mg	*3–7 years*: 100 mg *8–12 years*: 200 mg	200–600 mg	3–4 times daily
		Notes: Maximum of 20 mg/kg/day up to 2.4 g/day Avoid where there is a history of hypersensitivity and in dehydration (risk of renal failure)			

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
INSULIN		**Birth to 18 years**	
Primary treatment for patients with type 1 and 2 diabetes uncontrolled by other means	IV infusion in ketoacidosis	0.05–0.1 units/kg/h Notes: Adjust dose according to blood glucose level	Continuous

IPRATROPIUM		Birth to 1 month	1 month to 2 years	2–12 years	12–18 years	
Treatment of chronic reversible airways obstruction. May be used with a β_2-agonist in the treatment of severe, acute asthma	Nebulised	25 mcg/kg	125 mcg	250 mcg	500 mcg	Single dose
		Notes: Can be repeated every 20–30 minutes in the first 2 hours in acute severe asthma. Reduce dose frequency as clinical improvement occurs				

LABETALOL		Birth to 1 month	1 month to 12 years	12–18 years	
Hypertension and hypertensive crises	IV bolus	–	250–500 mcg/kg	50 mg	Single dose
		Notes: Loading dose			
	IV infusion	500 mcg/kg/h up to a max. of 4 mg/kg/h	1–3 mg/kg/h	120 mg/h	Continuous
		Notes: Start at low dose and titrate according to response, until the blood pressure has been reduced to an acceptable level Avoid in asthma, heart failure and heart block			

INDICATION	ROUTE	AGE/WEIGHT		FREQUENCY
LIDOCAINE		**Birth to 12 years**	**12–18 years**	
Antiarrhythmic VF or pulseless tachycardia Local anaesthetic	IV/IO	1 mg/kg (max. dose 100 mg)	50–100 mg	Single dose
		Notes: Repeat every 5 minutes if needed to a total maximum of 3 mg/kg In the 12–18-year age group give 50 mg in lighter patients or those whose circulation is impaired		
	IV infusion	Then: 600 mcg/kg to 3 mg/kg/h	4 mg/min for 30 minutes then 2 mg/min for 2 hours then 1 mg/min*	Continuous
		*In the 12–18-year group: reducing concentration further if infusion is continued beyond 24 hours *Maintenance dosing*: ECG monitoring with infusion		
	Local infiltration	Up to 3 mg/kg	Up to 200 mg	Single dose
		Notes: No more often than every 4 hours Use fine needles (27–29 gauge) It is less painful if buffered before use with 8.4% sodium bicarbonate 1 ml to every 10 ml lidocaine 1%		
	Intraurethral	3–4 mg/kg	–	Single dose
		Notes: Use prior to urinary catheterisation. Warm the solution to body temperature and inject it very slowly to reduce local stinging		

LORATADINE		Birth to 2 years	2–12 years: Less than 30 kg	2–12 years: Over 30 kg	12–18 years	
Symptoms of allergy in association with anaphylaxis	Oral	–	5 mg	10 mg	10 mg	Once daily
		Notes: Hepatic impairment: reduce dose frequency to alternate days in severe impairment				

LORAZEPAM		Birth to 12 years	12–18 years	
Status epilepticus	IV, rectal or sublingual	100 mcg/kg (max. dose 4 g)	4 mg	Single dose
		Notes: Generally given as a single dose; may be repeated once if initial dose is ineffective Limited experience in neonates May cause apnoea Flumazenil is an antidote		

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
MAGNESIUM SULPHATE		**Birth to 1 month**	**1 month to 2 years**	**2–18 years**	
Hypomagnesaemia in septicaemia	IV		0.2 ml/kg 50% $MgSO_4$ over 30 minutes (max. 10 ml)		Single dose
		Notes: Repeat later if serum magnesium remains low			
Treatment of asthma	IV	Not recommended	Limited experience	*Over 2 years*: 40 mg/kg	Single dose over 20 minutes
		Notes: Has been used in infants but experience is limited. Maximum of 2 g			
Treatment of torsades de pointes	IV	Not recommended	25–50 mg/kg		Single dose
		Notes: Maximum of 2 g			

MANNITOL		Birth to 18 years	
Treatment of oedematous states, including ascites and treatment of raised intracranial pressure	IV infusion over 30 minutes	250–500 mg/kg (1.25–2.5 ml/kg of 20% solution) Notes: Cerebral and ocular oedema May be repeated once or twice after an interval of 4–8 hours if necessary (if serum osmolality <310 mOsm/l)	Single dose

MIDAZOLAM		Birth to 1 month	1 month to 2 years	2–12 years	12–18 years	
Status epilepticus	Buccal/ intranasal	– –	0.5 mg/kg *<6 months* (dose by weight): 300 mcg/kg *>6 months* (dose by age): 2.5 mg	0.5 mg/kg *1–4 years*: 5 mg *5–9 years*: 7.5 mg *>10 years*: 10 mg	0.5 mg/kg 10 mg	Single dose Single dose
		Notes: Buccal administration is the preferred route over intranasal administration. The parenteral preparation can be used for this route The dose by weight for the buccal route is 0.5 mg/kg from 6 months. Maximum dose 10 mg				

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
MORPHINE		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Control of severe pain	IV infusion	*Pre-term*: 25–50 mcg/kg	–	–	–	Single dose Loading dose
		Then: 5 mcg/kg/h	–	–	–	Continuous
		Term: 50–100 mcg/kg	–	–	–	Single dose Loading dose
		Then: 10–20 mcg/kg/h	–	–	–	Continuous
	IV bolus	–	100–200 mcg/kg		2.5–10 mg	*<6 months*: up to 4 times in 24 hours *>6 months*: up to 6 times in 24 hours
		Notes: Respiratory monitoring is mandatory Give IV over at least 5–10 minutes <1 year: use the lower stated dose and consider oxygen saturation monitoring				
	IV infusion	–	10–30 mcg/kg/h			Continuous
			<6 months: initial rate is 10 mcg/kg/h			
			>6 months: initial rate is 20 mcg/kg/h			
		Notes: Use IV bolus as starting dose first 1 mg/kg body weight in 50 ml saline, infused at 1 ml/h = 20 mcg/kg/h				
	Oral	–	*<1 year*: 80 mcg/kg	200–500 mcg/kg	10–15 mg	Up to 6 times in 24 hours
			>1 year: 200–400 mcg/kg			
		Notes: Starting doses should be reviewed regularly and adjusted according to the patient's response				

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
NALOXONE		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Reversal of opioid-induced central and respiratory depression	IV infusion	10 mcg/kg	–	–	–	Continuous
	IM	200 mcg (60 mcg/kg)	–	–	–	Single dose
		Notes: Use 400 mcg/ml naloxone preparation Gradual onset of action (3–4 minutes) but the effect is prolonged				
	IV bolus	–	10 mcg/kg		10 mcg/kg (max. dose 800 mcg)	Single dose
		–	Then: 100 mcg/kg (max. dose 2 mg)		Then: 2 mg	Single dose
		Notes: Initial dose followed by another dose if no response. Due to short half-life of naloxone, repeat doses as necessary to maintain opioid reversal Observe for recurrence of central nervous system and respiratory depression				
	IV infusion	–	5–20 mcg/kg/h		Infuse a solution of 4 mcg/ml at a rate adjusted according to response	Continuous

Specifically indicated for the reversal of respiratory depression in a newborn infant whose mother has received narcotics within 4 hours of delivery. It is generally preferred to give an IM injection for a prolonged effect.

Do not administer to newborns whose mothers are suspected of narcotic abuse, as a withdrawal syndrome may be precipitated. *Always* establish and maintain adequate ventilation before administration of naloxone.

NIFEDIPINE		Birth to 1 month	1 month to 18 years	
Hypertensive crisis	Oral	–	250–500 mcg/kg	Single dose
		Notes: Bite the capsule releasing the contents into the mouth and then swallow. Aspirate liquid from the capsule for young children		

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
PARACETAMOL		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Analgesic/ antipyretic	Oral loading dose	20 mg/kg	20 mg/kg		1 g	Single dose
	Oral maintenance dose	*≤32 weeks gestation*: 15 mg/kg	*32 weeks gestation to 12 years*: dose by weight, 20 mg/kg	500 mg to 1 g	1 g	4–6 hourly *<32 weeks gestation*: 12 hourly *32 weeks gestation to 1 month*: 8 hourly
		Notes: Maximum *daily* dose 60 mg/kg (total 4 g) Preterm 28–32 weeks max. daily dose 30 mg/kg				
	Rectal loading dose	*<32 weeks gestation*: 20 mg/kg	*1–3 months:* 30 mg/kg *>3 months:* 40 mg/kg		1 g	Single dose
	Rectal maintenance dose	15 mg/kg	*32 weeks gestation to 12 years:* dose by weight, 20 mg/kg	500 mg to 1 g	1 g	4–6 hourly *<32 weeks gestation*: 12 hourly *32 weeks gestation to 1 month*: 8 hourly
		Notes: Maximum *daily* dose 60 mg/kg (total 4 g) Preterm 28–32 weeks max. daily dose 30 mg/kg				
	IV	*<10 kg*: 7.5 mg/kg	*10–50 kg:* 15 mg/kg *>50 kg:* 1 g	–	–	4–6 hourly *<32 weeks gestation*: 12 hourly *32 weeks gestation to 1 month*: 8 hourly
		Notes: <10 kg: max. *daily* dose 30 mg/kg 10–50 kg: max. *daily* dose 60 mg/kg >50 kg: max. *daily* dose 4 g				

INDICATION	ROUTE	AGE/WEIGHT			FREQUENCY
PARALDEHYDE		**Birth to 1 month**	**1 month to 12 years**	**12–18 years**	
Status epilepticus	Rectal	0.4 ml/kg		5–10 ml	Single dose
		Notes: Doses stated in ml/kg or as ml of paraldehyde Dilute with an equal volume of olive or sunflower oil before administration, or if using a ready-prepared 'special', remember that it is already diluted and dose accordingly			
	IM	0.2 ml/kg	0.1–0.15 ml/kg	5–10 ml	Single dose
		Notes: IM use should be avoided whenever possible May cause pain and sterile abscess Maximum 5 ml (1 ml in neonates) at one site Consider adding hyaluronidase 150 units			
PHENOBARBITAL (PHENOBARBITONE)		**Birth to 12 years**		**12–18 years**	
Status epilepticus Respiratory depression especially when used with benzodiazepines	IV slow bolus	20 mg/kg		20 mg/kg	Single
		Notes: Loading dose			
		Then: 2.5–5 mg/kg		Then: 300 mg dose	Twice daily (once daily in neonatal period)
		Notes: Maintenance			
PHENYTOIN		**Birth to 1 month**	**1 month to 12 years**	**12–18 years**	
Antiepileptic	IV	20 mg/kg	20 mg/kg		Single dose
		Notes: Loading dose (over 20 minutes) Monitor ECG and blood pressure			
		2.5–5 mg/kg		–	Twice daily
		–		100 mg	3–4 times a day
		Notes: Usual maintenance dose over 20 minutes			
Antiarrhythmic	IV	–	20 mg/kg		Single dose

Reduce dose in liver disease.

INDICATION	ROUTE	AGE/WEIGHT		FREQUENCY
POTASSIUM CHLORIDE		**Birth to 1 month**	**1 month to 18 years**	
Acute hypokalaemia	IV infusion	–	0.1–0.25 mmol/kg/h	Continuous
		Notes: Always check the dose carefully, as an overdose can be rapidly fatal; dilute with at least 50 times its volume and mix well Restrict to critical care areas, store in a locked cupboard and document as for controlled drugs Recheck the potassium level after 3 hours		

PREDNISOLONE		Birth to 1 month	1 month to 12 years	12–18 years	
Acute asthma	Oral	–	1 mg/kg (max. dose 40 mg)		Once daily
		Notes: Treat for 1–5 days and then stop (no need to taper doses)			
Suppression of inflammatory and allergic disorders	Oral	–	1–2 mg/kg		Once daily
		Notes: The daily dose can be given in 2–3 divided doses if necessary. Consider alternate day treatment in long term			
Croup requiring intubation	Oral	–	1 mg/kg	–	Twice daily
		Notes: Start within 24 hours of intubation, continuing until 24 hours after extubation			

PROPRANOLOL		Birth to 12 years	12–18 years	
Dysrhythmias	IV bolus	25–50 mcg/kg	1 mg	Single dose
		Notes: Repeat the injection as needed up to 4 times daily. Give injection slowly over 5 minutes under ECG control		

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
QUININE		**Birth to 18 years**	
Treatment of *Plasmodium falciparum* malaria	IV infusion at least over 4 hours	20 mg/kg (max. 1.4 g)	Single loading dose
		Notes: For seriously ill patients or those unable to take tablets	
		Then: 10 mg/kg (max. 700 mg)	Then after 8–12 hours maintenance dose
		Notes: Maintenance dose can be repeated every 8–12 hours but change to oral therapy as soon as possible	
		Then: 5–7 mg/kg	Maintenance dose after 48 hours IV therapy
		Notes: If IV therapy is required after 48 hours, use this maintenance dose every 8 hours	

Risk of arrhythmias with amiodarone and flecainide. Side effects are common: tinnitus, headache, visual disturbance and hypoglycaemia. Use glucose 5% to dilute to a concentration of 2 mg/ml (max. 30 mg/ml in fluid restriction).
Monitor ECG and blood sugar.

SALBUTAMOL		Birth to 1 month	1 month to 2 years	2–18 years	
Treatment of asthma	Aerosol inhaler	–	Up to 1 mg		Single dose
		Notes: Asthma reliever given as required; 1–2 hourly initially, then reduce frequency to 4–6 hourly; 1000 mcg = 10 sprays (each of 100 mcg)			
	Nebuliser solution	*<5 years:* 2.5 mg *>5 years:* 5 mg			Single dose
		Notes: Asthma reliever given as required according to severity and response			
	IV bolus over 5 minutes	5 mcg/kg		15 mcg/kg (max. 250 mcg)	Single dose
		Notes: Status asthmaticus: maximum concentration 50 mcg in 1 ml			
	IV infusion	1–5 mcg/kg/min			Continuous
		Notes: Status asthmaticus: doses up to 10 mcg/kg/min have been used Solution compatible with potassium but not with aminophylline			
Renal hyperkalaemia	IV bolus	4 mcg/kg			Single dose
		Notes: Repeat if necessary			
	Nebuliser	2.5–5 mg			Single dose
		Notes: Repeat if necessary			

INDICATION	ROUTE	AGE/WEIGHT	FREQUENCY
SODIUM BICARBONATE		**Birth to 18 years**	
Resuscitation	Slow IV	1 ml/kg of 8.4% initially if indicated Followed by 0.5 ml/kg of 8.4% if needed	
Metabolic acidosis	Slow IV	1–2 mmol/kg Notes: Only after attention to ventilation and perfusion Always infuse slowly If acidosis is persistent consider inborn errors or toxins	
Renal hyperkalaemia	Slow IV	1 mmol/kg Notes: Dose adjusted according to plasma bicarbonate level	Single dose
SODIUM CHLORIDE 3%		**Birth to 18 years**	
Management of raised intracranial pressure	IV	3–5 ml/kg IV over 15 minutes	Single dose
SODIUM NITROPRUSSIDE		**Birth to 18 years**	
Hypertensive crisis	IV infusion	500 nanograms/kg/min Notes: Initial dose: increase in increments of 200 nanograms/kg/min as necessary to a maximum of 8 mcg/kg/min Use only with expert advice	Continuous

INDICATION	ROUTE	AGE/WEIGHT				FREQUENCY
TERBUTALINE		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**12–18 years**	
Relief of bronchospasm in bronchial asthma	Nebulised	–	2.5–5 mg	*<5 years*: 2.5–5 mg *>5 years*: 5–10 mg	10 mg	Single dose
		Notes: Reliever doses are repeated as required				
VERAPAMIL		**Birth to 1 month**	**1 month to 2 years**	**2–12 years**	**2–18 years**	
Treatment for SVT (adenosine first line)	Slow IV bolus	–	*<1 year*: – *>1 year*: 100–300 mcg/kg (max. 5 mg)	100–300 mcg/kg (max. 5 mg)	5 mg	Single dose over 2–3 minutes
		Notes: ECG and blood pressure monitoring required Dose may be repeated after 30 minutes if necessary. Many cases are controlled by doses at the lower end of the range Caution in liver disease. Do not use with β-blockers Use only with expert advice				

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