European Resuscitation Council Guidelines for Resuscitation 2005
Section 6. Paediatric life support

Dominique Biarent, Robert Bingham, Sam Richmond, Ian Maconochie, Jonathan Wyllie, Sheila Simpson, Antonio Rodriguez Nunez, David Zideman

Introduction

The process

The European Resuscitation Council (ERC) issued guidelines for paediatric life support (PLS) in 1994, 1998 and 2000.[1—4] The last edition was based on the International Consensus on Science published by the American Heart Association in collaboration with the International Liaison Committee on Resuscitation (ILCOR), undertaking a series of evidence-based evaluations of the science of resuscitation which culminated in the publication of the Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care in August 2000.[5,6] This process was repeated in 2004/2005, and the resulting Consensus on Science and Treatment Recommendations were published simultaneously in *Resuscitation*, *Circulation* and *Pediatrics* in November 2005.[7,8] The PLS Working Party of the ERC has considered this document and the supporting scientific literature, and has recommended changes to the ERC PLS Guidelines. These are presented in this paper.

Guidelines changes

The approach to changes has been to alter the guidelines in response to convincing new scientific evidence and, where possible, to simplify them in order to assist teaching and retention. As before, there remains a paucity of good-quality evidence on paediatric resuscitation specifically and some conclusions have had to be drawn from animal work and extrapolated adult data.

The current guidelines have a strong focus on simplification based on the knowledge that many children receive no resuscitation at all because rescuers fear doing harm. This fear is fuelled by the knowledge that resuscitation guidelines for children are different. Consequently, a major area of study was the feasibility of applying the same guidance for all adults and children. Bystander resuscitation improves outcome significantly,[9,10] and there is good evidence from paediatric animal models that even doing chest compressions or expired air ventilation alone may be better than doing nothing at all.[11] It follows that outcomes could be improved if bystanders, who would otherwise do nothing, were encouraged to begin resuscitation, even if they do not follow an algorithm targeted specifically at children. There are, however, dis-

tinct differences between the predominantly adult arrest of cardiac origin and asphyxial arrest, which is most common in children,[12] so a separate paediatric algorithm is justified for those with a duty to respond to paediatric emergencies (usually healthcare professionals), who are also in a position to receive enhanced training.

Compression:ventilation ratios

The ILCOR treatment recommendation was that the compression:ventilation ratio should be based on whether one or more than one rescuers were present. ILCOR recommends that lay rescuers, who usually learn only single rescuer techniques, should be taught to use a ratio of 30 compressions to 2 ventilations, which is the same as the adult guidelines and enables anyone trained in BLS techniques to resuscitate children with minimal additional information. Two or more rescuers with a duty to respond should learn a different ratio (15:2), as this has been validated by animal and manikin studies.[13–17] This latter group, who would normally be healthcare professionals, should receive enhanced training targeted specifically at the resuscitation of children. Although there are no data to support the superiority of any particular ratio in children, ratios of between 5:1 and 15:2 have been studied in manikins, and animal and mathematical models, and there is increasing evidence that the 5:1 ratio delivers an inadequate number of compressions.[14,18] There is certainly no justification for having two separate ratios for children aged greater or less than 8 years, so a single ratio of 15:2 for multiple rescuers with a duty to respond is a logical simplification.

It would certainly negate any benefit of simplicity if lay rescuers were taught a different ratio for use if there were two of them, but those with a duty to respond can use the 30:2 ratio if they are alone, particularly if they are not achieving an adequate number of compressions because of difficulty in the transition between ventilation and compression.

Age definitions

The adoption of single compression:ventilation ratios for children of all ages, together with the change in advice on the lower age limit for the use of automated external defibrillators (AEDs), renders the previous guideline division between children above and below 8 years of age unnecessary. The differences between adult and paediatric resuscitation are based largely on differing aetiology, as primary cardiac arrest is more common in adults whereas children usually suffer from sec-

ondary cardiac arrest. The onset of puberty, which is the physiological end of childhood, is the most logical landmark for the upper age limit for use of paediatric guidance. This has the advantage of being simple to determine, in contrast to an age limit in years, as age may be unknown at the start of resuscitation. Clearly, it is inappropriate and unnecessary to establish the onset of puberty formally; if rescuers believe the victim to be a child they should use the paediatric guidelines. If a misjudgement is made and the victim turns out to be a young adult, little harm will accrue, as studies of aetiology have shown that the paediatric pattern of arrest continues into early adulthood.[19] An infant is a child under 1 year of age; a child is between 1 year and puberty. It is necessary to differentiate between infants and older children, as there are some important differences between these two groups.

Chest compression technique

The modification to age definitions enables a simplification of the advice on chest compression. Advice for determining the landmarks for infant compression is now the same as for older children, as there is evidence that the previous recommendation could result in compression over the upper abdomen.[20] Infant compression technique remains the same: two-finger compression for single rescuers and two-thumb, encircling technique for two or more rescuers,[21–25] but for older children there is no division between the one- or two-hand technique.[26] The emphasis is on achieving an adequate depth of compression with minimal interruptions, using one or two hands according to rescuer preference.

Automated external defibrillators

Case reports published since International Guidelines 2000 have reported safe and successful use of AEDs in children less than 8 years of age.[27,28] Furthermore, recent studies have shown that AEDs are capable of identifying arrhythmias in children accurately and that, in particular, they are extremely unlikely to advise a shock inappropriately.[29,30] Consequently, advice on the use of AEDs has been revised to include all children aged greater than 1 year.[31] Nevertheless, if there is any possibility that an AED may need to be used in children, the purchaser should check that the performance of the particular model has been tested against paediatric arrhythmias.

Many manufacturers now supply purpose-made paediatric pads or programmes, which typically attenuate the output of the machine to 50–75 J.[32]

These devices are recommended for children aged 1—8 years.[33,34] If no such system or manually adjustable machine is available, an unmodified adult AED may be used in children older than 1 year.[35] There is currently insufficient evidence to support a recommendation for or against the use of AEDs in children aged less than 1 year.

Manual defibrillators

The 2005 Consensus Conference treatment recommendation for paediatric ventricular fibrillation (VF) or paediatric pulseless ventricular tachycardia (VT) is to defibrillate promptly. In adult ALS, the recommendation is to give a single shock and then resume CPR immediately without checking for a pulse or reassessing the rhythm (see Section 3). As a consequence of this single-shock strategy, when using a monophasic defibrillator in adults a higher initial energy dose than used previously is recommended (360 J versus 200 J) (see Section 3). The ideal energy dose for safe and effective defibrillation in children is unknown, but animal models and small paediatric series show that doses larger than 4 J kg^{-1} defibrillate effectively with negligible side effects.[27,34,36,37] Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic shocks.[33,34,37—40] For simplicity of sequence and consistency with adult BLS and ALS, we recommend a single-shock strategy using a non-escalating dose of 4 J kg^{-1} (monophasic or biphasic) for defibrillation in children.

Foreign-body airway obstruction sequence

The guidance for managing foreign-body airway obstruction (FBAO) in children has been simplified and brought into closer alignment to the adult sequence. These changes are discussed in detail at the end of this section.

In the following text the masculine includes the feminine and 'child' refers to both infants and children unless noted otherwise.

6a Paediatric basic life support

Sequence of action

Rescuers who have been taught adult BLS and have no specific knowledge of paediatric resuscitation may use the adult sequence, with the exception that they should perform 5 initial breaths followed by approximately 1 min of CPR before they go for help (Figure 6.1; also see adult BLS guideline).
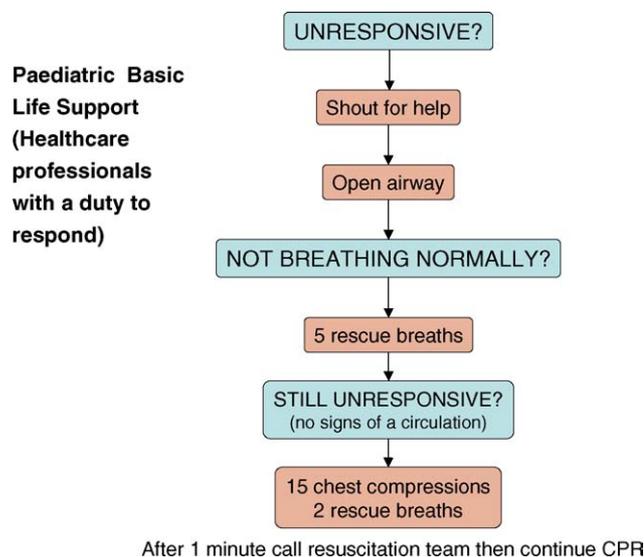

Paediatric Basic Life Support (Healthcare professionals with a duty to respond)

UNRESPONSIVE?
↓
Shout for help
↓
Open airway
↓
NOT BREATHING NORMALLY?
↓
5 rescue breaths
↓
STILL UNRESPONSIVE?
(no signs of a circulation)
↓
15 chest compressions
2 rescue breaths

After 1 minute call resuscitation team then continue CPR

Figure 6.1 Paediatric basic life support algorithm.

The following sequence is to be observed by those with a duty to respond to paediatric emergencies (usually health professionals).

1. Ensure the safety of rescuer and child.
2. Check the child's responsiveness.
   - Gently stimulate the child and ask loudly: ''Are you all right?''
   - Do not shake infants or children with suspected cervical spinal injuries.

3a If the child responds by answering or moving
   - leave the child in the position in which you find him (provided he is not in further danger)
   - check his condition and get help if needed
   - reassess him regularly

3b If the child does not respond
   - shout for help;
   - open the child's airway by tilting the head and lifting the chin, as follows:
     o initially with the child in the position in which you find him, place your hand on his forehead and gently tilt his head back;
     o at the same time, with your fingertip(s) under the point of the child's chin, lift the chin. Do not push on the soft tissues under the chin as this may block the airway;
     o if you still have difficulty in opening the airway, try the jaw thrust method. Place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward;
     o both methods may be easier if the child is turned carefully onto his back.

If you suspect that there may have been an injury to the neck, try to open the airway using chin lift or jaw thrust alone. If this is unsuccessful, add head tilt a small amount at a time until the airway is open.

4. Keeping the airway open, look, listen and feel for normal breathing by putting your face close to the child's face and looking along the chest.

- Look for chest movements.
- Listen at the child's nose and mouth for breath sounds.
- Feel for air movement on your cheek.

Look, listen and feel for no more than 10 s before deciding.

5a If the child is breathing normally
  - turn the child on his side into the recovery position (see below)
  - check for continued breathing

5b If the child is *not* breathing or is making agonal gasps (infrequent, irregular breaths)
  - carefully remove any obvious airway obstruction;
  - give five initial rescue breaths;
  - while performing the 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 a circulation, which will be described later.

Rescue breaths for a child over 1 year are performed as follows (Figure 6.2).
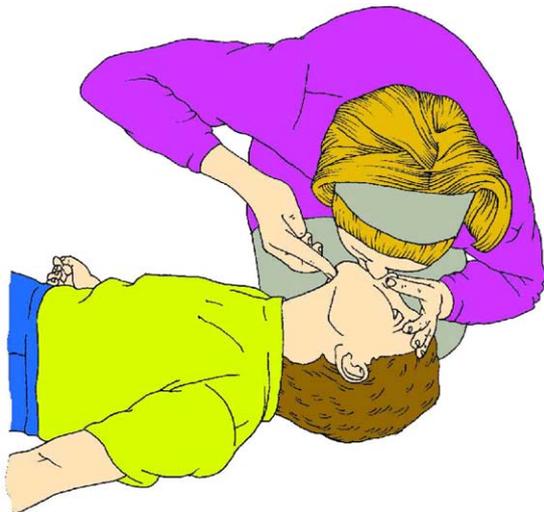
- Ensure head tilt and chin lift.

- Pinch the soft part of the nose closed with the index finger and thumb of your hand on his forehead.
- Open his mouth a little, but maintain the chin upwards.
- Take a breath and place your lips around the mouth, making sure that you have a good seal.
- Blow steadily into the mouth over about 1−1.5 s, watching for chest rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air is expelled.
- Take another breath and repeat this sequence five times. Identify effectiveness by seeing that the child's chest has risen and fallen in a similar fashion to the movement produced by a normal breath.

Rescue breaths for an infant are performed as follows (Figure 6.3).

- Ensure a neutral position of the head and a chin lift.
- Take a breath and cover the mouth and nasal apertures of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot be covered in the older infant, the rescuer may attempt to seal only the infant's nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape).
- Blow steadily into the infant's mouth and nose over 1−1.5 s, sufficient to make the chest visibly rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air is expelled.
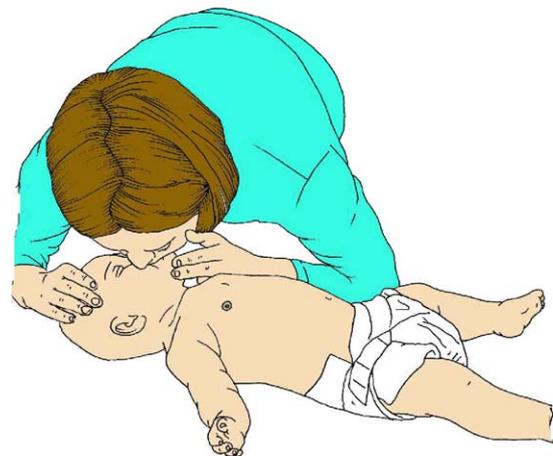- Take another breath and repeat this sequence five times.

Figure 6.2 Mouth-to-mouth ventilation— child. © 2005 ERC.

Figure 6.3 Mouth-to-mouth and nose ventilation— infant. © 2005 ERC.

If you have difficulty achieving an effective breath, the airway may be obstructed.

- Open the child's mouth and remove any visible obstruction. Do not perform a blind finger sweep.
- Ensure that there is adequate head tilt and chin lift but also that the neck is not over-extended.
- If head tilt and chin lift have not opened the airway, try the jaw thrust method.
- Make up to five attempts to achieve effective breaths; if still unsuccessful, move on to chest compressions.

6. Assess the child's circulation. Take no more than 10 s to
   - look for signs of a circulation. This includes any movement, coughing or normal breathing (not agonal gasps, which are infrequent, irregular breaths);
   - check the pulse (if you are a health care provider) but ensure you take no more than 10 s.

If the child is aged over 1 year, feel for the carotid pulse in the neck.

In an infant, feel for the brachial pulse on the inner aspect of the upper arm.

7a If you are confident that you can detect signs of a circulation within 10 s
   - continue rescue breathing, if necessary, until the child starts breathing effectively on his own
   - turn the child onto his side (into the recovery position) if he remains unconscious
   - re-assess the child frequently
7b If there are no signs of a circulation, or no pulse or a slow pulse (less than 60 min^{-1} with poor perfusion), or you are not sure
   - start chest compressions
   - combine rescue breathing and chest compressions

Chest compressions are performed as follows. For all children, compress the lower third of the sternum. To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger's breadth above this; the compression should be sufficient to depress the sternum by approximately one third of the depth of the chest. Release the pressure and repeat at a rate of about 100 min^{-1}. After 15 compressions, tilt the head, lift the chin, and give two effective breaths. Continue compressions and breaths in a ratio of 15:2. Lone rescuers may use a ratio of 30:2, particularly if having difficulty with the transition between compression and ventilation. Although the
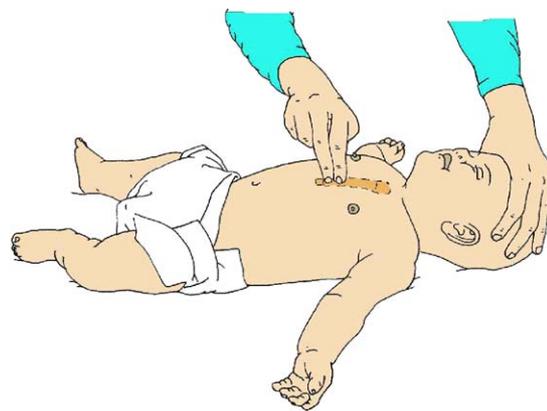

Figure 6.4 Chest compression — infant. © 2005 ERC.

rate of compressions will be 100 min^{-1}, the actual number delivered per minute will be less than 100 because of pauses to give breaths. The best method for compression varies slightly between infants and children.

To perform chest compression in infants, the lone rescuer compresses the sternum with the tips of two fingers (Figure 6.4). If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower third of the sternum (as above) with the tips pointing towards the infant's head. Spread the rest of both hands with the fingers together to encircle the lower part of the infant's rib cage with the tips of the fingers supporting the infant's back. Press down on the lower sternum with the two thumbs to depress it approximately one third of the depth of the infant's chest.

To perform chest compression in children over 1 year of age, place the heel of one hand over the lower third of the sternum (as above) (Figures 6.5 and 6.6). Lift the fingers to ensure that pressure is not applied over the child's ribs. Position yourself vertically above the victim's chest and, with your arm straight, compress the sternum to depress it by approximately one third of the depth of the chest. In larger children or for small rescuers, this is achieved most easily by using both hands with the fingers interlocked.

8. Continue resuscitation until
   - the child shows signs of life (spontaneous respiration, pulse, movement)
   - qualified help arrives
   - you become exhausted

When to call for assistance

It is vital for rescuers to get help as quickly as possible when a child collapses.

- When more than one rescuer is available, one starts resuscitation while another rescuer goes for assistance.
- If only one rescuer is present, undertake resuscitation for about 1 min before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child while summoning help.
- The only exception to performing 1 min of CPR before going for help is in the case of a child with a witnessed, sudden collapse when the rescuer is alone. In this case cardiac arrest is likely to be arrhythmogenic in origin and the child will need defibrillation. Seek help immediately if there is no one to go for you.

Recovery position

An unconscious child whose airway is clear, and who is breathing spontaneously, should be turned on his side into the recovery position. There are several

Figure 6.5 Chest compression with one hand — child. © 2005 ERC.

Figure 6.6 Chest compression with two hands — child. © 2005 ERC.

recovery positions; each has its advocates. There are important principles to be followed.

- Place the child in as near true lateral position as possible, with his mouth dependent to enable free drainage of fluid.
- The position should be stable. In an infant this may require the support of a small pillow or a rolled-up blanket placed behind the back to maintain the position.
- Avoid any pressure on the chest that impairs breathing.
- It should be possible to turn the child onto his side and to return him back easily and safely, taking into consideration the possibility of cervical spine injury.
- Ensure the airway can be observed and accessed easily.
- The adult recovery position is suitable for use in children.

Foreign-body airway obstruction (FBAO)

No new evidence on this subject was presented during the 2005 Consensus Conference. Back blows, chest thrusts and abdominal thrusts all increase intrathoracic pressure and can expel foreign bodies from the airway. In half of the episodes, more than one technique is needed to relieve the obstruction.[41] There are no data to indicate which measure should be used first or in which order they should be applied. If one is unsuccessful, try the others in rotation until the object is cleared.

The International Guidelines 2000 algorithm is difficult to teach and knowledge retention poor. The FBAO algorithm for children has been simplified and aligned with the adult version (Figure 6.7). This should improve skill retention and encourage people, who might otherwise have been reluctant, to perform FBAO manoeuvres on children.
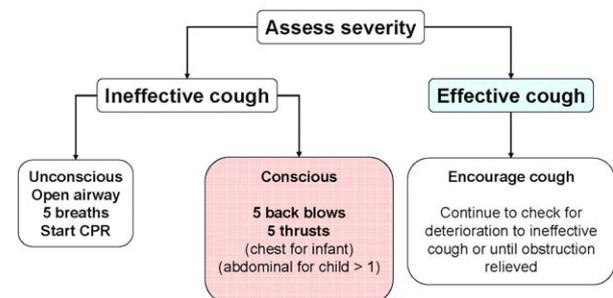
Figure 6.7 Paediatric foreign body airway obstruction algorithm.

The most significant difference from the adult algorithm is that abdominal thrusts should not be used to treat choking infants. Although abdominal thrusts have caused injuries in all age groups, the risk is particularly high in infants and very young children. This is because of the horizontal position of the ribs, which leaves the upper abdominal viscera much more exposed to trauma. For this reason, the guidelines for the treatment of FBAO are different between infants and children.

Recognition of FBAO

When a foreign body enters the airway, the child reacts immediately by coughing in an attempt to expel it. A spontaneous cough is likely to be more effective and safer than any manoeuvre a rescuer might perform. However, if coughing is absent or ineffective and the object completely obstructs the airway, the child will rapidly become asphyxiated. Active interventions to relieve FBAO are therefore required only when coughing becomes ineffective, but they then need to be commenced rapidly and confidently.

The majority of choking events in infants and children occur during play or eating episodes when a carer is usually present; thus, the events are frequently witnessed and interventions are usually initiated when the child is conscious.

Foreign-body airway obstruction is characterized by the sudden onset of respiratory distress associated with coughing, gagging or stridor. Similar signs and symptoms may be associated with other causes of airway obstruction, such as laryngitis or epiglottitis, which require different management. Suspect FBAO if the onset was very sudden and there are no other signs of illness and if there are clues to alert the rescuer, e.g. a history of eating or playing with small items immediately before the onset of symptoms.

General signs of FBAO	
Witnessed episode	
Coughing/choking	
Sudden onset	
Recent history of playing with / eating small objects	
Ineffective coughing	**Effective cough**
Unable to vocalise	Crying or verbal response to questions
Quiet or silent cough	Loud cough
Unable to breathe	Able to take a breath before coughing
Cyanosis	Fully responsive
Decreasing level of consciousness	

Relief of FBAO

1. Safety and summoning assistance

Safety is paramount: rescuers must not place themselves in danger and should consider the safest treatment of the choking child.

- If the child is coughing effectively, no external manoeuvre is necessary. Encourage the child to cough, and monitor continually.
- If the child's coughing is (or is becoming) ineffective, shout for help immediately and determine the child's conscious level.

2. Conscious child with FBAO

- If the child is still conscious but has absent or ineffective coughing, give back blows.
- If back blows do not relieve the FBAO, give chest thrusts to infants or abdominal thrusts to children. These manoeuvres create an 'artificial cough' to increase intrathoracic pressure and dislodge the foreign body.

Back blows. Back blows in the infant are performed as follows.

- Support the infant in a head downwards, prone position, to enable gravity to assist removal of the foreign body.
- A seated or kneeling rescuer should be able to support the infant safely across their lap.
- Support the infant's head by placing the thumb of one hand at the angle of the lower jaw, and one or two fingers from the same hand at the same point on the other side of the jaw.
- Do not compress the soft tissues under the infant's jaw, as this will exacerbate the airway obstruction.
- Deliver up to five sharp back blows with the heel of one hand in the middle of the back between the shoulder blades.
- The aim is to relieve the obstruction with each blow rather than to give all five blows.

Back blows in the child over 1 year of age are performed as follows.

- Back blows are more effective if the child is positioned head down.
- A small child may be placed across the rescuer's lap, as with the infant.
- If this is not possible, support the child in a forward-leaning position and deliver the back blows from behind.

If back blows fail to dislodge the object, and the child is still conscious, use chest thrusts for infants or abdominal thrusts for children. Do not use abdominal thrusts (Heimlich manoeuvre) in infants.

Chest thrusts for infants.

- Turn the infant into a head-downwards supine position. This is achieved safely by placing the free arm along the infant's back and encircling the occiput with the hand.
- Support the infant down your arm, which is placed down (or across) your thigh.
- Identify the landmark for chest compressions (lower sternum approximately a finger's breadth above the xiphisternum).
- Give five chest thrusts; these are similar to chest compressions but sharper and delivered at a slower rate.

Abdominal thrusts for children over 1 year.

- Stand or kneel behind the child; place your arms under the child's arms and encircle his torso.
- Clench your fist and place it between the umbilicus and xiphisternum.
- Grasp this hand with the other hand and pull sharply inwards and upwards.
- Repeat up to five times.
- Ensure that pressure is not applied to the xiphoid process or the lower rib cage; this might cause abdominal trauma.

Following the chest or abdominal thrusts, reassess the child. If the object has not been expelled and the victim is still conscious, continue the sequence of back blows and chest (for infant) or abdominal (for children) thrusts. Call out, or send, for help if it is still not available. Do not leave the child at this stage.

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance. Abdominal thrusts may cause internal injuries, and all victims so treated should be examined by a medical practitioner.[42]

3. Unconscious child with FBAO

If the child with FBAO is, or becomes, unconscious, place him on a firm, flat surface. Call out, or send, for help if it is still not available. Do not leave the child at this stage; proceed as follows.

- Open the mouth and look for any obvious object. If an object is seen, make an attempt to remove it with a single finger sweep. Do not attempt blind or repeated finger sweeps; these can impact the object more deeply into the pharynx and cause injury.
- Open the airway using a head tilt and/or chin lift and attempt five rescue breaths. Assess the effectiveness of each breath; if a breath does not make the chest rise, reposition the head before making the next attempt.
- Attempt five rescue breaths and, if there is no response (moving, coughing, spontaneous breaths), proceed to chest compressions without further assessment of the circulation.
- Follow the sequence for single-rescuer CPR (step 7b above) for approximately 1 min before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breaths, look to see if the foreign body can be seen in the mouth.
- If an object is seen, attempt to remove it with a single finger sweep.
- If it appears the obstruction has been relieved, open and check the airway as above; deliver rescue breaths if the child is not breathing.
- If the child regains consciousness and exhibits spontaneous effective breathing, place him in a safe position lying on his side and monitor breathing and conscious level while awaiting the arrival of the EMS.

6b Paediatric advanced life support

Prevention of cardiopulmonary arrest

In children, secondary cardiopulmonary arrests, caused by either circulatory or respiratory failure, are more frequent than primary arrests caused by arrhythmias.[9,12,43–46] So-called 'asphyxial arrests' or respiratory arrests are also more common in young adulthood (e.g., trauma, drowning, poisoning).[47,48] The outcome from cardiopulmonary arrests in children is poor; identification of the antecedent stages of cardiac or respiratory failure is a priority, as effective early intervention may be life saving.

The order of assessment and intervention for any seriously ill or injured child follows the ABC principles.

- A indicates airway (Ac for airway and cervical spine stabilisation for the injured child).
- B indicates breathing.
- C indicates circulation.

Interventions are made at each step of the assessment as abnormalities are identified; the next

step of the assessment is not started until the preceding abnormality has been managed and corrected if possible.

Diagnosing respiratory failure: assessment of A and B

The first steps in the assessment of the seriously ill or injured child are the management of the airway and breathing. Abnormalities in airway patency and breathing lead to respiratory failure. Signs of respiratory failure are

- respiratory rate outside the normal range for the child's age—either too fast or too slow
- initially increasing work of breathing which may progress to inadequate/decreased work of breathing, additional noises such as stridor, wheeze or grunting, or the loss of breath sounds
- cyanosis (without/with supplemental oxygen)

There may be associated signs in other organ systems affected by inadequate ventilation and oxygenation; these are detectable in the C steps of assessment, such as

- increasing tachycardia progressing to bradycardia (this latter sign being an ominous indicator of the loss of compensatory mechanisms)
- alteration in the level of consciousness

Diagnosing circulatory failure: assessment of C

Shock is characterised by a mismatch between metabolic tissue demand and delivery of oxygen and nutrients by the circulation.[49] Physiological compensatory mechanisms lead to changes in the heart rate, in the systemic vascular resistance (which commonly increases as an adaptive response) and in tissue and organ perfusion. Signs of circulatory failure are

- increased heart rate (bradycardia is an ominous sign, heralding physiological decompensation)
- decreased systemic blood pressure
- decreased peripheral perfusion (prolonged capillary refill time, decreased skin temperature, pale or mottled skin)
- weak or absent peripheral pulses
- decreased or increased preload
- decreased urine output and metabolic acidosis

Other systems may be affected, for example

- respiratory rate may be increased initially, becoming bradypnoeic with decompensated shock

- level of consciousness may decrease because of poor cerebral perfusion

Diagnosing cardiopulmonary arrest

Signs of cardiopulmonary arrest include

- unresponsiveness
- apnoea or gasping respiratory pattern
- absent circulation
- pallor or deep cyanosis

In the absence of 'signs of life', search for a central pulse or cardiac sounds (by direct chest auscultation) for a maximum of 10 s, before starting CPR. If there is any doubt, start CPR.[50—53]

Management of respiratory and circulatory failure

A and B

Open the airway and ensure adequate ventilation and oxygenation.

- Deliver high-flow oxygen.
- Achieving adequate ventilation and oxygenation may include the use of airway adjuncts, bag-mask ventilation (BMV), use of a laryngeal mask airway (LMA), securing a definitive airway by tracheal intubation and positive pressure ventilation.
- In rare, extreme circumstances, a surgical airway may be required.

C

Establish cardiac monitoring.

- Secure vascular access to the circulation. This may be via peripheral or central intravenous (IV) or by intraosseous (IO) cannulation.
- Give a fluid bolus and/or inotropes as required.

Assess and re-assess the child continuously, each time commencing at Airway before Breathing, thereafter moving onto the Circulation

Airway

Open the airway using basic life support techniques. Oropharyngeal and nasopharyngeal airways adjuncts can help maintain the airway. Use the oropharyngeal airway only in the unconscious child, in whom there is no gag reflex. Use the appropriate size, to avoid pushing the tongue backward and obstructing the epiglottis, or directly compressing the glottic area. The soft palate in

the child can be damaged by insertion of the oropharygneal airway; avoid this by inserting the oropharygneal airway under direct vision and passing it over a tongue depressor or laryngoscope. The nasopharyngeal airway is tolerated better in the conscious child (who has an effective gag reflex), but should not be used if there is a basal skull fracture or a coagulopathy. These simple airway adjuncts do not protect the airway from aspiration of secretions, blood or stomach contents.

Laryngeal mask airway

The LMA is an acceptable initial airway device for providers experienced in its use. It may be particularly helpful in airway obstruction caused by upper airway abnormalities. The LMA does not, however, protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required. LMA use is associated with a higher incidence of complications in small children compared with adults.[54]

Tracheal intubation

Tracheal intubation is the most secure and effective way to establish and maintain the airway, prevent gastric distension, protect the lungs against pulmonary aspiration, enable optimal control of the airway pressure and provide positive end expiratory pressure (PEEP). The oral route is preferable during resuscitation. Oral intubation is usually quicker and is associated with fewer complications than nasal placement. The judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is indicated in the conscious child to avoid multiple intubation attempts or intubation failure.[55–65] The anatomy of a child's airway differs significantly from that of an adult; hence, intubation of a child requires special training and experience. Check that tracheal tube placement is correct by clinical examination and end-tidal capnography. The tracheal tube must be secured, and monitoring of the vital signs is essential.[66]

It is also essential to plan an alternative airway management technique in case the trachea cannot be intubated.

Rapid sequence induction and intubation. The child who is in cardiopulmonary arrest and deep coma does not require sedation or analgesia to be intubated; otherwise, intubation must be preceded by oxygenation, rapid sedation, analgesia and the use of neuromuscular blocking drugs to minimise intubation complications and failure.[63] The intuba-

tor must be experienced and familiar with rapid-sequence induction drugs.

Tracheal tube sizes. The tracheal tube internal diameters (ID) for different ages are

- for neonates, 2.5—3.5 mm according to the formula (gestational age in weeks 10)
- for infants, 4 or 4.5 mm
- for children older than 1 year, according to the formula [(age in years/4) + 4]

Tracheal tube size estimation according the length of the child's body as measured by resuscitation tapes is more accurate than using the above formulae.[67]

Cuffed versus uncuffed tracheal tubes. In the prehospital setting, an uncuffed tracheal tube may be preferable when using sizes of up to 5.5 mm ID (i.e., for children up to 8 years). In hospital, a cuffed tracheal tube may be useful in certain circumstances, e.g. in cases of poor lung compliance, high airway resistance or large glottic air leak.[68–70] The correctly sized cuffed tracheal tube is as safe as an uncuffed tube for infants and children (not for neonates), provided attention is paid to its placement, size and cuff inflation pressure; excessive cuff pressure can lead to ischaemic necrosis of the surrounding laryngeal tissue and stenosis. Maintain the cuff inflation pressure below 20 cmH_2O and check it regularly.[71]

Confirmation of correct tracheal tube placement. Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with increased risk of death.[72,73] No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.[74–76] Assessment of the correct tracheal tube position is made by

- observation of the tube passing beyond the vocal cords
- observation of symmetrical chest wall movement during positive pressure ventilation
- observation of mist in the tube during the expiratory phase of ventilation
- absence of gastric distension
- equal air entry heard on bilateral auscultation of both axillae and apices of the chest
- absence of air entry into the stomach on auscultation
- detection of end-tidal CO_2 if the child has a perfusing rhythm (this may be seen with effective CPR)
- improvement or stabilisation of SpO_2 to the expected range

- improvement of heart rate towards the age-expected value (or remaining within the normal range)

If the child is in cardiopulmonary arrest and exhaled CO_2 is not detected, or if there is any doubt, confirm tracheal tube position by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and reassess its position. Maintain the child's head in neutral position; flexion of the head drives the tube further into the trachea whereas extension may pull it out of the airway.[77] Confirm the position of the tracheal tube at mid trachea by plain chest radiograph; the tracheal tube tip should be at the level of the 2nd or 3rd thoracic vertebra.

DOPES is a useful acronym for the causes of sudden deterioration in an intubated child

- D: displacement of the tracheal tube
- O: obstruction of the tracheal tube
- P: pneumothorax
- E: equipment failure (source of gas, BMV, ventilator, etc.)
- S: stomach (gastric distension may alter diaphragm mechanics)

Breathing

Oxygenation

Use oxygen at the highest concentration (i.e., 100%) during resuscitation. Once circulation is restored, give sufficient oxygen to maintain peripheral oxygen saturation at or above 95%.[78,79]

Studies in neonates suggest some advantages to using room air during resuscitation, but the evidence as yet is inconclusive (see Section 6c).[80–83] In the older child, there is no evidence for any such advantages, so use 100% oxygen for resuscitation.

Ventilation

Healthcare providers commonly provide excessive ventilation to victims of cardiopulmonary or respiratory arrest, and this may be detrimental. Hyperventilation causes increased thoracic pressure, decreased cerebral and coronary perfusion, and poorer survival rates in animals and adults.[84–89] The ideal tidal volume should achieve modest chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations (a lone rescuer may use 30:2); the correct compression rate is 100 min^{-1}.

Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 12–20 breaths min^{-1} without interrupting chest compressions. Take care to ensure that lung infla-

tion is adequate during chest compressions. When circulation is restored, or if the child still has a perfusing rhythm, ventilate at 12–20 breaths min^{-1} to achieve a normal pCO_2. Hyperventilation is harmful.

Bag-mask ventilation. BMV is effective and safe for a child requiring assisted ventilation for a short period, i.e. in the prehospital setting or in an emergency department.[73,90–92] Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate, auscultating for breath sounds and measuring peripheral oxygen saturation (SpO_2). Any healthcare provider dealing with children must be able to deliver BMV effectively.

Prolonged ventilation. If prolonged ventilation is required, the benefits of a secured airway probably outweigh the potential risks associated with tracheal intubation.

Monitoring of breathing and ventilation

End tidal CO_2. Monitoring end-tidal CO_2 with a colorimetric detector or capnometer confirms tracheal tube placement in the child weighing more than 2 kg, and may be used in pre- and in-hospital settings, as well as during any transportation of the child.[93–97] A colour change or the presence of a capnographic waveform indicates that the tube is in the tracheobronchial tree, both in the presence of a perfusing rhythm and during cardiopulmonary arrest. Capnography does not rule out intubation of the right mainstem bronchus. The absence of exhaled CO_2 during cardiopulmonary arrest may not be caused by tube misplacement, since a low or absent end-tidal CO_2 may reflect low or absent pulmonary blood flow.[98–101]

Oesophageal detector devices. The self-inflating bulb or aspirating syringe (oesophageal detector device, ODD) may be used for the secondary confirmation of tracheal tube placement in children with a perfusing rhythm.[102,103] There are no studies on the use of ODD in children who are in cardiopulmonary arrest.

Pulse oximetry. Clinical evaluation of the oxygen level is unreliable; therefore monitor the child's peripheral oxygen saturation continuously by pulse oximetry. Pulse oximetry can be unreliable under certain conditions, e.g. if the child is in shock, in cardiopulmonary arrest or has poor peripheral perfusion. Although pulse oximetry is relatively simple, it is a poor guide to tracheal tube displacement; capnography detects tracheal tube dislodgement more rapidly than pulse oximetry.[104]

Circulation

Vascular access

Vascular access is essential to give drugs and fluids and obtain blood samples. Venous access can be difficult to establish during resuscitation of an infant or child.[105] Limit the maximum number of attempts to obtain IV access to three; thereafter, insert an IO needle.[106]

Intraosseous access. IO access is a rapid, safe, and effective route to give drugs, fluids and blood products.[107–113] The onset of action and time to achieve adequate plasma drug concentrations are similar to those provided by central venous access.[114,115] Bone marrow samples can be used to cross-match for blood type or group,[116] for chemical analysis,[117,118] and for blood gas measurement (the values are comparable to central venous blood gases).[117,119,120] Flush each drug with a bolus of normal saline to ensure dispersal beyond the marrow cavity and to achieve faster distribution to the central circulation. Inject large boluses of fluid using manual pressure. Intraosseous access can be maintained until definitive IV access has been established.

Intravenous access. Peripheral IV access provides plasma concentrations of drugs and clinical responses equivalent to central or IO access.[121–125] Central lines provide more secure long-term access[121,122,124,125] but offer no advantages during resuscitation, compared with IO or peripheral IV access.

Tracheal tube access

IV and IO access are better than the tracheal route for giving drugs.[126] Lipid-soluble drugs, such as lidocaine, atropine, adrenaline and naloxone are absorbed via the lower airway.[127–131] Optimal tracheal tube drug doses are unknown because of the great variability of alveolar drug absorption, but the following dosages have been recommended as guidance

- adrenaline, 100 mcg kg^{-1}
- lidocaine, 2–3 mg kg^{-1}
- atropine, 30 mcg kg^{-1}

The optimal dose of naloxone is not known.

Dilute the drug in 5 ml of normal saline and follow administration with five ventilations.[132–134] Do not give non-lipid soluble medications (e.g., glucose, bicarbonate, calcium) via the tracheal tube because they will damage the airway mucosa.

Fluids and drugs

Volume expansion is indicated when a child shows signs of shock in the absence of volume overload.[135] If systemic perfusion is inadequate, give a bolus of 20 ml kg^{-1} of an isotonic crystalloid, even if the systemic blood pressure is normal. Following every bolus, re-assess the child's clinical state using ABC, to decide whether a further bolus or other treatment is required.

There are insufficient data to make recommendations about the use of hypertonic saline for shock associated with head injuries or hypovolaemia.[136] There are also insufficient data to recommend delayed fluid resuscitation in the hypotensive child with blunt trauma.[137] Avoid dextrose-containing solutions unless there is hypoglycaemia.[138–141] However, hypoglycaemia must actively be sought and avoided, particularly in the small child or infant.

Adenosine

Adenosine is an endogenous nucleotide which causes a brief atrioventricular (AV) block and impairs accessory bundle re-entry at the level of the AV node. Adenosine is recommended for the treatment of supraventricular tachycardia (SVT).[142] It is safe to use, as it has a short half-life (10 s); give it intravenously via upper limb or central veins, to minimise the time taken to reach the heart. Give adenosine rapidly, followed by a flush of 3–5 ml of normal saline.[143]

Adrenaline (epinephrine)

Adrenaline is an endogenous catecholamine with potent alpha, beta-1 and beta-1 adrenergic actions. It is the essential medication in cardiopulmonary arrest, and is placed prominently in the treatment algorithms for non-shockable and shockable rhythms. Adrenaline induces vasoconstriction, increases diastolic pressure and thereby improves coronary artery perfusion pressure, enhances myocardial contractility, stimulates spontaneous contractions and increases the amplitude and frequency of VF, so increasing the likelihood of successful defibrillation. The recommended IV/IO dose of adrenaline in children is 10 mcg kg^{-1}. The dose of adrenaline given via the tracheal tube is ten times this (100 mcg kg^{-1}).[127,144–146] If needed, give further doses of adrenaline every 3–5 min. The use of higher doses of adrenaline via the IV or IO route is not recommended routinely, as it does not improve survival or neurological outcome after cardiopulmonary arrest.[147–150]

Once spontaneous circulation is restored, a continuous infusion of adrenaline may be required. Its haemodynamic effects are dose related; there is also considerable variability between children in response, therefore, titrate the infusion dose to the desired effect. High infusion rates may cause excessive vasoconstriction, compromising extremity, mesenteric, and renal blood flow. High-dose adrenaline may cause severe hypertension and tachyarrhythmias.[151]

To avoid tissue damage it is essential to give adrenaline through a secure intravascular line (IV or IO). Adrenaline and other catecholamines are inactivated by alkaline solutions and should never be mixed with sodium bicarbonate.[152]

Amiodarone

Amiodarone is a non-competitive inhibitor of adrenergic receptors; it depresses conduction in myocardial tissue and therefore slows AV conduction and prolongs the QT interval and the refractory period. Except when given for the treatment of refractory VF/pulseless VT, amiodarone must be injected slowly (over 10–20 min) with systemic blood pressure and ECG monitoring to avoid fast-infusion-related hypotension. This side effect is less common with the aqueous solution.[153] Other rare but significant adverse effects are bradycardia and polymorphic VT.[154]

Atropine

Atropine accelerates sinus and atrial pacemakers by blocking the parasympathetic response. It may also increase AV conduction. Small doses (<100 mcg) may cause paradoxical bradycardia.[155]

Calcium

Calcium is essential for myocardial contraction[156,157] but routine use of calcium does not improve the outcome from cardiopulmonary arrest.[158–160]

Glucose

Neonatal, child and adult data show that both hyperglycaemia and hypoglycaemia are associated with poor outcome after cardiopulmonary arrest,[161–163] but it is uncertain if this is causative or merely an association.[164] Check blood or plasma glucose concentration and monitor closely in any ill or injured child, including after cardiac arrest. Do not give glucose-containing fluids during CPR unless hypoglycaemia is present. Avoid hyperglycaemia and hypoglycaemia following return of spontaneous circulation (ROSC).

Magnesium

There is no evidence for giving magnesium routinely during cardiopulmonary arrest.[165] Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsades de pointes VF, regardless of the cause.[166]

Sodium bicarbonate

Giving sodium bicarbonate routinely during cardiopulmonary arrest and CPR or after ROSC is not recommended.[167,168] After effective ventilation and chest compressions have been achieved and adrenaline given, sodium bicarbonate may be considered for the child who has had a prolonged cardiopulmonary arrest and severe metabolic acidosis. Sodium bicarbonate may also be considered in the case of haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic overdose. Excessive quantities of sodium bicarbonate may impair tissue oxygen delivery, produce hypokalaemia, hypernatraemia and hyperosmolality and inactivate catecholamines.

Lidocaine

Lidocaine is less effective than amiodarone for defibrillation-resistant VF/VT in adults,[169] and therefore is not the first-line treatment in defibrillation-resistant VF/VT in children.

Procainamide

Procainamide slows intra-atrial conduction and prolongs the QRS and QT intervals; it can be used in SVT[170,171] or VT[172] resistant to other medications, in the haemodynamically stable child. However, paediatric data are sparse and procainamide should be used cautiously.[173,174] Procainamide is a potent vasodilator and can cause hypotension; infuse it slowly with careful monitoring.[170,175,176]

Vasopressin

Vasopressin is an endogenous hormone that acts at specific receptors, mediating systemic vasoconstriction (via V1 receptor) and the reabsorption of water in the renal tubule (by the V2 receptor).[177] The use of vasopressin for the treatment of cardiac arrest in adults is discussed in detail in Section 4e. There is currently insufficient evidence to support

or refute the use of vasopressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm in adults. Thus, there is currently insufficient evidence to recommend the routine use of vasopressin in the child with cardiopulmonary arrest.[178—180]

Defibrillators

Defibrillators are either automatically (such as the AED) or manually operated, and may be capable of delivering either monophasic or biphasic shocks. Manual defibrillators capable of delivering the full energy requirements from neonates upwards must be available within hospitals and in other health-care facilities caring for children at risk of cardiopulmonary arrest. Automated external defibrillators are preset for all variables, including the energy dose.

Pad/paddle size for defibrillation. The largest possible available paddles should be chosen to provide good contact with the chest wall. The ideal size is unknown, but there should be good separation between the pads.[181,182] Recommended sizes are

- 4.5 cm diameter for infants and children weighing <10 kg
- 8—12 cm diameter for children >10 kg (older than 1 year)

To decrease skin and thoracic impedance, an electrically conducting interface is required between the skin and the paddles. Preformed gel pads or self-adhesive defibrillation electrodes are effective. Do not use ultrasound gel, saline-soaked gauze, alcohol-soaked gauze/pads or ultrasound gel.

Position of the paddles. Apply the paddles firmly to the bare chest in the anterolateral position, one paddle placed below the right clavicle and the other in the left axilla (Figure 6.8). If the paddles are too large, and there is a danger of charge arcing across the paddles, one should be placed on the upper back, below the left scapula, and the other on the front, to the left of the sternum. This is known as the anteroposterior position.

Optimal paddle force. To decrease transthoracic impedance during defibrillation, apply a force of 3 kg for children weighing <10 kg, and 5 kg for larger children.[183,184]

Energy dose in children. The ideal energy dose for safe and effective defibrillation is unknown.
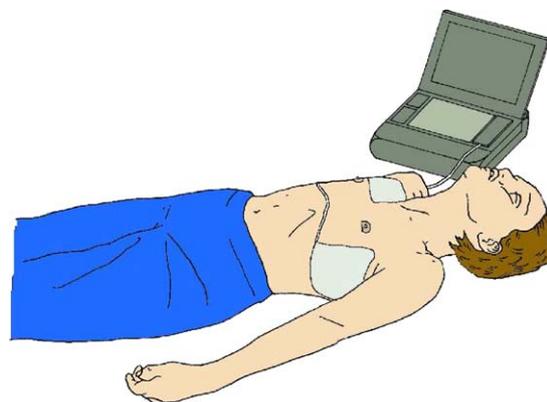
Figure 6.8 Paddle positions for defibrillation — child. © 2005 ERC.

Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic shocks.[33,34,37—40] Animal models show better results with paediatric doses of 3—4 J kg^{-1} than with lower doses,[34,37] or adult doses.[35] Doses larger than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.[27,36] When using a manual defibrillator, use 4 J kg^{-1} (biphasic or monophasic waveform) for the first and subsequent shocks.

If no manual defibrillator is available, use an AED that can recognise paediatric shockable rhythms.[29,30,185] This AED should be equipped with a dose attenuator which decreases the delivered energy to a lower dose more suitable for children aged 1—8 years (50—75 J).[31] If such an AED in not available, in an emergency use a standard AED and the preset adult energy levels. For children weighing more than 25 kg (above 8 years), use a standard AED with standard paddles. There is currently insufficient evidence to support a recommendation for or against the use of AEDs in children less than 1 year.

Management of cardiopulmonary arrest

A B C

Commence and continue with basic life support (Figure 6.9).

A and B

Oxygenate and ventilate with BMV.

- Provide positive pressure ventilation with a high inspired oxygen concentration.
- Give five rescue breaths followed by external chest compression and positive pressure ventila-
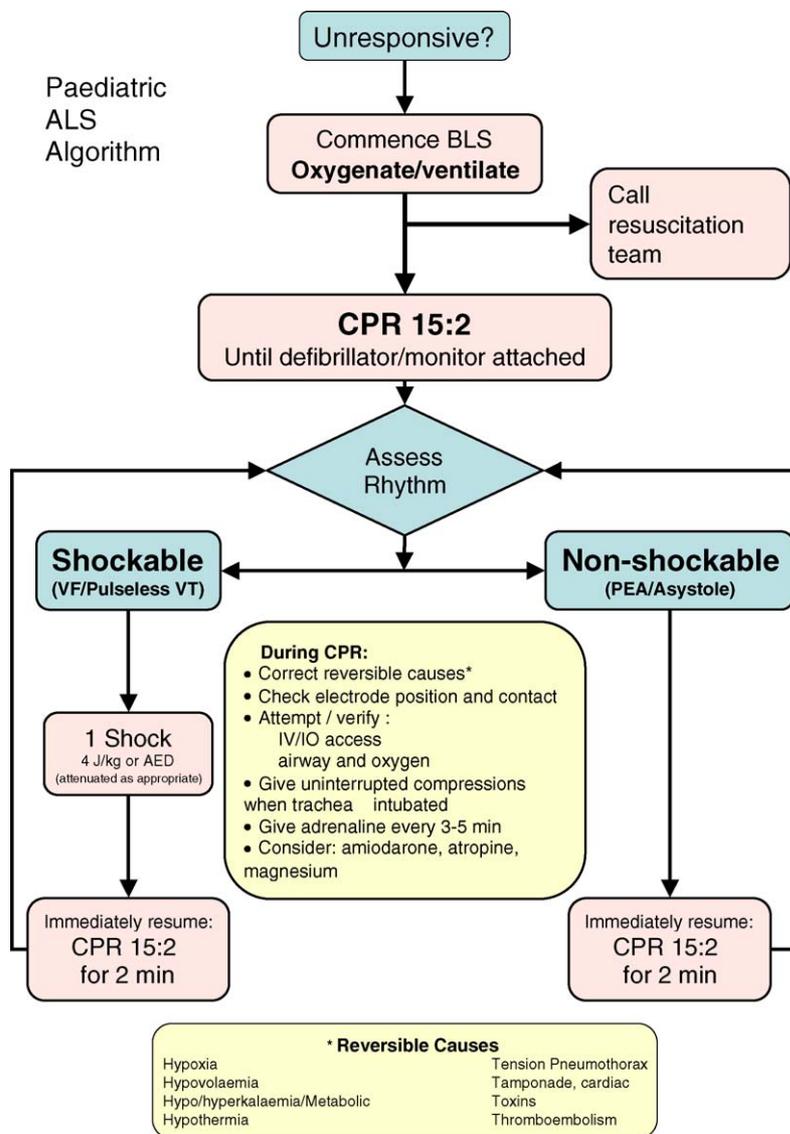
Figure 6.9 Paediatric advanced life support algorithm.

tion in the ratio of 15:2 (lone rescuer may use 30:2).
- Avoid rescuer fatigue by changing the rescuer performing chest compressions frequently.
- Establish cardiac monitoring.

C

Assess cardiac rhythm and signs of circulation (±check for a central pulse for no more than 10 s).

Asystole, pulseless electrical activity (PEA)—non-shockable

- Give adrenaline, 10 mcg kg^{-1} IV or IO, and repeat every 3—5 min.

- If no vascular access is available and a tracheal tube is in situ, give adrenaline, 100 mcg kg^{-1}, via this route until IV/IO access is obtained.
- Identify and treat any reversible causes (4Hs & 4Ts).

VF/pulseless VT—shockable

- Attempt defibrillation immediately (4 J kg^{-1} for all shocks).
- Resume CPR as soon as possible.
- After 2 min, check the cardiac rhythm on the monitor.
- Give second shock if still in VF/pulseless VT.

- Immediately resume CPR for 2 min and check monitor; if no change, give adrenaline followed immediately by a 3rd shock.
- CPR for 2 min.
- Give amiodarone if still in VF/pulseless VT followed immediately by a 4th shock.
- Give adrenaline every 3—5 min during CPR.
- If the child remains in VF/pulseless VT, continue to alternate shocks with 2 min of CPR.
- If signs of life become evident, check the monitor for an organised rhythm; if this is present, check for a central pulse.
- Identify and treat any reversible causes (4Hs & 4Ts).
- If defibrillation was successful but VF/pulseless VT recurs, resume CPR, give amiodarone and defibrillate again at the dose that was effective previously. Start a continuous infusion of amiodarone.

Reversible causes of cardiac arrest (4 Hs and 4 Ts)

- Hypoxia
- Hypovolaemia
- Hyper/hypokalaemia
- Hypothermia
- Tension pneumothorax
- Tamponade (coronary or pulmonary)
- Toxic/therapeutic disturbances
- Thrombosis (coronary or pulmonary)

Sequence of events in cardiopulmonary arrest

- When a child becomes unresponsive, without signs of life (no breathing, cough or any detectable movement), start CPR immediately.
- Provide BMV with 100% oxygen.
- Commence monitoring. Send for a manual or automatic external defibrillator (AED) to identify and treat shockable rhythms as quickly as possible.

In the less common circumstance of a witnessed sudden collapse, early activation of emergency services and getting an AED may be more appropriate; start CPR as soon as possible.

Rescuers must perform CPR with minimal interruption until attempted defibrillation.

Cardiac monitoring

Position the cardiac monitor leads or defibrillation paddles as soon as possible, to enable differentiation between a shockable and a non-shockable cardiac rhythm. Invasive monitoring of systemic blood pressure may help to improve effectiveness of chest compression,[186] but must not delay the provision of basic or advanced resuscitation.

Shockable rhythms comprise pulseless VT and VF. These rhythms are more likely in the child who presents with sudden collapse. Non-shockable rhythms comprise PEA, bradycardia (<60 beats min^{-1} with no signs of circulation) and asystole. PEA and bradycardia often have wide QRS complexes.

Non-shockable rhythms

Most cardiopulmonary arrests in children and adolescents are of respiratory origin.[19,44,187—189] A period of immediate CPR is therefore mandatory in this age group, before searching for an AED or manual defibrillator, as their immediate availability will not improve the outcome of a respiratory arrest.[11,13] Bystander CPR is associated with a better neurological outcome in adults and children.[9,10,190] The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by organised, wide complex electrical activity, usually at a slow rate, and absent pulses. PEA commonly follows a period of hypoxia or myocardial ischaemia, but occasionally can have a reversible cause (i.e., one of the 4 H's and 4 T'S) that led to a sudden impairment of cardiac output.

Shockable rhythms

VF occurs in 3.8—19% of cardiopulmonary arrests in children[9,45,188,189]; the incidence of VF/pulseless VT increases with age.[185,191] The primary determinant of survival from VF/pulseless VT cardiopulmonary arrest is the time to defibrillation. Prehospital defibrillation within the first 3 min of witnessed adult VF arrest results in >50% survival. However, the success of defibrillation decreases dramatically as the time to defibrillation increases; for every minute delay in defibrillation (without any CPR), survival decreases by 7—10%. Survival after more than 12 min of VF in adult victims is <5%.[192] Cardiopulmonary resuscitation provided before defibrillation for response intervals longer than 5 min improved outcome in some studies,[193,194] but not in others.[195]

Drugs in shockable rhythms

Adrenaline is given every 3—5 min by the IV or IO route in preference to the tracheal tube route. Amiodarone is indicated in defibrillation-resistant

VF/pulseless VT. Experimental and clinical experience with amiodarone in children is scarce; evidence from adult studies[169,196,197] demonstrates increased survival to hospital admission, but not to hospital discharge. One paediatric case series demonstrates the effectiveness of amiodarone for life-threatening ventricular arrhythmias.[198] Therefore, IV amiodarone has a role in the treatment of defibrillation refractory or recurrent VF/pulseless VT in children.

Arrhythmias

Unstable arrhythmias

Check the central pulse of any child with an arrhythmia; if the pulse is absent, proceed to treating the child as being in cardiopulmonary arrest. If the child has a central pulse, evaluate his haemodynamic status. Whenever the haemodynamic status is compromised, the first steps are as follows.

- Open the airway.
- Assist ventilation and give oxygen.
- Attach ECG monitor or defibrillator and assess the cardiac rhythm.
- Evaluate if the rhythm is slow or fast for the child's age.
- Evaluate if the rhythm is regular or irregular.
- Measure QRS complex (narrow complexes, <0.08 s duration; large complexes, >0.08 s).
- The treatment options are dependent on the child's haemodynamic stability.

Bradycardia

Bradycardia is caused commonly by hypoxia, acidosis and severe hypotension; it may progress to cardiopulmonary arrest. Give 100% oxygen, and positive pressure ventilation if required, to any child presenting with bradyarrhythmia and circulatory failure.

If a poorly perfused child has a heart rate <60 beats min^{-1}, and does not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline. If the bradycardia is caused by vagal stimulation, provide ventilation with 100% oxygen and give atropine, before giving adrenaline.

A cardiac pacemaker is useful only in cases of AV block or sinus node dysfunction unresponsive to oxygenation, ventilation, chest compressions and other medications; the pacemaker is not effective in asystole or arrhythmias caused by hypoxia or ischaemia.[199]

Tachycardia

Narrow complex tachycardia. If supraventricular tachycardia (SVT) is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. The manoeuvres can be used in unstable children if they do not delay chemical or electrical cardioversion.[200] If the child is haemodynamically unstable, omit vagal manoeuvres and attempt electrical cardioversion immediately. Adenosine is usually effective in converting SVT into sinus rhythm. Adenosine is given by rapid IV injection as closely as practical to the heart (see above), followed immediately by a bolus of normal saline.

Electrical cardioversion (synchronised with R wave) is indicated in the haemodynamically compromised child, in whom vascular access is not available, or in whom adenosine has failed to convert the rhythm. The first energy dose for electrical cardioversion of SVT is $0.5-1\,J\,kg^{-1}$ and the second dose is $2\,J\,kg^{-1}$. If unsuccessful, give amiodarone or procainamide under guidance from a paediatric cardiologist or intensivist before the third attempt.

Amiodarone has been shown to be effective in the treatment of SVT in several paediatric studies.[198,201–207] However, since most studies of the use of amiodarone in narrow-complex tachycardias have been for junctional ectopic tachycardia in postoperative children, the applicability of its use in all cases of SVT may be limited. If the child is haemodynamically stable, early consultation with an expert is recommended before giving amiodarone.

Wide complex tachycardia. In children, wide-QRS-complex tachycardia is more likely to be supraventricular than ventricular in origin.[208] However, wide-QRS-complex tachycardia, although uncommon, must be considered to be VT in haemodynamically unstable children until proven otherwise. VT occurs most often in the child with underlying heart disease (e.g., after cardiac surgery, cardiomyopathy, myocarditis, electrolyte disorders, prolonged QT interval, central intracardiac catheter). Synchronised cardioversion is the treatment of choice for unstable VT with a pulse. Consider antiarrhythmic therapy if a second cardioversion dose is unsuccessful or if VT recurs. Amiodarone has been shown to be safe and effective in treating paediatric arrhythmias.[198,202,203,209]

Stable arrhythmias

Contact an expert before initiating therapy, while maintaining the child's ABC. Depending on the

child's clinical history, presentation and ECG diagnosis, a child with stable, wide-QRS-complex tachycardia may be treated for SVT and be given vagal manoeuvres or adenosine. Otherwise, consider amiodarone as a treatment option; similarly, consider amiodarone if the diagnosis of VT is confirmed by ECG. Procainamide may also be considered in stable SVT refractory to vagal manoeuvres and adenosine[210–212] as well as in stable VT.[172,213,214] Do not give procainamide with amiodarone.

Post-arrest management

Myocardial dysfunction is common after cardiopulmonary resuscitation.[215,216] Vasoactive drugs may improve the child's post-arrest haemodynamic values, but the drugs must be titrated according to the clinical condition. They must be given continuously through an IV line.

Temperature control and management

Hypothermia is common in the child following cardiopulmonary resuscitation.[217] Central hypothermia (32–34 °C) may be beneficial, whereas fever may be detrimental to the injured brain of survivors. Although there are no paediatric studies, mild hypothermia has an acceptable safety profile in adults[218,219] and neonates;[220–224] it could increase the number of neurologically intact survivors.

A child who regains a spontaneous circulation but remains comatose after cardiopulmonary arrest may benefit from being cooled to a core temperature of 32–34 °C for 12–24 h. The successfully resuscitated child with hypothermia and ROSC should not be actively rewarmed unless the core temperature is below 32 °C. Following a period of mild hypothermia, rewarm the child slowly at 0.25–0.5 °C h^{-1}.

There are several methods to induce, monitor and maintain body temperature in children. External and/or internal cooling techniques can be used to initiate cooling.[225–227] Shivering can be prevented by deep sedation and neuromuscular blockade. Complications can occur and include an increased risk of infection, cardiovascular instability, coagulopathy, hyperglycaemia and electrolyte abnormalities.[228,229]

The optimum target temperature, rate of cooling, duration of hypothermia and rate of rewarming after deliberate cooling have yet to be determined; currently, no specific protocol for children can be recommended.

Fever is common following cardiopulmonary resuscitation; it is associated with a poor neurological outcome,[230–232] the risk of which increases with each degree of body temperature greater than 37 °C.[230] There are limited experimental data suggesting that the treatment of fever with antipyretics and/or physical cooling reduces neuronal damage.[233,234] Antipyretics and accepted drugs to treat fever are safe; therefore, use them to treat fever aggressively.

Prognosis of cardiopulmonary arrest

There are no simple guidelines to determine when resuscitative efforts become futile. After 20 min of resuscitation, the team leader of the resuscitation team should consider whether or not to stop.[187,235–239] The relevant considerations in the decision to continue the resuscitation include the cause of arrest,[45,240] pre-existing conditions, whether the arrest was witnessed, the duration of untreated cardiopulmonary arrest (''no flow''), the effectiveness and duration of CPR (''low flow''), the promptness of extracorporeal life support for a reversible disease process[241–243] and associated special circumstances (e.g, icy water drowning,[9,244] exposure to toxic drugs).

Parental presence

The majority of parents would like to be present during resuscitation and when any procedure is carried out on their child.[245–255]. Parents witnessing their child's resuscitation can see that everything possible has been attempted.[256–260] Furthermore, they may have the opportunity to say goodbye to their child; allowing parents to be at the side of their child has been shown to help them gain a realistic view of the attempted resuscitation and the child's death.[261] Families who were present at their child's death showed less anxiety and depression, better adjustment and had an improved grieving process when assessed several months later.[260] Parental presence in the resuscitation room may help healthcare providers maintain their professional behaviour while also helping them to see the child as a human being and a family member.[261]

Family presence guidelines

A dedicated member of the resuscitation team should be present with the parents to explain the process in an empathetic manner, ensuring that

the parents do not interfere with or distract the resuscitation. If the presence of the parents is impeding the progress of the resuscitation, they should be sensitively asked to leave. When appropriate, physical contact with the child should be allowed and, wherever possible, the parents should be allowed to be with their dying child at the final moment.[256,261−264]

The leader of the resuscitation team, not the parents, will decide when to stop the resuscitation; this should be expressed with sensitivity and understanding. After the event the team should be debriefed, to enable any concerns to be expressed and for the team to reflect on their clinical practice in a supportive environment.

6c Resuscitation of babies at birth

Introduction

The following guidelines for resuscitation at birth have been developed during the process that culminated in the 2005 International Consensus Conference on Emergency Cardiovascular Care (ECC) and Cardiopulmonary Resuscitation (CPR) Science with Treatment Recommendations.[265] They are an extension of the guidelines already published by the ERC,[2] and take into account recommendations made by other national[266] and international organisations.[267]

The guidelines that follow do not define the only way that resuscitation at birth should be achieved; they merely represent a widely accepted view of how resuscitation at birth can be carried out both safely and effectively.

Preparation

Relatively few babies need any resuscitation at birth. Of those that do need help, the overwhelming majority will require only assisted lung aeration. A small minority may need a brief period of chest compressions in addition to lung aeration. Of 100,000 babies born in Sweden in 1 year, only 10 per 1000 (1%) babies weighing 2.5 kg or more appeared to need resuscitation at delivery.[268] Of those babies receiving resuscitation, 8 per 1000 responded to mask inflation and only 2 per 1000 appeared to need intubation.[268] The same study tried to assess the unexpected need for resuscitation at birth, and found that for low-risk babies, i.e. those born after 32 weeks' gestation and following an apparently normal labour, about 2 per 1000 (0.2%) appeared to need resuscitation at delivery. Of these, 90% responded to mask inflation alone, whereas the remaining 10% appeared not to respond to mask inflation and therefore were intubated at birth.

Resuscitation or specialist help at birth is more likely to be needed by babies with intrapartum evidence of significant fetal compromise, babies delivering before 35 weeks' gestation, babies delivering vaginally by the breech and multiple pregnancies. Although it is often possible to predict the need for resuscitation before a baby is born, this is not always the case. Therefore, personnel trained in newborn life support should be easily available at every delivery and, should there be any need for resuscitation, the care of the baby should be their sole responsibility. One person experienced in tracheal intubation of the newborn should also be easily available for normal low-risk deliveries and, ideally, in attendance for deliveries associated with a high risk for neonatal resuscitation. Local guidelines indicating who should attend deliveries should be developed based on current practice and clinical audit.

An organised programme educating in the standards and skills required for resuscitation of the newborn is therefore essential for any institution in which deliveries occur.

Planned home deliveries

The recommendations for those who should attend a planned home delivery vary from country to country, but the decision to undergo a planned home delivery, once agreed by the medical and midwifery staff, should not compromise the standard of initial resuscitation at birth. There will inevitably be some limitations to resuscitation of a newborn baby in the home because of the distance from further assistance, and this must be made clear to the mother at the time plans for home delivery are made. Ideally, two trained professionals should be present at all home deliveries;[269] one of these must be fully trained and experienced in providing mask ventilation and chest compressions in the newborn.

Equipment and environment

Resuscitation at birth is often a predictable event. It is therefore simpler to prepare the environment and the equipment before delivery of the baby than is the case in adult resuscitation. Resuscitation should ideally take place in a warm, well-lit, draught-free area with a flat resuscitation surface placed below a radiant heater and other resusci-

tation equipment immediately available. All equipment must be checked daily.

When a birth takes place in a non-designated delivery area, the recommended minimum set of equipment includes a device for safe, assisted lung aeration of an appropriate size for the newborn, warm dry towels and blankets, a clean (sterile) instrument for cutting the umbilical cord and clean gloves for the attendant. It may also be helpful to have a suction device with a suitably sized suction catheter and a tongue depressor (or laryngoscope), to enable the oropharynx to be examined.

Temperature control

Naked, wet, newborn babies cannot maintain their body temperature in a room that feels comfortably warm for adults. Compromised babies are particularly vulnerable.[270] Exposure of the newborn to cold stress will lower arterial oxygen tension[271] and increase metabolic acidosis.[272] Prevent heat loss by

- protecting the baby from draughts
- keeping the delivery room warm
- drying the term baby immediately after delivery. Cover the head and body of the baby, apart from the face, with a warm towel to prevent further heat loss. Alternatively, place the baby skin to skin with the mother and cover both with a towel
- placing the baby on a warm surface under a pre-heated radiant warmer if resuscitation is needed

In very preterm babies (especially below 28 weeks' gestation), drying and wrapping may not be sufficiently effective. A more effective method of keeping these babies warm is to cover the head and body of the baby (apart from the face) with plastic wrapping, without drying the baby beforehand, and then to place the baby so covered under radiant heat.

Initial assessment

The Apgar scoring system was not designed to identify prospectively babies needing resuscitation.[273] Several studies have also suggested that it is highly subjective.[274] However, components of the score, namely respiratory rate, heart rate and colour, if assessed rapidly, can identify babies needing resuscitation.[275] Furthermore, repeated assessment of these components can indicate whether the baby is responding or whether further efforts are needed.

Respiratory activity

Check whether the baby is breathing. If so, evaluate the rate, depth and symmetry of respiration, together with any abnormal breathing pattern such as gasping or grunting.

Heart rate

This is best evaluated by listening to the apex beat with a stethoscope. Feeling the pulse in the base of the umbilical cord is often effective but can be misleading; cord pulsation is only reliable if found to be more than 100 beats min^{-1}.[276]

Colour

A healthy baby is born blue but becomes pink within 30 s of the onset of effective breathing. Observe whether the baby is centrally pink, cyanosed or pale. Peripheral cyanosis is common and does not, by itself, indicate hypoxaemia.

Tone

A very floppy baby is likely to be unconscious and is likely to need respiratory support.

Tactile stimulation

Drying the baby usually produces enough stimulation to induce effective respiration. Avoid more vigorous methods of stimulation. If the baby fails to establish spontaneous and effective respirations following a brief period of stimulation, further support will be required.

Classification according to initial assessment

On the basis of the initial assessment, the babies can usually be divided into four groups.

Group 1: vigorous breathing or crying
good tone
rapidly becoming pink
heart rate higher than 100 beats min^{-1}

This baby requires no intervention other than drying, wrapping in a warm towel and, where appropriate, handing to the mother. The baby will remain warm through skin-to-skin contact with mother under a cover, and may be put to the breast at this stage.

Group 2:   breathing inadequately or apnoeic
                    remaining centrally blue
                    normal or reduced tone
                    heart rate less than 100 beats min^{-1}

This baby may respond to tactile stimulation and/or facial oxygen, but may need mask inflation.

Group 3:   breathing inadequately or apnoeic
                    blue or pale
                    floppy
                    heart rate less than 100 beats min^{-1}

This baby may improve with mask inflation but may also require chest compressions.

Group 4:   breathing inadequately or apnoeic
                    pale
                    floppy
                    no detectable heart rate

This baby will require immediate airway control, lung inflation and ventilation. Once this has been successfully accomplished, the baby may also need chest compressions and perhaps drugs.

There remains a very rare group of babies who, though breathing adequately and with a good heart rate, remain blue. This group includes a range of possible diagnoses such as diaphragmatic hernia, surfactant deficiency, congenital pneumonia, pneumothorax or cyanotic congenital heart disease.

Newborn life support

Commence newborn life support (Figure 6.10) if assessment demonstrates that the baby has failed to establish adequate regular normal breathing, or has a heart rate of less than 100 beats min^{-1}. Opening the airway and aerating the lungs is usually all that is necessary. Furthermore, more complex interventions will be futile unless these two first steps have been successfully completed.

Airway

The baby should be on his or her back with the head in a neutral position (Figure 6.11). A 2-cm thickness of the blanket or towel placed under the baby's shoulder may be helpful in maintaining proper head position. In floppy babies, application of jaw thrust or the use of an appropriately sized oropharyngeal airway may be helpful in opening the airway.

Suction is needed only if there is particulate matter or blood obstructing the airway. Aggressive pharyngeal suction can delay the onset of spontaneous breathing and cause laryngeal spasm and vagal

bradycardia.[277] The presence of thick meconium in a non-vigorous baby is the only indication for considering immediate suction. If suction is required, it is best done under direct vision. Connect a 12—14 FG suction catheter, or a Yankauer sucker, to a suction source not exceeding −100 mmHg.

Breathing

There is currently insufficient evidence to specify the concentration of oxygen to be used when starting resuscitation. After initial steps at birth, if respiratory efforts are absent or inadequate, lung aeration is the priority (Figure 6.12). The primary measure of adequate initial lung inflation is a prompt improvement in heart rate; assess chest wall movement if the heart rate does not improve.
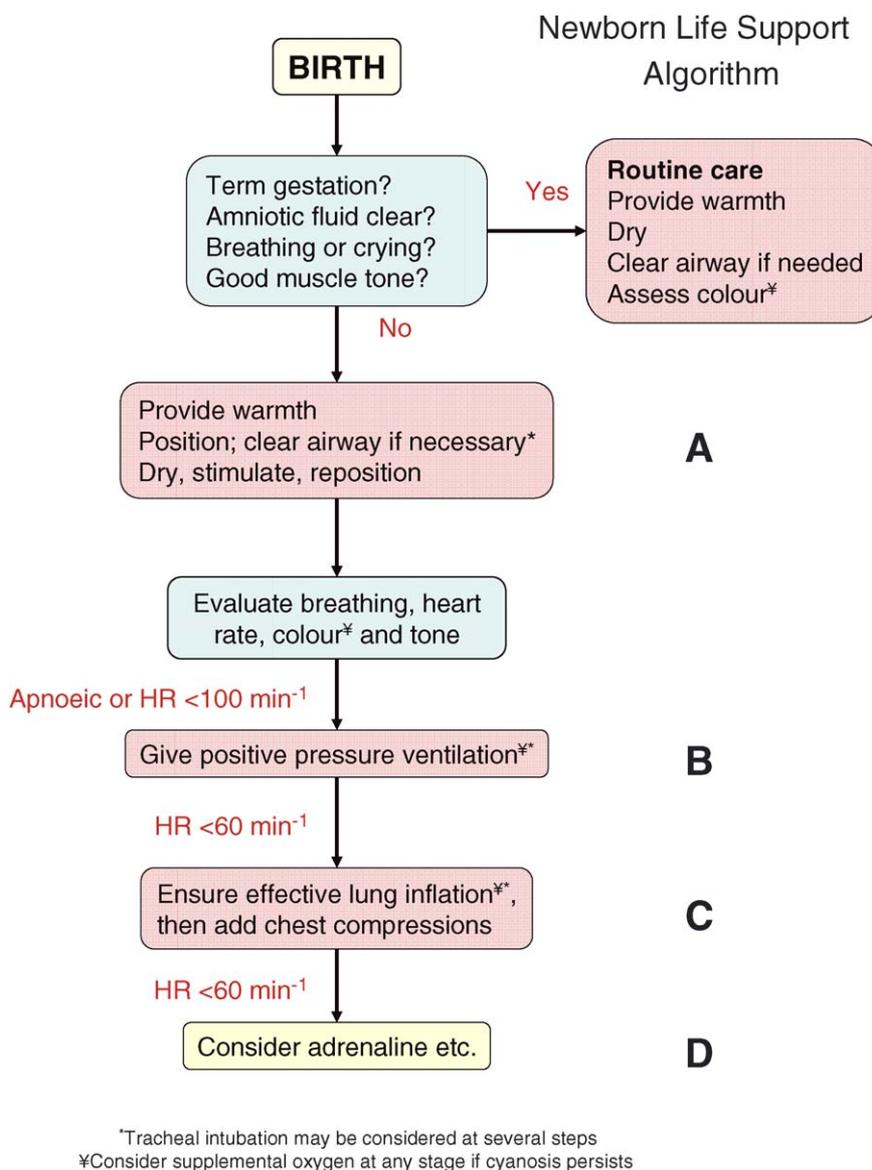
For the first few breaths maintain the initial inflation pressure for 2—3 s. This will help lung expansion. Most babies needing resuscitation at birth will respond with a rapid increase in heart rate within 30 s of lung inflation. If the heart rate increases but the baby is not breathing adequately, continue ventilation at a rate of about 30 breaths min^{-1}, allowing approximately 1 s for each inflation, until there is adequate spontaneous breathing.

Adequate passive ventilation is usually indicated by either a rapidly increasing heart rate or a heart rate that is maintained faster than 100 beats min^{-1}. If the baby does not respond in this way, the most likely reason is inadequate airway control or ventilation. Look for passive chest movement in time with inflation efforts; if these are present, then lung aeration has been achieved. If these are absent, then airway control and lung aeration have not been confirmed. Without adequate lung aeration chest compressions will be ineffective; therefore, confirm lung aeration before progressing to circulatory support. Some practitioners will ensure lung aeration by tracheal intubation, but this requires training and experience to be achieved effectively. If this skill is not available and the heart rate is decreasing, re-evaluate airway position and deliver aeration breaths while summoning a colleague with intubation skills.

Continue ventilatory support until the baby has established normal regular breathing.

Circulatory support

Circulatory support with chest compressions is effective only if the lungs have first been successfully inflated. Give chest compressions if the heart rate is less than 60 beats min^{-1} despite adequate ventilation. The optimal technique is to place the

Newborn Life Support
Algorithm

BIRTH

Term gestation?
Amniotic fluid clear?   Yes
Breathing or crying?
Good muscle tone?

Routine care
Provide warmth
Dry
Clear airway if needed
Assess colour¥

No

Provide warmth
Position; clear airway if necessary*   **A**
Dry, stimulate, reposition

Evaluate breathing, heart
rate, colour¥ and tone

Apnoeic or HR <100 min^{-1}

Give positive pressure ventilation¥*   **B**

HR <60 min^{-1}

Ensure effective lung inflation¥*,
then add chest compressions   **C**

HR <60 min^{-1}

Consider adrenaline etc.   **D**

*Tracheal intubation may be considered at several steps
¥Consider supplemental oxygen at any stage if cyanosis persists

Figure 6.10 Newborn life support algorithm.

two thumbs side by side over the lower third of the sternum, with the fingers encircling the torso and supporting the back (Figure 6.13).[21,22,25,278,279] The lower third of the sternum is compressed to a
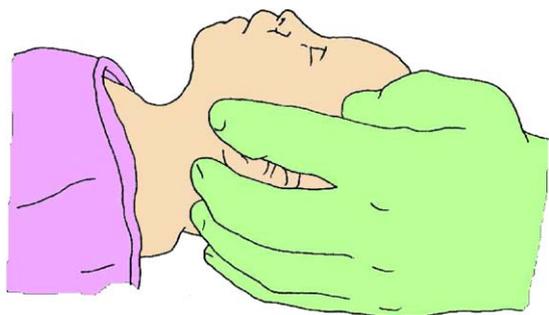
Figure 6.11 Newborn head in neutral position. © 2005 Resuscitation Council (UK).

depth of approximately one third of the anterior-posterior diameter of the chest. A compression to relaxation ratio with a slightly shorter compression than relaxation phase offers theoretical advantages for blood flow in the very young infant.[280] Do not lift the thumbs off the sternum during the relaxation phase, but allow the chest wall to return to its relaxed position between compressions. Use a 3:1 ratio of compressions to ventilations, aiming to achieve approximately 120 events min^{-1}, i.e. approximately 90 compressions and 30 breaths. However, the quality of the compressions and breaths are more important than the rate.[281]

Check the heart rate after about 30 s and periodically thereafter. Discontinue chest compressions when the spontaneous heart rate is faster then 60 beats min^{-1}.
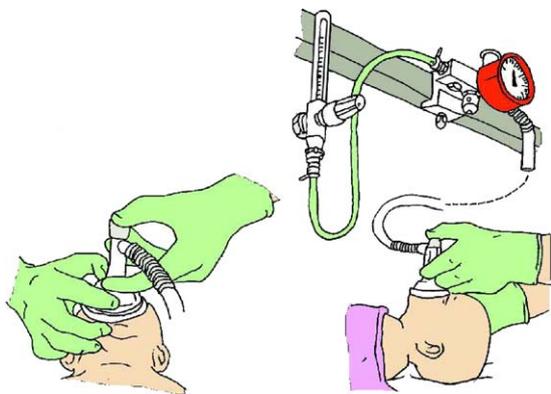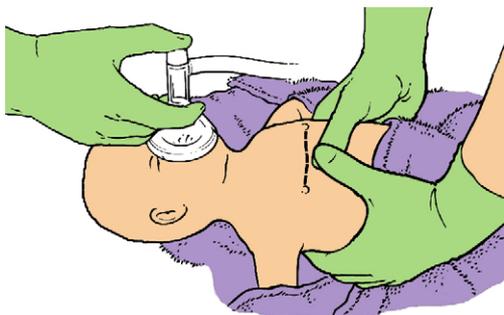
Figure 6.12 Airway and ventilation — newborn. © 2005 Resuscitation Council (UK).

Drugs

Drugs are rarely indicated in resuscitation of the newborn infant. Bradycardia in the newborn infant is usually caused by inadequate lung inflation or profound hypoxia, and establishing adequate ventilation is the most important step to correct it. However, if the heart rate remains less than 60 beats min^{-1} despite adequate ventilation and chest compressions, drugs may be needed. These drugs are presumed to exert their effect by their action on the heart and are being given because cardiac function is inadequate. It is therefore necessary to give them as close to the heart as possible, ideally via a rapidly inserted umbilical venous catheter (Figure 6.14).

Adrenaline

Despite the lack of human data, it is reasonable to continue to use adrenaline when adequate ventilation and chest compressions have failed to increase the heart rate above 60 beats min^{-1}. Use the IV route as soon as venous access is established. The recommended IV dose is 10—30 mcg kg^{-1}. The tracheal route is not recommended (see below) but, if

Figure 6.13 Ventilation and chest compression — newborn. © 2005 Resuscitation Council (UK).
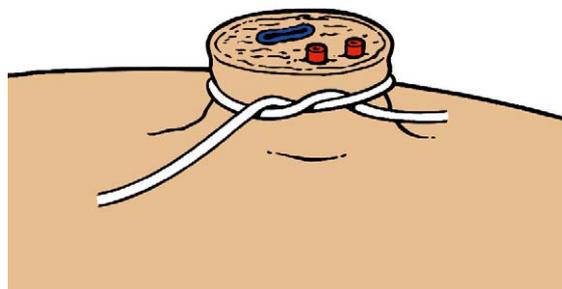
Figure 6.14 Newborn umbilical cord showing the arteries and veins. © 2005 Resuscitation Council (UK).

it is used, it is highly likely that doses of 30 mcg kg^{-1} or less are ineffective. Try a higher dose (up to 100 mcg kg^{-1}). The safety of these higher tracheal doses has not been studied. Do not give high IV doses.

Bicarbonate

If effective spontaneous cardiac output is not restored despite adequate ventilation and adequate chest compressions, reversing intracardiac acidosis may improve myocardial function and achieve a spontaneous circulation. Give 1—2 mmol kg^{-1} IV.

Fluids

Consider volume expansion when there has been suspected blood loss or the infant appears to be in shock (pale, poor perfusion, weak pulse) and has not responded adequately to other resuscitative measures. In the absence of suitable blood (i.e., irradiated and leucocyte-depleted group O Rh-negative blood) isotonic crystalloid rather than albumin is the solution of choice for restoring intravascular volume in the delivery room. Give a bolus of 10—20 ml kg^{-1}.

Stopping resuscitation

Local and national committees will determine the indications for stopping resuscitation. However, data from infants without signs of life from birth lasting at least 10 min or longer show either high mortality or severe neurodevelopmental disability. After 10 min of continuous and adequate resuscitation efforts, discontinuation of resuscitation may be justified if there are no signs of life.

Communication with the parents

It is vitally important that the team caring for the newborn baby informs the parents of the baby's progress. At delivery adhere to the routine local plan and, if possible, hand the baby to the mother at the earliest opportunity. If resuscitation is required, inform the parents of the procedures being undertaken and why they are required.

Decisions to discontinue resuscitation ideally should involve senior paediatric staff. Whenever possible, the decision to attempt resuscitation of an extremely preterm baby should be taken in close consultation with the parents and senior paediatric and obstetric staff. Where a difficulty has been foreseen, for example in the case of severe congenital malformation, the options and prognosis should be discussed with the parents, midwives, obstetricians and birth attendants before delivery.

All discussions and decisions should be carefully recorded in the mother's notes before delivery and also in the baby's records after birth.

Specific questions addressed at the 2005 Consensus Conference

Maintaining normal temperature in preterm infants

Significantly, preterm babies are likely to become hypothermic despite careful application of the traditional techniques for keeping them warm (drying, wrapping and placing under radiant heat).[282] Several randomised controlled trials and observational studies have shown that placing preterm babies under radiant heat and then covering the babies with food-grade plastic wrapping, without drying them, significantly improves temperature on admission to intensive care compared with traditional techniques.[283–285] The baby's temperature must be monitored closely because of the small but described risk of hyperthermia with this technique.[286] All resuscitation procedures, including intubation, chest compression and insertion of lines, can be achieved with the plastic cover in place.

Infants born to febrile mothers have been reported to have a higher incidence of perinatal respiratory depression, neonatal seizures, early mortality and cerebral palsy.[286–288] Animal studies indicate that hyperthermia during or following ischaemia is associated with a progression of cerebral injury.[233,289] Hyperthermia should be avoided.

Meconium

Five years ago, a large randomised controlled study showed that attempting to intubate and aspirate inhaled meconium from the tracheas of vigorous infants at birth was not beneficial.[290] A more recent large multicentre randomised controlled study has now shown that suctioning meconium from the baby's nose and mouth before delivery of the baby's chest (intrapartum suctioning) does not reduce the incidence or severity of meconium aspiration syndrome.[291] Intrapartum suctioning is therefore no longer recommended. Intubation and suction of meconium from the trachea of non-vigorous infants born through meconium-stained liquor is still recommended.

Air or 100% oxygen

Several studies in recent years have raised concerns about the potential adverse effects of 100% oxygen on respiratory physiology and cerebral circulation, and the potential tissue damage from oxygen free radicals. There are also concerns about tissue damage from oxygen deprivation during and following asphyxia. Studies examining blood pressure, cerebral perfusion, and various biochemical measures of cell damage in asphyxiated animals resuscitated with 100% versus 21% oxygen, have shown conflicting results.[292–296] One study of preterm infants (below 33 weeks' gestation) exposed to 80% oxygen found lower cerebral blood flow when compared with those stabilised with 21% oxygen.[297] Some animal data indicate the opposite effect, i.e. reduced blood pressure and cerebral perfusion with air versus 100% oxygen.[292] Meta-analysis of four human studies demonstrated a reduction in mortality and no evidence of harm in infants resuscitated with air versus those resuscitated with 100% oxygen. However, there are several significant concerns about the methodology of these studies, and these results should be interpreted with caution.[80,298]

At present, the standard approach to resuscitation is to use 100% oxygen. Some clinicians may elect to start resuscitation with an oxygen concentration less than 100%, including some who may start with air. Evidence suggests that this approach may be reasonable. However, where possible, ensure supplemental oxygen is available for use if there is no rapid improvement following successful lung aeration. If supplemental oxygen is not readily available, ventilate the lungs with air. Supplemental oxygen is recommended for babies who are breathing but have central cyanosis.

Monitoring the oxygen saturation of babies undergoing resuscitation may be useful, but studies have shown that term healthy newborns may take more than 10 min to achieve a preductal oxygen saturation above 95% and nearly an hour to achieve this post-ductally.[299–301] Giving a variable concentration of oxygen guided by pulse oximetry may improve the ability to achieve 'normal' oxygen saturation values while more quickly avoiding 'hyperoxia', but the definition of these two terms in the baby at birth are undetermined. Oxygen is a drug, and oxidant injury is theoretically more likely in preterm infants.

Initial breaths and assisted ventilation

In term infants, spontaneous or assisted initial inflations create a functional residual capacity (FRC).[302–309] The optimum pressure, inflation time and flow required to establish an effective FRC have not been determined. Average initial peak inflating pressures of 30–40 cmH$_2$O (inflation time undefined) usually ventilate unresponsive term infants successfully.[305–307,309] Assisted ventilation rates of 30–60 breaths min^{-1} are used commonly, but the relative efficacy of various rates has not been investigated.

The primary measure of adequate initial ventilation is prompt increase in heart rate; assess passive chest wall movement if the heart rate does not increase. The initial peak inflating pressures needed are variable and unpredictable, and should be individualised to achieve an increase in heart rate or movement of the chest with each breath. Where pressure is being monitored, an initial inflation pressure of 20 cmH$_2$O may be effective, but 30–40 cmH$_2$O or higher may be required in some term babies. If pressure is not being monitored but merely limited by a non-adjustable 'blow-off' valve, use the minimum inflation required to achieve an increase in heart rate. There is insufficient evidence to recommend an optimum inflation time. In summary, provide artificial ventilation at 30–60 breaths min^{-1} to achieve or maintain a heart rate higher than 100 beats min^{-1} promptly.

Assisted ventilation of preterm infants

Animal studies show that preterm lungs are easily damaged by large volume inflations immediately after birth,[310] and that maintaining a positive end-expiratory pressure (PEEP) immediately after birth protects against lung damage. PEEP also improves lung compliance and gas exchange.[311,312] Human case series show that most apnoeic preterm infants can be ventilated with an initial inflation pressure of 20–25 cmH$_2$O, though some infants appear to require a higher pressure.[313,314]

When ventilating preterm infants, very obvious passive chest wall movement may indicate excessive tidal volumes and should be avoided. Monitoring of pressure may help to provide consistent inflations and avoid high pressures. If positive-pressure ventilation is required, an initial inflation pressure of 20–25 cmH$_2$O is adequate for most preterm infants. If a prompt increase in heart rate or chest movement is not obtained, higher pressures may be needed. If continued positive-pressure ventilation is required, PEEP may be beneficial. Continuous positive airway pressure (CPAP) in spontaneously breathing preterm infants following resuscitation may also be beneficial.[314]

Devices

Effective ventilation can be achieved with either a flow-inflating or self-inflating bag or with a T-piece mechanical device designed to regulate pressure.[315–317] The blow-off valves of self-inflating bags are flow dependent, and pressures generated may exceed the value specified by the manufacturer.[318] Target inflation pressures and long inspiratory times are achieved more consistently in mechanical models when using T-piece devices than when using bags,[319] although the clinical implications are not clear. More training is required to provide an appropriate pressure using flow-inflating bags compared with self-inflating bags.[320] A self-inflating bag, a flow-inflating bag or a T-piece mechanical device, all designed to regulate pressure or limit pressure applied to the airway, can be used to ventilate a newborn.

Laryngeal mask airways (LMAs) are effective for ventilating newborn near-term and full-term infants.[321,322] There are few data on the use of these devices in small preterm infants.[323,324] Three case series show that the LMA can provide effective ventilation in a time frame consistent with current resuscitation guidelines, although the babies being studied were not being resuscitated.[322,325,326] A randomised controlled trial found no clinically significant difference between the LMA and tracheal intubation when bag-mask ventilation was unsuccessful.[321] It is unclear whether the conclusions of this study can be generalized, since the LMA was inserted by experienced providers. Case reports suggest that when bag-mask ventilation has been unsuccessful and tracheal intubation is unfeasible or unsuccessful, the LMA may provide effective ventilation.[327–329] There is insufficient evidence to support the routine use of the LMA as the primary airway device for resuscitation at birth.

Table 6.1 Calculation of tracheal tube size and depth of insertion[a]

Child's weight (kg)	Gestation (weeks)	Tube size (mm ID)	Depth of insertion (cm)[a]
<1	<28	2.5	6.5—7
1—2	28—34	3.0	7—8
2—3	34—38	3.0/3.5	8—9
>3	>38	3.5/4.0	>9

[a] Depth of insertion from the upper lip can be estimated as insertion depth at lip (cm) = weight in kg + 6 cm.

There are also reservations concerning its effectiveness in the following situations

- when chest compressions are required
- for very low birth weight (VLBW) babies
- when the amniotic fluid is meconium stained

Confirming tracheal tube placement

Tracheal intubation may be considered at several points during neonatal resuscitation

- when suctioning to remove meconium or other tracheal blockage is required
- if bag-mask ventilation is ineffective or prolonged
- when chest compressions are performed
- in special circumstances (e.g., congenital diaphragmatic hernia or birth weight below 1000 g)

The use and timing of tracheal intubation will depend on the skill and experience of the available resuscitators.

Following tracheal intubation and intermittent positive pressure, a prompt increase in heart rate is the best indicator that the tube is in the tracheo-bronchial tree.[330] Exhaled CO_2 detection is effective for confirmation of tracheal tube placement in infants, including VLBW infants.[331–334] Detection of exhaled CO_2 in patients with adequate cardiac output confirms placement of the tube within the trachea, whereas failure to detect exhaled CO_2 strongly suggests oesophageal intubation.[331,333] Poor or absent pulmonary blood flow or tracheal obstruction may prevent detection of exhaled CO_2 despite correct tracheal tube placement. Tracheal tube placement is identified correctly in nearly all patients who are not in cardiac arrest[99]; however, in critically ill infants with poor cardiac output, inability to detect exhaled CO_2 despite correct placement may lead to unnecessary extubation. Other clinical indicators of correct tracheal tube placement include evaluation of condensed humidified gas during exhalation and presence or absence of chest movement, but these have not been evaluated systematically in newborn babies.

Tracheal tube placement (Table 6.1) must be assessed visually during intubation and, in most cases, will be confirmed by a rapid increase in heart rate on ventilating via the tracheal tube. If the heart rate remains slow, incorrect tube placement is the most likely cause. Check tube placement either visually or by detection of exhaled CO_2.

Route and dose of adrenaline

There are no placebo-controlled studies that have evaluated the use of adrenaline at any stage in human neonatal resuscitation. A paediatric study[148] and newborn animal studies[335,336] showed no benefit and a trend toward reduced survival and worse neurological status after high-dose IV adrenaline (100 mcg kg^{-1}) during resuscitation. Animal and adult human studies demonstrate that, when given via the trachea, considerably higher doses of adrenaline than currently recommended are required to achieve adequate plasma concentrations.[337–339] One neonatal animal study using the currently recommended dose of tracheal adrenaline (10 mcg kg^{-1}) showed no benefit.[126] One neonatal cohort study of nine preterm babies requiring resuscitation showed that tracheal adrenaline was absorbed, but these workers used 7—25 times the dose recommended currently.[340]

Post-resuscitation care

Babies who have required resuscitation may deteriorate. Once adequate ventilation and circulation are established, the infant should be maintained in or transferred to an environment in which close monitoring and anticipatory care can be provided.

Glucose

Hypoglycaemia was associated with adverse neurological outcome in a neonatal animal model of asphyxia and resuscitation.[341] Newborn animals which were hypoglycaemic at the time of

an anoxic or hypoxic-ischaemic insult had larger areas of cerebral infarction and/or decreased survival compared with controls.[342,343] One clinical study demonstrated an association between hypoglycaemia and poor neurological outcome following perinatal asphyxia.[344] No clinical neonatal studies have investigated the relationship between hyperglycaemia and neurological outcome, although in adults hyperglycaemia is associated with a worse outcome.[345] The range of blood glucose concentration that is associated with the least brain injury following asphyxia and resuscitation cannot be defined on available evidence. Infants who require significant resuscitation should be monitored and treated to maintain blood glucose within the normal range.

Induced hypothermia

In a multicentre trial involving newborns with suspected asphyxia (indicated by need for resuscitation at birth, metabolic acidosis and early encephalopathy), selective head cooling (34.5 °C) was associated with a non-significant reduction in the number of survivors with severe disability at 18 months, but a significant benefit in the subgroup with moderate encephalopathy as judged by amplitude-integrated electroencephalogram.[220] Infants with severe electroencephalographic suppression and seizures did not benefit from treatment.[346] A second, small, controlled pilot study in asphyxiated infants with early induced systemic hypothermia resulted in fewer deaths and disabilities at 12 months. Modest hypothermia is associated with bradycardia and elevated blood pressure that do not usually require treatment, but a rapid increase in body temperature may cause hypotension.[347] Profound hypothermia (core temperature below 33 °C) may cause arrhythmia, bleeding, thrombosis and sepsis, but studies so far have not reported these complications in infants treated with modest hypothermia.[220,348]

There are insufficient data to recommend routine use of modest systemic or selective cerebral hypothermia following resuscitation of infants with suspected asphyxia. Further clinical trials are needed to determine which infants benefit most and which method of cooling is most effective.

Withholding or discontinuing resuscitation

Mortality and morbidity for newborns varies according to region and to availability of resources.[349] Social science studies indicate that parents desire a larger role in decisions to resuscitate and to continue life support in severely compromised babies.[350] There is considerable variability among providers about the benefits and disadvantages of aggressive therapies in such babies.[351,352]

Withholding resuscitation

It is possible to identify conditions associated with high mortality and poor outcome, where withholding resuscitation may be considered reasonable, particularly when there has been the opportunity for discussion with parents.[282,353] A consistent and coordinated approach to individual cases by the obstetric and neonatal teams and the parents is an important goal. Withholding resuscitation and discontinuation of life-sustaining treatment during or following resuscitation are considered by many to be ethically equivalent, and clinicians should not be hesitant to withdraw support when the possibility of functional survival is highly unlikely. The following guidelines must be interpreted according to current regional outcomes.

- Where gestation, birth weight and/or congenital anomalies are associated with almost certain early death, and unacceptably high morbidity is likely among the rare survivors, resuscitation is not indicated. Examples from the published literature include extreme prematurity (gestational age <23 weeks and/or birthweight <400 g), and anomalies such as anencephaly and confirmed trisomy 13 or 18.
- Resuscitation is nearly always indicated in conditions associated with a high survival rate and acceptable morbidity. This will generally include babies with gestational age of 25 weeks or above (unless there is evidence of fetal compromise such as intrauterine infection or hypoxia-ischaemia) and those with most congenital malformations.
- In conditions associated with uncertain prognosis, where there is borderline survival and a relatively high rate of morbidity, and where the anticipated burden to the child is high, parental desires regarding resuscitation should be supported.

Withdrawing resuscitation efforts

Data from infants without signs of life from birth, lasting at least 10 min or longer, show either high mortality or severe neurodevelopmental disability.[354,355] After 10 min of uninterrupted and adequate resuscitation efforts, discontinuation of resuscitation may be justified if there are no signs of life.

References

1. Zideman D, Bingham R, Beattie T, et al. Guidelines for paediatric life support: a Statement by the Paediatric Life Support Working Party of the European Resuscitation Council. Resuscitation 1994;27:91—105.
2. European Resuscitation Council. Paediatric life support: (including the recommendations for resuscitation of babies at birth). Resuscitation 1998;37:95—6.
3. Phillips B, Zideman D, Garcia-Castrillo L, Felix M, Shwarz-Schwierin U. European Resuscitation Council Guidelines 2000 for Basic Paediatric Life Support. A statement from the Paediatric Life Support Working Group and approved by the Executive Committee of the European Resuscitation Council. Resuscitation 2001;48:223—9.
4. Phillips B, Zideman D, Garcia-Castrillo L, Felix M, Shwarz-Schwierin V. European Resuscitation Council Guidelines 2000 for Advanced Paediatric Life Support. A statement from Paediatric Life Support Working Group and approved by the Executive Committee of the European Resuscitation Council. Resuscitation 2001;48:231—4.
5. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care—an international consensus on science. Resuscitation 2000;46:3—430.
6. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science. Circulation 2000;102(Suppl. I):I-1—I-370.
7. International Liaison Committee on Resuscitation. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2005;67:157—341.
8. International Liaison Committee on Resuscitation. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Circulation, in press.
9. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. Resuscitation 1995;30:141—50.
10. Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury. Pediatrics 1994;94:137—42.
11. Berg RA, Hilwig RW, Kern KB, Ewy GA. Bystander'' chest compressions and assisted ventilation independently improve outcome from piglet asphyxial pulseless ''cardiac arrest''. Circulation 2000;101:1743—8.
12. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. Ann Emerg Med 1999;33:195—205.
13. Berg RA, Hilwig RW, Kern KB, Babar I, Ewy GA. Simulated mouth-to-mouth ventilation and chest compressions (bystander cardiopulmonary resuscitation) improves outcome in a swine model of prehospital pediatric asphyxial cardiac arrest. Crit Care Med 1999;27:1893—9.
14. Dorph E, Wik L, Steen PA. Effectiveness of ventilation-compression ratios 1:5 and 2:15 in simulated single rescuer paediatric resuscitation. Resuscitation 2002;54:259—64.
15. Turner I, Turner S, Armstrong V. Does the compression to ventilation ratio affect the quality of CPR: a simulation study. Resuscitation 2002;52:55—62.
16. Babbs CF, Kern KB. Optimum compression to ventilation ratios in CPR under realistic, practical conditions: a physiological and mathematical analysis. Resuscitation 2002;54:147—57.
17. Babbs CF, Nadkarni V. Optimizing chest compression to rescue ventilation ratios during one-rescuer CPR by professionals and lay persons: children are not just little adults. Resuscitation 2004;61:173—81.
18. Whyte SD, Wyllie JP. Paediatric basic life support: a practical assessment. Resuscitation 1999;41:153—217.
19. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. Ann Emerg Med 1992;21:1102—6.
20. Clements F, McGowan J. Finger position for chest compressions in cardiac arrest in infants. Resuscitation 2000;44:43—6.
21. Houri PK, Frank LR, Menegazzi JJ, Taylor R. A randomized, controlled trial of two-thumb vs two-finger chest compression in a swine infant model of cardiac arrest. Prehosp Emerg Care 1997;1:65—7.
22. David R. Closed chest cardiac massage in the newborn infant. Pediatrics 1988;81:552—4.
23. Dorfsman ML, Menegazzi JJ, Wadas RJ, Auble TE. Two-thumb vs two-finger chest compression in an infant model of prolonged cardiopulmonary resuscitation. Acad Emerg Med 2000;7:1077—82.
24. Whitelaw CC, Slywka B, Goldsmith LJ. Comparison of a two-finger versus two-thumb method for chest compressions by healthcare providers in an infant mechanical model. Resuscitation 2000;43:213—6.
25. Menegazzi JJ, Auble TE, Nicklas KA, Hosack GM, Rack L, Goode JS. Two-thumb versus two-finger chest compression during CRP in a swine infant model of cardiac arrest. Ann Emerg Med 1993;22:240—3.
26. Stevenson AG, McGowan J, Evans AL, Graham CA. CPR for children: one hand or two? Resuscitation 2005;64:205—8.
27. Gurnett CA, Atkins DL. Successful use of a biphasic waveform automated external defibrillator in a high-risk child. Am J Cardiol 2000;86:1051—3.
28. Konig B, Benger J, Goldsworthy L. Automatic external defibrillation in a 6 year old. Arch Dis Child 2005;90:310—1.
29. Atkinson E, Mikysa B, Conway JA, et al. Specificity and sensitivity of automated external defibrillator rhythm analysis in infants and children. Ann Emerg Med 2003;42:185—96.
30. Cecchin F, Jorgenson DB, Berul CI, et al. Is arrhythmia detection by automatic external defibrillator accurate for children? Sensitivity and specificity of an automatic external defibrillator algorithm in 696 pediatric arrhythmias. Circulation 2001;103:2483—8.
31. Samson R, Berg R, Bingham R. Pediatric Advanced Life Support Task Force ILCoR. Use of automated external defibrillators for children: an update. An advisory statement from the Pediatric Advanced Life Support Task Force of the International Liaison Committee on Resuscitation. Resuscitation 2003;57:237—43.
32. Jorgenson D, Morgan C, Snyder D, et al. Energy attenuator for pediatric application of an automated external defibrillator. Crit Care Med 2002;30:S145—7.
33. Tang W, Weil MH, Jorgenson D, et al. Fixed-energy biphasic waveform defibrillation in a pediatric model of cardiac arrest and resuscitation. Crit Care Med 2002;30:2736—41.
34. Berg RA, Chapman FW, Berg MD, et al. Attenuated adult biphasic shocks compared with weight-based monophasic shocks in a swine model of prolonged pediatric ventricular fibrillation. Resuscitation 2004;61:189—97.
35. Berg RA, Samson RA, Berg MD, et al. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. J Am Coll Cardiol 2005;45:786—9.

36. Rossano JQ, Schiff L, Kenney MA, Atkins DL. Survival is not correlated with defibrillation dosing in pediatric out-of-hospital ventricular fibrillation. Circulation 2003;108:320—1.

37. Clark CB, Zhang Y, Davies LR, Karlsson G, Kerber RE. Pediatric transthoracic defibrillation: biphasic versus monophasic waveforms in an experimental model. Resuscitation 2001;51:159—63.

38. Schneider T, Martens PR, Paschen H, et al. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. Optimized Response to Cardiac Arrest (ORCA) Investigators. Circulation 2000;102:1780—7.

39. Faddy SC, Powell J, Craig JC. Biphasic and monophasic shocks for transthoracic defibrillation: a meta analysis of randomised controlled trials. Resuscitation 2003;58:9—16.

40. van Alem AP, Chapman FW, Lank P, Hart AA, Koster RW. A prospective, randomised and blinded comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. Resuscitation 2003;58:17—24.

41. Redding JS. The choking controversy: critique of evidence on the Heimlich maneuver. Crit Care Med 1979;7:475—9.

42. International Liaison Committee on Resuscitation. Part 2. Adult Basic Life Support. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2005;67:187—200.

43. Sirbaugh PE, Pepe PE, Shook JE, et al. A prospective, population-based study of the demographics, epidemiology, management, and outcome of out-of-hospital pediatric cardiopulmonary arrest. Ann Emerg Med 1999;33:174—84.

44. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. Ann Emerg Med 1995;25:495—501.

45. Reis AG, Nadkarni V, Perondi MB, Grisi S, Berg RA. A prospective investigation into the epidemiology of in-hospital pediatric cardiopulmonary resuscitation using the international Utstein reporting style. Pediatrics 2002;109:200—9.

46. Young KD, Gausche-Hill M, McClung CD, Lewis RJ. A prospective, population-based study of the epidemiology and outcome of out-of-hospital pediatric cardiopulmonary arrest. Pediatrics 2004;114:157—64.

47. Richman PB, Nashed AH. The etiology of cardiac arrest in children and young adults: special considerations for ED management. Am J Emerg Med 1999;17:264—70.

48. Engdahl J, Bang A, Karlson BW, Lindqvist J, Herlitz J. Characteristics and outcome among patients suffering from out of hospital cardiac arrest of non-cardiac aetiology. Resuscitation 2003;57:33—41.

49. Carcillo JA. Pediatric septic shock and multiple organ failure. Crit Care Clin 2003;19:413—40, viii.

50. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation 1996;33:107—16.

51. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. Resuscitation 2000;44:195—201.

52. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. Acad Emerg Med 2004;11:878—80.

53. Frederick K, Bixby E, Orzel MN, Stewart-Brown S, Willett K. Will changing the emphasis from 'pulseless' to 'no signs of circulation' improve the recall scores for effective life support skills in children? Resuscitation 2002;55:255—61.

54. Park C, Bahk JH, Ahn WS, Do SH, Lee KH. The laryngeal mask airway in infants and children. Can J Anaesth 2001;48:413—7.

55. Hedges JR, Dronen SC, Feero S, Hawkins S, Syverud SA, Shultz B. Succinylcholine-assisted intubations in prehospital care. Ann Emerg Med 1988;17:469—72.

56. Murphy-Macabobby M, Marshall WJ, Schneider C, Dries D. Neuromuscular blockade in aeromedical airway management. Ann Emerg Med 1992;21:664—8.

57. Sayre M, Weisgerber I. The use of neuromuscular blocking agents by air medical services. J Air Med Transp 1992;11:7—11.

58. Rose W, Anderson L, Edmond S. Analysis of intubations. Before and after establishment of a rapid sequence intubation protocol for air medical use. Air Med J 1994;13:475—8.

59. Sing RF, Reilly PM, Rotondo MF, Lynch MJ, McCans JP, Schwab CW. Out-of-hospital rapid-sequence induction for intubation of the pediatric patient. Acad Emerg Med 1996;3:41—5.

60. Ma OJ, Atchley RB, Hatley T, Green M, Young J, Brady W. Intubation success rates improve for an air medical program after implementing the use of neuromuscular blocking agents. Am J Emerg Med 1998;16:125—7.

61. Tayal V, Riggs R, Marx J, Tomaszewski C, Schneider R. Rapid-sequence intubation at an emergency medicine residency: success rate and adverse events during a two-year period. Acad Emerg Med 1999;6:31—7.

62. Wang HE, Sweeney TA, O'Connor RE, Rubinstein H. Failed prehospital intubations: an analysis of emergency department courses and outcomes. Prehosp Emerg Care 2001;5:134—41.

63. Sagarin MJ, Chiang V, Sakles JC, et al. Rapid sequence intubation for pediatric emergency airway management. Pediatr Emerg Care 2002;18:417—43.

64. Kaye K, Frascone RJ, Held T. Prehospital rapid-sequence intubation: a pilot training program. Prehosp Emerg Care 2003;7:235—40.

65. Wang HE, Kupas DF, Paris PM, Bates RR, Costantino JP, Yealy DM. Multivariate predictors of failed prehospital endotracheal intubation. Acad Emerg Med 2003;10:717—24.

66. Pepe P, Zachariah B, Chandra N. Invasive airway technique in resuscitation. Ann Emerg Med 1991;22:393—403.

67. Luten RC, Wears RL, Broselow J, et al. Length-based endotracheal tube and emergency equipment in pediatrics. Ann Emerg Med 1992;21:900—4.

68. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr 1994;125:57—62.

69. Khine HH, Corddry DH, Kettrick RG, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. Anesthesiology 1997;86:627—31.

70. Newth CJ, Rachman B, Patel N, Hammer J. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. J Pediatr 2004;144:333—7.

71. Mhanna MJ, Zamel YB, Tichy CM, Super DM. The ''air leak'' test around the endotracheal tube, as a predictor of postextubation stridor, is age dependent in children. Crit Care Med 2002;30:2639—43.

72. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. Ann Emerg Med 2001;37:32—7.

73. Gausche M, Lewis RJ, Stratton SJ, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. JAMA 2000;283:783—90.

74. Kelly JJ, Eynon CA, Kaplan JL, de Garavilla L, Dalsey WC. Use of tube condensation as an indicator of endotracheal tube placement. Ann Emerg Med 1998;31:575—8.

75. Andersen KH, Hald A. Assessing the position of the tracheal tube: the reliability of different methods. Anaesthesia 1989;44:984—5.

76. Andersen KH, Schultz-Lebahn T. Oesophageal intubation can be undetected by auscultation of the chest. Acta Anaesthesiol Scand 1994;38:580—2.

77. Hartrey R, Kestin IG. Movement of oral and nasal tracheal tubes as a result of changes in head and neck position. Anaesthesia 1995;50:682—7.

78. Van de Louw A, Cracco C, Cerf C, et al. Accuracy of pulse oximetry in the intensive care unit. Intensive Care Med 2001;27:1606—13.

79. Seguin P, Le Rouzo A, Tanguy M, Guillou YM, Feuillu A, Malledant Y. Evidence for the need of bedside accuracy of pulse oximetry in an intensive care unit. Crit Care Med 2000;28:703—6.

80. Tan A, Schulze A, O'Donnell CP, Davis PG. Air versus oxygen for resuscitation of infants at birth. Cochrane Database Syst Rev 2004:CD002273.

81. Ramji S, Rasaily R, Mishra PK, et al. Resuscitation of asphyxiated newborns with room air or 100% oxygen at birth: a multicentric clinical trial. Indian Pediatr 2003;40:510—7.

82. Vento M, Asensi M, Sastre J, Garcia-Sala F, Pallardo FV, Vina J. Resuscitation with room air instead of 100% oxygen prevents oxidative stress in moderately asphyxiated term neonates. Pediatrics 2001;107:642—7.

83. Saugstad OD. Resuscitation of newborn infants with room air or oxygen. Semin Neonatol 2001;6:233—9.

84. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. Crit Care Med 2004;32:S345—51.

85. Aufderheide TP, Sigurdsson G, Pirrallo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circulation 2004;109:1960—5.

86. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA 2005;293:299—304.

87. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. AMA 2005;293:305—10.

88. Abella BS, Sandbo N, Vassilatos P, et al. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. Circulation 2005;111:428—34.

89. Borke WB, Munkeby BH, Morkrid L, Thaulow E, Saugstad OD. Resuscitation with 100% O(2) does not protect the myocardium in hypoxic newborn piglets. Arch Dis Child Fetal Neonatal Ed 2004;89:F156—60.

90. Stockinger ZT, McSwain Jr NE. Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation. J Trauma 2004;56:531—6.

91. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care 2002;6:283—90.

92. Cooper A, DiScala C, Foltin G, Tunik M, Markenson D, Welborn C. Prehospital endotracheal intubation for severe head injury in children: a reappraisal. Semin Pediatr Surg 2001;10:3—6.

93. Bhende MS, Thompson AE, Cook DR, Saville AL. Validity of a disposable end-tidal CO_2 detector in verifying endotracheal tube placement in infants and children. Ann Emerg Med 1992;21:142—5.

94. Bhende MS, Thompson AE, Orr RA. Utility of an end-tidal carbon dioxide detector during stabilization and transport of critically ill children. Pediatrics 1992;89(pt 1):1042—4.

95. Bhende MS, LaCovey DC. End-tidal carbon dioxide monitoring in the prehospital setting. Prehosp Emerg Care 2001;5:208—13.

96. Ornato JP, Shipley JB, Racht EM, et al. Multicenter study of a portable, hand-size, colorimetric end-tidal carbon dioxide detection device. Ann Emerg Med 1992;21:518—23.

97. Gonzalez del Rey JA, Poirier MP, Digiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO_2 system in the detection of airway mishaps: an animal trial. Pediatr Emerg Care 2000;16:121—3.

98. Bhende MS, Thompson AE. Evaluation of an end-tidal CO_2 detector during pediatric cardiopulmonary resuscitation. Pediatrics 1995;95:395—9.

99. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. Am J Emerg Med 1996;14:349—50.

100. DeBehnke DJ, Hilander SJ, Dobler DW, Wickman LL, Swart GL. The hemodynamic and arterial blood gas response to asphyxiation: a canine model of pulseless electrical activity. Resuscitation 1995;30:169—75.

101. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. Ann Emerg Med 1990;19:1104—6.

102. Sharieff GQ, Rodarte A, Wilton N, Silva PD, Bleyle D. The self-inflating bulb as an esophageal detector device in children weighing more than twenty kilograms: a comparison of two techniques. Ann Emerg Med 2003;41:623—9.

103. Sharieff GQ, Rodarte A, Wilton N, Bleyle D. The self-inflating bulb as an airway adjunct: is it reliable in children weighing less than 20 kilograms? Acad Emerg Med 2003;10:303—8.

104. Poirier MP, Gonzalez Del-Rey JA, McAneney CM, DiGiulio GA. Utility of monitoring capnography, pulse oximetry, and vital signs in the detection of airway mishaps: a hyperoxemic animal model. Am J Emerg Med 1998;16:350—2.

105. Lillis KA, Jaffe DM. Prehospital intravenous access in children. Ann Emerg Med 1992;21:1430—4.

106. Kanter RK, Zimmerman JJ, Strauss RH, Stoeckel KA. Pediatric emergency intravenous access. Evaluation of a protocol. Am J Dis Child 1986;140:132—4.

107. Banerjee S, Singhi SC, Singh S, Singh M. The intraosseous route is a suitable alternative to intravenous route for fluid resuscitation in severely dehydrated children. Indian Pediatr 1994;31:1511—20.

108. Glaeser PW, Hellmich TR, Szewczuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. Ann Emerg Med 1993;22:1119—24.

109. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. J Pediatr Surg 1993;28:158—61.

110. Orlowski JP, Julius CJ, Petras RE, Porembka DT, Gallagher JM. The safety of intraosseous infusions: risks of fat and bone marrow emboli to the lungs. Ann Emerg Med 1989;18:1062—7.

111. Orlowski JP, Porembka DT, Gallagher JM, Lockrem JD, VanLente F. Comparison study of intraosseous, central intravenous, and peripheral intravenous infusions of emergency drugs. Am J Dis Child 1990;144:112—7.

112. Abe KK, Blum GT, Yamamoto LG. Intraosseous is faster and easier than umbilical venous catheterization in new-

born emergency vascular access models. Am J Emerg Med 2000;18:126—9.

113. Ellemunter H, Simma B, Trawoger R, Maurer H. Intraosseous lines in preterm and full term neonates. Arch Dis Child Fetal Neonatal Ed 1999;80:F74—5.

114. Cameron JL, Fontanarosa PB, Passalaqua AM. A comparative study of peripheral to central circulation delivery times between intraosseous and intravenous injection using a radionuclide technique in normovolemic and hypovolemic canines. J Emerg Med 1989;7:123—7.

115. Warren DW, Kissoon N, Sommerauer JF, Rieder MJ. Comparison of fluid infusion rates among peripheral intravenous and humerus, femur, malleolus, and tibial intraosseous sites in normovolemic and hypovolemic piglets. Ann Emerg Med 1993;22:183—6.

116. Brickman KR, Krupp K, Rega P, Alexander J, Guinness M. Typing and screening of blood from intraosseous access. Ann Emerg Med 1992;21:414—7.

117. Johnson L, Kissoon N, Fiallos M, Abdelmoneim T, Murphy S. Use of intraosseous blood to assess blood chemistries and hemoglobin during cardiopulmonary resuscitation with drug infusions. Crit Care Med 1999;27:1147—52.

118. Ummenhofer W, Frei FJ, Urwyler A, Drewe J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? Resuscitation 1994;27:123—8.

119. Kissoon N, Idris A, Wenzel V, Murphy S, Rush W. Intraosseous and central venous blood acid—base relationship during cardiopulmonary resuscitation. Pediatr Emerg Care 1997;13:250—3.

120. Abdelmoneim T, Kissoon N, Johnson L, Fiallos M, Murphy S. Acid-base status of blood from intraosseous and mixed venous sites during prolonged cardiopulmonary resuscitation and drug infusions. Crit Care Med 1999;27:1923—8.

121. Venkataraman ST, Orr RA, Thompson AE. Percutaneous infraclavicular subclavian vein catheterization in critically ill infants and children. J Pediatr 1988;113:480—5.

122. Fleisher G, Caputo G, Baskin M. Comparison of external jugular and peripheral venous administration of sodium bicarbonate in puppies. Crit Care Med 1989;17:251—4.

123. Hedges JR, Barsan WB, Doan LA, et al. Central versus peripheral intravenous routes in cardiopulmonary resuscitation. Am J Emerg Med 1984;2:385—90.

124. Neufeld JD, Marx JA, Moore EE, Light AI. Comparison of intraosseous, central, and peripheral routes of crystalloid infusion for resuscitation of hemorrhagic shock in a swine model. J Trauma 1993;34:422—8.

125. Stenzel JP, Green TP, Fuhrman BP, Carlson PE, Marchessault RP. Percutaneous femoral venous catheterizations: a prospective study of complications. J Pediatr 1989;114:411—5.

126. Kleinman ME, Oh W, Stonestreet BS. Comparison of intravenous and endotracheal epinephrine during cardiopulmonary resuscitation in newborn piglets. Crit Care Med 1999;27:2748—54.

127. Efrati O, Ben-Abraham R, Barak A, et al. Endobronchial adrenaline: should it be reconsidered? Dose response and haemodynamic effect in dogs. Resuscitation 2003;59:117—22.

128. Howard RF, Bingham RM. Endotracheal compared with intravenous administration of atropine. Arch Dis Child 1990;65:449—50.

129. Prengel AW, Lindner KH, Hahnel J, Ahnefeld FW. Endotracheal and endobronchial lidocaine administration: effects on plasma lidocaine concentration and blood gases. Crit Care Med 1991;19:911—5.

130. Crespo SG, Schoffstall JM, Fuhs LR, Spivey WH. Comparison of two doses of endotracheal epinephrine in a cardiac arrest model. Ann Emerg Med 1991;20:230—4.

131. Lee PL, Chung YT, Lee BY, Yeh CY, Lin SY, Chao CC. The optimal dose of atropine via the endotracheal route. Ma Zui Xue Za Zhi 1989;27:35—8.

132. Hahnel JH, Lindner KH, Schurmann C, Prengel A, Ahnefeld FW. Plasma lidocaine levels and PaO$_2$ with endobronchial administration: dilution with normal saline or distilled water? Ann Emerg Med 1990;19:1314—7.

133. Jasani MS, Nadkarni VM, Finkelstein MS, Mandell GA, Salzman SK, Norman ME. Effects of different techniques of endotracheal epinephrine administration in pediatric porcine hypoxic-hypercarbic cardiopulmonary arrest. Crit Care Med 1994;22:1174—80.

134. Steinfath M, Scholz J, Schulte am Esch J, Laer S, Reymann A, Scholz H. The technique of endobronchial lidocaine administration does not influence plasma concentration profiles and pharmacokinetic parameters in humans. Resuscitation 1995;29:55—62.

135. Carcillo JA, Fields AI. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. Crit Care Med 2002;30:1365—78.

136. Simma B, Burger R, Falk M, Sacher P, Fanconi S. A prospective, randomized, and controlled study of fluid management in children with severe head injury: lactated Ringer's solution versus hypertonic saline. Crit Care Med 1998;26:1265—70.

137. Rocha e Silva M. Hypertonic saline resuscitation. Medicina (B Aries) 1998; 58:393-402.

138. Katz LM, Wang Y, Ebmeyer U, Radovsky A, Safar P. Glucose plus insulin infusion improves cerebral outcome after asphyxial cardiac arrest. Neuroreport 1998;9:3363—7.

139. Longstreth Jr WT, Copass MK, Dennis LK, Rauch-Matthews ME, Stark MS, Cobb LA. Intravenous glucose after out-of-hospital cardiopulmonary arrest: a community-based randomized trial. Neurology 1993;43:2534—41.

140. Chang YS, Park WS, Ko SY, et al. Effects of fasting and insulin-induced hypoglycemia on brain cell membrane function and energy metabolism during hypoxia-ischemia in newborn piglets. Brain Res 1999;844:135—42.

141. Cherian L, Goodman JC, Robertson CS. Hyperglycemia increases brain injury caused by secondary ischemia after cortical impact injury in rats. Crit Care Med 1997;25:1378—83.

142. Paul T, Bertram H, Bokenkamp R, Hausdorf G. Supraventricular tachycardia in infants, children and adolescents: diagnosis, and pharmacological and interventional therapy. Paediatr Drugs 2000;2:171—81.

143. Losek JD, Endom E, Dietrich A, Stewart G, Zempsky W, Smith K. Adenosine and pediatric supraventricular tachycardia in the emergency department: multicenter study and review. Ann Emerg Med 1999;33:185—91.

144. Roberts JR, Greenburg MI, Knaub M, Baskin SI. Comparison of the pharmacological effects of epinephrine administered by the intravenous and endotracheal routes. JACEP 1978;7:260—4.

145. Zaritsky A. Pediatric resuscitation pharmacology. Members of the Medications in Pediatric Resuscitation Panel. Ann Emerg Med 1993;22(pt 2):445—55.

146. Manisterski Y, Vaknin Z, Ben-Abraham R, et al. Endotracheal epinephrine: a call for larger doses. Anesth Analg 2002;95:1037—41 [table of contents].

147. Patterson MD, Boenning DA, Klein BL, et al. The use of high-dose epinephrine for patients with out-of-hospital cardiopulmonary arrest refractory to prehospital interventions. Pediatr Emerg Care 2005;21:227—37.

148. Perondi MB, Reis AG, Paiva EF, Nadkarni VM, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. N Engl J Med 2004;350:1722—30.

149. Carpenter TC, Stenmark KR. High-dose epinephrine is not superior to standard-dose epinephrine in pediatric in-hospital cardiopulmonary arrest. Pediatrics 1997;99:403—8.

150. Dieckmann R, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. Pediatrics 1995;95:901—13.

151. Berg RA, Otto CW, Kern KB, et al. High-dose epinephrine results in greater early mortality after resuscitation from prolonged cardiac arrest in pigs: a prospective, randomized study. Crit Care Med 1994;22:282—90.

152. Rubertsson S, Wiklund L. Hemodynamic effects of epinephrine in combination with different alkaline buffers during experimental, open-chest, cardiopulmonary resuscitation. Crit Care Med 1993;21:1051—7.

153. Somberg JC, Timar S, Bailin SJ, et al. Lack of a hypotensive effect with rapid administration of a new aqueous formulation of intravenous amiodarone. Am J Cardiol 2004;93:576—81.

154. Yap S-C, Hoomtje T, Sreeram N. Polymorphic ventricular tachycardia after use of intravenous amiodarone for postoperative junctional ectopic tachycardia. Int J Cardiol 2000;76:245—7.

155. Dauchot P, Gravenstein JS. Effects of atropine on the electrocardiogram in different age groups. Clin Pharmacol Ther 1971;12:274—80.

156. Stulz PM, Scheidegger D, Drop LJ, Lowenstein E, Laver MB. Ventricular pump performance during hypocalcemia: clinical and experimental studies. J Thorac Cardiovasc Surg 1979;78:185—94.

157. van Walraven C, Stiell IG, Wells GA, Hebert PC, Vandemheen K, The OTAC Study Group. Do advanced cardiac life support drugs increase resuscitation rates from in-hospital cardiac arrest? Ann Emerg Med 1998;32:544—53.

158. Paraskos JA. Cardiovascular pharmacology III: atropine, calcium, calcium blockers, and (beta)-blockers. Circulation 1986;74:IV-86—9.

159. Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. The effectiveness of calcium chloride in refractory electromechanical dissociation. Ann Emerg Med 1985;14:626—9.

160. Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. Lack of effectiveness of calcium chloride in refractory asystole. Ann Emerg Med 1985;14:630—2.

161. Srinivasan V, Spinella PC, Drott HR, Roth CL, Helfaer MA, Nadkarni V. Association of timing, duration, and intensity of hyperglycemia with intensive care unit mortality in critically ill children. Pediatr Crit Care Med 2004;5:329—36.

162. Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. Mayo Clin Proc 2004;79:992—1000.

163. Losek JD. Hypoglycemia and the ABC'S (sugar) of pediatric resuscitation. Ann Emerg Med 2000;35:43—6.

164. Finney SJ, Zekveld C, Elia A, Evans TW. Glucose control and mortality in critically ill patients. Jama 2003;290:2041—7.

165. Allegra J, Lavery R, Cody R, et al. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. Resuscitation 2001;49:245—9.

166. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. Circulation 1988;77:392—7.

167. Lokesh L, Kumar P, Murki S, Narang A. A randomized controlled trial of sodium bicarbonate in neonatal resuscitation-effect on immediate outcome. Resuscitation 2004;60:219—23.

168. Bar-Joseph G, Abramson NS, Kelsey SF, Mashiach T, Craig MT, Safar P. Improved resuscitation outcome in emergency medical systems with increased usage of sodium bicarbonate during cardiopulmonary resuscitation. Acta Anaesthesiol Scand 2005;49:6—15.

169. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med 2002;346:884—90.

170. Walsh EP, Saul JP, Sholler GF, et al. Evaluation of a staged treatment protocol for rapid automatic junctional tachycardia after operation for congenital heart disease. J Am Coll Cardiol 1997;29:1046—53.

171. Wang L. Congenital long QT syndrome: 50 years of electrophysiological research from cell to bedside. Acta Cardiologica 2003;58:133—8.

172. Singh BN, Kehoe R, Woosley RL, Scheinman M, Quart B, Sotalol Multicenter Study Group. Multicenter trial of sotalol compared with procainamide in the suppression of inducible ventricular tachycardia: a double-blind, randomized parallel evaluation. Am Heart J 1995;129:87—97.

173. Luedtke SA, Kuhn RJ, McCaffrey FM. Pharmacologic management of supraventricular tachycardias in children, part 2: atrial flutter, atrial fibrillation, and junctional and atrial ectopic tachycardia. Ann Pharmacother 1997;31:1347—59.

174. Luedtke SA, Kuhn RJ, McCaffrey FM. Pharmacologic management of supraventricular tachycardias in children. Part 1: Wolff-Parkinson-White and atrioventricular nodal reentry. Ann Pharmacother 1997;31:1227—43.

175. Mandapati R, Byrum CJ, Kavey RE, et al. Procainamide for rate control of postsurgical junctional tachycardia. Pediatr Cardiol 2000;21:123—8.

176. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. J Formos Med Assoc 2000;99:766—70.

177. Holmes CL, Landry DW, Granton JT. Science review: Vasopressin and the cardiovascular system part 1—receptor physiology. Crit Care 2003;7:427—34.

178. Voelckel WG, Lurie KG, McKnite S, et al. Effects of epinephrine and vasopressin in a piglet model of prolonged ventricular fibrillation and cardiopulmonary resuscitation. Crit Care Med 2002;30:957—62.

179. Voelckel WG, Lurie KG, McKnite S, et al. Comparison of epinephrine and vasopressin in a pediatric porcine model of asphyxial cardiac arrest. Crit Care Med 2000;28:3777—83.

180. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. Resuscitation 2002;52:149—56.

181. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using ''adult'' electrode paddles. Pediatrics 1994;94:90—3.

182. Atkins DL, Sirna S, Kieso R, Charbonnier F, Kerber RE. Pediatric defibrillation: importance of paddle size in determining transthoracic impedance. Pediatrics 1988;82:914—8.

183. Bennetts SH, Deakin CD, Petley GW, Clewlow F. Is optimal paddle force applied during paediatric external defibrillation? Resuscitation 2004;60:29—32.

184. Deakin C, Bennetts S, Petley G, Clewlow F. What is the optimal paddle force for paediatric defibrillation? Resuscitation 2002;55:59.

185. Atkins DL, Hartley LL, York DK. Accurate recognition and effective treatment of ventricular fibrillation by automated external defibrillators in adolescents. Pediatrics 1998;101(pt 1):393—7.

186. Pierpont GL, Kruse JA, Nelson DH. Intra-arterial monitoring during cardiopulmonary resuscitation. Cathet Cardiovasc Diagn 1985;11:513—20.
187. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. Ann Emerg Med 1987;16:1107—11.
188. Mogayzel C, Quan L, Graves JR, Tiedeman D, Fahrenbruch C, Herndon P. Out-of-hospital ventricular fibrillation in children and adolescents: causes and outcomes. Ann Emerg Med 1995;25:484—91.
189. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. Characteristics and outcome among children suffering from out of hospital cardiac arrest in Sweden. Resuscitation 2005;64:37—40.
190. Berg RA. Role of mouth-to-mouth rescue breathing in bystander cardiopulmonary resuscitation for asphyxial cardiac arrest. Crit Care Med 2000;28(Suppl.):N193—5.
191. Appleton GO, Cummins RO, Larson MP, Graves JR. CPR and the single rescuer: at what age should you ''call first'' rather than ''call fast''? Ann Emerg Med 1995;25:492—4.
192. Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. Ann Emerg Med 1993;22:1652—8.
193. Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA 1999;281:1182—8.
194. Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. JAMA 2003;289:1389—95.
195. Jacobs IG, Finn JC, Oxer HF, Jelinek GA. CPR before defibrillation in out-of-hospital cardiac arrest: a randomized trial. Emerg Med Australas 2005;17:39—45.
196. Somberg JC, Bailin SJ, Haffajee CI, et al. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. Am J Cardiol 2002;90:853—9.
197. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. N Engl J Med 1999;341:871—8.
198. Perry JC, Fenrich AL, Hulse JE, Triedman JK, Friedman RA, Lamberti JJ. Pediatric use of intravenous amiodarone: efficacy and safety in critically ill patients from a multicenter protocol. J Am Coll Cardiol 1996;27:1246—50.
199. Cummins RO, Graves JR, Larsen MP, et al. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. N Engl J Med 1993;328:1377—82.
200. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. Arch Dis Child 1990;65:127—9.
201. Bianconi L, Castro AMD, Dinelli M, et al. Comparison of intravenously administered dofetilide versus amiodarone in the acute termination of atrial fibrillation and flutter. A multicentre, randomized, double-blind, placebo-controlled study. Eur Heart J 2000;21:1265—73.
202. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. Eur J Pediatr 2003;162:880—4.
203. Celiker A, Ceviz N, Ozme S. Effectiveness and safety of intravenous amiodarone in drug-resistant tachyarrhythmias of children. Acta Paediatrica Japonica 1998;40:567—72.
204. Dodge-Khatami A, Miller O, Anderson R, Gil-Jaurena J, Goldman A, de Leval M. Impact of junctional ectopic tachycardia on postoperative morbidity following repair of congenital heart defects. Eur J Cardio-Thoracic Surg 2002;21:255—9.
205. Figa FH, Gow RM, Hamilton RM, Freedom RM. Clinical efficacy and safety of intravenous Amiodarone in infants and children. Am J Cardiol 1994;74:573—7.
206. Hoffman TM, Bush DM, Wernovsky G, et al. Postoperative junctional ectopic tachycardia in children: incidence, risk factors, and treatment. Ann Thorac Surg 2002;74:1607—11.
207. Soult JA, Munoz M, Lopez JD, Romero A, Santos J, Tovaruela A. Efficacy and safety of intravenous amiodarone for short-term treatment of paroxysmal supraventricular tachycardia in children. Pediatr Cardiol 1995;16:16—9.
208. Benson Jr D, Smith W, Dunnigan A, Sterba R, Gallagher J. Mechanisms of regular wide QRS tachycardia in infants and children. Am J Cardiol 1982;49:1778—88.
209. Drago F, Mazza A, Guccione P, Mafrici A, Di Liso G, Ragonese P. Amiodarone used alone or in combination with propranolol: a very effective therapy for tachyarrhythmias in infants and children. Pediatr Cardiol 1998;19:445—9.
210. Benson DJ, Dunnigan A, Green T, Benditt D, Schneider S. Periodic procainamide for paroxysmal tachycardia. Circulation 1985;72:147—52.
211. Komatsu C, Ishinaga T, Tateishi O, Tokuhisa Y, Yoshimura S. Effects of four antiarrhythmic drugs on the induction and termination of paroxysmal supraventricular tachycardia. Jpn Circ J 1986;50:961—72.
212. Mandel WJ, Laks MM, Obayashi K, Hayakawa H, Daley W. The Wolff-Parkinson-White syndrome: pharmacologic effects of procaine amide. Am Heart J 1975;90:744—54.
213. Meldon SW, Brady WJ, Berger S, Mannenbach M. Pediatric ventricular tachycardia: a review with three illustrative cases. Pediatr Emerg Care 1994;10:294—300.
214. Shih JY, Gillette PC, Kugler JD, et al. The electrophysiologic effects of procainamide in the immature heart. Pediatr Pharmacol (New York) 1982;2:65—73.
215. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. Crit Care Med 1988;16:331—5.
216. Checchia PA, Sehra R, Moynihan J, Daher N, Tang W, Weil MH. Myocardial injury in children following resuscitation after cardiac arrest. Resuscitation 2003;57:131—7.
217. Hickey RW, Ferimer H, Alexander HL, et al. Delayed, spontaneous hypothermia reduces neuronal damage after asphyxial cardiac arrest in rats. Crit Care Med 2000;28:3511—6.
218. Hypothermia After Cardiac Arrest Study Aroup. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549—56.
219. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. N Engl J Med 2002;346:557—63.
220. Gluckman PD, Wyatt JS, Azzopardi D, et al. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. Lancet 2005;365:663—70.
221. Battin MR, Penrice J, Gunn TR, Gunn AJ. Treatment of term infants with head cooling and mild systemic hypothermia (35.0 degrees C and 34.5 degrees C) after perinatal asphyxia. Pediatrics 2003;111:244—51.
222. Compagnoni G, Pogliani L, Lista G, Castoldi F, Fontana P, Mosca F. Hypothermia reduces neurological damage in asphyxiated newborn infants. Biol Neonate 2002;82:222—7.
223. Gunn AJ, Gluckman PD, Gunn TR. Selective head cooling in newborn infants after perinatal asphyxia: a safety study. Pediatrics 1998;102:885—92.

224. Debillon T, Daoud P, Durand P, et al. Whole-body cooling after perinatal asphyxia: a pilot study in term neonates. Dev Med Child Neurol 2003;45:17—23.

225. Hachimi-Idrissi S, Corne L, Ebinger G, Michotte Y, Huyghens L. Mild hypothermia induced by a helmet device: a clinical feasibility study. Resuscitation 2001;51:275—81.

226. Bernard S, Buist M, Monteiro O, Smith K. Induced hypothermia using large volume, ice-cold intravenous fluid in comatose survivors of out-of-hospital cardiac arrest: a preliminary report. Resuscitation 2003;56:9—13.

227. Kliegel A, Losert H, Sterz F, et al. Cold simple intravenous infusions preceding special endovascular cooling for faster induction of mild hypothermia after cardiac arrest—a feasibility study. Resuscitation 2005;64:347—51.

228. Polderman KH, Peerdeman SM, Girbes AR. Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. J Neurosurg 2001;94:697—705.

229. Polderman KH. Application of therapeutic hypothermia in the intensive care unit. Opportunities and pitfalls of a promising treatment modality—Part 2. Practical aspects and side effects. Intensive Care Med 2004;30:757—69.

230. Zeiner A, Holzer M, Sterz F, et al. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. Arch Intern Med 2001;161:2007—12.

231. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. Intensive Care Med 1991;17:419—20.

232. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? Resuscitation 2001;49:273—7.

233. Coimbra C, Boris-Moller F, Drake M, Wieloch T. Diminished neuronal damage in the rat brain by late treatment with the antipyretic drug dipyrone or cooling following cerebral ischemia. Acta Neuropathol (Berl) 1996;92:447—53.

234. Coimbra C, Drake M, Boris-Moller F, Wieloch T. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an anti-inflammatory/antipyretic drug: evidence for chronic encephalopathic processes following ischemia. Stroke 1996;27:1578—85.

235. Gillis J, Dickson D, Rieder M, Steward D, Edmonds J. Results of inpatient pediatric resuscitation. Crit Care Med 1986;14:469—71.

236. Schindler MB, Bohn D, Cox PN, et al. Outcome of out-of-hospital cardiac or respiratory arrest in children. N Engl J Med 1996;335:1473—9.

237. Suominen P, Korpela R, Kuisma M, Silfvast T, Olkkola KT. Paediatric cardiac arrest and resuscitation provided by physician-staffed emergency care units. Acta Anaesthesiol Scand 1997;41:260—5.

238. Lopez-Herce J, Garcia C, Rodriguez-Nunez A, et al. Long-term outcome of paediatric cardiorespiratory arrest in Spain. Resuscitation 2005;64:79—85.

239. Lopez-Herce J, Garcia C, Dominguez P, et al. Characteristics and outcome of cardiorespiratory arrest in children. Resuscitation 2004;63:311—20.

240. Hazinski MF, Chahine AA, Holcomb III GW, Morris Jr JA. Outcome of cardiovascular collapse in pediatric blunt trauma. Ann Emerg Med 1994;23:1229—35.

241. Morris MC, Wernovsky G, Nadkarni VM. Survival outcomes after extracorporeal cardiopulmonary resuscitation instituted during active chest compressions following refractory in-hospital pediatric cardiac arrest. Pediatr Crit Care Med 2004;5:440—6.

242. Duncan BW, Ibrahim AE, Hraska V, et al. Use of rapid-deployment extracorporeal membrane oxygenation for the resuscitation of pediatric patients with heart disease after cardiac arrest. J Thorac Cardiovasc Surg 1998;116:305—11.

243. Parra DA, Totapally BR, Zahn E, et al. Outcome of cardiopulmonary resuscitation in a pediatric cardiac intensive care unit. Crit Care Med 2000;28:3296—300.

244. Idris AH, Berg RA, Bierens J, et al. Recommended guidelines for uniform reporting of data from drowning: the ''Utstein style''. Resuscitation 2003;59:45—57.

245. Bauchner H, Waring C, Vinci R. Parental presence during procedures in an emergency room: results from 50 observations. Pediatrics 1991;87:544—8.

246. Bauchner H, Vinci R, Waring C. Pediatric procedures: do parents want to watch? Pediatrics 1989;84:907—9 [comment].

247. Bauchner H, Zuckerman B. Cocaine, sudden infant death syndrome, and home monitoring. J Pediatr 1990;117:904—6.

248. Haimi-Cohen Y, Amir J, Harel L, Straussberg R, Varsano Y. Parental presence during lumbar puncture: anxiety and attitude toward the procedure. Clin Pediatr (Phila) 1996;35:2—4.

249. Sacchetti A, Lichenstein R, Carraccio CA, Harris RH. Family member presence during pediatric emergency department procedures. Pediatr Emerg Care 1996;12:268—71.

250. Boie ET, Moore GP, Brummett C, Nelson DR. Do parents want to be present during invasive procedures performed on their children in the emergency department? A survey of 400 parents. Ann Emerg Med 1999;34:70—4.

251. Taylor N, Bonilla L, Silver P, Sagy M. Pediatric procedure: do parents want to be present? Crit Care Med 1996;24:A131.

252. Powers KS, Rubenstein JS. Family presence during invasive procedures in the pediatric intensive care unit: a prospective study. Arch Pediatr Adolesc Med 1999;153:955—8.

253. Cameron JA, Bond MJ, Pointer SC. Reducing the anxiety of children undergoing surgery: parental presence during anaesthetic induction. J Paediatr Child Health 1996;32:51—6.

254. Merritt KA, Sargent JR, Osborn LM. Attitudes regarding parental presence during medical procedures. Am J Dis Child 1990;144:270—1.

255. Wolfram RW, Turner ED. Effects of parental presence during children's venipuncture. Acad Emerg Med 1996;3:58—64.

256. Jarvis AS. Parental presence during resuscitation: attitudes of staff on a paediatric intensive care unit. Intensive Crit Care Nurs 1998;14:3—7.

257. Meyers TA, Eichhorn DJ, Guzzetta CE. Do families want to be present during CPR? A retrospective survey. J Emerg Nurs 1998;24:400—5.

258. Doyle CJ, Post H, Burney RE, Maino J, Keefe M, Rhee KJ. Family participation during resuscitation: an option. Ann Emerg Med 1987;16:673—5.

259. Hanson C, Strawser D. Family presence during cardiopulmonary resuscitation: Foote Hospital emergency department's nine-year perspective. J Emerg Nurs 1992;18:104—6.

260. Robinson SM, Mackenzie-Ross S, Campbell Hewson GL, Egleston CV, Prevost AT. Psychological effect of witnessed resuscitation on bereaved relatives. Lancet 1998;352:614—7.

261. Meyers TA, Eichhorn DJ, Guzzetta CE, et al. Family presence during invasive procedures and resuscitation. Am J Nurs 2000;100:32—42 [quiz 3].

262. Beckman AW, Sloan BK, Moore GP, et al. Should parents be present during emergency department procedures on children, and who should make that decision? A survey of emergency physician and nurse attitudes. Acad Emerg Med 2002;9:154—8.

263. Eppich WJ, Arnold LD. Family member presence in the pediatric emergency department. Curr Opin Pediatr 2003;15:294—8.

264. Eichhorn DJ, Meyers TA, Mitchell TG, Guzzetta CE. Opening the doors: family presence during resuscitation. J Cardiovasc Nurs 1996;10:59—70.

265. International Liaison Committee on Resuscitation. Part 7. Neonatal Life Support. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations. Resuscitation 2005;67:293—303.

266. Resuscitation Council (UK). Resuscitation at birth. Newborn life support course provider manual. London, Resuscitation Council (UK); 2001.

267. Niermeyer S, Kattwinkel J, Van Reempts P, et al. International Guidelines for Neonatal Resuscitation: an excerpt from the Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science. Contributors and Reviewers for the Neonatal Resuscitation Guidelines. Pediatrics E2000;106:29.

268. Palme-Kilander C. Methods of resuscitation in low-Apgar-score newborn infants—a national survey. Acta Paediatr 1992;81:739—44.

269. British Paediatric Association Working Party. Neonatal Resuscitation. London: British Paediatric Association; 1993.

270. Dahm LS, James LS. Newborn temperature and calculated heat loss in the delivery room. Pediatrics 1972;49:504—13.

271. Stephenson J, Du J, Tk O. The effect if cooling on blood gas tensions in newborn infants. J Pediatr 1970;76:848—52.

272. Gandy GM, Adamsons Jr K, Cunningham N, Silverman WA, James LS. Thermal environment and acid—base homeostasis in human infants during the first few hours of life. J Clin Invest 1964;43:751—8.

273. Apgar V. A proposal for a new method of evaluation of the newborn infant. Curr Res Anesth Analg 1953:32.

274. Anonymous. Is the Apgar score outmoded? Lancet 1989;i:591—2.

275. Chamberlain G, Banks J. Assessment of the Apgar score. Lancet 1974;2:1225—8.

276. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. Resuscitation 2004;60:213—7.

277. Cordero Jr L, Hon EH. Neonatal bradycardia following nasopharyngeal stimulation. J Pediatr 1971;78:441—7.

278. Thaler MM, Stobie GH. An improved technique of external caridac compression in infants and young children. N Engl J Med 1963;269:606—10.

279. Todres ID, Rogers MC. Methods of external cardiac massage in the newborn infant. J Pediatr 1975;86:781—2.

280. Dean JM, Koehler RC, Schleien CL, et al. Improved blood flow during prolonged cardiopulmonary resuscitation with 30% duty cycle in infant pigs. Circulation 1991;84:896—904.

281. Whyte SD, Sinha AK, Wyllie JP. Neonatal resuscitation: a practical assessment. Resuscitation 1999;40:21—5.

282. Costeloe K, Hennessy E, Gibson AT, Marlow N, Wilkinson AR. The EPICure study: outcomes to discharge from hospital for infants born at the threshold of viability. Pediatrics 2000;106:659—71.

283. Vohra S, Frent G, Campbell V, Abbott M, Whyte R. Effect of polyethylene occlusive skin wrapping on heat loss in very low birth weight infants at delivery: a randomized trial. J Pediatr 1999;134:547—51.

284. Lenclen R, Mazraani M, Jugie M, et al. Use of a polyethylene bag: a way to improve the thermal environment of the premature newborn at the delivery room. Arch Pediatr 2002;9:238—44.

285. Bjorklund LJ, Hellstrom-Westas L. Reducing heat loss at birth in very preterm infants. J Pediatr 2000;137:739—40.

286. Vohra S, Roberts RS, Zhang B, Janes M, Schmidt B. Heat loss prevention (HeLP) in the delivery room: a randomized controlled trial of polyethylene occlusive skin wrapping in very preterm infants. J Pediatr 2004;145:750—3.

287. Lieberman E, Eichenwald E, Mathur G, Richardson D, Heffner L, Cohen A. Intrapartum fever and unexplained seizures in term infants. Pediatrics 2000;106:983—8.

288. Grether JK, Nelson KB. Maternal infection and cerebral palsy in infants of normal birth weight. JAMA 1997;278:207—11.

289. Dietrich WD, Alonso O, Halley M, Busto R. Delayed posttraumatic brain hyperthermia worsens outcome after fluid percussion brain injury: a light and electron microscopic study in rats. Neurosurgery 1996;38:533—41 [discussion 41].

290. Wiswell TE, Gannon CM, Jacob J, et al. Delivery room management of the apparently vigorous meconium-stained neonate: results of the multicenter, international collaborative trial. Pediatrics 2000;105:1—7.

291. Vain NE, Szyld EG, Prudent LM, Wiswell TE, Aguilar AM, Vivas NI. Oropharyngeal and nasopharyngeal suctioning of meconium-stained neonates before delivery of their shoulders: multicentre, randomised controlled trial. Lancet 2004;364:597—602.

292. Solas AB, Kutzsche S, Vinje M, Saugstad OD. Cerebral hypoxemia-ischemia and reoxygenation with 21% or 100% oxygen in newborn piglets: effects on extracellular levels of excitatory amino acids and microcirculation. Pediatr Crit Care Med 2001;2:340—5.

293. Solas AB, Kalous P, Saugstad OD. Reoxygenation with 100 or 21% oxygen after cerebral hypoxemia-ischemia-hypercapnia in newborn piglets. Biol Neonate 2004;85:105—11.

294. Solas AB, Munkeby BH, Saugstad OD. Comparison of short- and long-duration oxygen treatment after cerebral asphyxia in newborn piglets. Pediatr Res 2004;56:125—31.

295. Huang CC, Yonetani M, Lajevardi N, Delivoria-Papadopoulos M, Wilson DF, Pastuszko A. Comparison of postasphyxial resuscitation with 100% and 21% oxygen on cortical oxygen pressure and striatal dopamine metabolism in newborn piglets. J Neurochem 1995;64:292—8.

296. Kutzsche S, Ilves P, Kirkeby OJ, Saugstad OD. Hydrogen peroxide production in leukocytes during cerebral hypoxia and reoxygenation with 100% or 21% oxygen in newborn piglets. Pediatr Res 2001;49:834—42.

297. Lundstrom KE, Pryds O, Greisen G. Oxygen at birth and prolonged cerebral vasoconstriction in preterm infants. Arch Dis Child Fetal Neonatal Ed 1995;73:F81—6.

298. Davis PG, Tan A, O'Donnell CP, Schulze A. Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. Lancet 2004;364:1329—33.

299. Harris AP, Sendak MJ, Donham RT. Changes in arterial oxygen saturation immediately after birth in the human neonate. J Pediatr 1986;109:117—9.

300. Reddy VK, Holzman IR, Wedgwood JF. Pulse oximetry saturations in the first 6 hours of life in normal term infants. Clin Pediatr (Phila) 1999;38:87—92.

301. Toth B, Becker A, Seelbach-Gobel B. Oxygen saturation in healthy newborn infants immediately after birth measured by pulse oximetry. Arch Gynecol Obstet 2002;266:105—7.

302. Karlberg P, Koch G. Respiratory studies in newborn infants. III. Development of mechanics of breathing during the first week of life. A longitudinal study Acta Paediatr 1962;(Suppl. 135):121—9.

303. Vyas H, Milner AD, Hopkins IE. Intrathoracic pressure and volume changes during the spontaneous onset of respiration in babies born by cesarean section and by vaginal delivery. J Pediatr 1981;99:787—91.

304. Mortola JP, Fisher JT, Smith JB, Fox GS, Weeks S, Willis D. Onset of respiration in infants delivered by cesarean section. J Appl Physiol 1982;52:716—24.

305. Hull D. Lung expansion and ventilation during resuscitation of asphyxiated newborn infants. J Pediatr 1969;75:47—58.

306. Upton CJ, Milner AD. Endotracheal resuscitation of neonates using a rebreathing bag. Arch Dis Child 1991;66:39—42.

307. Vyas H, Milner AD, Hopkin IE, Boon AW. Physiologic responses to prolonged and slow-rise inflation in the resuscitation of the asphyxiated newborn infant. J Pediatr 1981;99:635—9.

308. Vyas H, Field D, Milner AD, Hopkin IE. Determinants of the first inspiratory volume and functional residual capacity at birth. Pediatr Pulmonol 1986;2:189—93.

309. Boon AW, Milner AD, Hopkin IE. Lung expansion, tidal exchange, and formation of the functional residual capacity during resuscitation of asphyxiated neonates. J Pediatr 1979;95:1031—6.

310. Ingimarsson J, Bjorklund LJ, Curstedt T, et al. Incomplete protection by prophylactic surfactant against the adverse effects of large lung inflations at birth in immature lambs. Intensive Care Med 2004;30:1446—53.

311. Nilsson R, Grossmann G, Robertson B. Bronchiolar epithelial lesions induced in the premature rabbit neonate by short periods of artificial ventilation. Acta Pathol Microbiol Scand [A] 1980;88:359—67.

312. Probyn ME, Hooper SB, Dargaville PA, et al. Positive end expiratory pressure during resuscitation of premature lambs rapidly improves blood gases without adversely affecting arterial pressure. Pediatr Res 2004;56:198—204.

313. Hird MF, Greenough A, Gamsu HR. Inflating pressures for effective resuscitation of preterm infants. Early Hum Dev 1991;26:69—72.

314. Lindner W, Vossbeck S, Hummler H, Pohlandt F. Delivery room management of extremely low birth weight infants: spontaneous breathing or intubation? Pediatrics 1999;103:961—7.

315. Allwood AC, Madar RJ, Baumer JH, Readdy L, Wright D. Changes in resuscitation practice at birth. Arch Dis Child Fetal Neonatal Ed 2003;88:F375—9.

316. Cole AF, Rolbin SH, Hew EM, Pynn S. An improved ventilator system for delivery-room management of the newborn. Anesthesiology 1979;51:356—8.

317. Hoskyns EW, Milner AD, Hopkin IE. A simple method of face mask resuscitation at birth. Arch Dis Child 1987;62:376—8.

318. Ganga-Zandzou PS, Diependaele JF, Storme L, et al. Is Ambu ventilation of newborn infants a simple question of finger-touch? Arch Pediatr 1996;3:1270—2.

319. Finer NN, Rich W, Craft A, Henderson C. Comparison of methods of bag and mask ventilation for neonatal resuscitation. Resuscitation 2001;49:299—305.

320. Kanter RK. Evaluation of mask-bag ventilation in resuscitation of infants. Am J Dis Child 1987;141:761—3.

321. Esmail N, Saleh M, et al. Laryngeal mask airway versus endotracheal intubation for Apgar score improvement in neonatal resuscitation. Egypt J Anesthesiol 2002;18:115—21.

322. Gandini D, Brimacombe JR. Neonatal resuscitation with the laryngeal mask airway in normal and low birth weight infants. Anesth Analg 1999;89:642—3.

323. Brimacombe J, Gandini D. Airway rescue and drug delivery in an 800 g neonate with the laryngeal mask airway. Paediatr Anaesth 1999;9:178.

324. Lonnqvist PA. Successful use of laryngeal mask airway in low-weight expremature infants with bronchopulmonary dysplasia undergoing cryotherapy for retinopathy of the premature. Anesthesiology 1995;83:422—4.

325. Paterson SJ, Byrne PJ, Molesky MG, Seal RF, Finucane BT. Neonatal resuscitation using the laryngeal mask airway. Anesthesiology 1994;80:1248—53.

326. Trevisanuto D, Ferrarese P, Zanardo V, Chiandetti L. Laryngeal mask airway in neonatal resuscitation: a survey of current practice and perceived role by anaesthesiologists and paediatricians. Resuscitation 2004;60:291—6.

327. Hansen TG, Joensen H, Henneberg SW, Hole P. Laryngeal mask airway guided tracheal intubation in a neonate with the Pierre Robin syndrome. Acta Anaesthesiol Scand 1995;39:129—31.

328. Osses H, Poblete M, Asenjo F. Laryngeal mask for difficult intubation in children. Paediatr Anaesth 1999;9:399—401.

329. Stocks RM, Egerman R, Thompson JW, Peery M. Airway management of the severely retrognathic child: use of the laryngeal mask airway. Ear Nose Throat J 2002;81:223—6.

330. Palme-Kilander C, Tunell R, Chiwei Y. Pulmonary gas exchange immediately after birth in spontaneously breathing infants. Arch Dis Child 1993;68:6—10.

331. Aziz HF, Martin JB, Moore JJ. The pediatric disposable end-tidal carbon dioxide detector role in endotracheal intubation in newborns. J Perinatol 1999;19:110—3.

332. Bhende MS, LaCovey D. A note of caution about the continuous use of colorimetric end-tidal CO_2 detectors in children. Pediatrics 1995;95:800—1.

333. Repetto JE, Donohue P-CP, Baker SF, Kelly L, Nogee LM. Use of capnography in the delivery room for assessment of endotracheal tube placement. J Perinatol 2001;21:284—7.

334. Roberts WA, Maniscalco WM, Cohen AR, Litman RS, Chhibber A. The use of capnography for recognition of esophageal intubation in the neonatal intensive care unit. Pediatr Pulmonol 1995;19:262—8.

335. Berg RA, Otto CW, Kern KB, et al. A randomized, blinded trial of high-dose epinephrine versus standard-dose epinephrine in a swine model of pediatric asphyxial cardiac arrest. Crit Care Med 1996;24:1695—700.

336. Burchfield DJ, Preziosi MP, Lucas VW, Fan J. Effects of graded doses of epinephrine during asphxia-induced bradycardia in newborn lambs. Resuscitation 1993;25:235—44.

337. Ralston SH, Voorhees WD, Babbs CF. Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: improved regional blood flow and resuscitation in dogs. Ann Emerg Med 1984;13:79—86.

338. Ralston SH, Tacker WA, Showen L, Carter A, Babbs CF. Endotracheal versus intravenous epinephrine during electromechanical dissociation with CPR in dogs. Ann Emerg Med 1985;14:1044—8.

339. Redding JS, Asuncion JS, Pearson JW. Effective routes of drug administration during cardiac arrest. Anesth Analg 1967;46:253—8.

340. Schwab KO, von Stockhausen HB. Plasma catecholamines after endotracheal administration of adrenaline during postnatal resuscitation. Arch Dis Child Fetal Neonatal Ed 1994;70:F213—7.

341. Brambrink AM, Ichord RN, Martin LJ, Koehler RC, Traystman RJ. Poor outcome after hypoxia-ischemia in newborns is associated with physiological abnormalities during early recovery. Possible relevance to secondary brain injury after head trauma in infants. Exp Toxicol Pathol 1999;51:151—62.

342. Vannucci RC, Vannucci SJ. Cerebral carbohydrate metabolism during hypoglycemia and anoxia in newborn rats. Ann Neurol 1978;4:73—9.

343. Yager JY, Heitjan DF, Towfighi J, Vannucci RC. Effect of insulin-induced and fasting hypoglycemia on peri-

natal hypoxic-ischemic brain damage. Pediatr Res 1992;31:138—42.

344. Salhab WA, Wyckoff MH, Laptook AR, Perlman JM. Initial hypoglycemia and neonatal brain injury in term infants with severe fetal acidemia. Pediatrics 2004;114:361—6.

345. Kent TA, Soukup VM, Fabian RH. Heterogeneity affecting outcome from acute stroke therapy: making reperfusion worse. Stroke 2001;32:2318—27.

346. Eicher DJ, Wagner CL, Katikaneni LP, et al. Moderate hypothermia in neonatal encephalopathy: efficacy outcomes. Pediatr Neurol 2005;32:11—7.

347. Thoresen M, Whitelaw A. Cardiovascular changes during mild therapeutic hypothermia and rewarming in infants with hypoxic-ischemic encephalopathy. Pediatrics 2000;106:92—9.

348. Shankaran S, Laptook A, Wright LL, et al. Whole-body hypothermia for neonatal encephalopathy: animal observations as a basis for a randomized, controlled pilot study in term infants. Pediatrics 2002;110:377—85.

349. De Leeuw R, Cuttini M, Nadai M, et al. Treatment choices for extremely preterm infants: an international perspective. J Pediatr 2000;137:608—16.

350. Lee SK, Penner PL, Cox M. Comparison of the attitudes of health care professionals and parents toward active treatment of very low birth weight infants. Pediatrics 1991;88:110—4.

351. Kopelman LM, Irons TG, Kopelman AE. Neonatologists judge the ''Baby Doe'' regulations. N Engl J Med 1988;318:677—83.

352. Sanders MR, Donohue PK, Oberdorf MA, Rosenkrantz TS, Allen MC. Perceptions of the limit of viability: neonatologists' attitudes toward extremely preterm infants. J Perinatol 1995;15:494—502.

353. Draper ES, Manktelow B, Field DJ, James D. Tables for predicting survival for preterm births are updated. BMJ 2003;327:872.

354. Jain L, Ferre C, Vidyasagar D, Nath S, Sheftel D. Cardiopulmonary resuscitation of apparently stillborn infants: survival and long-term outcome. J Pediatr 1991;118:778—82.

355. Haddad B, Mercer BM, Livingston JC, Talati A, Sibai BM. Outcome after successful resuscitation of babies born with apgar scores of 0 at both 1 and 5 minutes. Am J Obstet Gynecol 2000;182:1210—4.