

European Resuscitation Council Guidelines for Resuscitation 2010 Section 6. Paediatric life support

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Introduction

These guidelines on paediatric life support are based on two main principles: (1) the incidence of critical illness, particularly cardiopulmonary arrest, and injury in children is much lower than in adults; (2) most paediatric emergencies are served primarily by providers who are not paediatric specialists and who have limited paediatric emergency medical experience. Therefore, guidelines on paediatric life support must incorporate the best available scientific evidence but must also be simple and feasible. Finally, international guidelines need to acknowledge the variation in national and local emergency medical infrastructures and allow flexibility when necessary.

The process

The European Resuscitation Council (ERC) published guidelines for paediatric life support (PLS) in 1994, 1998, 2000 and 2005.^{1–5} The latter two were based on the International Consensus on Science published by the International Liaison Committee on Resuscitation (ILCOR).^{6–8} This process was repeated in 2009/2010, and the resulting Consensus on Science with Treatment Recommendations (CoSTR) was published simultaneously in Resuscitation, Circulation and Pediatrics.^{9,10} The PLS Working Party of the ERC has developed the ERC PLS Guidelines based on the 2010 CoSTR and supporting scientific literature. The guidelines for resuscitation of babies at birth are now covered in Section 7.¹¹

Summary of changes since the 2005 Guidelines

Guideline changes have been made in response to convincing new scientific evidence and to simplify teaching and retention. As before, there remains a paucity of good-quality evidence on paediatric resuscitation. Therefore to facilitate and support dissemination and implementation of the PLS Guidelines, changes have been made only if there is new, high-level scientific evidence or to ensure consistency with the adult guidelines. The feasibility of applying the same guidance for all adults and children remains a major topic of study. Major changes in these new guidelines include:

Recognition of cardiac arrest

Healthcare providers cannot reliably determine the presence or absence of a pulse in less than 10 s in infants or children.^{12,13} Therefore pulse palpation cannot be the sole determinant of cardiac arrest and the need for chest compressions. If the victim is unresponsive, not breathing normally, and there are no signs of life, lay rescuers should begin CPR. Healthcare providers should look for signs of life and if they are confident in the technique, they may add pulse palpation for diagnosing cardiac arrest and decide whether they should begin chest compressions or not. The decision to begin CPR must be taken in less than 10 s. According to the child's age, carotid (children), brachial (infants) or femoral pulse (children and infants) checks may be used.^{14,15}

Compression ventilation ratios

The compression ventilation (CV) ratio used for children should be based on whether one, or more than one rescuer is present.¹⁶ Lay rescuers, who usually learn only single-rescuer techniques, should

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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 basic life support (BLS) to resuscitate children with minimal additional information. Rescuers with a duty to respond should learn and use a 15:2 CV ratio as this has been validated in animal and manikin studies.^{17–21} This latter group, who would normally be healthcare professionals, should receive enhanced training targeted specifically at the resuscitation of children. For them, simplicity would be lost if a different ratio was taught for the scenario when one or two or more rescuers were present. However, 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. Ventilation remains a very important component of CPR in asphyxial arrests.²² Rescuers who are unable or unwilling to provide mouth-to-mouth ventilation should be encouraged to perform at least compression-only CPR.

CPR quality

The compression technique for infants includes two-finger compression for single rescuers and the two-thumb encircling technique for two or more rescuers.^{23–27} For older children, a one- or two-hand technique can be used, according to rescuer preference.²⁸ The emphasis is on achieving an adequate depth of compression: at least 1/3 of the anterior-posterior chest diameter in all children (i.e., approximately 4 cm in infants and approximately 5 cm in children). Subsequent complete release should also be emphasised. Chest compressions must be performed with minimal interruptions to minimise no-flow time. For both infants and children, the compression rate should be at least 100 but not greater than 120 min⁻¹.

Defibrillation

Automated external defibrillators

Case reports indicate that automated external defibrillators (AEDs) are safe and successful when used in children older than 1 year of age.^{29,30} Automated external defibrillators are capable of identifying arrhythmias in children accurately; in particular, they are extremely unlikely to advise a shock inappropriately.^{31–33} Consequently, the use of AEDs is indicated in all children aged greater than 1 year.³⁴ 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 software, which typically attenuate the output of the machine to 50–75 J³⁵ and these are recommended for children aged 1–8 years.^{36,37} If an attenuated shock or a manually adjustable machine is not available, an unmodified adult AED may be used in children older than 1 year.³⁸ The evidence to support a recommendation for the use of AEDs in children aged less than 1 year is limited to case reports.^{39,40} The incidence of shockable rhythms in infants is very low except when they suffer from cardiac disease.^{41–43} In these rare cases, the risk/benefit ratio may be favourable and use of an AED (preferably with dose attenuator) should be considered.

Manual defibrillators

The treatment recommendation for paediatric ventricular fibrillation (VF) or paediatric pulseless ventricular tachycardia (VT) remains immediate defibrillation. In adult advanced life support (ALS), the recommendation is to give a single shock and then resume CPR immediately without checking for a pulse or re-

assessing the rhythm (see Section 4).^{44–47} To reduce the no-flow time, chest compressions should be continued while applying and charging the paddles or self-adhesive pads (if the size of the child's chest allows this). Chest compressions should be briefly paused once the defibrillator is charged to deliver the shock. The ideal energy dose for safe and effective defibrillation in children is unknown, but animal models and small paediatric case series show that doses larger than 4 J kg⁻¹ defibrillate effectively with negligible side effects.^{29,37,48,49} Clinical studies in children indicate that doses of 2 J kg⁻¹ are insufficient in most cases.^{13,42,50} Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic shocks.^{36,37,49,51–53}

Therefore, for simplicity and consistency with adult BLS and ALS guidance, a single-shock strategy using a non-escalating dose of 4 J kg⁻¹ (preferably biphasic but monophasic is acceptable) is recommended for defibrillation in children. Use the largest size paddles or pads that fit on the infant or child's chest in the antero-lateral or antero-posterior position without the pads/paddles touching each other.¹³

Airway

Cuffed tracheal tubes

Cuffed tracheal tubes can be used safely in infants and young children. The size should be selected by applying a validated formula.

Cricoid pressure

The safety and value of using cricoid pressure during tracheal intubation is not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.

Capnometry

Monitoring exhaled carbon dioxide (CO₂), ideally by capnography, is helpful to confirm correct tracheal tube position and recommended during CPR to help assess and optimize its quality.

Titration of oxygen

Based on increasing evidence of potential harm from hyperoxaemia after cardiac arrest, once spontaneous circulation is restored, inspired oxygen should be titrated to limit the risk of hyperoxaemia.

Rapid response systems

Implementation of a rapid response system in a paediatric in-patient setting may reduce rates of cardiac and respiratory arrest and in-hospital mortality.

New topics

New topics in the 2010 guidelines include channelopathies (i.e., the importance of autopsy and subsequent family testing) and several new special circumstances: trauma, single ventricle pre- and post-1st stage repair, post-Fontan circulation, and pulmonary hypertension.

Terminology

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

The term *newly born* refers to a neonate immediately after delivery. A *neonate* is a child within 4 weeks of age. An *infant* is a child under 1 year of age, and the term *child* refers to children between 1 year and onset of puberty. From puberty children are referred to as *adolescents* for whom the adult guidelines apply. Furthermore, it is necessary to differentiate between infants and older children, as there are some important differences with respect to diagnostic and interventional techniques between these two groups. 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. 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 cardiac arrest continues into early adulthood.⁵⁴

A. Paediatric basic life support

Sequence of actions

Rescuers who have been taught adult BLS and have no specific knowledge of paediatric resuscitation may use the adult sequence, as outcome is worse if they do nothing. Non-specialists who wish to learn paediatric resuscitation because they have responsibility for children (e.g., teachers, school nurses, lifeguards), should be taught that it is preferable to modify adult BLS and perform five initial breaths followed by approximately 1 min of CPR before they go for help (see adult BLS guideline).

The following sequence is to be followed by *those with a duty to respond to paediatric emergencies* (usually health professional teams) (Fig. 6.1).

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?
- 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.
 - Re-assess him regularly.
- 3B. If the child does not respond:
 - Shout for help.
 - Turn carefully the child on his back.
 - Open the child's airway by tilting the head and lifting the chin.
 - Place your hand on his forehead and gently tilt his head back.
 - 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 obstruct the airway.
 - If you still have difficulty in opening the airway, try a jaw thrust: place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward.

Have a low threshold for suspecting an injury to the neck; if so, try to open the airway by jaw thrust alone. If jaw thrust alone does not enable adequate airway patency, 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.

Paediatric basic life support

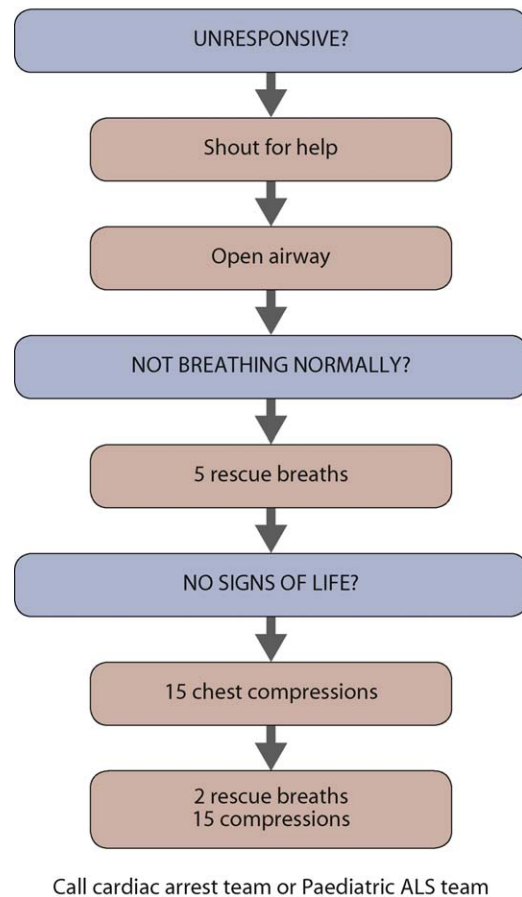


Fig. 6.1. Paediatric basic life support algorithm for those with a duty to respond to paediatric emergencies.

In the first few minutes after a cardiac arrest a child may be taking slow infrequent gasps. Look, listen and feel for no more than 10 s before deciding – if you have any doubt whether breathing is normal, act as if it is not normal:

- 5A. If the child is breathing normally:
 - Turn the child on his side into the recovery position (see below).
 - Send or go for help – call the local emergency number for an ambulance.
 - Check for continued breathing.
- 5B. If breathing is not normal or absent:
 - 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 life', which will be described later.

Rescue breaths for a child over 1 year of age (Fig. 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.
- Allow the mouth to open, but maintain chin lift.
- Take a breath and place your lips around the mouth, making sure that you have a good seal.



Fig. 6.2. Mouth-to-mouth ventilation – child.

- 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 comes out.
- 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 (Fig. 6.3):

- Ensure a neutral position of the head (as an infant's head is usually flexed when supine, this may require some extension) and a chin lift.
- Take a breath and cover the mouth and nose 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 position and chin lift, take your mouth away from the victim and watch for his chest to fall as air comes out.
- Take another breath and repeat this sequence five times.



Fig. 6.3. Mouth-to-mouth and nose ventilation – infant.

For both infants and children, 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 has 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 life – this includes any movement, coughing or normal breathing (not abnormal gasps or infrequent, irregular breaths).

If you check the pulse, ensure you take no more than 10 s.

In a child 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.

The femoral pulse in the groin, which is half way between the anterior superior iliac spine and the symphysis pubis, can also be used in infant and children.

- If you are confident that you can detect signs of life within 10 s:
 - Continue rescue breathing, if necessary, until the child starts breathing effectively on his own.
 - Turn the child on to his side (into the recovery position) if he remains unconscious.
 - Re-assess the child frequently.
- If there are no signs of life, unless you are CERTAIN you can feel a definite pulse of greater than 60 beats min^{-1} within 10 s:
 - Start chest compressions.
 - Combine rescue breathing and chest compressions:

Chest compressions:

For all children, compress the lower half 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 at least one third of the depth of the chest. Don't be afraid to push too hard: "Push Hard and Fast". Release the pressure completely and repeat at a rate of at least 100 min^{-1} (but not exceeding 120 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. The best method for compression varies slightly between infants and children.

Chest compression in infants (Fig. 6.4): The lone rescuer compresses the sternum with the tips of two fingers. If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower half 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. For both methods, depress the lower sternum by at least one third of the depth of the infant's chest.

Chest compression in children over 1 year of age (Figs. 6.5 and 6.6): Place the heel of one hand over the lower half of the sternum (as above). 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 at least 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.

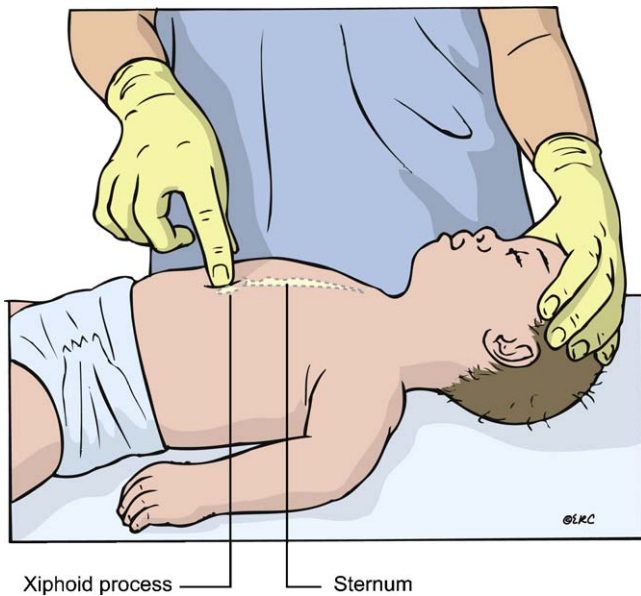


Fig. 6.4. Chest compression – infant.

8. Do not interrupt resuscitation until:

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally or a definite pulse of greater than 60 min^{-1} is palpated).
- Further qualified help arrives and takes over.
- 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 whilst summoning help.



Fig. 6.5. Chest compression with one hand – child.

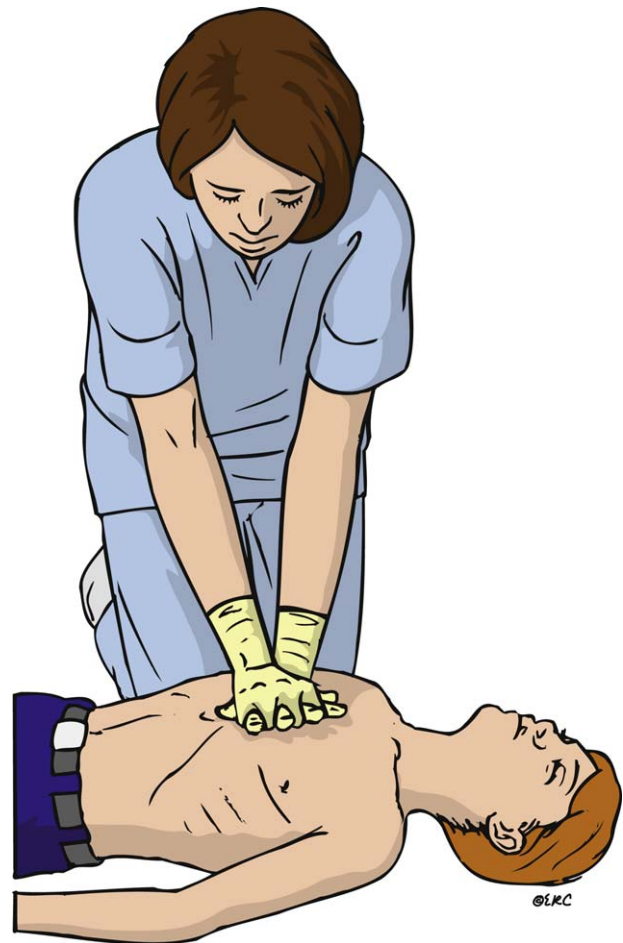


Fig. 6.6. Chest compression with two hands – child.

- 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 caused by an arrhythmia 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 normally, should be turned on his side into the recovery position.

There are several recovery positions; they all aim to prevent airway obstruction and reduce the likelihood of fluids such as saliva, secretions or vomit from entering into the upper airway.

There are important principles to be followed.

- Place the child in as near true lateral position as possible, with his mouth dependent, which should enable the free drainage of fluid.
- The position should be stable. In an infant, this may require a small pillow or a rolled-up blanket to be placed along his back to maintain the position, so preventing the infant from rolling into either the supine or prone position.
- Avoid any pressure on the child's chest that may impair breathing.
- It should be possible to turn the child onto his side and back again to the recovery position easily and safely, taking into consideration the possibility of cervical spine injury by in-line cervical stabilisation techniques.

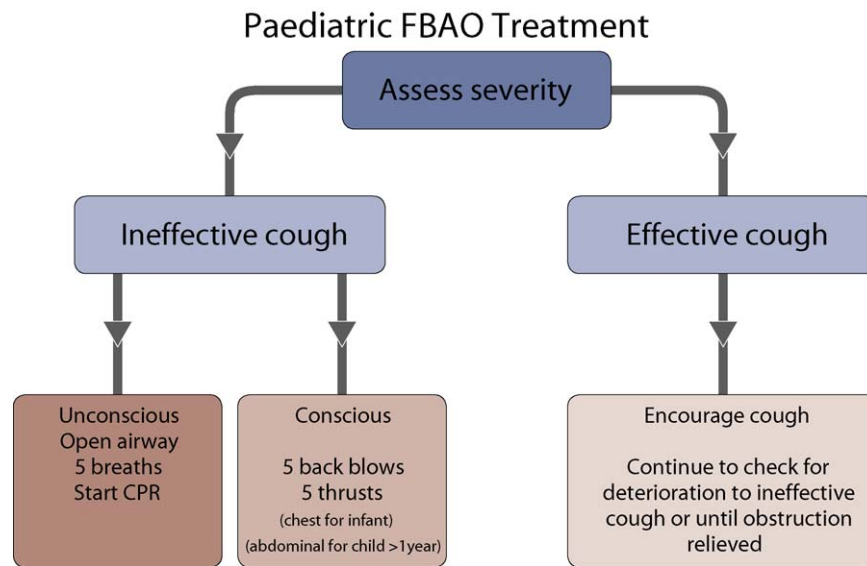


Fig. 6.7. Paediatric foreign body airway obstruction algorithm.

- Regularly change side to avoid pressure points (i.e., every 30 min).
- The adult recovery position is suitable for use in children.

Foreign body airway obstruction

No new evidence on this subject was presented during the 2010 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.⁵⁵ 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 foreign body airway obstruction (FBAO) algorithm for children was simplified and aligned with the adult version in 2005 guidelines; this continues to be the recommended sequence for managing FBAO (Fig. 6.7).

The most significant difference from the adult algorithm is that abdominal thrusts should not be used for 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 foreign body airway obstruction

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 characterised by the sudden onset of respiratory distress associated with coughing, gagging

or stridor (Table 6.1). Similar signs and symptoms may be associated with other causes of airway obstruction such as laryngitis or epiglottitis; these conditions are managed differently to that of FBAO. Suspect FBAO if the onset was very sudden and there are no other signs of illness; there may be clues to alert the rescuer, e.g., a history of eating or playing with small items immediately before the onset of symptoms.

Relief of FBAO (Fig. 6.7)

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, increasing intrathoracic pressure and dislodging the foreign body.

Table 6.1
Sign of foreign body airway obstruction.

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	

Back blows in infants.

- Support the infant in a head downward, 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.

Back blows in children over 1 year.

- 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 downward 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 (on the lower half of the 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 may cause abdominal trauma.

Following the chest or abdominal thrusts, re-assess 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 treated with abdominal thrusts should be examined by a doctor.⁵

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:

Airway opening. Open the mouth and look for any obvious object. If one 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.

Rescue breaths. Open the airway using a head tilt/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.

Chest compressions and CPR.

- 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 a minute 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 on his side (recovery position) and monitor breathing and conscious level whilst awaiting the arrival of the EMS.

B. Paediatric advanced life support**Prevention of cardiopulmonary arrest**

In children, secondary cardiopulmonary arrests, caused by either respiratory or circulatory failure, are more frequent than primary arrests caused by arrhythmias.^{56–61} So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g., trauma, drowning, poisoning).^{62,63} 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 (with haemorrhage control in injured child).

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. Summoning a paediatric rapid response team or medical emergency team may reduce the risk of respiratory and/or cardiac arrest in hospitalised children outside the intensive care setting.^{64–69} This team should include at least one paediatrician with specific knowledge in the field and one specialised nurse, and should be called to evaluate a potentially critically ill child who is

not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).

Diagnosing respiratory failure: assessment of A and B

Assessment of a potentially critically ill child starts with assessment of airway (A) and breathing (B). Abnormalities in airway patency or gas exchange in the lungs can lead to respiratory failure.

Signs of respiratory failure include:

- 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 as the patient tires or compensatory mechanisms fail, additional noises such as stridor, wheeze, grunting, or the loss of breath sounds.
- Decreased *tidal volume* marked by shallow breathing, decreased chest expansion or decreased air entry at auscultation.
- *Hypoxaemia* (without/with supplemental oxygen) generally identified by cyanosis but best evaluated by pulse oximetry.

There may be associated signs in other organ systems that are either affected by inadequate ventilation and oxygenation or act to compensate the respiratory problem. These are detectable in step C of the assessment and include:

- Increasing tachycardia (compensatory mechanism in an attempt to increase oxygen delivery).
- Pallor.
- Bradycardia (ominous indicator of the loss of compensatory mechanisms).
- Alteration in the level of consciousness (a sign that compensatory mechanisms are overwhelmed).

Diagnosing circulatory failure: assessment of C

Circulatory failure (or shock) is characterised by a mismatch between metabolic demand by the tissues and delivery of oxygen and nutrients by the circulation.⁷⁰ 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 include:

- Increased *heart rate* (bradycardia is an ominous sign of 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 *intravascular volume*.
- Decreased urine output and metabolic acidosis.

Other systems may be affected, for example:

- Respiratory frequency may be increased initially, in an attempt to improve oxygen delivery, later becoming slow and accompanied by decompensated circulatory failure.
- Level of consciousness may decrease because of poor cerebral perfusion.

Diagnosing cardiopulmonary arrest

Signs of cardiopulmonary arrest include:

- Unresponsiveness to pain (coma).
- Apnoea or gasping respiratory pattern.
- Absent circulation.
- Pallor or deep cyanosis.

Palpation of a pulse is not reliable as the sole determinant of the need for chest compressions.^{71,72} If cardiac arrest is suspected, and in the absence of signs of life, rescuers (lay and professional) should begin CPR unless they are certain they can feel a central pulse within 10 s (infants – brachial or femoral artery; children – carotid or femoral artery). If there is any doubt, start CPR.^{72–75} If personnel skilled in echocardiography are available, this investigation may help to detect cardiac activity and potentially treatable causes for the arrest.⁷⁶ However, echocardiography must not interfere with the performance of chest compressions.

Management of respiratory and circulatory failure

In children, there are many causes of respiratory and circulatory failure and they may develop gradually or suddenly. Both may be initially compensated but will normally decompensate without adequate treatment. Untreated decompensated respiratory or circulatory failure will lead to cardiopulmonary arrest. Hence, the aim of paediatric life support is early and effective intervention in children with respiratory and circulatory failure to prevent progression to full arrest.

Airway and breathing

- Open the airway and ensure adequate ventilation and oxygenation. Deliver high-flow oxygen.
- Establish respiratory monitoring (first line – pulse oximetry/SpO₂).
- Achieving adequate ventilation and oxygenation may require 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.
- Very rarely, a surgical airway may be required.

Circulation

- Establish cardiac monitoring (first line – pulse oximetry/SpO₂, electrocardiography/ECG and non-invasive blood pressure/NIBP).
- Secure intravascular access. This may be by peripheral intravenous (IV) or by intraosseous (IO) cannulation. If already in situ, a central intravenous catheter should be used.
- Give a fluid bolus (20 ml kg⁻¹) and/or drugs (e.g., inotropes, vasopressors, anti-arrhythmics) as required.
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.^{77–80}
- Assess and re-assess the child continuously, commencing each time with the airway before proceeding to breathing and then the circulation.
- During treatment, capnography, invasive monitoring of arterial blood pressure, blood gas analysis, cardiac output monitoring, echocardiography and central venous oxygen saturation (ScvO₂) may be useful to guide the treatment of respiratory and/or circulatory failure.

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 (from the incisors to the angle of the mandible), to avoid pushing the tongue backward and obstructing the epiglottis, or directly compressing the glottis. The soft palate in the child can be damaged by insertion of the oropharyngeal airway – avoid this by inserting the oropharyngeal airway with care; do not use any force. The nasopharyngeal airway is usually tolerated better in the conscious or semi-conscious child (who has an effective gag reflex), but should not be used if there is a basal skull fracture or a coagulopathy. The correct insertion depth should be sized from the nostrils to the angle of the mandible but must be re-assessed after insertion. These simple airway adjuncts do not protect the airway from aspiration of secretions, blood or stomach contents.

Laryngeal mask airway (LMA)

Although bag-mask ventilation remains the recommended first line method for achieving airway control and ventilation in children, the LMA is an acceptable airway device for providers trained in its use.^{81,82} It is particularly helpful in airway obstruction caused by supraglottic airway abnormalities or if bag-mask ventilation is not possible. The LMA does not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required. Use of the LMA is associated with a higher incidence of complications in small children compared with adults.^{83,84} Other supraglottic airway devices (e.g., laryngeal tube), which have been used successfully in children's anaesthesia, may also be useful in an emergency but there are few data on the use of these devices in paediatric emergencies.⁸⁵

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 quicker and simpler, and is associated with fewer complications than nasal placement. In the conscious child, the judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is essential in order to avoid multiple intubation attempts or intubation failure.^{86–95} The anatomy of a child's airway differs significantly from that of an adult; hence, intubation of a child requires special training and experience. Clinical examination and capnography must be used to confirm correct tracheal tube placement. The tracheal tube must be secured and vital signs monitored.⁹⁶ It is also essential to plan an alternative airway management technique in case the trachea cannot be intubated.

There is currently no evidence-based recommendation defining the setting-, patient- and operator-related criteria for prehospital tracheal intubation of children. Prehospital tracheal intubation of children may be considered if:

- (1) the airway and/or breathing is seriously compromised or threatened;
- (2) the mode and duration of transport require the airway to be secured early (e.g., air transport); and
- (3) if the operator is adequately skilled in advanced paediatric airway management including the use of drugs to facilitate tracheal intubation.⁹⁷

Table 6.2

General recommendation for cuffed and uncuffed tracheal tube sizes (internal diameter in mm).

	Uncuffed	Cuffed
Neonates <i>premature</i>	Gestational age in weeks/10	Not used
Neonates <i>Full term</i>	3.5	Not usually used
Infants	3.5–4.0	3.0–3.5
Child 1–2 years	4.0–4.5	3.5–4.0
Child >2 years	Age/4 + 4	Age/4 + 3.5

Rapid-sequence induction and intubation

The child who is in cardiopulmonary arrest and/or deep coma does not require sedation or analgesia to be intubated; otherwise, intubation must be preceded by oxygenation (gentle BMV is sometimes required to avoid hypoxia), rapid sedation, analgesia and the use of neuromuscular blocking drugs to minimise intubation complications and failure.⁹⁸ The intubator must be experienced and familiar with drugs used for rapid-sequence induction. The use of cricoid pressure may prevent or limit regurgitation of gastric contents^{99,100} but it may distort the airway and make laryngoscopy and intubation more difficult.¹⁰¹ Cricoid pressure should not be used if either intubation or oxygenation is compromised.

Tracheal tube sizes

A general recommendation for tracheal tube internal diameters (ID) for different ages is shown in Table 6.2.^{102–107} This is a guide only and tubes one size larger and smaller should always be available. Tracheal tube size can also be estimated from the length of the child's body as measured by resuscitation tapes.¹⁰⁸

Cuffed versus uncuffed tracheal tubes

Uncuffed tracheal tubes have been used traditionally in children up to 8 years of age but cuffed tubes may offer advantages in certain circumstances e.g., when lung compliance is poor, airway resistance is high or if there is a large air leak from the glottis.^{102,109,110} The use of cuffed tubes also makes it more likely that the correct tube size will be chosen on the first attempt.^{102,103,111} 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.^{109,110,112} As excessive cuff pressure may lead to ischaemic damage to the surrounding laryngeal tissue and stenosis, cuff inflation pressure should be monitored and maintained at less than 25 cm H₂O.¹¹²

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.^{113,114} No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.^{115–117}

Assessment of the correct tracheal tube position is made by:

- laryngoscopic observation of the tube passing beyond the vocal cords;
- detection of end-tidal CO₂ (by colorimetry or capnometry/-graphy) if the child has a perfusing rhythm (this may also be seen with effective CPR, but it is not completely reliable);
- 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 in the axillae and apices of the chest;
- absence of air entry into the stomach on auscultation;

- improvement or stabilisation of SpO₂ in the expected range (delayed sign!);
- improvement of heart rate towards the age-expected value (or remaining within the normal range) (delayed sign!).

If the child is in cardiopulmonary arrest and exhaled CO₂ is not detected despite adequate chest compressions, or if there is any doubt, confirm tracheal tube position by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and re-assess its position. Maintain the child's head in the neutral position. Flexion of the head drives the tube further into the trachea whereas extension may pull it out of the airway.¹¹⁸ Confirm the position of the tracheal tube at the mid-trachea by chest X-ray; 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:

- Displacement of the tracheal tube.
- Obstruction of the tracheal tube or of the heat and moisture exchanger (HME).
- Pneumothorax.
- Equipment failure (source of gas, bag-mask, ventilator, etc.).
- Stomach (gastric distension may alter diaphragm mechanics).

Breathing

Oxygenation

Give oxygen at the highest concentration (i.e., 100%) during initial resuscitation. Once circulation is restored, give sufficient oxygen to maintain an arterial oxygen saturation (SaO₂) in the range of 94–98%.^{119,120}

Studies in neonates suggest some advantages of using room air during resuscitation (see Section 7).^{11,121–124} In the older child, there is no evidence of benefit for air instead of oxygen, so use 100% oxygen for initial resuscitation and after return of a spontaneous circulation (ROSC) titrate the fraction inspired oxygen (FiO₂) to achieve a SaO₂ in the range of 94–98%. In smoke inhalation (carbon monoxide poisoning) and severe anaemia however a high FiO₂ should be maintained until the problem has been solved because in these circumstances dissolved oxygen plays an important role in oxygen transport.

Ventilation

Healthcare providers commonly provide excessive ventilation during CPR and this may be harmful. Hyperventilation causes increased intrathoracic pressure, decreased cerebral and coronary perfusion, and poorer survival rates in animals and adults.^{125–131} Although normoventilation is the objective during resuscitation, it is difficult to know the precise minute volume that is being delivered. A simple guide to deliver an acceptable tidal volume is to achieve modest chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a compression rate of 100–120 min⁻¹.¹²⁵ Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the victim's age and, as soon as possible, by monitoring end-tidal CO₂ and blood gas values.

Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10–12 breaths min⁻¹ without interrupting chest compressions. Take care to ensure that lung inflation 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 arterial carbon dioxide tension (PaCO₂). Hyperventilation and hypoventilation are harmful.

Bag-mask ventilation (BMV)

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.^{114,132–135} Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring peripheral oxygen saturation (SpO₂). Any healthcare provider with a responsibility for treating 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. For emergency intubation, both cuffed and uncuffed tracheal tubes are acceptable.

Monitoring of breathing and ventilation

End-tidal CO₂

Monitoring end-tidal CO₂ (ETCO₂) 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.^{136–139} A colour change or the presence of a capnographic waveform for more than four ventilated breaths 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 a bronchus. The absence of exhaled CO₂ during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent ETCO₂ may reflect low or absent pulmonary blood flow.^{140–143}

Capnography may also provide information on the efficiency of chest compressions and can give an early indication of ROSC.^{144,145} Efforts should be made to improve chest compression quality if the ETCO₂ remains below 15 mmHg (2 kPa). Care must be taken when interpreting ETCO₂ values especially after the administration of adrenaline or other vasoconstrictor drugs when there may be a transient decrease in values,^{146–150} or after the use of sodium bicarbonate when there may be a transient increase.¹⁵¹ Current evidence does not support the use of a threshold ETCO₂ value as an indicator for the discontinuation of resuscitation efforts.

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.^{152,153} There are no studies on the use of the ODD in children who are in cardiopulmonary arrest.

Pulse oximetry

Clinical evaluation of the oxygen saturation of arterial blood (SaO₂) is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry (SpO₂). Pulse oximetry can be unreliable under certain conditions, for example, if the child is in circulatory failure, 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.¹⁵⁴

Circulation

Vascular access

Vascular access is essential to enable drugs and fluids to be given, and blood samples obtained. Venous access can be dif-

difficult to establish during resuscitation of an infant or child. In critically ill children, whenever venous access is not readily attainable intraosseous access should be considered early, especially if the child is in cardiac arrest or decompensated circulatory failure.^{155–157} In any case, in critically ill children, if attempts at establishing intravenous (IV) access are unsuccessful after 1 min, insert an intraosseous (IO) needle instead.^{155,158}

Intraosseous access

Intraosseous access is a rapid, safe, and effective route to give drugs, fluids and blood products.^{159–168} The onset of action and time to achieve adequate plasma drug concentrations are similar to that achieved via the central venous route.^{169,170} Bone marrow samples can be used to cross match for blood type or group¹⁷¹ for chemical analysis^{172,173} and for blood gas measurement (the values are comparable to central venous blood gases if no drug has been injected in the cavity).^{172,174–176} However samples can damage autoanalysers and should be used preferably in cartridge analyser. 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. The benefits of semi-automated IO devices remain to be seen but preliminary experiences show them to be rapid and effective for obtaining circulatory access.^{167,168,177,178}

Intravenous access

Peripheral IV access provides plasma concentrations of drugs and clinical responses equivalent to central or IO access.^{156,157,179–181} Central venous lines provide more secure long-term access but, compared with IO or peripheral IV access, offer no advantages during resuscitation.^{156,179–181}

Tracheal tube access

Intraosseous or IV access should be definitely preferred to the tracheal route for giving drugs.¹⁸² Drugs given via the trachea have highly variable absorption but, for guidance, the following dosages have been recommended:

Adrenaline	100 $\mu\text{g kg}^{-1}$
Lidocaine	2–3 mg kg^{-1}
Atropine	30 $\mu\text{g kg}^{-1}$

The optimal dose of naloxone is not known.

Dilute the drug in 5 ml of normal saline and follow administration with five ventilations.^{183–185} 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 circulatory failure in the absence of volume overload.¹⁸⁶ Isotonic crystalloids are recommended as the initial resuscitation fluid for infants and children with any type of circulatory failure.

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 circulatory failure associated with head injuries or hypovolaemia.^{187,188}

There are also insufficient data to recommend delayed fluid resuscitation in the hypotensive child with blunt trauma.¹⁸⁹ Avoid dextrose containing solutions unless there

is hypoglycaemia.^{190–193} Monitor glucose levels and avoid hypoglycaemia; infants and small children are particularly prone to hypoglycaemia.

Adenosine

Adenosine is an endogenous nucleotide that 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).¹⁹⁴ It is safe because 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.¹⁹⁵ Adenosine must be used with caution in asthmatics, second or third degree AV block, long QT syndromes and in cardiac transplant recipients.

Adrenaline (epinephrine)

Adrenaline is an endogenous catecholamine with potent α , β_1 and β_2 adrenergic actions. It is placed prominently in the cardiac arrest 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 for the first and for subsequent doses is 10 $\mu\text{g kg}^{-1}$. The maximum single dose is 1 mg. If needed, give further doses of adrenaline every 3–5 min. Intratracheal adrenaline is no longer recommended,^{196–199} but if this route is ever used, the dose is ten times this (100 $\mu\text{g kg}^{-1}$).

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.^{200–203}

Once spontaneous circulation is restored, a continuous infusion of adrenaline may be required. Its haemodynamic effects are dose related; there is also considerable variability in response between children; 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 can cause severe hypertension and tachyarrhythmias.²⁰⁴

To avoid tissue damage it is essential to give adrenaline through a secure intravascular line (IV or IO). Adrenaline (and other catecholamines) is inactivated by alkaline solutions and should never be mixed with sodium bicarbonate.²⁰⁵

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 causing hypotension. This side effect is less common with the aqueous solution.²⁰⁶ Other rare but significant adverse effects are bradycardia and polymorphic VT.²⁰⁷

Atropine

Atropine accelerates sinus and atrial pacemakers by blocking the parasympathetic response. It may also increase AV conduction. Small doses (< 100 μg) may cause paradoxical bradycardia.²⁰⁸ In

bradycardia with poor perfusion that is unresponsive to ventilation and oxygenation, the first line drug is adrenaline, not atropine.

Atropine is recommended for bradycardia caused by increased vagal tone or cholinergic drug toxicity.^{209–212}

Calcium

Calcium is essential for myocardial function^{213,214} but routine use of calcium does not improve the outcome from cardiopulmonary arrest.^{215–217}

Calcium is indicated in the presence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia and hyperkalaemia.^{218–220}

Glucose

Data from neonates, children and adults indicate that both hyper- and hypo-glycaemia are associated with poor outcome after cardiopulmonary arrest,^{221–223} but it is uncertain if this is causative or merely an association.²²⁴ 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 hyper- and hypo-glycaemia following ROSC. Strict glucose control has not shown survival benefits in adults when compared with moderate glucose control^{225,226} and it increases the risk of hypoglycaemia in neonates, children and adults.^{227–231}

Magnesium

There is no evidence for giving magnesium routinely during cardiopulmonary arrest.²³² Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsades de pointes VT regardless of the cause.²³³

Sodium bicarbonate

Do not give sodium bicarbonate routinely during cardiopulmonary arrest or after ROSC.^{220,234,235} After effective ventilation and chest compressions have been achieved and adrenaline given, sodium bicarbonate may be considered for the child with prolonged cardiopulmonary arrest and/or severe metabolic acidosis. Sodium bicarbonate may also be considered in case of haemodynamic instability and co-existing hyperkalaemia, or in the management of tricyclic antidepressant drug overdose. Excessive quantities of sodium bicarbonate may impair tissue oxygen delivery, produce hypokalaemia, hypernatraemia, hyperosmolality, and inactivate catecholamines.

Lidocaine

Lidocaine is less effective than amiodarone for defibrillation-resistant VF/pulseless VT in adults²³⁶ and therefore is not the first line treatment in defibrillation-resistant VF/pulseless VT in children.

Procainamide

Procainamide slows intra-atrial conduction and prolongs the QRS and QT intervals. It can be used in SVT^{237–239} or VT²⁴⁰ resistant to other medications in the haemodynamically stable child. However, paediatric data are sparse and procainamide should be used cautiously.^{241,242} Procainamide is a potent vasodilator and can cause hypotension: infuse it slowly with careful monitoring.^{243–245}

Vasopressin – terlipressin

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).²⁴⁶ There is currently insufficient evidence to support or refute the use of vasopressin or terlipressin as an alternative to, or in combination with, adrenaline in any cardiac arrest rhythm in adults or children.^{247–258}

Some studies have reported that terlipressin (a long-acting analogue of vasopressin with comparable effects) improves haemodynamics in children with refractory, vasodilatory septic shock, but its impact on survival is less clear.^{255–257,259,260} Two paediatric series suggested that terlipressin could be effective in refractory cardiac arrest.^{258,261}

These drugs could be used in cardiac arrest refractory to several adrenaline doses.

Defibrillators

Defibrillators are either automatically 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 healthcare facilities caring for children at risk of cardiopulmonary arrest. Automated external defibrillators (AEDs) are preset for all variables including the energy dose.

Pad/paddle size for defibrillation

Select the largest possible available paddles to provide good contact with the chest wall. The ideal size is unknown but there should be good separation between the pads.^{13,262,263}

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 antero-lateral position, one paddle placed below the right clavicle and the other in the left axilla (Fig. 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 antero-posterior position and is also acceptable.

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.^{264,265} In practice, this means that the paddles should be applied firmly.

Energy dose in children

The ideal energy dose for safe and effective defibrillation is unknown. Biphasic shocks are at least as effective and produce less post-shock myocardial dysfunction than monophasic

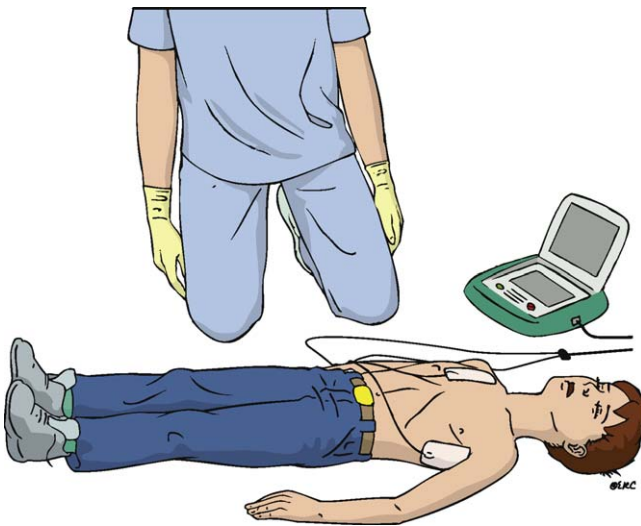


Fig. 6.8. Paddle positions for defibrillation – child.

Shockable – VF/pulseless VT

Attempt defibrillation immediately (4 J kg^{-1}):

- Charge the defibrillator while another rescuer continues chest compressions.
- Once the defibrillator is charged, pause the chest compressions, ensure that all rescuers are clear of the patient. Minimise the delay between stopping chest compressions and delivery of the shock – even 5–10 s delay will reduce the chances of the shock being successful.^{268,269}
- Give one shock.
- Resume CPR as soon as possible without re-assessing the rhythm.
- After 2 min, check briefly the cardiac rhythm on the monitor.
- Give second shock (4 J kg^{-1}) if still in VF/pulseless VT.
- Give CPR for 2 min as soon as possible without re-assessing the rhythm.
- Pause briefly to assess the rhythm; if still in VF/pulseless VT give a third shock at 4 J kg^{-1} .
- Give adrenaline $10\text{ }\mu\text{g kg}^{-1}$ and amiodarone 5 mg kg^{-1} after the third shock once CPR has been resumed.
- Give adrenaline every alternate cycle (i.e., every 3–5 min during CPR).
- Give a second dose of amiodarone 5 mg/kg ²⁷⁰ if still in VF/pulseless VT after the fifth shock.

If the child remains in VF/pulseless VT, continue to alternate shocks of 4 J kg^{-1} with 2 min of CPR. If signs of life become evident, check the monitor for an organised rhythm; if this is present, check for signs of life and a central pulse and evaluate the haemodynamics of the child (blood pressure, peripheral pulse, capillary refill time).

Identify and treat any reversible causes (4 Hs and 4 Ts) remembering that the first 2 Hs (hypoxia and hypovolaemia) have the highest prevalence in critically ill or injured children (Fig. 6.11).

If defibrillation was successful but VF/pulseless VT recurs, resume CPR, give amiodarone and defibrillate again at 4 J kg^{-1} . Start a continuous infusion of amiodarone.

Reversible causes of cardiac arrest

The reversible causes of cardiac arrest can be considered quickly by recalling the 4 Hs and 4 Ts:

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

Sequence of events in cardiopulmonary arrest

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

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

shocks.^{36,49,51–53,266} Animal models show better results with paediatric doses of $3–4\text{ J kg}^{-1}$ than with lower doses,⁴⁹ or adult doses.³⁸ Clinical studies in children indicate that doses of 2 J kg^{-1} are insufficient in most cases.^{12,38,42} Doses larger than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.^{29,48} When using a manual defibrillator, use 4 J kg^{-1} (preferably biphasic but monophasic waveform is also acceptable) for the first and subsequent shocks.

If no manual defibrillator is available, use an AED that can recognise paediatric shockable rhythms.^{31,32,267} The 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).^{34,37} If such an AED is not available, use a standard AED and the preset adult energy levels. For children above 8 years, use a standard AED with standard paddles. Although the evidence to support a recommendation for the use of AEDs (preferably with dose attenuator) in children less than 1 year is limited to case reports,^{39,40} it is acceptable if no other option is available.

Advanced management of cardiopulmonary arrest (Fig. 6.9)

ABC

Commence and continue with basic life support
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 ventilation in the ratio of 15:2
- Avoid rescuer fatigue by frequently changing the rescuer performing chest compressions
- Establish cardiac monitoring

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

Non-shockable – asystole, pulseless electrical activity (PEA)

- Give adrenaline IV or IO ($10\text{ }\mu\text{g kg}^{-1}$) and repeat every 3–5 min.
- Identify and treat any reversible causes (4 Hs and 4 Ts) (Fig. 6.10).

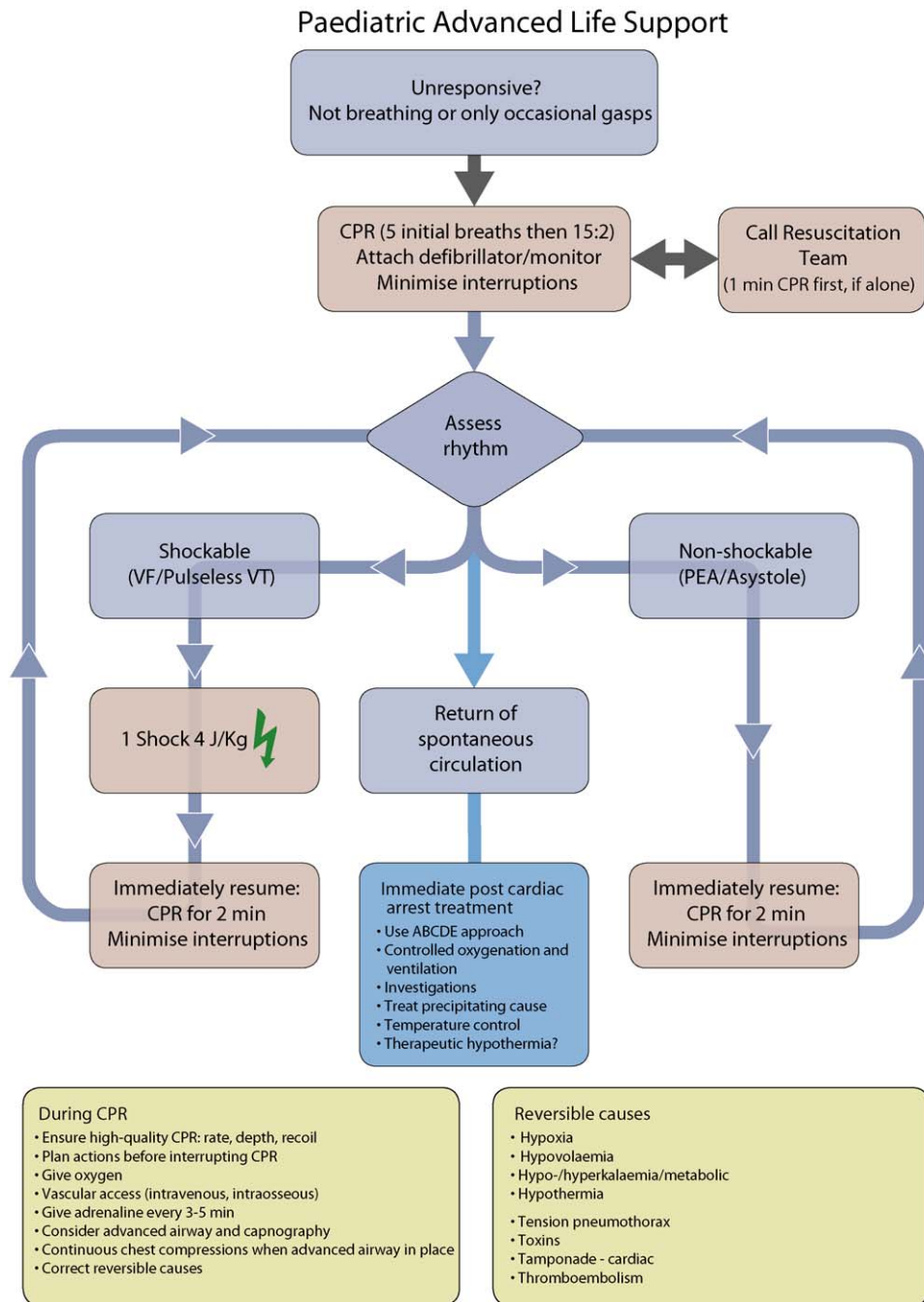


Fig. 6.9. Paediatric advanced life support algorithm.

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 compression²⁷¹ but must not delay the provision of basic or advanced resuscitation.

Shockable rhythms are pulseless VT and VF. These rhythms are more likely after sudden collapse in children with heart disease or adolescents.^{41–43} Non-shockable rhythms are pulseless electrical activity (PEA), bradycardia ($<60 \text{ min}^{-1}$ with no signs of circulation), and asystole. PEA and bradycardia often have wide-QRS complexes.

Echocardiography may be used to identify potentially treatable causes of cardiac arrest in children. Cardiac activity can be rapidly visualised⁷⁶ and pericardial tamponade diagnosed.²⁷² However, appropriately skilled operators must be available and its use should be balanced against the interruption to chest compressions during examination.

Non-shockable rhythms

Most cardiopulmonary arrests in children and adolescents are of respiratory origin.^{54,58,273–275} A period of immediate CPR is therefore mandatory in this age group before searching for an AED or manual defibrillator, as its immediate availability will not improve the outcome of a respiratory arrest.^{17,276}

CARDIAC ARREST: NON SHOCKABLE RHYTHM

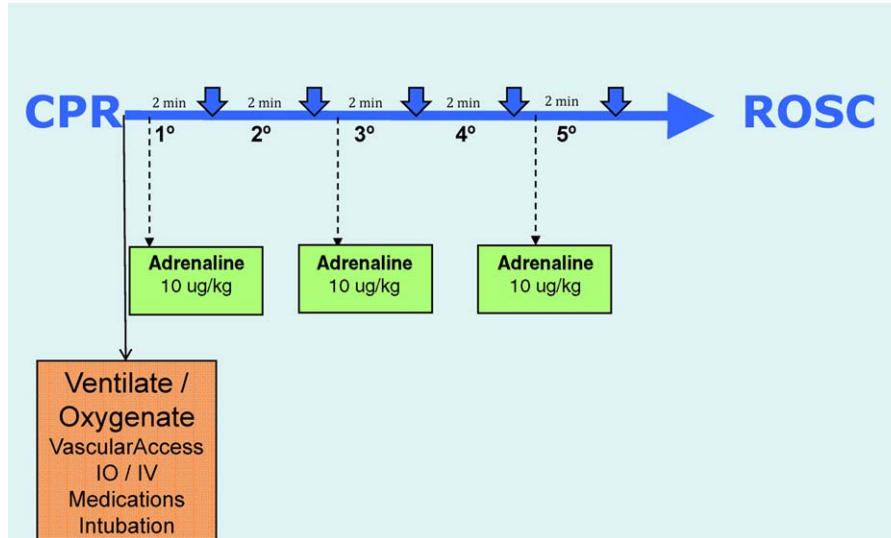


Fig. 6.10. Paediatric algorithm for non-shockable rhythm.

CARDIAC ARREST – SHOCKABLE RHYTHM

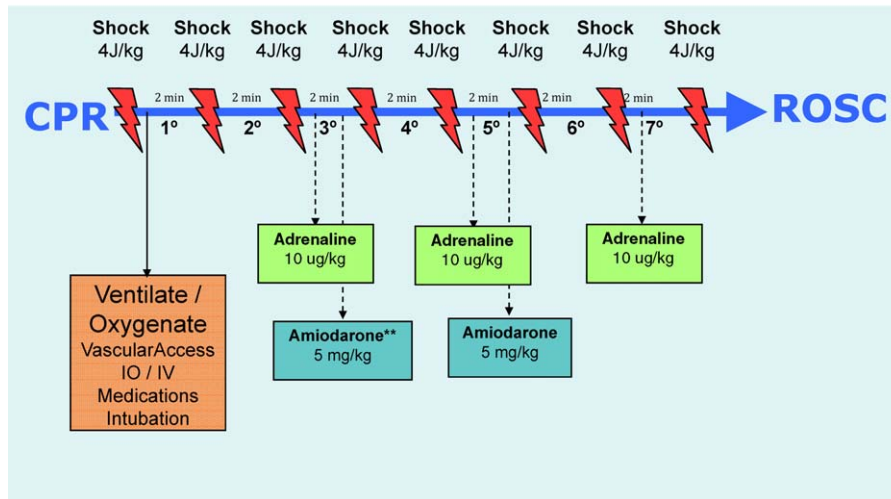


Fig. 6.11. Paediatric algorithm for shockable rhythm.

Bystander CPR is associated with a better neurological outcome in adults and children.^{277–279} The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by organised, wide or narrow complex electrical activity, usually (but not always) at a slow rate, and absent pulses. It commonly follows a period of hypoxia or myocardial ischaemia, but occasionally can have a reversible cause (i.e., one of the 4 Hs and 4 Ts) that led to a sudden impairment of cardiac output.

Shockable rhythms

Primary VF occurs in 3.8–19% of cardiopulmonary arrests in children.^{13,41–43,60,274,275,277} The incidence of VF/pulseless VT increases with age.^{267,280} The primary determinant of survival from VT/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 the longer the time until defibrillation:

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%.²⁸¹ Cardiopulmonary resuscitation provided before defibrillation for response intervals longer than 5 min improved outcome in some studies,^{282,283} but not in others.²⁸⁴

Secondary VF is present at some point in up to 27% of in-hospital resuscitation events. It has a much poorer prognosis than primary VF.⁴³

Drugs in shockable rhythms

Adrenaline (epinephrine)

Adrenaline is given every 3–5 min by the IV or IO route in preference to the tracheal tube route.

Amiodarone in VF/pulseless VT

Amiodarone is indicated in defibrillation-resistant VF/pulseless VT. Experimental and clinical experience with amiodarone in children is scarce; evidence from adult studies^{236,285,286} demonstrates increased survival to hospital admission, but not to hospital dis-

charge. One paediatric case series demonstrates the effectiveness of amiodarone for life-threatening ventricular arrhythmias.²⁸⁷ Therefore, IV amiodarone has a role in the treatment of defibrillation refractory or recurrent VF/pulseless VT in children.

Extracorporeal life support

Extracorporeal life support should be considered for children with cardiac arrest refractory to conventional CPR, if the arrest occurs in a highly supervised environment and available expertise and equipment to rapidly initiate extracorporeal life support (ECLS).

Arrhythmias

Unstable arrhythmias

Check for signs of life and the central pulse of any child with an arrhythmia; if signs of life are absent, treat as for cardiopulmonary arrest. If the child has signs of life and a central pulse, evaluate the haemodynamic status. Whenever the haemodynamic status is compromised, the first steps are:

1. Open the airway.
2. Give oxygen and assist ventilation as necessary.
3. Attach ECG monitor or defibrillator and assess the cardiac rhythm.
4. Evaluate if the rhythm is slow or fast for the child's age.
5. Evaluate if the rhythm is regular or irregular.
6. Measure QRS complex (narrow complexes: <0.08 s duration; wide complexes: >0.08 s).
7. The treatment options are dependent on the child's haemodynamic stability.

Bradycardia

Bradycardia is caused commonly by hypoxia, acidosis and/or 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 they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline. If the bradycardia is caused by vagal stimulation (such as after passing a nasogastric tube), atropine may be effective.

Cardiac pacing (either transvenous or external) is generally not useful during resuscitation. It may be considered in cases of AV block or sinus node dysfunction unresponsive to oxygenation, ventilation, chest compressions and other medications; pacing is not effective in asystole or arrhythmias caused by hypoxia or ischaemia.²⁸⁸

Tachycardia

Narrow complex tachycardia

If SVT is the likely rhythm, vagal manoeuvres (Valsalva or diving reflex) may be used in haemodynamically stable children. They can also be used in haemodynamically unstable children, but only if they do not delay chemical or electrical cardioversion.²⁸⁹ If the child is unstable with a depressed conscious level, attempt synchronised electrical cardioversion immediately.

Adenosine is usually effective in converting SVT into sinus rhythm. It is given by rapid, intravenous injection as close as practicable to the heart (see above), and followed immediately by a bolus of normal saline. If the child is too haemodynamically unsta-

ble, omit vagal manoeuvres and adenosine and attempt electrical cardioversion immediately.

Electrical cardioversion (synchronised with R wave) is also indicated when vascular access is not available, or when 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. Verapamil may be considered as an alternative therapy in older children but should not be routinely used in infants.

Amiodarone has been shown to be effective in the treatment of SVT in several paediatric studies.^{270,287,290–297} However, since most studies of amiodarone use 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. An expert should also be consulted about alternative treatment strategies because the evidence to support other drugs in the treatment of SVT is limited and inconclusive.^{298,299} If amiodarone is used in this circumstance, avoid rapid administration because hypotension is common.

Wide complex tachycardia

In children, wide-QRS complex tachycardia is uncommon and more likely to be supraventricular than ventricular in origin.³⁰⁰ Nevertheless, in haemodynamically unstable children, it must be considered to be VT until proven otherwise. Ventricular tachycardia 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 anti-arrhythmic therapy if a second cardioversion attempt is unsuccessful or if VT recurs.

Amiodarone has been shown to be effective in treating paediatric arrhythmias,²⁹¹ although cardiovascular side effects are common.^{270,287,292,297,301}

Stable arrhythmias

Whilst maintaining the child's airway, breathing and circulation, contact an expert before initiating therapy. 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. Amiodarone may be considered as a treatment option if this fails or if the diagnosis of VT is confirmed on an ECG. Procainamide may also be considered in stable SVT refractory to vagal manoeuvres and adenosine.^{239,302–304} and in stable VT.^{239,240,305,306} Do not give procainamide with amiodarone.

Special circumstances

Channelopathy

When sudden unexplained cardiac arrest occurs in children and young adults, obtain a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents/drownings, or sudden death) and review any available previous ECGs. All infants, children, and young adults with sudden, unexpected death should, if possible, have an unrestricted, complete autopsy, performed preferably by pathologists with training and expertise in cardiovascular pathology.^{307–316} Consideration should be given to preservation and genetic analysis of tissue to determine the presence of a channelopathy. Refer families of

patients whose cause of death is not found on autopsy to a health care provider/centre with expertise in cardiac rhythm disturbances.

Life support for blunt or penetrating trauma

There is a very high mortality associated with cardiac arrest from major (blunt or penetrating) trauma.^{317–320} There is little evidence to support any additional specific interventions that are different from the routine management of cardiac arrest; however, the use of resuscitative thoracotomy may be considered in children with penetrating injuries.^{321–325}

Single ventricle post-stage 1 repair

The incidence of cardiac arrest in infants following single ventricle stage 1 repair is approximately 20%, with a survival to discharge of 33%.³²⁶ There is no evidence that anything other than routine resuscitative protocols should be followed. Diagnosis of the pre-arrest state is difficult but it may be assisted by monitoring the oxygen extraction (superior vena caval ScvO₂) or near infrared spectroscopy (cerebral and splanchnic circulations).^{327–329} Treatment of high systemic vascular resistance with alpha-adrenergic receptor blockade may improve systemic oxygen delivery,³³⁰ reduce the incidence of cardiovascular collapse,³³¹ and improve survival.³³²

Single ventricle post-Fontan

Children in the pre-arrest state who have Fontan or hemi-Fontan anatomy may benefit from increased oxygenation and an improved cardiac output by instituting negative pressure ventilation.^{333,334} Extracorporeal membrane oxygenation (ECMO) may be useful rescue for children with failing Fontan circulations but no recommendation can be made in favour or against ECMO in those with hemi-Fontan physiology or for rescue during resuscitation.³³⁵

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.^{336,337} Follow routine resuscitation protocols in these patients with emphasis on high FiO₂ and alkalosis/hyperventilation because this may be as effective as inhaled nitric oxide in reducing pulmonary vascular resistance.³³⁸ Resuscitation is most likely to be successful in patients with a reversible cause who are treated with intravenous epoprostenol or inhaled nitric oxide.³³⁹ If routine medications that reduce pulmonary artery pressure have been stopped, they should be restarted and the use of aerosolised epoprostenol or inhaled nitric oxide considered.³⁴⁰ Right ventricular support devices may improve survival.^{341–344}

Post-arrest management

After prolonged, complete, whole-body hypoxia-ischaemia ROSC has been described as an unnatural pathophysiological state, created by successful CPR.³⁴⁵ Post-arrest management must be a multidisciplinary activity and include all the treatments needed for complete neurological recovery. The main goals are to reverse brain injury and myocardial dysfunction, and to treat the systemic ischaemia/reperfusion response and any persistent precipitating pathology.

Myocardial dysfunction

Myocardial dysfunction is common after cardiopulmonary resuscitation.^{345–348} Vasoactive drugs (adrenaline, dobutamine,

dopamine and noradrenaline) may improve the child's post-arrest haemodynamic values but the drugs must be titrated according to the clinical condition.^{349–359}

Temperature control and management

Hypothermia is common in the child following cardiopulmonary resuscitation.³⁶⁰ Central hypothermia (32–34 °C) may be beneficial, whereas fever may be detrimental to the injured brain. Mild hypothermia has an acceptable safety profile in adults^{361,362} and neonates.^{363–368} Whilst it may improve neurological outcome in children, an observational study neither supports nor refutes the use of therapeutic hypothermia in paediatric cardiac arrest.³⁶⁹

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 at least 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.^{370–372} 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.^{373–375}

These guidelines are based on evidence from the use of therapeutic hypothermia in neonates and adults. At the time of writing, there are ongoing, prospective, multicentre trials of therapeutic hypothermia in children following in- and out-of-hospital cardiac arrest (www.clinicaltrials.gov; NCT00880087 and NCT00878644).

Fever is common following cardiopulmonary resuscitation and is associated with a poor neurological outcome,^{376–378} the risk increasing for each degree of body temperature greater than 37 °C.³⁷⁶ There are limited experimental data suggesting that the treatment of fever with antipyretics and/or physical cooling reduces neuronal damage.^{379,380} Antipyretics and accepted drugs to treat fever are safe; therefore, use them to treat fever aggressively.

Glucose control

Both hyper- and hypo-glycaemia may impair outcome of critically ill adults and children and should be avoided,^{228–230,381–383} but tight glucose control may also be harmful.^{231,384} Although there is insufficient evidence to support or refute a specific glucose management strategy in children with ROSC after cardiac arrest,^{225,226,345} it is appropriate to monitor blood glucose and avoid hypoglycaemia as well as sustained hyperglycaemia.

Prognosis of cardiopulmonary arrest

Although several factors are associated with outcome after cardiopulmonary arrest and resuscitation^{41,60,385–389} there are no simple guidelines to determine when resuscitative efforts become futile.

After 20 min of resuscitation, the resuscitation team leader should consider whether or not to stop.^{273,390–394} The relevant considerations in the decision to continue the resuscitation include the cause of arrest,^{60,395} pre-existing medical conditions, age,^{41,389} site of arrest, whether the arrest was witnessed,^{60,394} the duration of untreated cardiopulmonary arrest ('no flow'), number of doses of adrenaline, the ETCO₂ value, the presence of a shockable rhythm as the first or subsequent rhythm,^{386,387} the

promptness of extracorporeal life support for a reversible disease process,^{396–398} and associated special circumstances (e.g., icy water drowning,^{277,399,400} exposure to toxic drugs).

Parental presence

In some Western societies, the majority of parents prefer to be present during the resuscitation of their child.^{401–410} Parental presence has neither been perceived as disruptive,^{403,411–415} nor stressful for the staff.^{401,403,412} Parents witnessing their child's resuscitation believe their presence to be beneficial to the child.^{401–403,410,414–417} Allowing parents to be at the side of their child helps them to gain a realistic view of the attempted resuscitation and the child's death. Furthermore, they may have the opportunity to say goodbye to their child. Families who are present at their child's death show better adjustment and undergo a better grieving process.^{402–404,414,415,417,418}

Parental presence in the resuscitation room may help healthcare providers maintain their professional behaviour, whilst helping them to see the child as a human being and a family member.⁴¹¹ However in out-of-hospital resuscitation, some EMS providers may feel threatened by the presence of relatives or are concerned that relatives may interfere with their resuscitation efforts.⁴¹⁹ Evidence about parental presence during resuscitation comes from selected countries and can probably not be generalised to all of Europe, where there could be different socio-cultural and ethical considerations.

Family presence guidelines

When relatives are allowed in the resuscitation room, 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 process. 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.^{411,420–423}

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.

References

- European Resuscitation Council. Paediatric life support: (including the recommendations for resuscitation of babies at birth). *Resuscitation* 1998;37:95–6.
- 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, 1993. *Resuscitation* 1994;27:91–105.
- Phillips B, Zideman D, Wyllie J, Richmond S, van Reempts P. European Resuscitation Council Guidelines 2000 for newly born life support. A statement from the Paediatric Life Support Working Group and approved by the Executive Committee of the European Resuscitation Council. *Resuscitation* 2001;48:235–9.
- Phillips B, Zideman D, Garcia-Castrillo L, Felix M, Shwarz-Schwier 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.
- Biarent D, Bingham R, Richmond S, et al. European Resuscitation Council Guidelines for Resuscitation 2005. Section 6. Paediatric life support. *Resuscitation* 2005;67:S97–133.
- 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.
- 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. 1):I-46–8.
- 2005 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Part 6: Paediatric basic and advanced life support. *Resuscitation* 2005;67:271–91.
- 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation*, 2010; in press.
- 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Resuscitation*, 2010; in press.
- Richmond S, Wyllie J. European Resuscitation Council Guidelines for Resuscitation 2010. Section 7. Resuscitation of babies at birth. *Resuscitation* 2010;81:1389–99.
- Tibballs J, Weeraratna C. The influence of time on the accuracy of healthcare personnel to diagnose paediatric cardiac arrest by pulse palpation. *Resuscitation* 2010;81:671–5.
- Tibballs J, Carter B, Kiraly NJ, Ragg P, Clifford M. External and internal biphasic direct current shock doses for pediatric ventricular fibrillation and pulseless ventricular tachycardia. *Pediatr Crit Care Med* 2010.
- Sarti A, Savron F, Ronfani L, Pelizzo G, Barbi E. Comparison of three sites to check the pulse and count heart rate in hypotensive infants. *Paediatr Anaesth* 2006;16:394–8.
- Sarti A, Savron F, Casotto V, Cuttini M. Heartbeat assessment in infants: a comparison of four clinical methods. *Pediatr Crit Care Med* 2005;6:212–5.
- de Caen AR, Kleinman ME, Chameides L, et al. International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Part 10: Pediatric basic and advanced life support. *Resuscitation*, 2010; doi:10.1016/j.resuscitation.2010.08.028; in press.
- Berg RA, Hilwig RW, Kern KB, Babar J, 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.
- 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.
- 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.
- 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.
- 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.
- Kitamura T, Iwami T, Kawamura T, et al. Conventional and chest-compression-only cardiopulmonary resuscitation by bystanders for children who have out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. *Lancet* 2010.
- 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 [see comment]. *Prehosp Emerg Care* 1997;1:65–7.
- David R. Closed chest cardiac massage in the newborn infant. *Pediatrics* 1988;81:552–4.
- Dorfman 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.
- 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.
- 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.
- Stevenson AG, McGowan J, Evans AL, Graham CA. CPR for children: one hand or two? *Resuscitation* 2005;64:205–8.
- 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.
- Konig B, Bengler J, Goldsworthy L. Automatic external defibrillation in a 6 year old. *Arch Dis Child* 2005;90:310–1.
- 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.
- 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.
- Atkins DL, Scott WA, Blaufox AD, et al. Sensitivity and specificity of an automated external defibrillator algorithm designed for pediatric patients. *Resuscitation* 2008;76:168–74.
- 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, International Liaison Committee on Resuscitation. *Resuscitation* 2003;57:237–43.
- 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.

36. 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.
37. 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.
38. 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.
39. Bar-Cohen Y, Walsh EP, Love BA, Cecchin F. First appropriate use of automated external defibrillator in an infant. *Resuscitation* 2005;67:135–7.
40. Divekar A, Soni R. Successful parental use of an automated external defibrillator for an infant with long-QT syndrome. *Pediatrics* 2006;118:e526–9.
41. Atkins DL, Everson-Stewart S, Sears GK, et al. Epidemiology and outcomes from out-of-hospital cardiac arrest in children: the Resuscitation Outcomes Consortium Epistry-Cardiac Arrest. *Circulation* 2009;119:1484–91.
42. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Bellon JM. Pediatric defibrillation after cardiac arrest: initial response and outcome. *Crit Care* 2006;10:R113.
43. Samson RA, Nadkarni VM, Meaney PA, Carey SM, Berg MD, Berg RA. Outcomes of in-hospital ventricular fibrillation in children. *N Engl J Med* 2006;354:2328–39.
44. Rea TD, Helbock M, Perry S, et al. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. *Circulation* 2006;114:2760–5.
45. Menegazzi JJ, Hsieh M, Niemann JT, Swor RA. Derivation of clinical predictors of failed rescue shock during out-of-hospital ventricular fibrillation. *Prehosp Emerg Care* 2008;12:347–51.
46. Rea TD, Shah S, Kudenchuk PJ, Copass MK, Cobb LA. Automated external defibrillators: to what extent does the algorithm delay CPR? *Ann Emerg Med* 2005;46:132–41.
47. Becker L, Gold LS, Eisenberg M, White L, Hearne T, Rea T. Ventricular fibrillation in King County, Washington: a 30-year perspective. *Resuscitation* 2008;79:22–7.
48. Rossano J, Quan L, Schiff M, MA K, DL A. Survival is not correlated with defibrillation dosing in pediatric out-of-hospital ventricular fibrillation. *Circulation* 2003;108. IV-320-1.
49. 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.
50. Berg MD, Samson RA, Meyer RJ, Clark LL, Valenzuela TD, Berg RA. Pediatric defibrillation doses often fail to terminate prolonged out-of-hospital ventricular fibrillation in children. *Resuscitation* 2005;67:63–7.
51. 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.
52. 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.
53. 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.
54. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. *Ann Emerg Med* 1992;21:1102–6.
55. Redding JS. The choking controversy: critique of evidence on the Heimlich maneuver. *Crit Care Med* 1979;7:475–9.
56. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests – epidemiology and outcome. *Resuscitation* 1995;30:141–50.
57. 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.
58. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. *Ann Emerg Med* 1995;25:495–501.
59. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. *Ann Emerg Med* 1999;33:195–205.
60. 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.
61. 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.
62. 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.
63. 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.
64. Tibballs J, Kinney S. Reduction of hospital mortality and of preventable cardiac arrest and death on introduction of a pediatric medical emergency team. *Pediatr Crit Care Med* 2009;10:306–12.
65. Chan PS, Jain R, Nallmothu BK, Berg RA, Sasson C. Rapid response teams: a systematic review and meta-analysis. *Arch Intern Med* 2010;170:18–26.
66. Hunt EA, Zimmer KP, Rinke ML, et al. Transition from a traditional code team to a medical emergency team and categorization of cardiopulmonary arrests in a children's center. *Arch Pediatr Adolesc Med* 2008;162:117–22.
67. Sharek PJ, Parast LM, Leong K, et al. Effect of a rapid response team on hospital-wide mortality and code rates outside the ICU in a Children's Hospital. *JAMA* 2007;298:2267–74.
68. Brilli RJ, Gibson R, Luria JW, et al. Implementation of a medical emergency team in a large pediatric teaching hospital prevents respiratory and cardiopulmonary arrests outside the intensive care unit. *Pediatr Crit Care Med* 2007;8:236–46, quiz 47.
69. Tibballs J, Kinney S, Duke T, Oakley E, Hennessy M. Reduction of paediatric inpatient cardiac arrest and death with a medical emergency team: preliminary results. *Arch Dis Child* 2005;90:1148–52.
70. Carcillo JA. Pediatric septic shock and multiple organ failure. *Crit Care Clin* 2010;9:413–40, viii.
71. Tibballs J, Russell P. Reliability of pulse palpation by healthcare personnel to diagnose paediatric cardiac arrest. *Resuscitation* 2009;80:61–4.
72. Eberle B, Dick WF, Schneider T, Wissner 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.
73. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. *Resuscitation* 2000;44:195–201.
74. Lapostolle F, Le Toumelin P, Agostinucci JM, Catoire 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.
75. 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.
76. Tsung JW, Blaivas M. Feasibility of correlating the pulse check with focused point-of-care echocardiography during pediatric cardiac arrest: a case series. *Resuscitation* 2008;77:264–9.
77. Dung NM, Day NPJ, Tam DTH, et al. Fluid replacement in dengue shock syndrome: a randomized, double-blind comparison of four intravenous-fluid regimens. *Clin Infect Dis* 1999;29:787–94.
78. Ngo NT, Cao XT, Kneen R, et al. Acute management of dengue shock syndrome: a randomized double-blind comparison of 4 intravenous fluid regimens in the first hour. *Clin Infect Dis* 2001;32:204–13.
79. Wills BA, Nguyen MD, Ha TL, et al. Comparison of three fluid solutions for resuscitation in dengue shock syndrome. *N Engl J Med* 2005;353:877–89.
80. Upadhyay M, Singhi S, Murlidharan J, Kaur N, Majumdar S. Randomized evaluation of fluid resuscitation with crystalloid (saline) and colloid (polymer from degraded gelatin in saline) in pediatric septic shock. *Indian Pediatr* 2005;42:223–31.
81. Rechner JA, Loach VJ, Ali MT, Barber VS, Young JD, Mason DG. A comparison of the laryngeal mask airway with facemask and oropharyngeal airway for manual ventilation by critical care nurses in children. *Anaesthesia* 2007;62:790–5.
82. Blevin AE, McDouall SF, Rechner JA, et al. A comparison of the laryngeal mask airway with the facemask and oropharyngeal airway for manual ventilation by first responders in children. *Anaesthesia* 2009;64:1312–6.
83. 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.
84. Harnett M, Kinirons B, Heffernan A, Motherway C, Casey W. Airway complications in infants: comparison of laryngeal mask airway and the facemask-oral airway. *Can J Anaesth* 2000;47:315–8.
85. Scheller B, Schalk R, Byhahn C, et al. Laryngeal tube suction II for difficult airway management in neonates and small infants. *Resuscitation* 2009;80:805–10.
86. Hedges JR, Mann NC, Meischke H, Robbins M, Goldberg R, Zapka J. Assessment of chest pain onset and out-of-hospital delay using standardized interview questions: the REACT Pilot Study. Rapid Early Action for Coronary Treatment (REACT) Study Group. *Acad Emerg Med* 1998;5:773–80.
87. Murphy-Macabobby M, Marshall WJ, Schneider C, Dries D. Neuromuscular blockade in aeromedical airway management. *Ann Emerg Med* 1992;21:664–8.
88. Sayre M, Weisgerber I. The use of neuromuscular blocking agents by air medical services. *J Air Med Transp* 1992;11:7–11.
89. 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.
90. 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.
91. 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.
92. 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.
93. 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.
94. Kaye K, Frascone RJ, Held T. Prehospital rapid-sequence intubation: a pilot training program. *Prehosp Emerg Care* 2003;7:235–40.
95. 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.
96. Pepe P, Zachariah B, Chandra N. Invasive airway technique in resuscitation. *Ann Emerg Med* 1991;22:393–403.

97. Eich C, Roessler M, Nemeth M, Russo SG, Heuer JF, Timmermann A. Characteristics and outcome of prehospital paediatric tracheal intubation attended by anaesthesia-trained emergency physicians. *Resuscitation* 2009;80:1371–7.
98. Sagarin MJ, Chiang V, Sakles JC, et al. Rapid sequence intubation for pediatric emergency airway management. *Pediatr Emerg Care* 2002;18:417–23.
99. Moynihan RJ, Brock-Utne JG, Archer JH, Feld LH, Kreitzman TR. The effect of cricoid pressure on preventing gastric insufflation in infants and children. *Anesthesiology* 1993;78:652–6.
100. Salem MR, Joseph NJ, Heyman HJ, Belani B, Paulissian R, Ferrara TP. Cricoid compression is effective in obliterating the esophageal lumen in the presence of a nasogastric tube. *Anesthesiology* 1985;63:443–6.
101. Walker RW, Ravi R, Haylett K. Effect of cricoid force on airway calibre in children: a bronchoscopic assessment. *Br J Anaesth* 2010;104:71–4.
102. Khine HH, Corrdry DH, Ketrick RG, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. *Anesthesiology* 1997;86:627–31, discussion 27A.
103. Weiss M, Dullenkopf A, Fischer JE, Keller C, Gerber AC. Prospective randomized controlled multi-centre trial of cuffed or uncuffed endotracheal tubes in small children. *Br J Anaesth* 2009;103:867–73.
104. Duracher C, Schmautz E, Martinon C, Faivre J, Carli P, Orliaguet G. Evaluation of cuffed tracheal tube size predicted using the Khine formula in children. *Paediatr Anaesth* 2008;18:113–8.
105. Dullenkopf A, Kretschmar O, Knirsch W, et al. Comparison of tracheal tube cuff diameters with internal transverse diameters of the trachea in children. *Acta Anaesthesiol Scand* 2006;50:201–5.
106. Dullenkopf A, Gerber AC, Weiss M. Fit and seal characteristics of a new paediatric tracheal tube with high volume-low pressure polyurethane cuff. *Acta Anaesthesiol Scand* 2005;49:232–7.
107. Salgo B, Schmitz A, Henze G, et al. Evaluation of a new recommendation for improved cuffed tracheal tube size selection in infants and small children. *Acta Anaesthesiol Scand* 2006;50:557–61.
108. Luten RC, Wears RL, Broselow J, et al. Length-based endotracheal tube and emergency equipment in pediatric. *Ann Emerg Med* 1992;21:900–4.
109. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. *J Pediatr* 1994;125:57–62.
110. 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.
111. Dorsey DP, Bowman SM, Klein MB, Archer D, Sharar SR. Perioperative use of cuffed endotracheal tubes is advantageous in young pediatric burn patients. *Burns* 2010.
112. 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.
113. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med* 2001;37:32–7.
114. 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.
115. 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.
116. Andersen KH, Hald A. Assessing the position of the tracheal tube: the reliability of different methods. *Anaesthesia* 1989;44:984–5.
117. Andersen KH, Schultz-Lebahn T. Oesophageal intubation can be undetected by auscultation of the chest. *Acta Anaesthesiol Scand* 1994;38:580–2.
118. 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.
119. 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.
120. 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.
121. Tan A, Schulze A, O'Donnell CP, Davis PG. Air versus oxygen for resuscitation of infants at birth. *Cochrane Database Syst Rev* 2004;CD002273.
122. 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.
123. 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.
124. Saugstad OD. Resuscitation of newborn infants with room air or oxygen. *Semin Neonatol* 2001;6:233–9.
125. Aufderheide TP, Sigurdsson G, Pirralo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation* 2004;109:1960–5.
126. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. *Crit Care Med* 2004;32:S345–51.
127. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA* 2005;293:299–304.
128. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA* 2005;293:305–10.
129. 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.
130. Borke WB, Munkeby BH, Morkrid L, Thaulow E, Saugstad OD. Resuscitation with 100% O₂ does not protect the myocardium in hypoxic newborn piglets. *Arch Dis Child Fetal Neonatal Ed* 2004;89:F156–60.
131. O'Neill JF, Deakin CD. Do we hyperventilate cardiac arrest patients? *Resuscitation* 2007;73:82–5.
132. 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.
133. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. *Prehosp Emerg Care* 2002;6:283–90.
134. 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.
135. DiRusso SM, Sullivan T, Risucci D, Nealon P, Slim M. Intubation of pediatric trauma patients in the field: predictor of negative outcome despite risk stratification. *J Trauma* 2005;59:84–90, discussion 1.
136. Bhende MS, Thompson AE, Orr RA. Utility of an end-tidal carbon dioxide detector during stabilization and transport of critically ill children. *Pediatrics* 1992;89:1042–4.
137. Bhende MS, LaCovey DC. End-tidal carbon dioxide monitoring in the prehospital setting. *Prehosp Emerg Care* 2001;5:208–13.
138. 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.
139. Gonzalez del Rey JA, Poirier MP, DiGiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO₂ system in the detection of airway mishaps: an animal trial. *Pediatr Emerg Care* 2000;16:121–3.
140. Bhende MS, Thompson AE. Evaluation of an end-tidal CO₂ detector during pediatric cardiopulmonary resuscitation. *Pediatrics* 1995;95:395–9.
141. 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.
142. 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.
143. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. *Ann Emerg Med* 1990;19:1104–6.
144. Mauer D, Schneider T, Elich D, Dick W. Carbon dioxide levels during pre-hospital active compression – decompression versus standard cardiopulmonary resuscitation. *Resuscitation* 1998;39:67–74.
145. Kolar M, Krizmaric M, Klemen P, Grmec S. Partial pressure of end-tidal carbon dioxide successfully predicts cardiopulmonary resuscitation in the field: a prospective observational study. *Crit Care* 2008;12:R115.
146. Callahan M, Barton C, Matthey M. Effect of epinephrine on the ability of end-tidal carbon dioxide readings to predict initial resuscitation from cardiac arrest. *Crit Care Med* 1992;20:337–43.
147. Cantineau JP, Merckx P, Lambert Y, Sorkine M, Bertrand C, Duvaldestin P. Effect of epinephrine on end-tidal carbon dioxide pressure during prehospital cardiopulmonary resuscitation. *Am J Emerg Med* 1994;12:267–70.
148. Chase PB, Kern KB, Sanders AB, Otto CW, Ewy GA. Effects of graded doses of epinephrine on both noninvasive and invasive measures of myocardial perfusion and blood flow during cardiopulmonary resuscitation. *Crit Care Med* 1993;21:413–9.
149. Gonzalez ER, Ornato JP, Garnett AR, Levine RL, Young DS, Racht EM. Dose-dependent vasopressor response to epinephrine during CPR in human beings. *Ann Emerg Med* 1989;18:920–6.
150. Lindberg L, Liao Q, Steen S. The effects of epinephrine/norepinephrine on end-tidal carbon dioxide concentration, coronary perfusion pressure and pulmonary arterial blood flow during cardiopulmonary resuscitation. *Resuscitation* 2000;43:129–40.
151. Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;318:607–11.
152. Sharieff GQ, Rodarte A, Wilton N, Bleyde 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.
153. Sharieff GQ, Rodarte A, Wilton N, Silva PD, Bleyde 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.
154. 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.
155. Lillis KA, Jaffe DM. Prehospital intravenous access in children. *Ann Emerg Med* 1992;21:1430–4.
156. 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.
157. Hedges JR, Barsan WB, Doan LA, et al. Central versus peripheral intravenous routes in cardiopulmonary resuscitation. *Am J Emerg Med* 1984;2:385–90.
158. Kanter RK, Zimmerman JJ, Strauss RH, Stoeckel KA. Pediatric emergency intravenous access. Evaluation of a protocol. *Am J Dis Child* 1986;140:132–4.
159. 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.
160. Glaeser PW, Hellmich TR, Szwecuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. *Ann Emerg Med* 1993;22:1119–24.

161. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr Surg* 1993;28:158–61.
162. Orłowski 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.
163. Orłowski 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.
164. Abe KK, Blum GT, Yamamoto LG. Intraosseous is faster and easier than umbilical venous catheterization in newborn emergency vascular access models. *Am J Emerg Med* 2000;18:126–9.
165. Ellemunter H, Simma B, Trawogger R, Maurer H. Intraosseous lines in preterm and full term neonates. *Arch Dis Child Fetal Neonatal Ed* 1999;80:F74–5.
166. Fiorito BA, Mirza F, Doran TM, et al. Intraosseous access in the setting of pediatric critical care transport. *Pediatr Crit Care Med* 2005;6:50–3.
167. Horton MA, Beamer C. Powered intraosseous insertion provides safe and effective vascular access for pediatric emergency patients. *Pediatr Emerg Care* 2008;24:347–50.
168. Frascione RJ, Jensen J, Wewerka SS, Salzman JG. Use of the pediatric EZ-IO needle by emergency medical services providers. *Pediatr Emerg Care* 2009;25:329–32.
169. Cameron JL, Fontanarosa PB, Passalacqua 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.
170. 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.
171. 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.
172. 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.
173. Ummehofer 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.
174. 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.
175. Voelckel WC, Lindner KH, Wenzel V, et al. Intraosseous blood gases during hypothermia: correlation with arterial, mixed venous, and sagittal sinus blood. *Crit Care Med* 2000;28:2915–20.
176. Kissoon N, Peterson R, Murphy S, Gayle M, Ceithaml E, Harwood-Nuss A. Comparison of pH and carbon dioxide tension values of central venous and intraosseous blood during changes in cardiac output. *Crit Care Med* 1994;22:1010–5.
177. Eisenkraft A, Gilat E, Chapman S, Baranes S, Egoz I, Levy A. Efficacy of the bone injection gun in the treatment of organophosphate poisoning. *Biopharm Drug Dispos* 2007;28:145–50.
178. Brenner T, Bernhard M, Helm M, et al. Comparison of two intraosseous infusion systems for adult emergency medical use. *Resuscitation* 2008;78:314–9.
179. Venkataraman ST, Orr RA, Thompson AE. Percutaneous infraclavicular subclavian vein catheterization in critically ill infants and children. *J Pediatr* 1988;113:480–5.
180. 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.
181. 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.
182. 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.
183. Hahnel JH, Lindner KH, Schurmann C, Prengel A, Ahnefeld FW. Plasma lidocaine levels and PaO₂ with endobronchial administration: dilution with normal saline or distilled water? *Ann Emerg Med* 1990;19:1314–7.
184. 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.
185. 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.
186. Carcillo JA, Fields AL. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. *Crit Care Med* 2002;30:1365–78.
187. 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.
188. Myburgh J, Cooper DJ, Finfer S, et al. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. *N Engl J Med* 2007;357:874–84.
189. Rocha E, Silva M. Hypertonic saline resuscitation. *Medicina* 1998;58:393–402.
190. 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.
191. 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.
192. 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.
193. 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.
194. 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.
195. Losek JD, Endom E, Dietrich A, Stewart G, Zempsky W, Smith K. Adenosine and pediatric supraventricular tachycardia in the emergency department: multi-center study and review. *Ann Emerg Med* 1999;33:185–91.
196. 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.
197. Zaritsky A. Pediatric resuscitation pharmacology. Members of the Medications in Pediatric Resuscitation Panel. *Ann Emerg Med* 1993;22:445–55.
198. 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.
199. 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.
200. 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.
201. 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.
202. 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.
203. Dieckmann RA, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. *Pediatrics* 1995;95:901–13.
204. 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.
205. 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.
206. 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.
207. 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.
208. Dauchot P, Gravenstein JS. Effects of atropine on the electrocardiogram in different age groups. *Clin Pharmacol Ther* 1971;12:274–80.
209. Yilmaz O, Eser M, Sahiner A, Altintop L, Yesildag O. Hypotension, bradycardia and syncope caused by honey poisoning. *Resuscitation* 2006;68:405–8.
210. Brady WJ, Swart G, DeBehnke DJ, Ma OJ, Aufderheide TP. The efficacy of atropine in the treatment of hemodynamically unstable bradycardia and atrioventricular block: prehospital and emergency department considerations. *Resuscitation* 1999;41:47–55.
211. Smith I, Monk TG, White PF. Comparison of transesophageal atrial pacing with anticholinergic drugs for the treatment of intraoperative bradycardia. *Anesth Analg* 1994;78:245–52.
212. Chadda KD, Lichstein E, Gupta PK, Kourtesis P. Effects of atropine in patients with bradyarrhythmia complicating myocardial infarction: usefulness of an optimum dose for overdrive. *Am J Med* 1977;63:503–10.
213. 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.
214. van Walraven C, Stiell IG, Wells GA, Hebert PC, Vandemheen K. Do advanced cardiac life support drugs increase resuscitation rates from in-hospital cardiac arrest? The OTAC Study Group. *Ann Emerg Med* 1998;32:544–53.
215. Paraskos JA. Cardiovascular pharmacology III: atropine, calcium, calcium blockers, and (beta)-blockers. *Circulation* 1986;74:IV-86–9.
216. 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.
217. 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.
218. Srinivasan V, Morris MC, Helfaer MA, Berg RA, Nadkarni VM. Calcium use during in-hospital pediatric cardiopulmonary resuscitation: a report from the National Registry of Cardiopulmonary Resuscitation. *Pediatrics* 2008;121:e1144–51.
219. de Mos N, van Litsenburg RR, McCrindle B, Bohn DJ, Parshuram CS. Pediatric intensive-care-unit cardiac arrest: incidence, survival, and predictive factors. *Crit Care Med* 2006;34:1209–15.

220. Meert KL, Donaldson A, Nadkarni V, et al. Multicenter cohort study of in-hospital pediatric cardiac arrest. *Pediatr Crit Care Med* 2009;10:544–53.
221. 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.
222. Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. *Mayo Clin Proc* 2004;79:992–1000.
223. Losek JD. Hypoglycemia and the ABC'S (sugar) of pediatric resuscitation. *Ann Emerg Med* 2000;35:43–6.
224. Finney SJ, Zekveld C, Elia A, Evans TW. Glucose control and mortality in critically ill patients. *JAMA* 2003;290:2041–7.
225. Losert H, Sterz F, Roine RO, et al. Strict normoglycaemic blood glucose levels in the therapeutic management of patients within 12 h after cardiac arrest might not be necessary. *Resuscitation* 2008;76:214–20.
226. Oksanen T, Skrifvars MB, Varpula T, et al. Strict versus moderate glucose control after resuscitation from ventricular fibrillation. *Intensive Care Med* 2007;33:2093–100.
227. Beardsall K, Vanhaesebrouck S, Ogilvy-Stuart AL, et al. Early insulin therapy in very-low-birth-weight infants. *N Engl J Med* 2008;359:1873–84.
228. Vlasselaers D, Milants I, Desmet L, et al. Intensive insulin therapy for patients in paediatric intensive care: a prospective, randomised controlled study. *Lancet* 2009;373:547–56.
229. Gandhi GY, Murad MH, Flynn DN, et al. Effect of perioperative insulin infusion on surgical morbidity and mortality: systematic review and meta-analysis of randomized trials. *Mayo Clin Proc* 2008;83:418–30.
230. Griesdale DE, de Souza RJ, van Dam RM, et al. Intensive insulin therapy and mortality among critically ill patients: a meta-analysis including NICE-SUGAR study data. *CMAJ* 2009;180:821–7.
231. Finfer S, Chittock DR, Su SY, et al. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med* 2009;360:1283–97.
232. 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.
233. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. *Circulation* 1988;77:392–7.
234. 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.
235. 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.
236. 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.
237. 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.
238. Wang JD, Fu YC, Jan SL, Chi CS. Verapamil sensitive idiopathic ventricular tachycardia in an infant. *Jpn Heart J* 2003;44:667–71.
239. Chang PM, Silka MJ, Moromisato DY, Bar-Cohen Y. Amiodarone versus procainamide for the acute treatment of recurrent supraventricular tachycardia in pediatric patients. *Circ Arrhythm Electrophysiol* 2010;3:134–40.
240. Singh BN, Kehoe R, Woosley RL, Scheinman M, Quart B. Multicenter trial of sotalol compared with procainamide in the suppression of inducible ventricular tachycardia: a double-blind, randomized parallel evaluation. Sotalol Multicenter Study Group. *Am Heart J* 1995;129:87–97.
241. 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.
242. 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.
243. Mandapati R, Byrum CJ, Kavey RE, et al. Procainamide for rate control of post-surgical junctional tachycardia. *Pediatr Cardiol* 2000;21:123–8.
244. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. *J Formos Med Assoc* 2000;99:766–70.
245. Wang R, Schuyler J, Raymond R. The role of the cell membrane bicarbonate exchanger in NaHCO₃ therapy of imipramine cardiac dysfunction. *J Toxicol Clin Toxicol* 1997;35:533.
246. Holmes CL, Landry DW, Granton JT. Science review: vasopressin and the cardiovascular system. Part 1 – Receptor physiology. *Crit Care* 2003;7:427–34.
247. 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.
248. 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.
249. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. *Resuscitation* 2002;52:149–56.
250. Duncan JM, Meaney P, Simpson P, Berg RA, Nadkarni V, Schexnayder S. Vasopressin for in-hospital pediatric cardiac arrest: results from the American Heart Association National Registry of Cardiopulmonary Resuscitation. *Pediatr Crit Care Med* 2009;10:191–5.
251. Callaway CW, Hostler D, Doshi AA, et al. Usefulness of vasopressin administered with epinephrine during out-of-hospital cardiac arrest. *Am J Cardiol* 2006;98:1316–21.
252. Gueugniaud PY, David JS, Chanzy E, et al. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. *N Engl J Med* 2008;359:21–30.
253. Mukoyama T, Kinoshita K, Nagao K, Tanjoh K. Reduced effectiveness of vasopressin in repeated doses for patients undergoing prolonged cardiopulmonary resuscitation. *Resuscitation* 2009;80:755–61.
254. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. *Arch Intern Med* 2005;165:17–24.
255. Matok I, Vardi A, Efrati O, et al. Terlipressin as rescue therapy for intractable hypotension due to septic shock in children. *Shock* 2005;23:305–10.
256. Peters MJ, Booth RA, Petros AJ. Terlipressin bolus induces systemic vasoconstriction in septic shock. *Pediatr Crit Care Med* 2004;5:112–5.
257. Rodriguez-Nunez A, Lopez-Herce J, Gil-Anton J, Hernandez A, Rey C. Rescue treatment with terlipressin in children with refractory septic shock: a clinical study. *Crit Care* 2006;10:R20.
258. Gil-Anton J, Lopez-Herce J, Morteruel E, Carrillo A, Rodriguez-Nunez A. Pediatric cardiac arrest refractory to advanced life support: is there a role for terlipressin? *Pediatr Crit Care Med* 2010;11:139–41.
259. Rodriguez-Nunez A, Fernandez-Sanmartin M, Martinon-Torres F, Gonzalez-Alonso N, Martinon-Sanchez JM. Terlipressin for catecholamine-resistant septic shock in children. *Intensive Care Med* 2004;30:477–80.
260. Yildizdas D, Yapicioglu H, Celik U, Sertdemir Y, Alhan E. Terlipressin as a rescue therapy for catecholamine-resistant septic shock in children. *Intensive Care Med* 2008;34:511–7.
261. Matok I, Vardi A, Augarten A, et al. Beneficial effects of terlipressin in prolonged pediatric cardiopulmonary resuscitation: a case series. *Crit Care Med* 2007;35:1161–4.
262. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using “adult” electrode paddles. *Pediatrics* 1994;94:90–3.
263. 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.
264. Deakin C, Sado D, Petley G, Clewlow F. Determining the optimal paddle force for external defibrillation. *Am J Cardiol* 2002;90:812–3.
265. Bennetts SH, Deakin CD, Petley GW, Clewlow F. Is optimal paddle force applied during paediatric external defibrillation? *Resuscitation* 2004;60:29–32.
266. Berg MD, Banville IL, Chapman FW, et al. Attenuating the defibrillation dosage decreases postresuscitation myocardial dysfunction in a swine model of pediatric ventricular fibrillation. *Pediatr Crit Care Med* 2008;9:429–34.
267. Atkins DL, Hartley LL, York DK. Accurate recognition and effective treatment of ventricular fibrillation by automated external defibrillators in adolescents. *Pediatrics* 1998;101:393–7.
268. Edelson DP, Abella BS, Kramer-Johansen J, et al. Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. *Resuscitation* 2006;71:137–45.
269. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. *Circulation* 2002;105:2270–3.
270. Saul JP, Scott WA, Brown S, et al. Intravenous amiodarone for incessant tachyarrhythmias in children: a randomized, double-blind, antiarrhythmic drug trial. *Circulation* 2005;112:3470–7.
271. Pierpont GL, Kruse JA, Nelson DH. Intra-arterial monitoring during cardiopulmonary resuscitation. *Catheterization Cardiovasc Diagn* 1985;11:513–20.
272. Steiger HV, Rimbach K, Muller E, Breikreutz R. Focused emergency echocardiography: lifesaving tool for a 14-year-old girl suffering out-of-hospital pulseless electrical activity arrest because of cardiac tamponade. *Eur J Emerg Med* 2009;16:103–5.
273. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. *Ann Emerg Med* 1987;16:1107–11.
274. 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.
275. 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.
276. 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.
277. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. *Resuscitation* 1995;30:141–50.
278. Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury. *Pediatrics* 1994;94:137–42.
279. 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.
280. 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.
281. 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.

282. 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.
283. 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.
284. 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.
285. 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.
286. 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.
287. 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.
288. Cummins RO, Graves JR, Larsen MP, et al. Out-of-hospital transcatheter pacing by emergency medical technicians in patients with asystolic cardiac arrest. *N Engl J Med* 1993;328:1377–82.
289. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. *Arch Dis Child* 1990;65:127–9.
290. Bianconi L, Castro A, 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.
291. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. *Eur J Pediatr* 2003;162:880–4.
292. Celiker A, Ceviz N, Ozme S. Effectiveness and safety of intravenous amiodarone in drug-resistant tachyarrhythmias of children. *Acta Paediatr Jpn* 1998;40:567–72.
293. 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 Cardiothorac Surg* 2002;21:255–9.
294. 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.
295. 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.
296. 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.
297. Haas NA, Camphausen CK. Acute hemodynamic effects of intravenous amiodarone treatment in pediatric patients with cardiac surgery. *Clin Res Cardiol* 2008;97:801–10.
298. Adamson PC, Rhodes LA, Saul JP, et al. The pharmacokinetics of esmolol in pediatric subjects with supraventricular arrhythmias. *Pediatr Cardiol* 2006;27:420–7.
299. Chrysostomou C, Beerman L, Shiderly D, Berry D, Morell VO, Munoz R. Dexmedetomidine: a novel drug for the treatment of atrial and junctional tachyarrhythmias during the perioperative period for congenital cardiac surgery: a preliminary study. *Anesth Analg* 2008;107:1514–22.
300. 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.
301. Drago F, Mazza A, Guccione P, Mafri 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.
302. Benson DJ, Dunnigan A, Green T, Benditt D, Schneider S. Periodic procainamide for paroxysmal tachycardia. *Circulation* 1985;72:147–52.
303. 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.
304. 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.
305. Meldon SW, Brady WJ, Berger S, Mannenbach M. Pediatric ventricular tachycardia: a review with three illustrative cases. *Pediatr Emerg Care* 1994;10:294–300.
306. 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.
307. Ackerman MJ, Siu BL, Sturmer WQ, et al. Postmortem molecular analysis of SCN5A defects in sudden infant death syndrome. *JAMA* 2001;286:2264–9.
308. Arnestad M, Crotti L, Rognum TO, et al. Prevalence of long-QT syndrome gene variants in sudden infant death syndrome. *Circulation* 2007;115:361–7.
309. Cronk LB, Ye B, Kaku T, et al. Novel mechanism for sudden infant death syndrome: persistent late sodium current secondary to mutations in caveolin-3. *Heart Rhythm* 2007;4:161–6.
310. Millat G, Kugener B, Chevalier P, et al. Contribution of long-QT syndrome genetic variants in sudden infant death syndrome. *Pediatr Cardiol* 2009;30:502–9.
311. Otogiri T, Kijima K, Osawa M, et al. Cardiac ion channel gene mutations in sudden infant death syndrome. *Pediatr Res* 2008;64:482–7.
312. Plant LD, Bowers PN, Liu Q, et al. A common cardiac sodium channel variant associated with sudden infant death in African Americans, SCN5A S1103Y. *J Clin Invest* 2006;116:430–5.
313. Tester DJ, Dura M, Carturan E, et al. A mechanism for sudden infant death syndrome (SIDS): stress-induced leak via ryanodine receptors. *Heart Rhythm* 2007;4:733–9.
314. Albert CM, Nam EG, Rimm EB, et al. Cardiac sodium channel gene variants and sudden cardiac death in women. *Circulation* 2008;117:16–23.
315. Chugh SS, Senashova O, Watts A, et al. Postmortem molecular screening in unexplained sudden death. *J Am Coll Cardiol* 2004;43:1625–9.
316. Tester DJ, Spoon DB, Valdivia HH, Makielski JC, Ackerman MJ. Targeted mutational analysis of the RyR2-encoded cardiac ryanodine receptor in sudden unexplained death: a molecular autopsy of 49 medical examiner/coroner's cases. *Mayo Clin Proc* 2004;79:1380–4.
317. Calkins CM, Bensard DD, Partrick DA, Karrer FM. A critical analysis of outcome for children sustaining cardiac arrest after blunt trauma. *J Pediatr Surg* 2002;37:180–4.
318. Crewdson K, Lockey D, Davies G. Outcome from paediatric cardiac arrest associated with trauma. *Resuscitation* 2007;75:29–34.
319. Lopez-Herce Cid J, Dominguez Sampedro P, Rodriguez Nunez A, et al. Cardiorespiratory arrest in children with trauma. *An Pediatr (Barc)* 2006;65:439–47.
320. Perron AD, Sing RF, Branas CC, Huynh T. Predicting survival in pediatric trauma patients receiving cardiopulmonary resuscitation in the prehospital setting. *Prehosp Emerg Care* 2001;5:6–9.
321. Sheikh A, Brogan T. Outcome and cost of open- and closed-chest cardiopulmonary resuscitation in pediatric cardiac arrests. *Pediatrics* 1994;93:392–8.
322. Beaver BL, Colombani PM, Buck JR, Dudgeon DL, Bohrer SL, Haller Jr JA. Efficacy of emergency room thoracotomy in pediatric trauma. *J Pediatr Surg* 1987;22:19–23.
323. Powell RW, Gill EA, Jurkovich GJ, Ramenofsky ML. Resuscitative thoracotomy in children and adolescents. *Am Surg* 1988;54:188–91.
324. Rothenberg SS, Moore EE, Moore FA, Baxter BT, Moore JB, Cleveland HC. Emergency department thoracotomy in children – a critical analysis. *J Trauma* 1989;29:1322–5.
325. Suominen P, Rasanen J, Kivioja A. Efficacy of cardiopulmonary resuscitation in pulseless paediatric trauma patients. *Resuscitation* 1998;36:9–13.
326. Graham EM, Forbus GA, Bradley SM, Shirali GS, Atz AM. Incidence and outcome of cardiopulmonary resuscitation in patients with shunted single ventricle: advantage of right ventricle to pulmonary artery shunt. *J Thorac Cardiovasc Surg* 2006;131:e7–8.
327. Charpie JR, Dekeon MK, Goldberg CS, Mosca RS, Bove EL, Kulik TJ. Postoperative hemodynamics after Norwood palliation for hypoplastic left heart syndrome. *Am J Cardiol* 2001;87:198–202.
328. Hoffman GM, Mussatto KA, Brosig CL, et al. Systemic venous oxygen saturation after the Norwood procedure and childhood neurodevelopmental outcome. *J Thorac Cardiovasc Surg* 2005;130:1094–100.
329. Johnson BA, Hoffman GM, Tweddell JS, et al. Near-infrared spectroscopy in neonates before palliation of hypoplastic left heart syndrome. *Ann Thorac Surg* 2009;87:571–7, discussion 7–9.
330. Hoffman GM, Tweddell JS, Ghanayem NS, et al. Alteration of the critical arteriovenous oxygen saturation relationship by sustained afterload reduction after the Norwood procedure. *J Thorac Cardiovasc Surg* 2004;127:738–45.
331. De Oliveira NC, Van Arsdell GS. Practical use of alpha blockade strategy in the management of hypoplastic left heart syndrome following stage one palliation with a Blalock-Taussig shunt. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu* 2004;7:11–5.
332. Tweddell JS, Hoffman GM, Mussatto KA, et al. Improved survival of patients undergoing palliation of hypoplastic left heart syndrome: lessons learned from 115 consecutive patients. *Circulation* 2002;106:182–9.
333. Shekerdemian LS, Shore DF, Lincoln C, Bush A, Redington AN. Negative-pressure ventilation improves cardiac output after right heart surgery. *Circulation* 1996;94:II49–55.
334. Shekerdemian LS, Bush A, Shore DF, Lincoln C, Redington AN. Cardiopulmonary interactions after Fontan operations: augmentation of cardiac output using negative pressure ventilation. *Circulation* 1997;96:3934–42.
335. Booth KL, Roth SJ, Thiagarajan RR, Almodovar MC, del Nido PJ, Laussen PC. Extracorporeal membrane oxygenation support of the Fontan and bidirectional Glenn circulations. *Ann Thorac Surg* 2004;77:1341–8.
336. Polderman FN, Cohen J, Blom NA, et al. Sudden unexpected death in children with a previously diagnosed cardiovascular disorder. *Int J Cardiol* 2004;95:171–6.
337. Sanatani S, Wilson G, Smith CR, Hamilton RM, Williams WG, Adatia I. Sudden unexpected death in children with heart disease. *Congenit Heart Dis* 2006;1:89–97.
338. Morris K, Beghetti M, Petros A, Adatia I, Bohn D. Comparison of hyperventilation and inhaled nitric oxide for pulmonary hypertension after repair of congenital heart disease. *Crit Care Med* 2000;28:2974–8.
339. Hooper MM, Galie N, Murali S, et al. Outcome after cardiopulmonary resuscitation in patients with pulmonary arterial hypertension. *Am J Respir Crit Care Med* 2002;165:341–4.
340. Rimensberger PC, Spahr-Schopfer I, Berner M, et al. Inhaled nitric oxide versus aerosolized iloprost in secondary pulmonary hypertension in children with congenital heart disease: vasodilator capacity and cellular mechanisms. *Circulation* 2001;103:544–8.

341. Liu KS, Tsai FC, Huang YK, et al. Extracorporeal life support: a simple and effective weapon for postcardiotomy right ventricular failure. *Artif Organs* 2009;33:504–8.
342. Dhillon R, Pearson GA, Firmin RK, Chan KC, Leanage R. Extracorporeal membrane oxygenation and the treatment of critical pulmonary hypertension in congenital heart disease. *Eur J Cardiothorac Surg* 1995;9:553–6.
343. Arpesella G, Loforte A, Mikus E, Mikus PM. Extracorporeal membrane oxygenation for primary allograft failure. *Transplant Proc* 2008;40:3596–7.
344. Strueber M, Hoepfer MM, Fischer S, et al. Bridge to thoracic organ transplantation in patients with pulmonary arterial hypertension using a pumpless lung assist device. *Am J Transplant* 2009;9:853–7.
345. Nolan JP, Neumar RW, Adrie C, et al. Post-cardiac arrest syndrome: epidemiology, pathophysiology, treatment, and prognostication. A Scientific Statement from the International Liaison Committee on Resuscitation; the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; the Council on Stroke. *Resuscitation* 2008;79:350–79.
346. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. *Crit Care Med* 1988;16:331–5.
347. 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.
348. Mayr V, Luckner G, Jochberger S, et al. Arginine vasopressin in advanced cardiovascular failure during the post-resuscitation phase after cardiac arrest. *Resuscitation* 2007;72:35–44.
349. Huang L, Weil MH, Sun S, Cammarata G, Cao L, Tang W. Levosimendan improves postresuscitation outcomes in a rat model of CPR. *J Lab Clin Med* 2005;146:256–61.
350. Huang L, Weil MH, Tang W, Sun S, Wang J. Comparison between dobutamine and levosimendan for management of postresuscitation myocardial dysfunction. *Crit Care Med* 2005;33:487–91.
351. Kern KB, Hilwig RW, Rhee KH, Berg RA. Myocardial dysfunction after resuscitation from cardiac arrest: an example of global myocardial stunning. *J Am Coll Cardiol* 1996;28:232–40.
352. Meyer RJ, Kern KB, Berg RA, Hilwig RW, Ewy GA. Post-resuscitation right ventricular dysfunction: delineation and treatment with dobutamine. *Resuscitation* 2002;55:187–91.
353. Studer W, Wu X, Siegemund M, Marsch S, Seeberger M, Filipovic M. Influence of dobutamine on the variables of systemic haemodynamics, metabolism, and intestinal perfusion after cardiopulmonary resuscitation in the rat. *Resuscitation* 2005;64:227–32.
354. Vasquez A, Kern KB, Hilwig RW, Heidenreich J, Berg RA, Ewy GA. Optimal dosing of dobutamine for treating post-resuscitation left ventricular dysfunction. *Resuscitation* 2004;61:199–207.
355. Hoffman TM, Wernovsky G, Atz AM, et al. Efficacy and safety of milrinone in preventing low cardiac output syndrome in infants and children after corrective surgery for congenital heart disease. *Circulation* 2003;107:996–1002.
356. Alvarez J, Bouzada M, Fernandez AL, et al. Hemodynamic effects of levosimendan compared with dobutamine in patients with low cardiac output after cardiac surgery. *Rev Esp Cardiol* 2006;59:338–45.
357. Jorgensen K, Bech-Hanssen O, Houltz E, Ricksten SE. Effects of levosimendan on left ventricular relaxation and early filling at maintained preload and afterload conditions after aortic valve replacement for aortic stenosis. *Circulation* 2008;117:1075–81.
358. Lobato EB, Willert JL, Looke TD, Thomas J, Urdaneta F. Effects of milrinone versus epinephrine on left ventricular relaxation after cardiopulmonary bypass following myocardial revascularization: assessment by color m-mode and tissue Doppler. *J Cardiothorac Vasc Anesth* 2005;19:334–9.
359. Nijhawan N, Nicolosi AC, Montgomery MW, Aggarwal A, Pagel PS, Warltire DC. Levosimendan enhances cardiac performance after cardiopulmonary bypass: a prospective, randomized placebo-controlled trial. *J Cardiovasc Pharmacol* 1999;34:219–28.
360. Hickey RW, Kochanek PM, Ferimer H, Graham SH, Safar P. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. *Pediatrics* 2000;106:118–22.
361. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346:549–56.
362. 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.
363. 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.
364. 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.
365. 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.
366. Gunn AJ, Gunn TR, Gunning MI, Williams CE, Gluckman PD. Neuroprotection with prolonged head cooling started before postischemic seizures in fetal sheep. *Pediatrics* 1998;102:1098–106.
367. 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.
368. Shankaran S, Laptook AR, Ehrenkranz RA, et al. Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. *N Engl J Med* 2005;353:1574–84.
369. Doherty DR, Parshuram CS, Gaboury I, et al. Hypothermia therapy after pediatric cardiac arrest. *Circulation* 2009;119:1492–500.
370. 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.
371. 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.
372. 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.
373. Polderman KH. Application of therapeutic hypothermia in the ICU: opportunities and pitfalls of a promising treatment modality. Part 1: Indications and evidence. *Intensive Care Med* 2004;30:556–75.
374. 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.
375. Polderman KH, Peerdeman SM, Girbes AR. Hypophosphatemia and hypomagnesemia induced by cooling in patients with severe head injury. *J Neurosurg* 2001;94:697–705.
376. 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.
377. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. *Intensive Care Med* 1991;17:419–20.
378. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? *Resuscitation* 2001;49:273–7.
379. 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* 1996;92:447–53.
380. 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.
381. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. *N Engl J Med* 2001;345:1359–67.
382. Van den Berghe G, Wilmer A, Hermans G, et al. Intensive insulin therapy in the medical ICU. *N Engl J Med* 2006;354:449–61.
383. Wiener RS, Wiener DC, Larson RJ. Benefits and risks of tight glucose control in critically ill adults: a meta-analysis. *JAMA* 2008;300:933–44.
384. Treggiari MM, Karir V, Yanez ND, Weiss NS, Daniel S, Deem SA. Intensive insulin therapy and mortality in critically ill patients. *Crit Care* 2008;12:R29.
385. Slonim AD, Patel KM, Ruttimann UE, Pollack MM. Cardiopulmonary resuscitation in pediatric intensive care units. *Crit Care Med* 1997;25:1951–5.
386. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, et al. Effectiveness and long-term outcome of cardiopulmonary resuscitation in paediatric intensive care units in Spain. *Resuscitation* 2006;71:301–9.
387. Nadkarni VM, Larkin GL, Peberdy MA, et al. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. *JAMA* 2006;295:50–7.
388. Meaney PA, Nadkarni VM, Cook EF, et al. Higher survival rates among younger patients after pediatric intensive care unit cardiac arrests. *Pediatrics* 2006;118:2424–33.
389. Tibbals J, Kinney S. A prospective study of outcome of in-patient paediatric cardiopulmonary arrest. *Resuscitation* 2006;71:310–8.
390. Gillis J, Dickson D, Rieder M, Steward D, Edmonds J. Results of inpatient pediatric resuscitation. *Crit Care Med* 1986;14:469–71.
391. 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.
392. 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.
393. Lopez-Herce J, Garcia C, Dominguez P, et al. Outcome of out-of-hospital cardiorespiratory arrest in children. *Pediatr Emerg Care* 2005;21:807–15.
394. Lopez-Herce J, Garcia C, Dominguez P, et al. Characteristics and outcome of cardiorespiratory arrest in children. *Resuscitation* 2004;63:311–20.
395. Hazinski MF, Chahine AA, Holcomb 3rd GW, Morris Jr JA. Outcome of cardiovascular collapse in pediatric blunt trauma. *Ann Emerg Med* 1994;23:1229–35.
396. 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.
397. 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.
398. 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.
399. 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.
400. Eich C, Brauer A, Timmermann A, et al. Outcome of 12 drowned children with attempted resuscitation on cardiopulmonary bypass: an analysis of variables based on the “Utstein Style for Drowning”. *Resuscitation* 2007;75:42–52.

401. Dudley NC, Hansen KW, Furnival RA, Donaldson AE, Van Wagenen KL, Scaife ER. The effect of family presence on the efficiency of pediatric trauma resuscitations. *Ann Emerg Med* 2009;53:777–84, e3.
402. Tinsley C, Hill JB, Shah J, et al. Experience of families during cardiopulmonary resuscitation in a pediatric intensive care unit. *Pediatrics* 2008;122:e799–804.
403. Mangurten J, Scott SH, Guzzetta CE, et al. Effects of family presence during resuscitation and invasive procedures in a pediatric emergency department. *J Emerg Nurs* 2006;32:225–33.
404. McGahey-Oakland PR, Lieder HS, Young A, et al. Family experiences during resuscitation at a children's hospital emergency department. *J Pediatr Health Care* 2007;21:217–25.
405. Jones M, Qazi M, Young KD. Ethnic differences in parent preference to be present for painful medical procedures. *Pediatrics* 2005;116:e191–7.
406. 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.
407. Andrews R, Andrews R. Family presence during a failed major trauma resuscitation attempt of a 15-year-old boy: lessons learned [see comment]. *J Emerg Nurs* 2004;30:556–8.
408. Dill K, Gance-Cleveland B, Dill K, Gance-Cleveland B. With you until the end: family presence during failed resuscitation. *J Specialists Pediatr Nurs: JSPN* 2005;10:204–7.
409. Gold KJ, Gorenflo DW, Schwenk TL, et al. Physician experience with family presence during cardiopulmonary resuscitation in children [see comment]. *Pediatr Crit Care Med* 2006;7:428–33.
410. Duran CR, Oman KS, Abel JJ, Koziel VM, Szymanski D. Attitudes toward and beliefs about family presence: a survey of healthcare providers, patients' families, and patients. *Am J Crit Care* 2007;16:270–9.
411. Meyers TA, Eichhorn DJ, Guzzetta CE, et al. Family presence during invasive procedures and resuscitation. *Am J Nurs* 2000;100:32–42, quiz 3.
412. O'Connell KJ, Farah MM, Spandorfer P, et al. Family presence during pediatric trauma team activation: an assessment of a structured program. *Pediatrics* 2007;120:e565–74.
413. Engel KG, Barnosky AR, Berry-Bovia M, et al. Provider experience and attitudes toward family presence during resuscitation procedures. *J Palliative Med* 2007;10:1007–9.
414. Holzhauser K, Finucane J, De Vries S. Family presence during resuscitation: a randomised controlled trial of the impact of family presence. *Australasian Emerg Nurs J* 2005;8:139–47.
415. 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.
416. Meyers TA, Eichhorn DJ, Guzzetta CE. Do families want to be present during CPR? A retrospective survey. *J Emerg Nurs* 1998;24:400–5.
417. Hanson C, Strawser D. Family presence during cardiopulmonary resuscitation: foote Hospital emergency department's nine-year perspective. *J Emerg Nurs* 1992;18:104–6.
418. 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.
419. Compton S, Madgy A, Goldstein M, et al. Emergency medical service providers' experience with family presence during cardiopulmonary resuscitation. *Resuscitation* 2006;70:223–8.
420. 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.
421. Eppich WJ, Arnold LD. Family member presence in the pediatric emergency department. *Curr Opin Pediatr* 2003;15:294–8.
422. Eichhorn DJ, Meyers TA, Mitchell TG, Guzzetta CE. Opening the doors: family presence during resuscitation. *J Cardiovasc Nurs* 1996;10:59–70.
423. Jarvis AS. Parental presence during resuscitation: attitudes of staff on a paediatric intensive care unit. *Intensive Crit Care Nurs* 1998;14:3–7.