Resuscitation 95 (2015) 223–248

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

European Resuscitation Council Guidelines for Resuscitation 2015 Section 6. Paediatric life support

Ian K. Maconochie[a,*], Robert Bingham[b], Christoph Eich[c], Jesús López-Herce[d], Antonio Rodríguez-Núñez[e], Thomas Rajka[f], Patrick Van de Voorde[g], David A. Zideman[h], Dominique Biarent[i], on behalf of the Paediatric life support section Collaborators[1]

[a] Paediatric Emergency Medicine Department, Imperial College Healthcare NHS Trust and BRC Imperial NIHR, Imperial College, London, UK
[b] Department of Paediatric Anaesthesia, Great Ormond Street Hospital for Children, London, UK
[c] Department of Anaesthesia, Paediatric Intensive Care and Emergency Medicine, Auf der Bult Children's Hospital, Hannover, Germany
[d] Paediatric Intensive Care Department, Hospital General Universitario Gregorio Marañón, Medical School, Complutense University of Madrid, Madrid, Spain
[e] Paediatric Emergency and Critical Care Division, Paediatric Area Hospital Clinico Universitario de Santiago de Compostela, Santiago de Compostela, Spain
[f] Paediatric Intensive Care Department, Womens and Childrens Division, Oslo University Hospital, Ulleval, Oslo, Norway
[g] Paediatric Intensive Care and Emergency Medicine Departments, University Hospital Ghent and Ghent University, EMS Dispatch 112 Eastern Flanders, Federal Department Health Belgium, Ghent, Belgium
[h] Anaesthesia Department, Imperial College Healthcare NHS Trust, London, UK
[i] Paediatric Intensive Care and Emergency Medicine Departments, Universite Libre de Bruxelles, Hôpital Universitaire des Enfants, Brussels, Belgium

Introduction

These guidelines on paediatric life support are based on three main principles: (1) the incidence of critical illness, particularly cardiopulmonary arrest, and injury in children is much lower than in adults; (2) the illnesses and pathophysiological responses of paediatric patients often differ from those seen in adults; (3) many paediatric emergencies are managed 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, 2005 and 2010.[1–5] The latter three were based on the paediatric work of the International Consensus on Science published by the International Liaison Committee on Resuscitation (ILCOR).[6–10] This process was repeated in 2014/2015, and the resulting Consensus on Science with Treatment Recommendations (CoSTR) was published simultaneously in Resuscitation, Circulation and Pediatrics using the GRADE process.[11–13] The PLS Writing Group of the ERC has developed the ERC PLS Guidelines based on the 2015 CoSTR and supporting scientific literature. The guidelines for resuscitation of Babies at Birth are covered in the ERC GL2015 Babies at Birth.[14] Information pertaining to children are also found in the ERC GL2015 First Aid,[15] the ERC GL2015 chapter on Education[16] and in the GL2015 chapter on the Ethics of Resuscitation and End-of-Life Decisions.[17]

Summary of changes since 2010 Guidelines

Guideline changes have been made in response to convincing new scientific evidence and, by using clinical, organisational and educational findings, they have been adapted to promote their use and ease for teaching.

The 2015 ILCOR process was informed by librarians who helped paediatric experts in performing in-depth systematic searches on 21 different key questions relating to paediatric resuscitation. Relevant adult literature was also considered and, in a few cases, extrapolated to the paediatric questions when they overlapped with other Task Forces, or when there were insufficient paediatric data. In rare circumstances, appropriate animal studies were incorporated into reviews of the literature. However, these data were considered only when higher levels of evidence were not available. The topic areas that the paediatric COSTR questions dealt with related to: pre-cardiac arrest care, basic life support care, advanced life support during cardiac arrest and post-resuscitation care.

As in previous ILCOR deliberations, there remains a paucity of good-quality evidence on paediatric resuscitation with many gaps in knowledge about paediatric resuscitation having been identified in this round of the CoSTR process.

* Corresponding author.
E-mail address: i.maconochie@imperial.ac.uk (I.K. Maconochie).
[1] The members of the Paediatric life support section Collaborators are listed in the Collaborators section.

http://dx.doi.org/10.1016/j.resuscitation.2015.07.028
0300-9572/© 2015 European Resuscitation Council. Published by Elsevier Ireland Ltd. All rights reserved.

These ERC GL2015 have included the recommendations from the ILCOR CoSTR 2015, updating the scientific base in addition to these recommendations and accompanied by points of clarification on matters about which there have been questions since 2010.[12,13]

This section of the ERC GL 2015 on Paediatric Life Support includes:

- Basic life support.
- Management of foreign bodies in the airway.
- Prevention of cardiac arrest.
- Advanced life support during cardiac arrest.
- Post resuscitation care.

New topics in the ERC GL2015 include those from CoSTR recommendations as well as the deliberations of the PLS Writing Group of the ERC.

These include:

In BLS

- The duration of delivering a breath is about 1 s, to coincide with adult practice.
- For chest compressions, the lower sternum should be depressed by at least one third the anterior–posterior diameter of the chest, or by 4 cm for the infant and 5 cm for the child.

In managing the seriously ill child

- If there are no signs of septic shock, then children with a febrile illness should receive fluid with caution and reassessment following its administration. In some forms of septic shock, restricting fluids with isotonic crystalloid may be better than the liberal use of fluids.
- For cardioversion of a supraventricular tachycardia (SVT), the initial dose has been revised to 1 J kg^{-1}.

In the paediatric cardiac arrest algorithm

- Many of the features are now common with adult practice.

In post resuscitation care

- Preventing fever in children who have return of spontaneous circulation (ROSC) from an out-of-hospital setting.
- Targeted temperature management of children post ROSC should comprise treatment with either normothermia or mild hypothermia.
- There is no single predictor for when to stop resuscitation.

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 an infant within 4 weeks of being born. An infant is a child under one year of age (but does not include newly borns) 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.[18]

The terms paediatrician and paediatric nurse are used in this text as a generic term to represent clinicians who routinely manage ill or injured children, and could apply to others trained in the delivery of paediatric care, such as emergency department clinicians, or Paediatric Intensive Care Unit (PICU) specialists/paediatric anaesthetists.

Healthcare professionals are those people who look after patients and should have a higher level of training than lay people. This term relates particularly to the delivery of basic life support.

Paediatric basic life support

From the ILCOR CoSTR statement on the sequence for manoeuvres in BLS, there was found to be equipoise between the CAB sequence (compression for circulation, airway and breathing) and the ABC sequence (airway, breathing and compression for circulation).[19–21] Given that the ABC sequence has become an established and well recognised method for the delivery of CPR to children in Europe, the ERC PLS Writing Group determined that the use of this sequence should continue, particularly as the previous guidelines have led to its instruction to many hundreds of thousands of healthcare providers and lay people. This position will continue to be reviewed on the basis of any new knowledge that may be forthcoming.

Sequence of actions in BLS

Bystander CPR is associated with a better neurological outcome in adults and children.[22–26]

Rescuers who have been taught adult BLS or the chest compression-only sequence and have no specific knowledge of paediatric resuscitation may use this, as the outcome is worse if they do nothing. However, it is better to provide rescue breaths as part of the resuscitation sequence when applied to children as the asphyxial nature of most paediatric cardiac arrests necessitates ventilation as part of effective CPR.[25,26]

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 one minute of CPR before they go for help (see adult BLS guidelines).

BLS for those with a duty to respond

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

Although the following sequence describes expired air ventilation, health professionals with a responsibility for treating children will usually have access to, and training in the use of bag mask ventilation systems (BMV), and these should be used to provide rescue breaths.

1. Ensure the safety of rescuer and child.
2. Check the child's responsiveness.

- Stimulate the child and ask loudly: Are you all right?

3A. If the child responds by answering, crying or moving:

- Leave the child in the position in which you find him (provided he is not in further danger).
- Check his condition and call for help.
- Reassess him regularly.

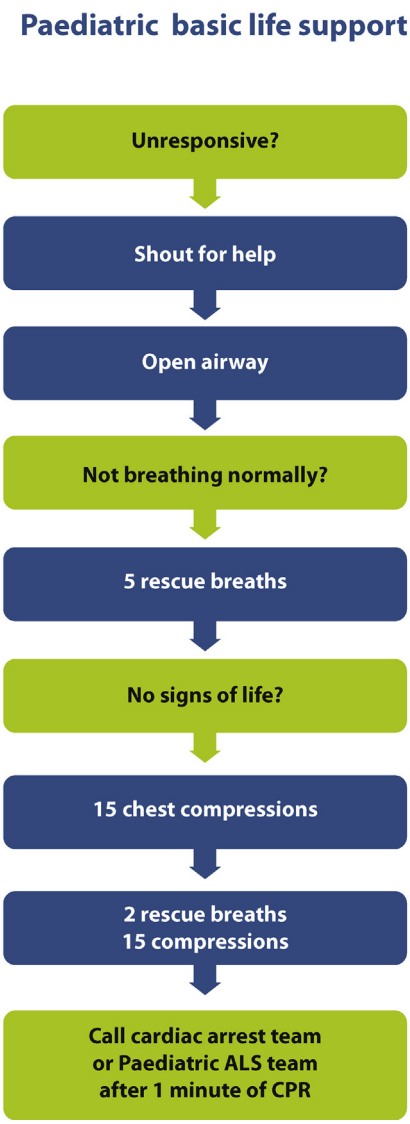

Fig. 6.1. Paediatric basic life support algorithm.

3B. If the child does not respond:

- Shout for help.
- Turn the child carefully 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. This is especially important in infants.
- 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:

Fig. 6.2. Mouth to mouth and nose ventilation–infant.

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

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). If there is a history of trauma, cervical spine injury should be considered.
- Send or go for help—call the emergency services.
- 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 an infant (Fig. 6.2)

- Ensure a neutral position of the head as an infant's head is usually flexed when supine, this may require some extension (a rolled towel/blanket under the upper part of the body may help to maintain the position) 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 for about 1 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.

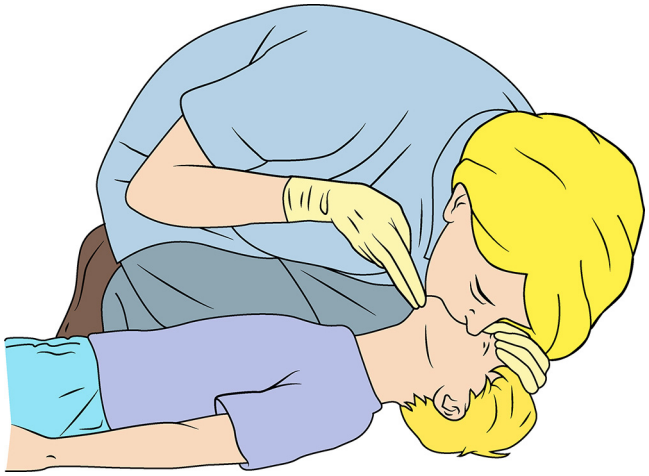

Fig. 6.3. Mouth to mouth ventilation–child.

Rescue breaths for a child over 1 year of age (Fig. 6.3):

- 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.
- Blow steadily into the mouth for about 1 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.
- 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.
- Reposition the head. 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 (gasps or infrequent, irregular breaths are abnormal). If you check the pulse, ensure that you take no more than 10 s. Pulse check is unreliable and therefore the complete picture of how the patient appears must guide whether BLS is required, i.e. if there are no signs of life, start BLS.[27,28]

7A. 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 his side (into the recovery position, with caution if there is a history of trauma) if he remains unconscious.
- Re-assess the child frequently.

7B. If there are no signs of life

- Start chest compressions.

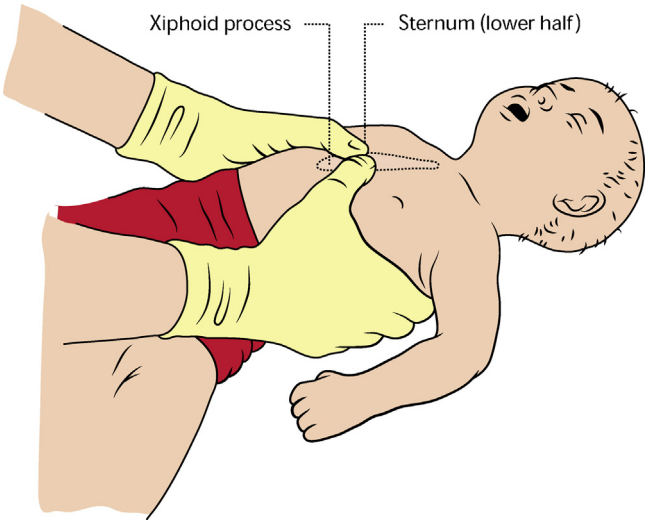

Fig. 6.4. Chest compression—infant.

- Combine rescue breathing and chest compressions at a ratio of 15 compressions to 2 ventilations.

Chest compressions

For all children, compress the lower half of the sternum. The compression should be sufficient to depress the sternum by at least one third of the anterior–posterior diameter of the chest. Release the pressure completely and repeat at a rate 100–120 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.

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 both hands with the fingers together to encircle the lower part of the infant's rib cage. The fingers should support the infant's back. For both methods, depress the lower sternum by at least one third the anterior–posterior dimension of the infant's chest or by 4 cm.[29]

Chest compression in children over 1 year of age (Figs. 6.5 and 6.6)

To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Place the heel of one hand on the sternum one finger's breadth above this. Lift the fingers to ensure that pressure is not applied onto the child's ribs. Position yourself above the victim's chest and, with your arm straight, compress the sternum to at least one third of the anterior–posterior dimension of the chest or by 5 cm.[29,30]

In larger children or for small rescuers, this is achieved most easily by using both hands, with the rescuer's fingers interlocked.

Do not interrupt resuscitation until

- The child shows signs of life (starts to wake up, to move, opens eyes and to breathe normally).
- More healthcare workers arrive and can either assist or take over.
- You become exhausted.

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

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.

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

- If only one rescuer is present, undertake resuscitation for about 1 min or 5 cycles of CPR before going for assistance. To minimise interruption in CPR, it may be possible to carry an infant or small child whilst summoning help.
- If you are on your own, witness a child suddenly collapse and you suspect a primary cardiac arrest, call for help first and then start CPR as the child will likely need urgent defibrillation. This is an uncommon situation.

AED and BLS

Continue with CPR until the AED arrives. Attach the AED and follow the instructions. For 1–8 year old, use attenuated pads if available, as explained in the chapter on Basic Life Support and Automated External Defibrillation.[31]

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.
- 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 (FBAO)

Back blows, chest thrusts and abdominal thrusts all increase intra-thoracic pressure and can expel foreign bodies from the airway. In half of the episodes more than one technique is needed to relieve the obstruction.[32] 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 (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 due to the horizontal position of the ribs, which leaves the upper abdominal viscera more exposed to traumatic injury. 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

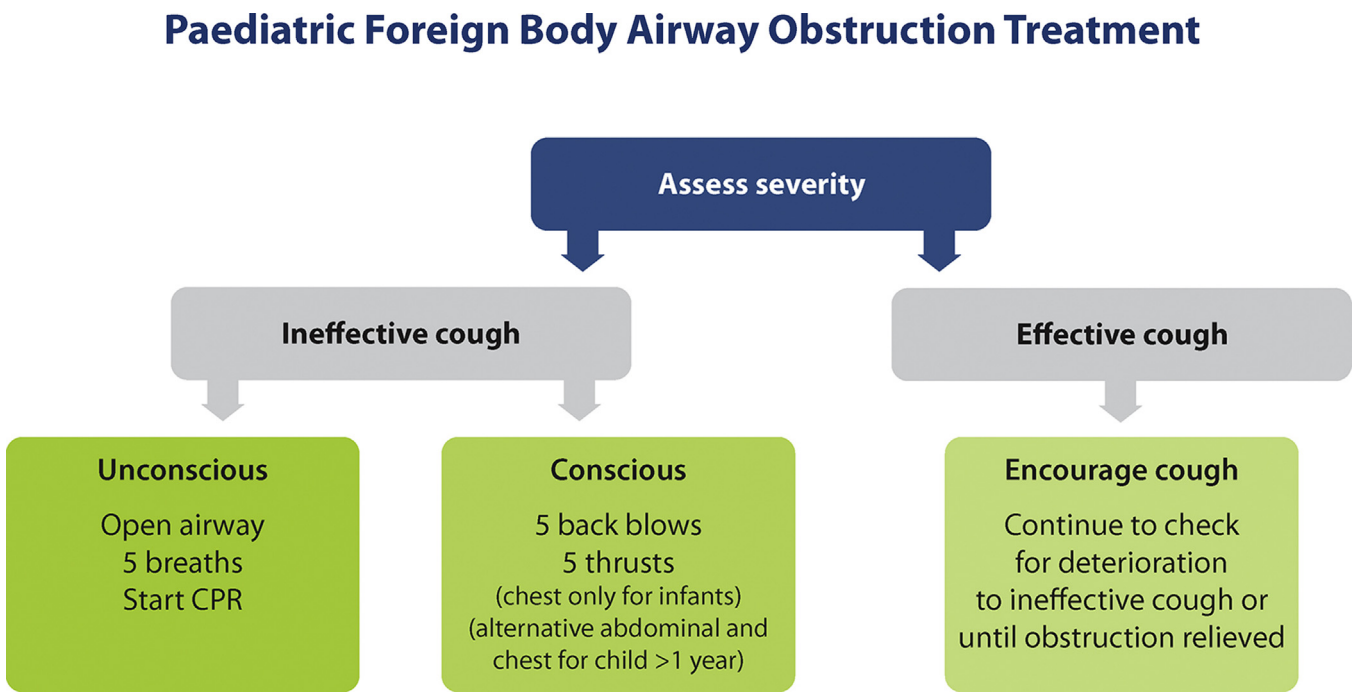

Fig. 6.7. Paediatric foreign body airway obstruction algorithm.

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)

Safety and summoning assistance. The principle of do no harm should be applied i.e. if the child is able to breath and cough, even with difficulty, encourage these spontaneous efforts. Do not intervene at this point as this may move the foreign body and worsen the problem, e.g. by causing full airway obstruction.

If the child is coughing effectively, no manoeuvre is necessary. Encourage the child to cough and continue monitoring the child's condition.

If the child's coughing is (or is becoming) ineffective, *shout for help* immediately and determine the child's conscious level.

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.

Back blows for 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 worsen 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 for 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.

Table 6.1
Signs 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	

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

If the object is expelled successfully, assess the child's clinical condition. It is possible that part of the object may remain in the respiratory tract and cause complications. If there is any doubt, seek medical assistance. Abdominal thrusts may cause internal injuries and all victims treated with abdominal thrusts should be examined by a doctor.[4]

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 could push the object deeper 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 or 5 cycles of 15 compressions to 2 ventilations before summoning the EMS (if this has not already been done by someone else).
- When the airway is opened for attempted delivery of rescue breath, check if the foreign body can be seen in the mouth.
- If an object is seen and can be reached, 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 the level of consciousness whilst awaiting the arrival of the EMS.

Paediatric advanced life support

Assessment of the seriously ill or injured child—The 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.[22,33–42] So-called asphyxial arrests or respiratory arrests are also more common in young adulthood (e.g. trauma, drowning and poisoning).[25,43–56]

Without treatment, the ill/injured child's initial physiological responses involve compensatory mechanisms. This means the affected system tries to adapt to the underlying physiological disturbance. So, for a circulatory problem, the initial physiological response will be in the circulatory system, and if there is a respiratory problem, then respiratory changes may take place. As things worsen, the other systems may become involved as part of the compensatory process. However, the child may continue to deteriorate, leading to decompensated respiratory or circulatory failure. Further physiological deterioration to cardiopulmonary failure may occur with the then inevitable progression to cardiopulmonary arrest. As the outcome from cardiopulmonary arrest in children is poor, identifying the preceding stages of circulatory or respiratory failure is a priority as effective early intervention in these stages may be lifesaving.

The order of assessment and intervention for any seriously ill child follows the ABCDE principles.

- A indicates airway.
- B indicates breathing.
- C indicates circulation.
- D indicates disability.
- E indicates exposure.

The topics of D (disability i.e. neurological status) and E (exposure with any subsequent conditions that may be found e.g. non-blanching rashes) are beyond the remit of these guidelines but are taught in paediatric life support courses.

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.

The role of the team leader is to co-ordinate care and to anticipate problems in the sequence. Each team member must be aware of the ABC principles.[57] Should deterioration occur, reassessment based on ABCDE is strongly recommended, starting at A again.

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 but the evidence is limited on this point as the literature tends not to separate out the team response alone from the other systems in place to

identify early deterioration.[58–69] This team should ideally include at least one physician experienced in acute paediatric care and a paediatric nurse (see the definitions in the terminology section above for the clinicians involved), and be called to evaluate a potentially critically ill child not already in a paediatric intensive care unit (PICU) or paediatric emergency department (ED).[70,71]

The ERC PLS writing group recognised that there is national and regional variation in countries as to the compositions of such a team but it is clear that processes to detect the early deterioration are key in reducing the morbidity and mortality of seriously ill and injured children. These processes with subsequent intervention by attending nurses and doctors have a higher priority for implementation than there solely being a rapid response or medical emergency team.[29,72–74]

Specific scores can be used (e.g. the paediatric early warning score, PEWS),[70,75–96] but there is no evidence that these improve the decision making process, or the clinical outcome.[29,71]

Diagnosing respiratory failure: Assessment of A and B

Assessment of a potentially critically ill child starts with the assessment of airway (A) and breathing (B).

Respiratory failure can be defined as the body's inability to maintain adequate blood levels of oxygen and carbon dioxide. Physiological compensatory mechanisms may be seen, such as an increase in respiratory rate and heart rate, and increased work of breathing, but these signs are not always present.

The signs of respiratory failure, as features of those physiological responses, may include:

- Respiratory rate outside the normal range for the child's age—either too fast or too slow.[97]
- Initially increased work of breathing, which may progress to inadequate/decreased work of breathing as the child tires or compensatory mechanisms fail.
- Additional noises such as stridor, wheeze, crackles, 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 it is often detectable prior to this by pulse oximetry.

There are uncommon conditions that can be associated with respiratory failure in which there is an inability of the body to raise these physiological compensatory signs. These are mostly due to abnormal neurological conditions (e.g. intoxication or coma) or muscular conditions (e.g. myopathy) where owing to muscle weakness, the child may not have the capacity to increase the work of breathing. A history or the presence of any features of these conditions is important to take into account when assessing the patient.

There may be associated signs in other organ systems. Even though the primary problem is respiratory, other organ systems will be involved to try to ameliorate the overall physiological disturbance.

These are detectable in step C of the assessment and include:

- Increasing tachycardia (compensatory mechanism to increase tissue oxygen delivery).
- Pallor.
- Bradycardia (an ominous indicator of the loss of compensatory mechanisms).
- Alteration in the level of consciousness (a sign that compensatory mechanisms are failing) owing to poor perfusion of the brain.

Diagnosing circulatory failure: Assessment of C

Circulatory failure is characterised by a mismatch between the metabolic demand by the tissues, and the delivery of oxygen and nutrients by the circulation.[97,98] Physiological compensatory mechanisms lead to changes in heart rate, in the systemic vascular resistance, and in tissue and organ perfusion. In some conditions, there may be vasodilation as part of the body's response to illness, e.g. toxic shock syndrome.

Signs of circulatory failure might include:

- Increased heart rate (bradycardia is an ominous sign of physiological decompensation).[97]
- Decreased systemic blood pressure.
- Decreased peripheral perfusion (prolonged capillary refill time, decreased skin temperature, pale or mottled skin)—signs of increased vascular resistance.
- Bounding pulses, vasodilation with widespread erythema may be seen in conditions with decreased vascular resistance.
- Weak or absent peripheral pulses.
- Decreased intravascular volume.
- Decreased urine output.

The transition from a compensatory state to decompensation may occur in an unpredictable way. Therefore, the child should be monitored, to detect and correct any deterioration in their physiological parameters promptly.

Other systems may be affected, for example:

- The respiratory rate may be increased initially, as an attempt to improve oxygen delivery, later becoming slower; this is usually accompanied by decompensated circulatory failure.
- The level of consciousness may decrease owing to poor cerebral perfusion.
- Poor cardiac functioning can lead to other signs, such as pulmonary oedema, enlarged liver, raised jugular veins.
- Poor tissue perfusion, metabolic acidosis and increased/increasing blood lactate levels may become progressively worse without correction.

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.[27,99–101] In the absence of signs of life, rescuers (lay and professional) should begin CPR unless they are certain that 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.[99,102–104] If personnel skilled in echocardiography are available, this investigation may help to detect cardiac activity and potentially treatable causes for the arrest.[100] However, echocardiography must not interfere with or delay 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 the early and effective intervention in children with respiratory and circulatory failure to prevent progression to full arrest.[105–110]

Airway and breathing

- Open the airway.
- Optimise ventilation.
- Ensure adequate oxygenation, start with 100% oxygen.
- Establish respiratory monitoring (first line – pulse oximetry/peripheral oxygen saturation – SpO_2).
- Achieving adequate ventilation and oxygenation—this may require the use of airway adjuncts ± bag-mask ventilation (BMV), the use of an LMA or other supraglottic airway, securing a definitive airway by tracheal intubation and positive pressure ventilation.
- For intubated children, it is standard practice that their end tidal carbon dioxide levels are monitored. End tidal carbon dioxide monitoring can be used in non-intubated critically ill patients.
- Very rarely, a surgical airway may be required.

Circulation

- Establish cardiac monitoring (first line—pulse oximetry/SpO_2, electrocardiography (ECG) and non-invasive blood pressure (NIBP)).
- Secure intravascular access. This may be achieved by peripheral intravenous (IV) or by intraosseous (IO) route. 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) to treat circulatory failure due to hypovolaemia, e.g. from fluid loss or maldistribution, as seen in septic shock and anaphylaxis.
- Consider carefully the use of fluid bolus in primary cardiac functioning disorders, e.g. myocarditis, cardiomyopathy.
- Do not give a fluid bolus in severe febrile illness when circulatory failure is absent.[29,111–113]
- Isotonic crystalloids are recommended as initial resuscitation fluid in infants and children with any type of shock, including septic shock.[29,114–119]
- Assess and re-assess the child repeatedly, beginning each time with the airway before proceeding to breathing and then the circulation. Blood gas and lactate measurement may be helpful.
- During treatment, capnography, invasive monitoring of arterial blood pressure, blood gas analysis, cardiac output monitoring, echocardiography and central venous oxygen saturation ($ScvO_2$) may be useful to guide the treatment of respiratory and/or circulatory failure.[120,121] Whilst the evidence for the use of these techniques is of low quality, the general principles of monitoring and assessing the impact of any interventions and those responses are key in managing seriously ill children.

Airway

Open the airway by using basic life support techniques. Oropharyngeal and nasopharyngeal airways adjuncts can help maintain the airway. An oropharyngeal airway may be helpful in the unconscious child, in whom there is no gag reflex. Use the appropriate size (as measured from the incisors to the angle of the mandible) to avoid pushing the tongue backward during insertion, as this may further obstruct the airway. The soft palate may be damaged by forceful insertion of the oropharyngeal airway—avoid this by inserting the oropharyngeal airway with care. Do not use force if the child resists.

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 and must be re-assessed after insertion. These simple airway adjuncts do not protect the airway from aspiration of secretions, blood or stomach contents.

Supraglottic airways devices (SADs) (including LMA)

Although BVM ventilation remains the recommended first line method for achieving airway control and ventilation in children, the SADs represent a range of acceptable airway devices that may assist providers trained in their use.[122,123] SADs may be particularly helpful in airway obstruction caused by supraglottic airway abnormalities, or if BVM ventilation is difficult or not possible.[124,125] SADs do not totally protect the airway from aspiration of secretions, blood or stomach contents, and therefore close observation is required.[126,127]

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 for tracheal intubation is preferable during resuscitation. Oral intubation is quicker and simpler, and is associated with fewer complications than nasal intubation. In the conscious child, the judicious use of anaesthetics, sedatives and neuromuscular blocking drugs is essential to avoid multiple intubation attempts or intubation failure.[128–137] Only skilled and experienced practitioners should perform intubation.

The anatomy of a child's airway differs significantly from that of an adult, and tube sizes and insertion depth vary considerably with age; hence, intubation of a child requires special training and ongoing experience. Clinical examination and capnography should be used to ensure that the tracheal tube remains secured and vital signs should be monitored.[136] It is also essential to anticipate potential cardiorespiratory problems and 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 pre-hospital tracheal intubation of children. Pre-hospital tracheal intubation of children may be considered if the airway and/or breathing is seriously compromised or threatened. The mode and duration of transport (e.g., air transport) may play a role in the decision to secure the airway before transport.

Anyone intending to intubate must be adequately skilled in advanced paediatric airway management including pre-oxygenation and the use of drugs to facilitate tracheal intubation.[138]

Intubation during cardiopulmonary arrest. The child who is in cardiopulmonary arrest does not require sedation or analgesia to be intubated. As previously stated, intubation of the seriously ill/injured child should be undertaken by an experienced and trained practitioner.

Tracheal tube sizes. Table 6.2 shows which tracheal tube internal diameters (ID) should be used for different ages.[139–144] 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 indicated by resuscitation tapes.[145,146]

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. in facial burns,[147] when lung compliance is poor, airway resistance is high or if there is a large air leak from the glottis.[139,148,149] The use of

Table 6.2
General recommendation for cuffed and uncuffed tracheal tube sizes (internal diameter in mm).

	Uncuffed	Cuffed
Premature neonates	Gestational age in weeks/10	Not used
Full term neonates	3.5	Not usually used
Infants	3.5–4.0	3.0–3.5
Child 1–2 y	4.0–4.5	3.5–4.0
Child >2 y	Age/4 + 4	Age/4 + 3.5

cuffed tubes also makes it more likely that the correct tube size will be chosen on the first attempt.[139,140,147] 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.[148–150] 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 H_2O.[150]

Confirmation of correct tracheal tube placement. Displaced, misplaced or obstructed tubes occur frequently in the intubated child and are associated with an increased risk of death.[151,152] No single technique is 100% reliable for distinguishing oesophageal from tracheal intubation.[153–155]

Assessment of the correct tracheal tube position is made by:

- Laryngoscopic observation of the tube passing through the vocal cords.
- Detection of end-tidal CO_2 (preferably by capno*graphy* or by capno*metry* or colorimetry) 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 SpO_2 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 CO_2 is not detected despite adequate chest compressions, or if there is any doubt as to the tube position, confirm the placement of the tracheal tube by direct laryngoscopy. After correct placement and confirmation, secure the tracheal tube and reassess 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.[156] 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. It is also helpful in the case of a child who requires intubation and thereafter fails to improve following being intubated. When the cause is found, steps should be taken to remedy the situation.

Displacement of the tracheal tube (in the oesophagus, pharynx or endobronchially). **O**bstruction of the tracheal tube, or of the heat and moisture exchanger (HME) or the respirator pipes. **P**neumothorax and other **p**ulmonary disorders (bronchospasm, oedema, pulmonary hypertension, etc.). **E**quipment failure (source of gas, bag-mask, ventilator, etc.). **S**tomach (gastric distension may alter diaphragm mechanics).

Breathing

Oxygenation

Give oxygen at the highest concentration (i.e. 100%) during initial resuscitation.

Studies in newly borns suggest advantages of using room air during resuscitation.[14] In infants and older children, however, there is no evidence of benefit for using air instead of oxygen so use 100% oxygen for the initial resuscitation. Once the child is stabilised and/or there is ROSC, titrate the fraction of inspired oxygen (FiO_2) to achieve normoxaemia, or at least (if arterial blood gas is not available), maintain SpO_2 in the range of 94–98%.[157,158] In smoke inhalation (carbon monoxide poisoning) and severe anaemia, however, high FiO_2 should be maintained until the underlying disorder is ameliorated as in these circumstances, dissolved oxygen in the blood plays an important role in oxygen transport to tissues.

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 there is some evidence of poorer survival rates in animals although other evidence suggests that survival rates are not worse.[159–166] A simple guide to deliver an appropriate tidal volume is to achieve normal chest wall rise. Use a ratio of 15 chest compressions to 2 ventilations and a compression rate of 100–120 min^{-1}.

Inadvertent hyperventilation during CPR occurs frequently, especially when the trachea is intubated and ventilations are given continuously along with asynchronous chest compressions.

Once the airway is protected by tracheal intubation, continue positive pressure ventilation at 10 breaths min^{-1} without interrupting the chest compressions. Take care to ensure that lung inflation is adequate during chest compressions. Once ROSC has been achieved, provide normal ventilation (rate/volume) based on the child's age, and by monitoring end-tidal CO_2 and blood gas values, to achieve a normal arterial carbon dioxide tension ($PaCO_2$) and arterial oxygen levels. Both hypocarbia and hypercarbia are associated with poor outcomes following cardiac arrest.[167] This means that the child with ROSC should usually be ventilated at 12–24 breaths min^{-1}, according to their age normal values.

In a few children the normal values for carbon dioxide and oxygenation levels may be different to that of the rest of the paediatric population; take care to restore the carbon dioxide and oxygen values to that child's normal levels, e.g. in children with chronic lung disease or congenital heart conditions.

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 pre-hospital setting or in an emergency department.[168,169] Assess the effectiveness of BMV by observing adequate chest rise, monitoring heart rate and auscultating for breath sounds, and measuring SpO_2. Any healthcare provider with a responsibility for treating children must be able to deliver BMV effectively.

Monitoring of breathing and ventilation

1.1.1.1. End-tidal CO_2. Monitoring end-tidal CO_2 ($ETCO_2$) with a colorimetric detector or capnometer confirms tracheal tube placement in the child weighing more than 2 kg, and may be used in pre- and in-hospital settings, as well as during any transportation of a child.[170–173] 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 CO_2 during cardiopulmonary arrest does not guarantee tube misplacement since a low or absent $ETCO_2$ may reflect low or absent pulmonary blood flow.[174–177]

In this circumstance, the tube placement should be checked by direct laryngoscopy and the chest auscultated for the sounds of air entry into the lungs.

Capnography may also provide information on the effectiveness of chest compressions and can give an early indication of ROSC.[178,179] Care must be taken when interpreting $ETCO_2$ values especially after the administration of adrenaline or other vasoconstrictor drugs when there may be a transient decrease in values[180–184] or after the use of sodium bicarbonate causing a transient increase.[185] Although an $ETCO_2$ higher than 2 kPa (15 mmHg) may be an indicator of adequate resuscitation, current evidence does not support the use of a threshold $ETCO_2$ value as an indicator for the quality of CPR or for the discontinuation of resuscitation.[29]

Peripheral pulse oximetry, SpO_2. Clinical evaluation to determine the degree of oxygenation in a child is unreliable; therefore, monitor the child's peripheral oxygen saturation continuously by pulse oximetry. Pulse oximetry can be unreliable under certain conditions, e.g. if the child is in circulatory failure, in cardiopulmonary arrest or has poor peripheral perfusion. In some circumstances the SpO_2 reading may not give a true assessment of the total amount of oxygen in the blood as it only measures the relative amount of oxygen bound to haemoglobin. Hence, in anaemia, methaemoglobinaemia or in carbon monoxide poisoning, SpO_2 values must be interpreted with caution.

Although pulse oximetry is relatively simple, it is a poor guide to tracheal tube displacement and must not be relied upon. Capnography detects tracheal tube dislodgement more rapidly than pulse oximetry and is the monitoring system of choice.[186]

Circulation

Vascular access

Vascular access is essential to enable drugs and fluids to be given, and blood samples obtained. Venous access can be difficult to establish during resuscitation of an infant or child. In critically ill children, whenever venous access is not readily attainable, intra-osseous access should be considered early, especially if the child is in cardiac arrest or decompensated circulatory failure.[187–193] In any case, in critically ill children, if attempts at establishing intravenous (IV) access are unsuccessful after one minute, insert an intra-osseous (IO) needle instead.[190,194]

IO access. IO access is a rapid, safe, and effective route to give drugs, fluids and blood products.[195–205] The onset of action and time to achieve adequate plasma drug concentrations are similar to that achieved via the central venous route.[206–209] Bone marrow samples can be used to cross match for blood type or group for chemical analysis[210–212] and for blood gas measurement (the values may be comparable to central venous blood gases if no drug has been injected in the cavity).[206,209,211,213–215] However, these bone marrow samples can damage auto-analysers and should be used preferably in a cartridge analyser.[216] After taking blood samples, flush each given 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 or a pressure bag.[217] Maintain IO access until definitive IV access has been established.[107,192,203,218,219]

Intravenous access and other routes. Peripheral IV access provides plasma concentrations of drugs and clinical responses equivalent to central or IO access.[220–222] The intramuscular route is preferred for the administration of adrenaline in anaphylaxis.[223,224] Other routes are useful for different circumstances e.g. intranasal, buccal etc. but are beyond the remit of these guidelines.[225] Central venous lines provide more secure long-term access but, compared with IO or peripheral IV access, offer no advantages during resuscitation.[190,191,221,226,227] The tracheal route for the administration of drugs is no longer recommended.[228,229]

Fluids and drugs

When a child shows signs of circulatory failure caused by hypovolaemia, controlled volume administration is indicated.[230] For children with febrile illness and not showing signs of circulatory failure, adopt a cautious approach to fluid therapy with frequent reassessment of the child.[29,111–113] Isotonic crystalloids are recommended as the initial resuscitation fluid for infants and children with any type of circulatory failure.[231,232] If there are signs that the 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 each bolus, re-assess the child's clinical state, using the ABCDE system of assessment, to decide whether a further bolus or other treatment is required (and how much and how fast). In some children, early inotropic or vasopressor support may be needed.[108,233] In addition, owing to decreased/decreasing consciousness or progressive respiratory failure, some patients will need intubation and mechanical ventilation, so be prepared in case this occurs.

There is growing evidence to prefer the use of balanced crystalloids as these induce less hyperchloraemic acidosis.[234–237]

In life-threatening hypovolaemic shock, as may be seen in rapid blood loss in trauma, limiting the use of crystalloids in favour of a regime of massive blood transfusion may be required. There are varying regimes of combining plasma, platelets and other blood products in delivering massive blood transfusion,[238,239] so the regime used should be according to local protocols. Similarly, in other types of shock, when multiple boluses of crystalloids are given, timely blood products should be considered to treat dilutional effects. Avoid glucose containing solutions unless there is hypoglycaemia.[240–244] Monitor blood glucose levels and avoid hypoglycaemia; infants and small children are particularly prone to hypoglycaemia.[245]

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).[246] 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. It causes asystole, which is usually short lived, hence adenosine must be given under ECG monitoring. Give adenosine rapidly, followed by a flush of 5 ml of normal saline.[247] 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 plays a central role 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

ventricular fibrillation (VF), so increasing the likelihood of successful defibrillation.

For cardiopulmonary resuscitation, the recommended IV/IO dose of adrenaline in children for the first and for subsequent doses is 10 micrograms kg^{-1}. The maximum single dose is 1 mg. If needed, give further doses of adrenaline every 3–5 min, i.e. every 2 cycles.

The use of single higher doses of adrenaline (above 10 micrograms kg^{-1}) is not recommended because it does not improve survival or neurological outcome after cardiopulmonary arrest.[248–252]

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, so compromising extremity, mesenteric, and renal blood flow. High-dose adrenaline can cause severe hypertension and tachyarrhythmias.[253] 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.[254]

Amiodarone for shock-resistant paediatric VF/pulseless VT

Amiodarone can be used to treat paediatric shock-resistant VF/pulseless VT (pVT). 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. Amiodarone can be given as part of the cardiac arrest algorithm in managing refractory VF/pVT. It is given after the third shock as a 5 mg kg^{-1} bolus (and can be repeated following the fifth shock). When treating other cardiac rhythm disturbances, amiodarone must be injected slowly (over 10–20 min) with systemic blood pressure and ECG monitoring to avoid causing hypotension.[255] This side effect is less common with the aqueous solution.[256] Other rare but significant adverse effects are bradycardia and polymorphic VT.[257]

Lidocaine has been suggested by COSTR as an alternative but most practitioners will have followed the guidance that has stated amiodarone is the drug of choice. The European Resuscitation Council advises that the clinician should use the drug with which they are familiar and for which they have knowledge of expected and unexpected listed side effects.

Lidocaine is a commonly used local anaesthetic as well as being a Class-1b antiarrhythmic drug. Lidocaine is an alternative to amiodarone in defibrillation-resistant VF/pulseless VT in children.[29,258–260] It can be used with a loading dose of 1 mg kg^{-1} (maximum dose 100 mg/dose) followed by continuous infusion at 20–50 micrograms kg^{-1} min^{-1}. Toxicity can occur if there is underlying renal or hepatic disease.

Atropine

Atropine accelerates sinus and atrial pacemakers by blocking the parasympathetic response. The commonly used dose is 20 micrograms kg^{-1}. It may also increase AV conduction. Small doses (<100 micrograms) may cause paradoxical bradycardia.[261] In bradycardia with poor perfusion unresponsive to ventilation and oxygenation, the first line drug is adrenaline, not atropine. Atropine is recommended only for bradycardia caused by increased vagal tone or cholinergic drug toxicity.[262–264] Its role in emergency intubation for the child is still unclear as there are no reported long-term benefits following ROSC.[29,265,266]

Calcium

Calcium is essential for myocardial function,[267] but the routine use of calcium does not improve the outcome from cardiopulmonary arrest.[268–272] Calcium is indicated in the presence of hypocalcaemia, calcium channel blocker overdose, hypermagnesaemia and hyperkalaemia.[46,272–274] Calcium supplementation may be required when massive blood transfusion is given, e.g. as in response to blood loss in trauma, or when any other large fluid volumes are given; the calcium levels must be monitored and replacement given to maintain normal blood levels.[238]

Glucose

Data from neonates, children and adults indicate that both hyper- and hypo- glycaemia are associated with poor outcome after cardiopulmonary arrest,[275,276] but it is uncertain if this is causative or merely an association.[241,276–278] 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.[245] Avoid hyper- and hypoglycaemia following ROSC.[279] In adults strict glucose control does not increase survival when compared with moderate glucose control[280,281] and it increases the risk of hypoglycaemia in neonates, children and adults.[282,283]

Magnesium

There is no evidence for giving magnesium routinely during cardiopulmonary arrest.[284,285] Magnesium treatment is indicated in the child with documented hypomagnesaemia or with torsade de pointes VT, (50 mg kg^{-1}) regardless of the cause.[286]

Sodium bicarbonate

There is no clear evidence for giving sodium bicarbonate routinely during cardiopulmonary arrest.[287–290] 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 and cause hypokalaemia, hypernatraemia, hyperosmolality and cerebral acidosis.

Procainamide

Procainamide slows intra-atrial conduction and prolongs the QRS and QT intervals. It can be used in supraventricular tachycardia (SVT)[291,292] or VT[293] resistant to other medications in the haemodynamically stable child. However, paediatric data are sparse and procainamide should be used cautiously.[294–297] Procainamide is a potent vasodilator and can cause hypotension: infuse it slowly with careful monitoring.[255,294]

Vasopressin—Terlipressin

Vasopressin is an endogenous hormone that acts at specific receptors, mediating systemic vasoconstriction (via V_1 receptor) and the reabsorption of water in the renal tubule (by the V_2 receptor).[298] 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.[299–306] These drugs may be considered in cardiac arrest refractory to adrenaline.

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.[307–309] Two paediatric case series suggested that terlipressin may be effective in refractory cardiac arrest.[303,310]

Defibrillators

Defibrillators are either automated 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 pre-set 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.[311,312]

Recommended sizes are:

- 4.5 cm diameter for infants and children weighing <10 kg.
- 8–12 cm diameter for children weighing >10 kg (older than one 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 and are recommended for maximal delivery of the energy. Self-adhesive pads facilitate continuous good quality CPR. Do not use saline-soaked gauze/pads, 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.[313,314] In practice, this means that the paddles should be applied firmly.

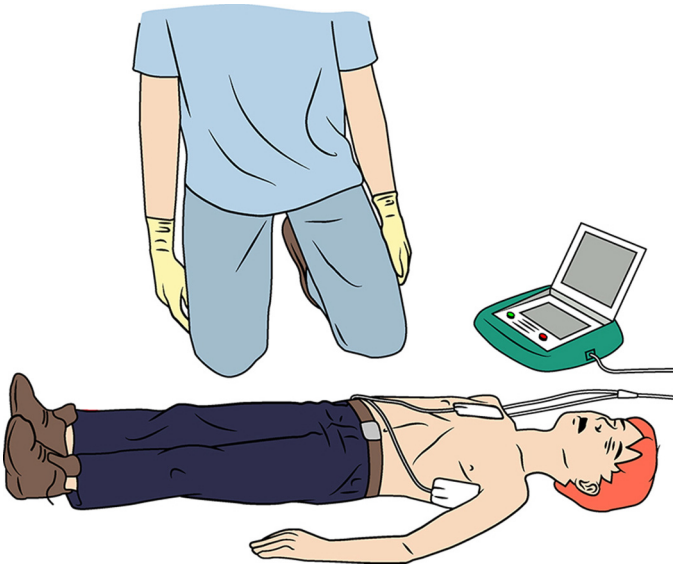

Fig. 6.8. Paddle positions for defibrillation—child.

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 shocks.[315] Animal models show better results with paediatric doses of 3–4 J kg^{-1} than with lower doses,[316] or adult doses,[317] but there are no data to support a different strategy to the current one of an initial dose of 2–4 J kg^{-1}. In Europe, for the sake of simplicity, we continue to recommend 4 J kg^{-1} for initial and subsequent defibrillation. Doses higher than 4 J kg^{-1} (as much as 9 J kg^{-1}) have defibrillated children effectively with negligible side effects.[318,319] 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.[320–322] The AED should be equipped with a dose attenuator that decreases the delivered energy to a lower dose more suitable for children aged 1–8 years (50–75 J).[317,323] If such an AED in not available, use a standard adult AED and the pre-set adult energy levels. For children older than 8 years, use a standard AED with standard paddles. Experience with the use of AEDs (preferably with dose attenuator) in children younger than 1 year is limited; its use is acceptable if no other option is available.

Advanced management of cardiopulmonary arrest (Fig. 6.9)

A, B and C: Commence and continue with basic life support.

A and B	Oxygenate and ventilate with BMV • Provide positive pressure ventilation with a high concentration of inspired oxygen (100%) • Establish cardiac monitoring • Avoid rescuer fatigue by frequently changing the rescuer performing chest compressions
C	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 micrograms kg^{-1}) and repeat every 3–5 min (every 2nd cycle) (Fig. 6.10).
- Identify and treat any reversible causes (4Hs & 4Ts).

Reversible causes of cardiac arrest

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

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

Shockable—VF/pulseless VT

Attempt defibrillation immediately (4 J kg^{-1}) (Fig. 6.11):

- Charge the defibrillator while another rescuer continues chest compressions
- Once the defibrillator is charged, pause the chest compressions and ensure that all rescuers are clear of the patient. Minimise the delay between stopping chest compressions and delivery of the

Paediatric Advanced Life Support

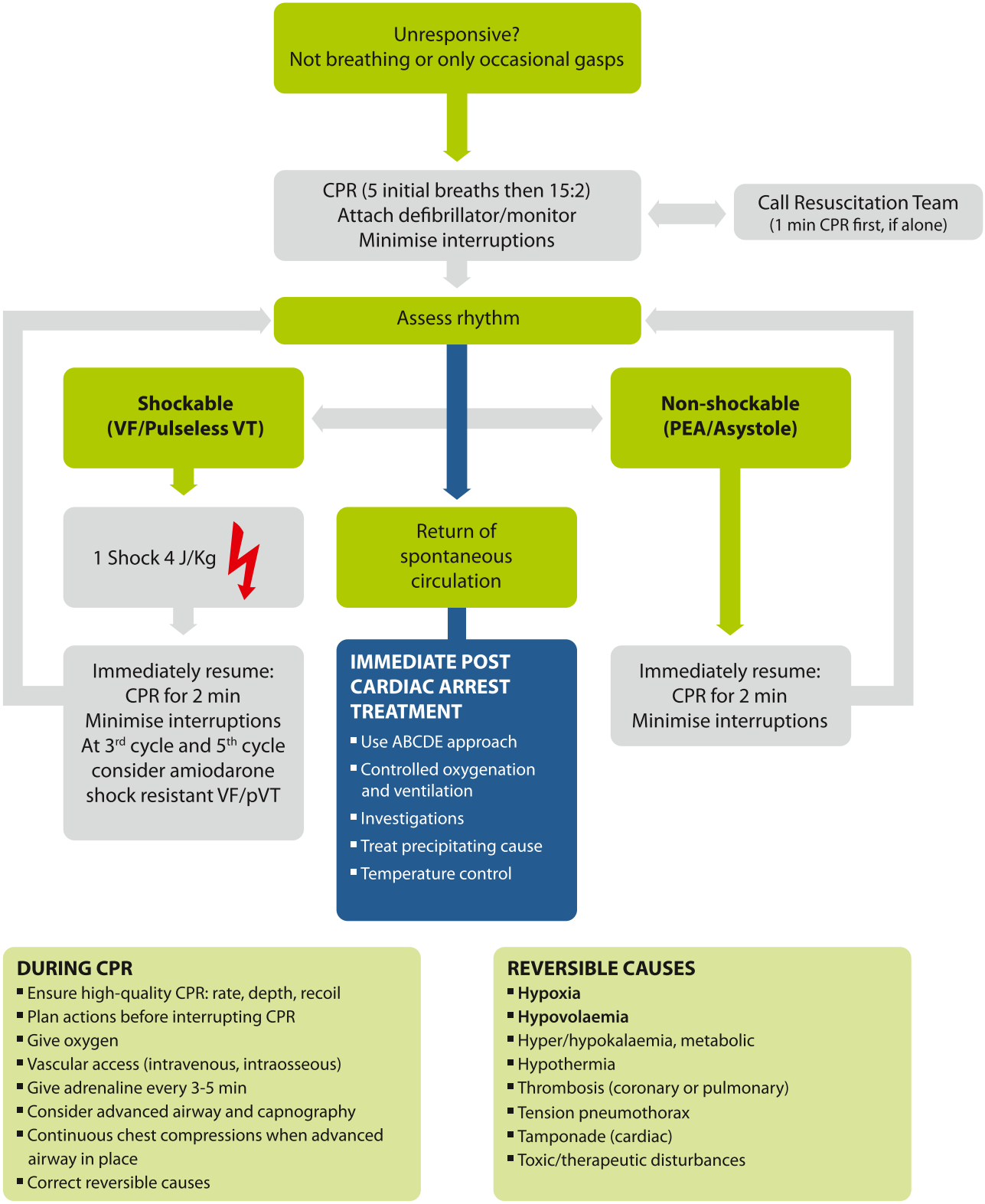

Fig. 6.9. Paediatric advanced life support algorithm.

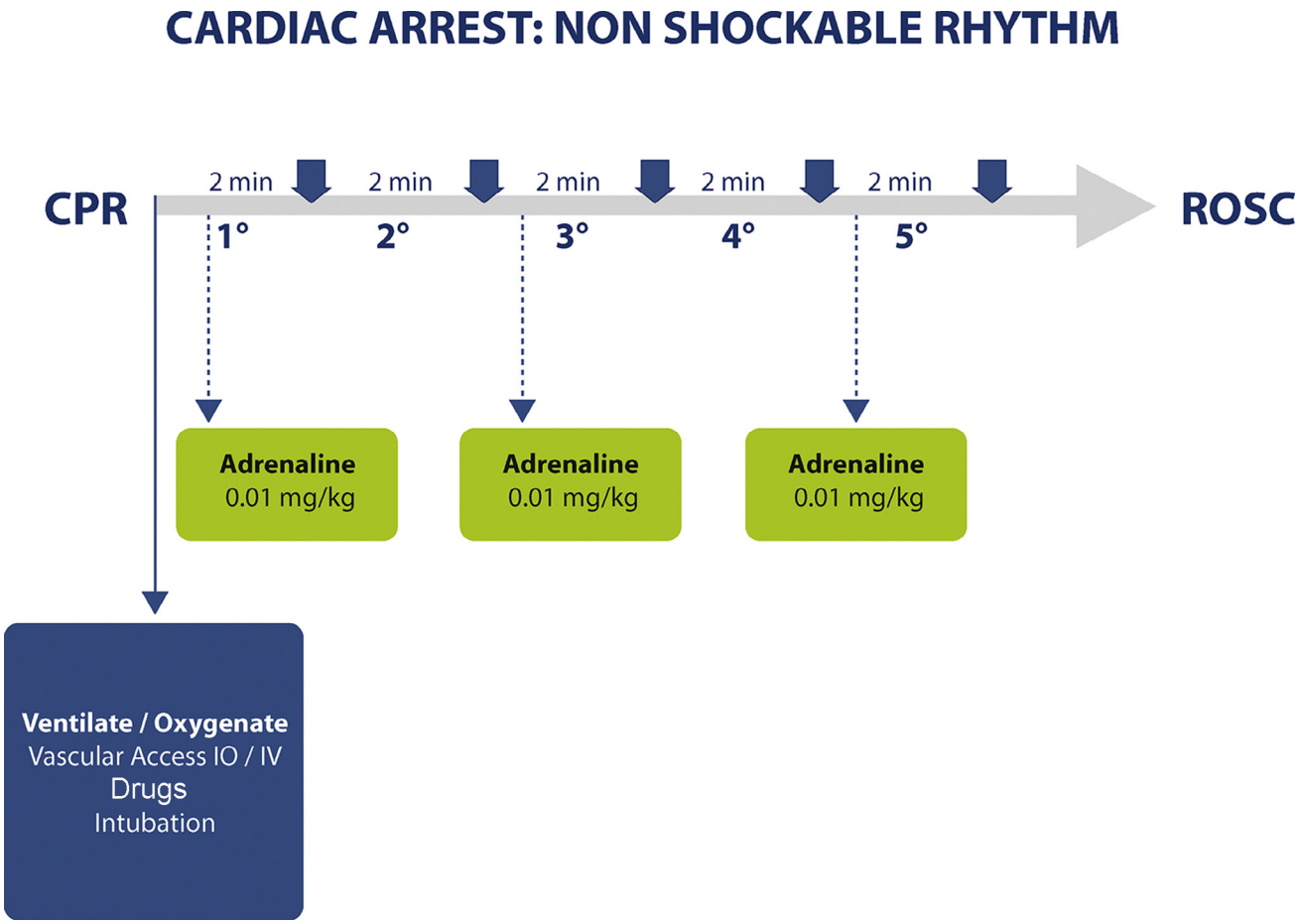

Fig. 6.10. Paediatric algorithm for non-shockable rhythm.

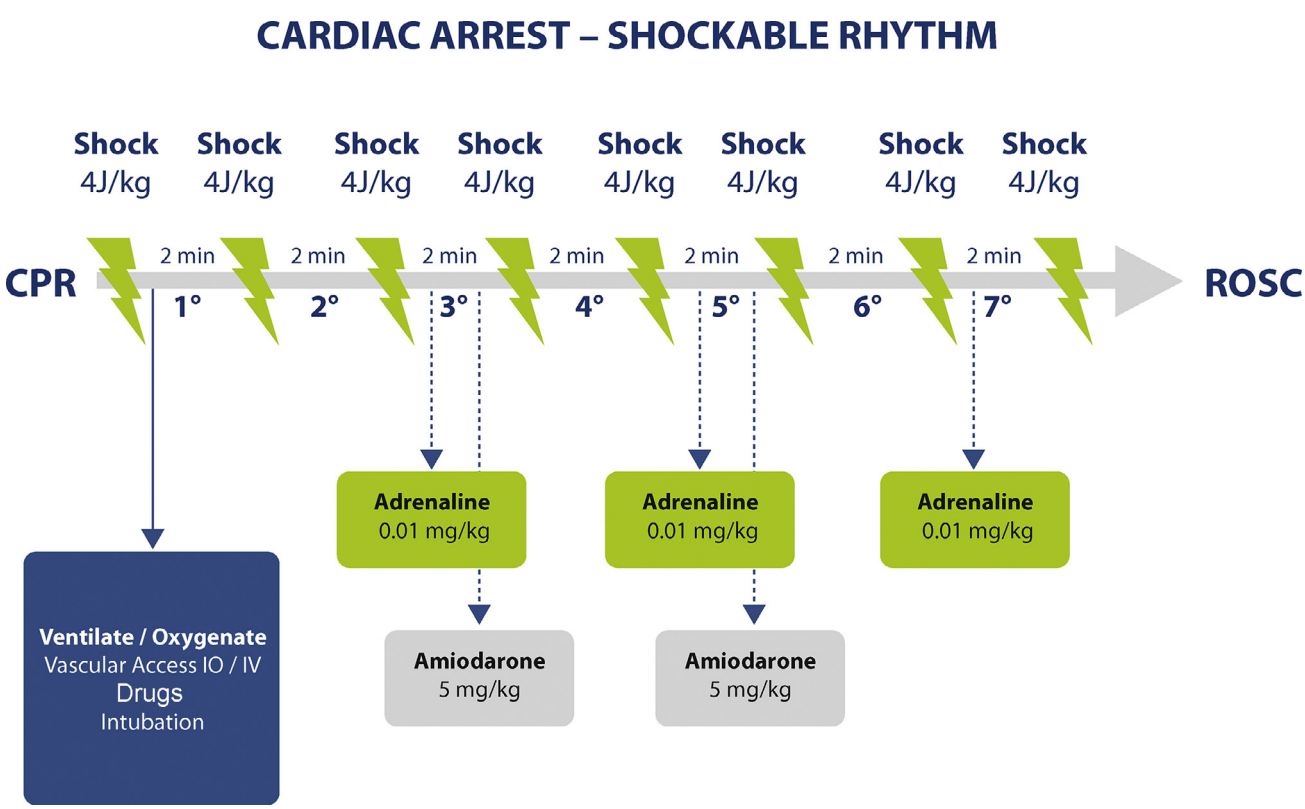

Fig. 6.11. Paediatric algorithm for shockable rhythm.

shock—even 5–10 s delay will reduce the chances of the shock being successful.
- Give one shock.
- Resume CPR as soon as possible without reassessing the rhythm.
- After 2 min, check briefly the cardiac rhythm on the monitor.
- Give second shock (4 J kg^{-1}) if still in VF/pVT.
- Give CPR for 2 min as soon as possible without reassessing the rhythm.
- Pause briefly to assess the rhythm; if still in VF/pVT give a third shock at 4 J kg^{-1}
- Give adrenaline 10 micrograms 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^{-1}[324] if still in VF/pVT after the fifth shock.

Lidocaine may be used as an alternative to amiodarone.

If the child remains in VF/pVT, 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 (4Hs & 4Ts) remembering that hypoxia and hypovolaemia have the highest prevalence in critically ill or injured children, and that electrolyte disturbances and toxicity are common causes of arrhythmia.

If defibrillation has been successful but VF/pVT recurs, resume CPR, give amiodarone or lidocaine and defibrillate again at the energy level that was effective previously.

Cardiac monitoring

Position the cardiac monitor leads or self-adhesive pads soon as possible to enable differentiation between a shockable and a non-shockable cardiac rhythm. Defibrillation paddles can be used to determine a rhythm if monitor leads or self-adhesive pads are not immediately available. Invasive monitoring of systemic blood pressure may help to improve the effectiveness of chest compression if present but it must never delay the provision or hamper the quality of basic or advanced resuscitation.

Non-shockable rhythms are pulseless electrical activity (PEA), bradycardia (<60 min^{-1} with no signs of circulation) and asystole. PEA and bradycardia often have wide QRS complexes.

Shockable rhythms are pVT and VF. These rhythms are more likely after sudden collapse in children with heart disease or in adolescents.

Non-shockable rhythms

Most cardiopulmonary arrests in children and adolescents are of respiratory origin.[325–327] 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. The most common ECG patterns in infants, children and adolescents with cardiopulmonary arrest are asystole and PEA. PEA is characterised by electrical activity on the ECG, 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 4Hs and 4Ts) that led to a sudden impairment of cardiac output.

Shockable rhythms

Primary VF occurs in 3.8% to 19% of cardiopulmonary arrests in children, the incidence of VF/pVT increases as the age increases.[48–56,328] The primary determinant of survival from VF/pVT 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%. 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.[329]

Drugs in shockable rhythms

Adrenaline (adrenaline). Adrenaline is given every 3–5 min, every 2 cycles by the IV or IO route.

Amiodarone or lidocaine. Either drug can be given in defibrillation-resistant VF/pVT.

Extracorporeal life support. Extracorporeal life support should be considered for children with cardiac arrest refractory to conventional CPR with a potentially reversible cause, if the arrest occurs where expertise, resources and sustainable systems are available 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 child with decompensated circulatory failure has a heart rate <60 beats min^{-1}, and they do not respond rapidly to ventilation with oxygen, start chest compressions and give adrenaline.

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.[330]

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.[331]

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 has signs of decompensated shock with depressed conscious level, 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 $1\,J\,kg^{-1}$ 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.[324,332–339] 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.[340,341] 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.[342] 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 signs of life. 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,[343] although cardiovascular side effects are common.[324,332,334,339,344]

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.

Special circumstances

Life support for blunt or penetrating trauma

Cardiac arrest from major (blunt or penetrating) trauma is associated with a very high mortality.[345–352] The 4Ts and 4Hs should be considered as potentially reversible causes. 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.[353–359]

Extracorporeal membrane oxygenation (ECMO)

For infants and children with a cardiac diagnosis and an in-hospital arrest ECMO should be considered as a useful rescue strategy if sufficient expertise and resources are available. There is insufficient evidence to suggest for or against the use of ECMO in non-cardiac arrest or for children with myocarditis or cardiomyopathy who are not in arrest.[29]

Pulmonary hypertension

There is an increased risk of cardiac arrest in children with pulmonary hypertension.[360,361] Follow routine resuscitation protocols in these patients with emphasis on high FiO_2 and alkalosis/hyperventilation because this may be as effective as inhaled nitric oxide in reducing pulmonary vascular resistance.[362] Resuscitation is most likely to be successful in patients with a reversible cause who are treated with intravenous epoprostenol or inhaled nitric oxide.[363] 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.[364–368] Right ventricular support devices may improve survival.[369–373]

Post-resuscitation care

After prolonged, complete, whole-body hypoxia-ischaemia ROSC has been described as an unnatural pathophysiological state, created by successful CPR.[374] Post-cardiac arrest care 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.[374–378] Parenteral fluids and vasoactive drugs (adrenaline, dobutamine, dopamine and noradrenaline) may improve the child's post-arrest haemodynamic status and should be titrated to maintain a systolic blood pressure of at least >5th centile for age.[29,379–390]

Although the measurement of blood pressure has limitations in determining perfusion of vital organs, it is a practical and valued measurement of haemodynamic status. Alternative perfusion endpoints (such as serum lactate levels, measures of cardiac output, mean blood pressure) can be targeted but the evidence for each of them individually is still equivocal. Ideally, they should be considered as a part of a global 'gestalt' observation. The optimal strategy to avoid hypotension i.e. the relative use of parenteral fluids versus inotropes and/or vasopressors in children post ROSC following cardiac arrest currently remains unclear. The need to use agents to maintain a normal blood pressure is a poor prognostic factor.[390]

Finally, subgroups of children might respond differently to components of the above interventions, such as cardiac patients or trauma patients who may be particularly sensitive to preload status and changes in afterload. Any interventions must be monitored and adapted according to the child's physiological responses. Reassessment of the child is key in improving their outcome.

Oxygenation and ventilation goals

Aim for a normal PaO_2 range (normoxaemia) post-ROSC once a patient is stabilised.[167,391–393] Balance the titration of oxygen delivery against the risk of inadvertent hypoxaemia.[29] Further challenges for paediatrics include identifying what the appropriate targets should be for specific patient subpopulations (e.g. infants and children with cyanotic heart disease).

There is insufficient paediatric evidence to suggest a specific $PaCO_2$ target, however, $PaCO_2$ should be measured post-ROSC and adjusted according to patient characteristics and needs.[29,167,394,395]

Adult data do not suggest any added benefit of either hypocapnia or hypercapnia; hypocapnia has even been associated with worse outcome. It is sensible to aim in general for normocapnia, although this decision might be in part influenced by context and disease. For instance, it is unclear if a strategy of permissive mild hypercapnia could be beneficial in ventilated children with respiratory failure.

Temperature control and management post ROSC

Mild hypothermia has an acceptable safety profile in adults[396,397] and neonates.[398–403] Recently the THAPCA out of hospital study showed that both hypothermia (32–34 °C) and controlled normothermia (36–37.5 °C) could be used in children.[404] The study did not show a significant difference for the primary outcome (neurologic status at one year) with either approach. The study was, however, underpowered to show a significant difference for survival, for which the lower 95% confidence interval approached 1. Furthermore, hyperthermia occurred frequently in the post-arrest period; hyperthermia is potentially harmful and should be avoided. After ROSC, a strict control of the temperature must be maintained to avoid hyperthermia (>37.5 °C) and severe hypothermia (<32 °C).[29]

Glucose control

Both hyper- and hypoglycaemia may impair outcome of critically ill adults and children and should be avoided,[405–407] but tight glucose control may also be harmful.[408] Although there is insufficient evidence to support or refute a specific glucose management strategy in children with ROSC after cardiac arrest, it is appropriate to monitor blood glucose and to avoid hypoglycaemia and hyperglycaemia.[280,281,374]

Prognosis of cardiopulmonary arrest

Although several factors are associated with outcome after cardiopulmonary arrest and resuscitation there are no simple guidelines to determine when resuscitative efforts become futile.[29,394,409–414]

The relevant considerations in the decision to continue the resuscitation include the duration of CPR, cause of arrest, pre-existing medical conditions, age, site of arrest, whether the arrest was witnessed,[36,415] the duration of untreated cardiopulmonary arrest ('no flow' time), the presence of a shockable rhythm as the first or subsequent rhythm, and associated special circumstances (e.g., icy water drowning[416,417] exposure to toxic drugs). The role of the EEG as a prognostic factor is still unclear. Problems with the literature in this area to identify individual factors are that the studies have largely not been designed in this context and therefore there may be bias as to its use in determining poor or good outcomes. Guidance on the termination of resuscitation attempts is discussed in the chapter on Ethics in Resuscitation and End-of-Life Decisions.[17]

Parental presence

In some Western societies, the majority of parents want to be present during the resuscitation of their child.[418–440] Parental presence has neither been perceived as disruptive nor stressful for the staff.[418,420,436,441] Parents witnessing their child's resuscitation believe their presence to be beneficial to the child.[418–420,427,438,442,443] 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.[419–421,438,439,443,444]

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.[435,440] However, in out-of-hospital resuscitation, some EMS providers may feel anxious owing to the presence of relatives or are concerned that relatives may interfere with their resuscitation efforts.[445] Evidence about parental presence during resuscitation comes from selected countries and can probably not be generalised to all of Europe, where there may be different socio-cultural and ethical considerations.[446,447]

Family presence guidelines

When parents are in the resuscitation room, a member of the resuscitation team should be allocated to them and explain the process in an empathetic manner, ensuring that they do not interfere with or distract the resuscitation process. If the presence of the relatives 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.[435,448–451] The number of relatives present should be at the discretion of the resuscitation team leader.

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.

Collaborators

Koenraad G. Monsieurs, Emergency Medicine, Faculty of Medicine and Health Sciences, University of Antwerp, Antwerp, Belgium and Faculty of Medicine and Health Sciences, University of Ghent, Ghent, Belgium.
Jerry P. Nolan, Anaesthesia and Intensive Care Medicine, Royal United Hospital, Bath, UK and University of Bristol, UK.

Conflict of interest statement

The authors declare no conflict of interest.

References

1. Zideman D, Bingham R, Beattie T, et al. Guidelines for paediatric life support: a statement by the paediatric life support working party of the European Resuscitation Council. Resuscitation 1994;27:91–105 (1993).
2. European Resuscitation Council. Paediatric life support: (including the recommendations for resuscitation of babies at birth). Resuscitation 1998;37:95–6.
3. Phillips B, Zideman D, 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.
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.
5. Biarent D, Bingham R, Eich C, et al. European Resuscitation Council guidelines for resuscitation 2010 section 6 paediatric life support. Resuscitation 2010;81:1364–88.
6. 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.
7. 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:I-46–8.
8. International Liaison Committee on Resuscitation. 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.
9. Kleinman ME, Chameides L, Schexnayder SM, et al. Special report—pediatric advanced life support: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Pediatrics 2010;5:1–9.
10. de Caen AR, Kleinman ME, Chameides L, et al. Part 10: Paediatric basic and advanced life support: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2010;81:e213–59.
11. Morley PT, Lang E, Aickin R, et al. Part 2: Evidence evaluation and management of conflict of interest for the ILCOR 2015 consensus on science and treatment recommendations. Resuscitation 2015;95:e33–41.
12. Maconochie I, de Caen A, Aickin R, et al. Part 6: Pediatric advanced life support: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2015;95:e149–70.
13. DeCaen A, et al. Part 6: Pediatric advanced life support: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Circulation (In press).
14. Wyllie J, Jos Bruinenberg J, Roehr CC, Rüdiger M, Trevisanuto D. B.U. European Resuscitation Council guidelines for resuscitation 2015 section 7 resuscitation and support of transition of babies at birth. Resuscitation 2015;95:248–62.
15. Zideman DA, De Buck EDJ, Singletary EM, et al. European Resuscitation Council guidelines for Resuscitation 2015 Section 9 First Aid. Resuscitation 2015;95:277–86.
16. Greif R, Lockey AS, Conaghan P, Lippert A, De Vries W, Monsieurs KG. European Resuscitation Council Guidelines For Resuscitation 2015 Section 10 Principles of Education In Resuscitation. Resuscitation 2015;95:287–300.
17. Bossaert L, Perkins GD, Askitopoulou H, et al. European Resuscitation Council guidelines for resuscitation 2015 section 11 the ethics of resuscitation and end-of-life decisions. Resuscitation 2015;95:301–10.
18. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. Ann Emerg Med 1992;21:1102–6.
19. Marsch S, Tschan F, Semmer NK, Zobrist R, Hunziker PR, Hunziker S. ABC versus CAB for cardiopulmonary resuscitation: a prospective, randomized simulator-based trial. Swiss Med Wkly 2013;143:w13856.
20. Lubrano R, Cecchetti C, Bellelli E, et al. Comparison of times of intervention during pediatric CPR maneuvers using ABC and CAB sequences: a randomized trial. Resuscitation 2012;83:1473–7.
21. Sekiguchi H, Kondo Y, Kukita I. Verification of changes in the time taken to initiate chest compressions according to modified basic life support guidelines. Am J Emerg Med 2013;31:1248–50.
22. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. Resuscitation 1995;30:141–50.
23. Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury. Pediatrics 1994;94:137–42.
24. 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.
25. 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;375:1347–54.
26. Goto Y, Maeda T, Goto Y. Impact of dispatcher-assisted bystander cardiopulmonary resuscitation on neurological outcomes in children with out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. J Am Heart Assoc 2014;3:e000499.
27. Tibballs J, Russell P. Reliability of pulse palpation by healthcare personnel to diagnose paediatric cardiac arrest. Resuscitation 2009;80:61–4.
28. Tibballs J, Weeranatna C. The influence of time on the accuracy of healthcare personnel to diagnose paediatric cardiac arrest by pulse palpation. Resuscitation 2010;81:671–5.
29. Maconochie I, de Caen A, Aickin R, et al. Part 6: Pediatric basic life support and pediatric advanced life support. 2015 International consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2015;95:e149–70.
30. Sutton RM, French B, Niles DE, et al. 2010 American Heart Association recommended compression depths during pediatric in-hospital resuscitations are associated with survival. Resuscitation 2014;85:1179–84.
31. Perkins GD, Handley AJ, Koster KW, et al. European Resuscitation Council guidelines for resuscitation 2015 section 2 adult basic life support and automated external defibrillation. Resuscitation 2015;95:81–98.
32. Redding JS. The choking controversy: critique of evidence on the Heimlich maneuver. Crit Care Med 1979;7:475–9.
33. 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.
34. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. Ann Emerg Med 1995;25:495–501.
35. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. Ann Emerg Med 1999;33:195–205.
36. 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.
37. 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.
38. Rajan S, Wissenberg M, Folke F, et al. Out-of-hospital cardiac arrests in children and adolescents: incidences, outcomes, and household socioeconomic status. Resuscitation 2015;88:12–9.
39. Gupta P, Tang X, Gall CM, Lauer C, Rice TB, Wetzel RC. Epidemiology and outcomes of in-hospital cardiac arrest in critically ill children across hospitals of varied center volume: a multi-center analysis. Resuscitation 2014;85:1473–9.
40. Nishiuchi T, Hayashino Y, Iwami T, et al. Epidemiological characteristics of sudden cardiac arrest in schools. Resuscitation 2014;85:1001–6.
41. Winkel BG, Risgaard B, Sadjadieh G, Bundgaard H, Haunso S, Tfelt-Hansen J. Sudden cardiac death in children (1–18 years): symptoms and causes of death in a nationwide setting. Eur Heart J 2014;35:868–75.
42. Pilmer CM, Kirsh JA, Hildebrandt D, Krahn AD, Gow RM. Sudden cardiac death in children and adolescents between 1 and 19 years of age. Heart Rhythm 2014;11:239–45.
43. 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.
44. 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.
45. Moler FW, Donaldson AE, Meert K, et al. Multicenter cohort study of out-of-hospital pediatric cardiac arrest. Crit Care Med 2011;39:141–9.
46. 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 (A journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
47. Donoghue AJ, Nadkarni V, Berg RA, et al. Out-of-hospital pediatric cardiac arrest: an epidemiologic review and assessment of current knowledge. Ann Emerg Med 2005;46:512–22.
48. Bray JE, Di Palma S, Jacobs I, Straney L, Finn J. Trends in the incidence of presumed cardiac out-of-hospital cardiac arrest in Perth, Western Australia, 1997–2010. Resuscitation 2014;85:757–61.
49. Mitani Y, Ohta K, Ichida F, et al. Circumstances and outcomes of out-of-hospital cardiac arrest in elementary and middle school students in the era of public-access defibrillation. Circ J 2014;78:701–7 (official journal of the Japanese Circulation Society).
50. Lin YR, Wu HP, Chen WL, et al. Predictors of survival and neurologic outcomes in children with traumatic out-of-hospital cardiac arrest during the early postresuscitative period. J Trauma Acute Care Surg 2013;75:439–47.
51. Zeng J, Qian S, Zheng M, Wang Y, Zhou G, Wang H. The epidemiology and resuscitation effects of cardiopulmonary arrest among hospitalized children and adolescents in Beijing: an observational study. Resuscitation 2013;84:1685–90.
52. Cheung W, Middleton P, Davies S, Tummala S, Thanakrishnan G, Gullick J. A comparison of survival following out-of-hospital cardiac arrest in Sydney, Australia, between 2004–2005 and 2009–2010. Crit Care Resusc 2013;15:241–6.
53. Nitta M, Kitamura T, Iwami T, et al. Out-of-hospital cardiac arrest due to drowning among children and adults from the Utstein Osaka Project. Resuscitation 2013;84:1568–73.
54. Dyson K, Morgans A, Bray J, Matthews B, Smith K. Drowning related out-of-hospital cardiac arrests: characteristics and outcomes. Resuscitation 2013;84:1114–8.
55. De Maio VJ, Osmond MH, Stiell IG, et al. Epidemiology of out-of hospital pediatric cardiac arrest due to trauma. Prehosp Emerg Care 2012;16:230–6 (official journal of the National Association of EMS Physicians and the National Association of State EMS Directors).
56. Deasy C, Bray J, Smith K, et al. Paediatric traumatic out-of-hospital cardiac arrests in Melbourne, Australia. Resuscitation 2012;83:471–5.
57. Knight LJ, Gabhart JM, Earnest KS, Leong KM, Anglemyer A, Franzon D. Improving code team performance and survival outcomes: implementation of pediatric resuscitation team training. Crit Care Med 2014;42:243–51.
58. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
59. 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.
60. 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.
61. 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.
62. 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, A journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).

63. Tibballs J, Kinney S, Duke T, Oakley E, Hennessy M. Reduction of paediatric in-patient cardiac arrest and death with a medical emergency team: preliminary results. Arch Dis Child 2005;90:1148–52.
64. Kotsakis A, Lobos AT, Parshuram C, et al. Implementation of a multicenter rapid response system in pediatric academic hospitals is effective. Pediatrics 2011;128:72–8.
65. Anwar-ul-Haque, Saleem AF, Zaidi S, Haider SR. Experience of pediatric rapid response team in a tertiary care hospital in Pakistan. Indian J Pediatr 2010;77:273–6.
66. Bonafide CP, Localio AR, Song L, et al. Cost–benefit analysis of a medical emergency team in a children's hospital. Pediatrics 2014;134:235–41.
67. Hayes LW, Dobyns EL, DiGiovine B, et al. A multicenter collaborative approach to reducing pediatric codes outside the ICU. Pediatrics 2012;129: e785–91.
68. Zenker P, Schlesinger A, Hauck M, et al. Implementation and impact of a rapid response team in a children's hospital. Jt Comm J Qual Patient Saf 2007;33:418–25.
69. Hanson CC, Randolph GD, Erickson JA, et al. A reduction in cardiac arrests and duration of clinical instability after implementation of a paediatric rapid response system. Qual Saf Health Care 2009;18:500–4.
70. Panesar R, Polikoff LA, Harris D, Mills B, Messina C, Parker MM. Characteristics and outcomes of pediatric rapid response teams before and after mandatory triggering by an elevated Pediatric Early Warning System (PEWS) score. Hosp Pediatr 2014;4:135–40.
71. Randhawa S, Roberts-Turner R, Woronick K, DuVal J. Implementing and sustaining evidence-based nursing practice to reduce pediatric cardiopulmonary arrest. West J Nurs Res 2011;33:443–56.
72. Harrison DA, Patel K, Nixon E, et al. Development and validation of risk models to predict outcomes following in-hospital cardiac arrest attended by a hospital-based resuscitation team. Resuscitation 2014;85:993–1000.
73. Tirkkonen J, Nurmi J, Olkkola KT, Tenhunen J, Hoppu S. Cardiac arrest teams and medical emergency teams in Finland: a nationwide cross-sectional postal survey. Acta Anaesthesiol Scand 2014;58:420–7.
74. Ludikhuize J, Borgert M, Binnekade J, Subbe C, Dongelmans D, Goossens A. Standardized measurement of the modified early warning score results in enhanced implementation of a rapid response system: a quasi-experimental study. Resuscitation 2014;85:676–82.
75. Chaiyakulsil C, Pandee U. Validation of pediatric early warning score in pediatric emergency department. Pediatr Int 2015 (In press).
76. Zuo C, Zhu Y. Development and applications of pediatric early warning score. Zhonghua Er Ke Za Zhi 2014;52:712–4.
77. Gold DL, Mihalov LK, Cohen DM. Evaluating the Pediatric Early Warning Score (PEWS) system for admitted patients in the pediatric emergency department. Acad Emerg Med 2014;21:1249–56 (official journal of the Society for Academic Emergency Medicine).
78. Watson A, Skipper C, Steury R, Walsh H, Levin A. Inpatient nursing care and early warning scores: a workflow mismatch. J Nurs Care Qual 2014;29: 215–22.
79. Breslin K, Marx J, Hoffman H, McBeth R, Pavuluri P. Pediatric early warning score at time of emergency department disposition is associated with level of care. Pediatr Emerg Care 2014;30:97–103.
80. Bonafide CP, Localio AR, Roberts KE, Nadkarni VM, Weirich CM, Keren R. Impact of rapid response system implementation on critical deterioration events in children. JAMA Pediatr 2014;168:25–33.
81. Seiger N, Maconochie I, Oostenbrink R, Moll HA. Validity of different pediatric early warning scores in the emergency department. Pediatrics 2013;132:e841–50.
82. Solevag AL, Eggen EH, Schroder J, Nakstad B. Use of a modified pediatric early warning score in a department of pediatric and adolescent medicine. PLoS ONE 2013;8:e72534.
83. McLellan MC, Gauvreau K, Connor JA. Validation of the Cardiac Children's Hospital Early Warning Score: an early warning scoring tool to prevent cardiopulmonary arrests in children with heart disease. Congenit Heart Dis 2014;9:194–202.
84. Bell D, Mac A, Ochoa Y, Gordon M, Gregurich MA, Taylor T. The Texas Children's Hospital Pediatric Advanced Warning Score as a predictor of clinical deterioration in hospitalized infants and children: a modification of the PEWS tool. J Pediatr Nurs 2013;28:e2–9.
85. Robson MA, Cooper CL, Medicus LA, Quintero MJ, Zuniga SA. Comparison of three acute care pediatric early warning scoring tools. J Pediatr Nurs 2013;28:e33–41.
86. Petrillo-Albarano T, Stockwell J, Leong T, Hebbar K. The use of a modified pediatric early warning score to assess stability of pediatric patients during transport. Pediatr Emerg Care 2012;28:878–82.
87. McLellan MC, Connor JA. The Cardiac Children's Hospital Early Warning Score (C-CHEWS). J Pediatr Nurs 2013;28:171–8.
88. Sweney JS, Poss WB, Grissom CK, Keenan HT. Comparison of severity of illness scores to physician clinical judgment for potential use in pediatric critical care triage. Disaster Med Public Health Prep 2012;6:126–30.
89. Bonafide CP, Holmes JH, Nadkarni VM, Lin R, Landis JR, Keren R. Development of a score to predict clinical deterioration in hospitalized children. J Hosp Med 2012;7:345–9.
90. Parshuram CS, Duncan HP, Joffe AR, et al. Multicentre validation of the bedside paediatric early warning system score: a severity of illness score to detect evolving critical illness in hospitalised children. Crit Care 2011;15:R184.
91. Akre M, Finkelstein M, Erickson M, Liu M, Vanderbilt L, Billman G. Sensitivity of the pediatric early warning score to identify patient deterioration. Pediatrics 2010;125:e763–9.
92. Parshuram CS, Hutchison J, Middaugh K. Development and initial validation of the Bedside Paediatric Early Warning System score. Crit Care 2009;13:R135.
93. Tucker KM, Brewer TL, Baker RB, Demeritt B, Vossmeyer MT. Prospective evaluation of a pediatric inpatient early warning scoring system. J Spec Pediatr Nurs 2009;14:79–85.
94. Egdell P, Finlay L, Pedley DK. The PAWS score: validation of an early warning scoring system for the initial assessment of children in the emergency department. Emerg Med J: EMJ 2008;25:745–9.
95. Edwards ED, Powell CV, Mason BW, Oliver A. Prospective cohort study to test the predictability of the Cardiff and Vale paediatric early warning system. Arch Dis Child 2009;94:602–6.
96. Duncan H, Hutchison J, Parshuram CS. The Pediatric Early Warning System score: a severity of illness score to predict urgent medical need in hospitalized children. J Crit Care 2006;21:271–8.
97. Fleming S, Thompson M, Stevens R, et al. Normal ranges of heart rate and respiratory rate in children from birth to 18 years of age: a systematic review of observational studies. Lancet 2011;377:1011–8.
98. Carcillo JA. Pediatric septic shock and multiple organ failure. Crit Care Clin 2003;19:413–40.
99. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation 1996;33:107–16.
100. 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.
101. Inagawa G, Morimura N, Miwa T, Okuda K, Hirata M, Hiroki K. A comparison of five techniques for detecting cardiac activity in infants. Paediatr Anaesth 2003;13:141–6.
102. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. Resuscitation 2000;44:195–201.
103. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. Acad Emerg Med 2004;11:878–80 (official journal of the Society for Academic Emergency Medicine).
104. 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.
105. Kus A, Gok CN, Hosten T, Gurkan Y, Solak M, Toker K. The LMA-Supreme versus the I-gel in simulated difficult airway in children: a randomised study. Eur J Anaesthesiol 2014;31:280–4.
106. Theiler LG, Kleine-Brueggeney M, Kaiser D, et al. Crossover comparison of the laryngeal mask supreme and the i-gel in simulated difficult airway scenario in anesthetized patients. Anesthesiology 2009;111:55–62.
107. Dolister M, Miller S, Borron S, et al. Intraosseous vascular access is safe, effective and costs less than central venous catheters for patients in the hospital setting. J Vasc Access 2013;14:216–24.
108. Levy B, Perez P, Perny J, Thivilier C, Gerard A. Comparison of norepinephrine-dobutamine to epinephrine for hemodynamics, lactate metabolism, and organ function variables in cardiogenic shock. A prospective, randomized pilot study. Crit Care Med 2011;39:450–5.
109. Rudiger A, Singer M. The heart in sepsis: from basic mechanisms to clinical management. Curr Vasc Pharmacol 2013;11:187–95.
110. Ohchi F, Komasawa N, Mihara R, Minami T. Comparison of mechanical and manual bone marrow puncture needle for intraosseous access: a randomized simulation trial. Springerplus 2015;4:211.
111. Maitland K, Kiguli S, Opoka RO, et al. Mortality after fluid bolus in African children with severe infection. N Engl J Med 2011;364:2483–95.
112. Maitland K, George EC, Evans JA, et al. Exploring mechanisms of excess mortality with early fluid resuscitation: insights from the FEAST trial. BMC Med 2013;11:68.
113. Kelm DJ, Perrin JT, Cartin-Ceba R, Gajic O, Schenck L, Kennedy CC. Fluid overload in patients with severe sepsis and septic shock treated with early goal-directed therapy is associated with increased acute need for fluid-related medical interventions and hospital death. Shock 2015;43:68–73.
114. Dung NM, Day NP, Tam DT, 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 (an official publication of the Infectious Diseases Society of America).
115. 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 (an official publication of the Infectious Diseases Society of America).
116. 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.
117. 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.
118. Santhanam I, Sangareddi S, Venkataraman S, Kissoon N, Thiruvengadamudayan V, Kasthuri RK. A prospective randomized controlled study of two fluid regimens in the initial management of septic shock in the emergency department. Pediatr Emerg Care 2008;24:647–55.

119. Carcillo JA, Davis AL, Zaritsky A. Role of early fluid resuscitation in pediatric septic shock. JAMA 1991;266:1242–5.
120. Sutton RM, Friess SH, Bhalala U, et al. Hemodynamic directed CPR improves short-term survival from asphyxia-associated cardiac arrest. Resuscitation 2013;84:696–701.
121. Friess SH, Sutton RM, Bhalala U, et al. Hemodynamic directed cardiopulmonary resuscitation improves short-term survival from ventricular fibrillation cardiac arrest. Crit Care Med 2013;41:2698–704.
122. 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.
123. 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.
124. Xue FS, Wang Q, Yuan YJ, Xiong J, Liao X. Comparison of the I-gel supraglottic airway as a conduit for tracheal intubation with the intubating laryngeal mask airway. Resuscitation 2010;81:910–1 (author reply 1).
125. Larkin C, King B, D'Agapeyeff A, Gabbott D. iGel supraglottic airway use during hospital cardiopulmonary resuscitation. Resuscitation 2012;83:e141.
126. 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.
127. 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.
128. 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 (official journal of the Society for Academic Emergency Medicine).
129. Murphy-Macabobby M, Marshall WJ, Schneider C, Dries D. Neuromuscular blockade in aeromedical airway management. Ann Emerg Med 1992;21:664–8.
130. Sayre M, Weisgerber I. The use of neuromuscular blocking agents by air medical services. J Air Med Transp 1992;11:7–11.
131. 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.
132. 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 (official journal of the Society for Academic Emergency Medicine).
133. 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.
134. 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 (official journal of the Society for Academic Emergency Medicine).
135. 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 (official journal of the Society for Academic Emergency Medicine).
136. Pepe P, Zachariah B, Chandra N. Invasive airway technique in resuscitation. Ann Emerg Med 1991;22:393–403.
137. Kaye K, Frascone RJ, Held T. Prehospital rapid-sequence intubation: a pilot training program. Prehosp Emerg Care 2003;7:235–40 (official journal of the National Association of EMS Physicians and the National Association of State EMS Directors).
138. 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.
139. Khine HH, Corddry DH, Kettrick RG, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. Anesthesiology 1997;86:627–31 (discussion 27A).
140. 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.
141. 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.
142. 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.
143. 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.
144. 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.
145. Luten RC, Wears RL, Broselow J, et al. Length-based endotracheal tube and emergency equipment in pediatrics. Ann Emerg Med 1992;21:900–4.
146. Sandell JM, Maconochie IK, Jewkes F. Prehospital paediatric emergency care: paediatric triage. Emerg Med J: EMJ 2009;26:767–8.
147. 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;36:856–60 (journal of the International Society for Burn Injuries).
148. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr 1994;125:57–62.
149. 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.
150. 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.
151. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. Ann Emerg Med 2001;37:32–7.
152. 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.
153. 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.
154. Andersen KH, Hald A. Assessing the position of the tracheal tube: the reliability of different methods. Anaesthesia 1989;44:984–5.
155. Andersen KH, Schultz-Lebahn T. Oesophageal intubation can be undetected by auscultation of the chest. Acta Anaesthesiol Scand 1994;38:580–2.
156. 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.
157. 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.
158. 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.
159. Aufderheide TP, Sigurdsson G, Pirrallo RG, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circulation 2004;109:1960–5.
160. Aufderheide TP, Lurie KG. Death by hyperventilation: a common and life-threatening problem during cardiopulmonary resuscitation. Crit Care Med 2004;32. S345-S51.
161. Wik L, Kramer-Johansen J, Myklebust H, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA 2005;293:299–304.
162. Abella BS, Alvarado JP, Myklebust H, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA 2005;293:305–10.
163. 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.
164. Borke WB, Munkeby BH, Morkrid L, Thaulow E, Saugstad OD. Resuscitation with 100% O(2) does not protect the myocardium in hypoxic newborn piglets. Arch Dis Child Fetal 2004;89. F156-F60 (neonatal edition).
165. O'Neill JF, Deakin CD. Do we hyperventilate cardiac arrest patients? Resuscitation 2007;73:82–5.
166. Gazmuri RJ, Ayoub IM, Radhakrishnan J, Motl J, Upadhyaya MP. Clinically plausible hyperventilation does not exert adverse hemodynamic effects during CPR but markedly reduces end-tidal PCO(2). Resuscitation 2012;83:259–64.
167. Del Castillo J, Lopez-Herce J, Matamoros M, et al. Hyperoxia, hypocapnia and hypercapnia as outcome factors after cardiac arrest in children. Resuscitation 2012;83:1456–61.
168. 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.
169. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care 2002;6:283–90 (official journal of the National Association of EMS Physicians and the National Association of State EMS Directors).
170. 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.
171. Bhende MS, LaCovey DC. End-tidal carbon dioxide monitoring in the prehospital setting. Prehosp Emerg Care 2001;5:208–13 (official journal of the National Association of EMS Physicians and the National Association of State EMS Directors).
172. 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.
173. 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.
174. Bhende MS, Thompson AE. Evaluation of an end-tidal CO_2 detector during pediatric cardiopulmonary resuscitation. Pediatrics 1995;95:395–9.
175. 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.
176. 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.
177. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. Ann Emerg Med 1990;19:1104–6.
178. 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.

179. Kolar M, Krizmaric M, Klemen P, Grmec S. Partial pressure of end-tidal carbon dioxide successful predicts cardiopulmonary resuscitation in the field: a prospective observational study. Crit Care 2008;12:R115.
180. Callaham M, Barton C, Matthay 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.
181. 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.
182. 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.
183. 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.
184. 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.
185. Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. N Engl J Med 1988;318:607–11.
186. 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.
187. Lillis KA, Jaffe DM. Prehospital intravenous access in children. Ann Emerg Med 1992;21:1430–4.
188. 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.
189. Hedges JR, Barsan WB, Doan LA, et al. Central versus peripheral intravenous routes in cardiopulmonary resuscitation. Am J Emerg Med 1984;2:385–90.
190. Reades R, Studnek JR, Vandeventer S, Garrett J. Intraosseous versus intravenous vascular access during out-of-hospital cardiac arrest: a randomized controlled trial. Ann Emerg Med 2011;58:509–16.
191. Paxton JH, Knuth TE, Klausner HA. Proximal humerus intraosseous infusion: a preferred emergency venous access. J Trauma 2009;67:606–11.
192. Santos D, Carron PN, Yersin B, Pasquier M. EZ-IO((R)) intraosseous device implementation in a pre-hospital emergency service: a prospective study and review of the literature. Resuscitation 2013;84:440–5.
193. Reiter DA, Strother CG, Weingart SD. The quality of cardiopulmonary resuscitation using supraglottic airways and intraosseous devices: a simulation trial. Resuscitation 2013;84:93–7.
194. Kanter RK, Zimmerman JJ, Strauss RH, Stoeckel KA. Pediatric emergency intravenous access. Evaluation of a protocol. Am J Dis Child 1986;140:132–4.
195. 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.
196. Anson JA. Vascular access in resuscitation: is there a role for the intraosseous route? Anesthesiology 2014;120:1015–31.
197. Glaeser PW, Hellmich TR, Szewczuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. Ann Emerg Med 1993;22:1119–24.
198. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. J Pediatr Surg 1993;28:158–61.
199. Orlowski JP, Julius CJ, Petras RE, Porembka DT, Gallagher JM. The safety of intraosseous infusions: risks of fat and bone marrow emboli to the lungs. Ann Emerg Med 1989;18:1062–7.
200. Orlowski JP, Porembka DT, Gallagher JM, Lockrem JD, VanLente F. Comparison study of intraosseous, central intravenous, and peripheral intravenous infusions of emergency drugs. Am J Dis Child 1990;144:112–7.
201. Ellemunter H, Simma B, Trawoger R, Maurer H. Intraosseous lines in preterm and full term neonates. Arch Dis Child 1999;80. F74-F5 (Fetal and Neonatal Edition).
202. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
203. Horton MA, Beamer C. Powered intraosseous insertion provides safe and effective vascular access for pediatric emergency patients. Pediatr Emerg Care 2008;24:347–50.
204. Frascone 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.
205. Neuhaus D, Weiss M, Engelhardt T, et al. Semi-elective intraosseous infusion after failed intravenous access in pediatric anesthesia. Paediatr Anaesth 2010;20:168–71.
206. Cameron JL, Fontanarosa PB, Passalaqua AM. A comparative study of peripheral to central circulation delivery times between intraosseous and intravenous injection using a radionuclide technique in normovolemic and hypovolemic canines. J Emerg Med 1989;7:123–7.
207. 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.
208. Buck ML, Wiggins BS, Sesler JM. Intraosseous drug administration in children and adults during cardiopulmonary resuscitation. Ann Pharmacother 2007;41:1679–86.
209. Hoskins SL, do Nascimento Jr P, Lima RM, Espana-Tenorio JM, Kramer GC. Pharmacokinetics of intraosseous and central venous drug delivery during cardiopulmonary resuscitation. Resuscitation 2012;83:107–12.
210. 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.
211. 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.
212. Ummenhofer W, Frei FJ, Urwyler A, Drewe J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? Resuscitation 1994;27:123–8.
213. 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.
214. Voelckel WG, 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.
215. 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.
216. Veldhoen ES, de Vooght KM, Slieker MG, Versluys AB, Turner NM. Analysis of bloodgas, electrolytes and glucose from intraosseous samples using an i-STAT((R)) point-of-care analyser. Resuscitation 2014;85:359–63.
217. Ong ME, Chan YH, Oh JJ, Ngo AS. An observational, prospective study comparing tibial and humeral intraosseous access using the EZ-IO. Am J Emerg Med 2009;27:8–15.
218. 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.
219. Brenner T, Bernhard M, Helm M, et al. Comparison of two intraosseous infusion systems for adult emergency medical use. Resuscitation 2008;78:314–9.
220. Turner DA, Kleinman ME. The use of vasoactive agents via peripheral intravenous access during transport of critically Ill infants and children. Pediatr Emerg Care 2010;26:563–6.
221. Venkataraman ST, Orr RA, Thompson AE. Percutaneous infraclavicular subclavian vein catheterization in critically ill infants and children. J Pediatr 1988;113:480–5.
222. 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.
223. Simons FE, Ardusso LR, Bilo MB, et al. International consensus on (ICON) anaphylaxis. World Allergy Organ J 2014;7:9.
224. Campbell RL, Bellolio MF, Knutson BD, et al. Epinephrine in anaphylaxis: higher risk of cardiovascular complications and overdose after administration of intravenous bolus epinephrine compared with intramuscular epinephrine. J Allergy Clin Immunol Pract 2015;3:76–80.
225. Del Pizzo J, Callahan JM. Intranasal medications in pediatric emergency medicine. Pediatr Emerg Care 2014;30:496–501 (quiz 2–4).
226. Leidel BA, Kirchhoff C, Bogner V, Braunstein V, Biberthaler P, Kanz KG. Comparison of intraosseous versus central venous vascular access in adults under resuscitation in the emergency department with inaccessible peripheral veins. Resuscitation 2012;83:40–5.
227. 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.
228. Quinton DN, O'Byrne G, Aitkenhead AR. Comparison of endotracheal and peripheral intravenous adrenaline in cardiac arrest: is the endotracheal route reliable? Lancet 1987;1:828–9.
229. 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.
230. Carcillo JA, Fields AI. Clinical practice parameters for hemodynamic support of pediatric and neonatal patients in septic shock. J Pediatr (Rio J) 2002;78:449–66.
231. Perel P, Roberts I, Ker K. Colloids versus crystalloids for fluid resuscitation in critically ill patients. Cochrane Database Syst Rev 2013;2:CD000567.
232. 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.
233. Dellinger RP, Levy MM, Rhodes A, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock, 2012. Intensive Care Med 2013;39:165–228.
234. Burdett E, Dushianthan A, Bennett-Guerrero E, et al. Perioperative buffered versus non-buffered fluid administration for surgery in adults. Cochrane Database Syst Rev 2012;12:CD004089.
235. Shaw AD, Raghunathan K, Peyerl FW, Munson SH, Paluszkiewicz SM, Schermer CR. Association between intravenous chloride load during resuscitation and in-hospital mortality among patients with SIRS. Intensive Care Med 2014;40:1897–905.
236. Yunos NM, Bellomo R, Bailey M. Chloride-restrictive fluid administration and incidence of acute kidney injury—reply. JAMA 2013;309:543–4.

237. Yunos NM, Bellomo R, Hegarty C, Story D, Ho L, Bailey M. Association between a chloride-liberal vs chloride-restrictive intravenous fluid administration strategy and kidney injury in critically ill adults. JAMA 2012;308:1566–72.
238. Elmer J, Wilcox SR, Raja AS. Massive transfusion in traumatic shock. J Emerg Med 2013;44:829–38.
239. Kua JP, Ong GY, Ng KC. Physiologically-guided balanced resuscitation: an evidence-based approach for acute fluid management in paediatric major trauma. Ann Acad Med Singapore 2014;43:595–604.
240. 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.
241. Peng TJ, Andersen LW, Saindon BZ, et al. The administration of dextrose during in-hospital cardiac arrest is associated with increased mortality and neurologic morbidity. Crit Care 2015;19:160.
242. 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.
243. 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.
244. 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.
245. Salter N, Quin G, Tracy E. Cardiac arrest in infancy: don't forget glucose! Emerg Med J: EMJ 2010;27:720–1.
246. 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.
247. Losek JD, Endom E, Dietrich A, Stewart G, Zempsky W, Smith K. Adenosine and pediatric supraventricular tachycardia in the emergency department: multicenter study and review. Ann Emerg Med 1999;33:185–91.
248. 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.
249. 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.
250. 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.
251. Dieckmann RA, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. Pediatrics 1995;95:901–13.
252. Enright K, Turner C, Roberts P, Cheng N, Browne G. Primary cardiac arrest following sport or exertion in children presenting to an emergency department: chest compressions and early defibrillation can save lives, but is intravenous epinephrine always appropriate? Pediatr Emerg Care 2012;28:336–9.
253. 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.
254. 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.
255. Saharan S, Balaji S. Cardiovascular collapse during amiodarone infusion in a hemodynamically compromised child with refractory supraventricular tachycardia. Ann Pediatr Cardiol 2015;8:50–2.
256. 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.
257. 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.
258. 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.
259. Valdes SO, Donoghue AJ, Hoyme DB, et al. Outcomes associated with amiodarone and lidocaine in the treatment of in-hospital pediatric cardiac arrest with pulseless ventricular tachycardia or ventricular fibrillation. Resuscitation 2014;85:381–6.
260. Kudenchuk PJ, Brown SP, Daya M, et al. Resuscitation Outcomes Consortium-Amiodarone Lidocaine or Placebo Study (ROC-ALPS): rationale and methodology behind an out-of-hospital cardiac arrest antiarrhythmic drug trial. Am Heart J 2014;167:e4, 653-9.
261. Dauchot P, Gravenstein JS. Effects of atropine on the electrocardiogram in different age Groups. Clin Pharmacol Ther 1971;12:274–80.
262. 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.
263. 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.
264. 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.
265. Fastle RK, Roback MG. Pediatric rapid sequence intubation: incidence of reflex bradycardia and effects of pretreatment with atropine. Pediatr Emerg Care 2004;20:651–5.
266. Jones P, Dauger S, Denjoy I, et al. The effect of atropine on rhythm and conduction disturbances during 322 critical care intubations. Pediatr Crit Care Med 2013;14:e289–97 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
267. 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.
268. Paraskos JA. Cardiovascular pharmacology III: atropine, calcium, calcium blockers, and (beta)-blockers. Circulation 1986;74. IV-IV86.
269. Gupta P, Tomar M, Radhakrishnan S, Shrivastava S. Hypocalcemic cardiomyopathy presenting as cardiogenic shock. Ann Pediatr Cardiol 2011;4:152–5.
270. 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.
271. Kette F, Ghuman J, Parr M. Calcium administration during cardiac arrest: a systematic review. Eur J Emerg Med 2013;20:72–8 (official journal of the European Society for Emergency Medicine).
272. 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.
273. de Mos N, van Litsenburg RR, McCrindle B, Bohn DJ, Parshuram CS. Pediatric in-intensive-care-unit cardiac arrest: incidence, survival, and predictive factors. Crit Care Med 2006;34:1209–15.
274. Dias CR, Leite HP, Nogueira PC, Brunow de Carvalho W. Ionized hypocalcemia is an early event and is associated with organ dysfunction in children admitted to the intensive care unit. J Crit Care 2013;28:810–5.
275. Krinsley JS. Effect of an intensive glucose management protocol on the mortality of critically ill adult patients. Mayo Clin Proc 2004;79:992–1000.
276. Losek JD. Hypoglycemia and the ABC'S (sugar) of pediatric resuscitation. Ann Emerg Med 2000;35:43–6.
277. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
278. Finney SJ, Zekveld C, Elia A, Evans TW. Glucose control and mortality in critically ill patients. JAMA 2003;290:2041–7.
279. Topjian AA, Berg RA, Bierens JJ, et al. Brain resuscitation in the drowning victim. Neurocrit Care 2012;17:441–67.
280. 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.
281. 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.
282. Macrae D, Grieve R, Allen E, et al. A randomized trial of hyperglycemic control in pediatric intensive care. N Engl J Med 2014;370:107–18.
283. Investigators N-SSFinfer S, Chittock DR, et al. Intensive versus conventional glucose control in critically ill patients. N Engl J Med 2009;360:1283–97.
284. 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.
285. Reis AG, Ferreira de Paiva E, Schvartsman C, Zaritsky AL. Magnesium in cardiopulmonary resuscitation: critical review. Resuscitation 2008;77:21–5.
286. Tzivoni D, Banai S, Schuger C, et al. Treatment of torsade de pointes with magnesium sulfate. Circulation 1988;77:392–7.
287. 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.
288. 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.
289. Weng YM, Wu SH, Li WC, Kuo CW, Chen SY, Chen JC. The effects of sodium bicarbonate during prolonged cardiopulmonary resuscitation. Am J Emerg Med 2013;31:562–5.
290. Raymond TT, Stromberg D, Stigall W, Burton G, Zaritsky A. American Heart Association's Get With The Guidelines—Resuscitation I. Sodium bicarbonate use during in-hospital pediatric pulseless cardiac arrest—a report from the American Heart Association Get With The Guidelines((R))-Resuscitation. Resuscitation 2015;89:106–13.
291. 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.
292. Wang JD, Fu YC, Jan SL, Chi CS. Verapamil sensitive idiopathic ventricular tachycardia in an infant. Jpn Heart J 2003;44:667–71.
293. 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.

294. Chang PM, Silka MJ, Moromisato DY, Bar-Cohen Y. Amiodarone versus procainamide for the acute treatment of recurrent supraventricular tachycardia in pediatric patients. Circ Arrhythmia Electrophysiol 2010;3:134–40.
295. Mandapati R, Byrum CJ, Kavey RE, et al. Procainamide for rate control of post-surgical junctional tachycardia. Pediatr Cardiol 2000;21:123–8.
296. 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.
297. 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.
298. Holmes CL, Landry DW, Granton JT. Science review: Vasopressin and the cardiovascular system part 1—receptor physiology. Crit Care 2003;7:427–34.
299. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
300. 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.
301. 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.
302. 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.
303. 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.
304. Mentzelopoulos SD, Malachias S, Chamos C, et al. Vasopressin, steroids, and epinephrine and neurologically favorable survival after in-hospital cardiac arrest: a randomized clinical trial. JAMA 2013;310:270–9.
305. Daley MJ, Lat I, Mieure KD, Jennings HR, Hall JB, Kress JP. A comparison of initial monotherapy with norepinephrine versus vasopressin for resuscitation in septic shock. Ann Pharmacother 2013;47:301–10.
306. Ong ME, Tiah L, Leong BS, et al. A randomised, double-blind, multi-centre trial comparing vasopressin and adrenaline in patients with cardiac arrest presenting to or in the Emergency Department. Resuscitation 2012;83:953–60.
307. 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.
308. 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.
309. Peters MJ, Booth RA, Petros AJ. Terlipressin bolus induces systemic vasoconstriction in septic shock. Pediatr Crit Care Med 2004;5:112–5 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
310. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
311. 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.
312. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using "adult" electrode paddles. Pediatrics 1994;94:90–3.
313. Deakin C, Sado D, Petley G, Clewlow F. Determining the optimal paddle force for external defibrillation. Am J Cardiol 2002;90:812–3.
314. Bennetts SH, Deakin CD, Petley GW, Clewlow F. Is optimal paddle force applied during paediatric external defibrillation? Resuscitation 2004;60:29–32.
315. 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 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
316. 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.
317. 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.
318. 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.
319. Rossano JQ, Schiff L, Kenney MA, et al. Survival is not correlated with defibrillation dosing in pediatric out-of-hospital ventricular fibrillation. Circulation 2003;108:320–1. IV (MA K, DL A).
320. 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.
321. 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.
322. 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.
323. 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.
324. 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.
325. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. Ann Emerg Med 1987;16:1107–11.
326. 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.
327. 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.
328. Johnson MA, Grahan BJ, Haukoos JS, et al. Demographics, bystander CPR, and AED use in out-of-hospital pediatric arrests. Resuscitation 2014;85:920–6.
329. 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.
330. Cummins RO, Graves JR, Larsen MP, et al. Out-of-hospital transcutaneous pacing by emergency medical technicians in patients with asystolic cardiac arrest. N Engl J Med 1993;328:1377–82.
331. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. Arch Dis Child 1990;65:127–9.
332. 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.
333. Bianconi L, Castro AMD, 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.
334. 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.
335. 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.
336. 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.
337. 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.
338. 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.
339. Haas NA, Camphausen CK. Acute hemodynamic effects of intravenous amiodarone treatment in pediatric patients with cardiac surgery. Clin Res Cardiol 2008;97:801–10 (official journal of the German Cardiac Society).
340. Adamson PC, Rhodes LA, Saul JP, et al. The pharmacokinetics of esmolol in pediatric subjects with supraventricular arrhythmias. Pediatr Cardiol 2006;27:420–7.
341. 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.
342. 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.
343. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. Eur J Pediatr 2003;162:880–4.
344. Drago F, Mazza A, Guccione P, Mafrici A, Di Liso G, Ragonese P. Amiodarone used alone or in combination with propranolol: a very effective therapy for tachyarrhythmias in infants and children. Pediatr Cardiol 1998;19:445–9.
345. 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.
346. Crewdson K, Lockey D, Davies G. Outcome from paediatric cardiac arrest associated with trauma. Resuscitation 2007;75:29–34.
347. 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.
348. 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 (official journal of the National Association of EMS Physicians and the National Association of State EMS Directors).
349. Brindis SL, Gausche-Hill M, Young KD, Putnam B. Universally poor outcomes of pediatric traumatic arrest: a prospective case series and review of the literature. Pediatr Emerg Care 2011;27:616–21.
350. Murphy JT, Jaiswal K, Sabella J, Vinson L, Megison S, Maxson RT. Prehospital cardiopulmonary resuscitation in the pediatric trauma patient. J Pediatr Surg 2010;45:1413–9.
351. Widdel L, Winston KR. Prognosis for children in cardiac arrest shortly after blunt cranial trauma. J Trauma 2010;69:783–8.

352. Duron V, Burke RV, Bliss D, Ford HR, Upperman JS. Survival of pediatric blunt trauma patients presenting with no signs of life in the field. J Trauma Acute Care Surg 2014;77:422–6.
353. Sheikh A, Brogan T. Outcome and cost of open- and closed-chest cardiopulmonary resuscitation in pediatric cardiac arrests. Pediatrics 1994;93:392–8.
354. 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.
355. Powell RW, Gill EA, Jurkovich GJ, Ramenofsky ML. Resuscitative thoracotomy in children and adolescents. Am Surg 1988;54:188–91.
356. 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.
357. Suominen P, Rasanen J, Kivioja A. Efficacy of cardiopulmonary resuscitation in pulseless paediatric trauma patients. Resuscitation 1998;36:9–13.
358. Easter JS, Vinton DT, Haukoos JS. Emergent pediatric thoracotomy following traumatic arrest. Resuscitation 2012;83:1521–4.
359. Hofbauer M, Hupfl M, Figl M, Hochtl-Lee L, Kdolsky R. Retrospective analysis of emergency room thoracotomy in pediatric severe trauma patients. Resuscitation 2011;82:185–9.
360. 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.
361. 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.
362. 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.
363. Hoeper 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.
364. 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.
365. Sablotzki A, Hentschel T, Gruenig E, et al. Hemodynamic effects of inhaled aerosolized iloprost and inhaled nitric oxide in heart transplant candidates with elevated pulmonary vascular resistance. Eur J Cardiothorac Surg 2002;22:746–52.
366. Kirbas A, Yalcin Y, Tanrikulu N, Gurer O, Isik O. Comparison of inhaled nitric oxide and aerosolized iloprost in pulmonary hypertension in children with congenital heart surgery. Cardiol J 2012;19:387–94.
367. Loukanov T, Bucsenez D, Springer W, et al. Comparison of inhaled nitric oxide with aerosolized iloprost for treatment of pulmonary hypertension in children after cardiopulmonary bypass surgery. Clin Res Cardiol 2011;100:595–602 (official journal of the German Cardiac Society).
368. Antoniou T, Koletsis EN, Prokakis C, et al. Hemodynamic effects of combination therapy with inhaled nitric oxide and iloprost in patients with pulmonary hypertension and right ventricular dysfunction after high-risk cardiac surgery. J Cardiothorac Vasc Anesth 2013;27:459–66.
369. 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.
370. 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.
371. Arpesella G, Loforte A, Mikus E, Mikus PM. Extracorporeal membrane oxygenation for primary allograft failure. Transplant Proc 2008;40:3596–7.
372. Strueber M, Hoeper MM, Fischer S, et al. Bridge to thoracic organ transplantation in patients with pulmonary arterial hypertension using a pumpless lung assist device. Am J Transpl 2009;9:853–7 (official journal of the American Society of Transplantation and the American Society of Transplant Surgeons).
373. Simon MA. Assessment and treatment of right ventricular failure. Nat Rev Cardiol 2013;10:204–18.
374. 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.
375. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. Crit Care Med 1988;16:331–5.
376. 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.
377. Conlon TW, Falkensammer CB, Hammond RS, Nadkarni VM, Berg RA, Topjian AA. Association of left ventricular systolic function and vasopressor support with survival following pediatric out-of-hospital cardiac arrest. Pediatr Crit Care Med 2015;16:146–54 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
378. Bougouin W, Cariou A. Management of postcardiac arrest myocardial dysfunction. Curr Opin Crit Care 2013;19:195–201.
379. 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.
380. 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.
381. 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.
382. 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.
383. 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.
384. 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.
385. 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.
386. 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.
387. 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.
388. 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.
389. Nijhawan N, Nicolosi AC, Montgomery MW, Aggarwal A, Pagel PS, Warltier DC. Levosimendan enhances cardiac performance after cardiopulmonary bypass: a prospective, randomized placebo-controlled trial. J Cardiovasc Pharmacol 1999;34:219–28.
390. Topjian AA, French B, Sutton RM, et al. Early postresuscitation hypotension is associated with increased mortality following pediatric cardiac arrest. Crit Care Med 2014;42:1518–23.
391. Guerra-Wallace MM, Casey 3rd FL, Bell MJ, Fink EL, Hickey RW. Hyperoxia and hypoxia in children resuscitated from cardiac arrest. Pediatr Crit Care Med 2013;14:e143–8 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
392. Ferguson LP, Durward A, Tibby SM. Relationship between arterial partial oxygen pressure after resuscitation from cardiac arrest and mortality in children. Circulation 2012;126:335–42.
393. Bennett KS, Clark AE, Meert KL, et al. Early oxygenation and ventilation measurements after pediatric cardiac arrest: lack of association with outcome. Crit Care Med 2013;41:1534–42.
394. Lopez-Herce J, del Castillo J, Matamoros M, et al. Post return of spontaneous circulation factors associated with mortality in pediatric in-hospital cardiac arrest: a prospective multicenter multinational observational study. Crit Care 2014;18:607.
395. Roberts BW, Kilgannon JH, Chansky ME, Mittal N, Wooden J, Trzeciak S. Association between postresuscitation partial pressure of arterial carbon dioxide and neurological outcome in patients with post-cardiac arrest syndrome. Circulation 2013;127:2107–13.
396. 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.
397. Hypothermia after Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med 2002;346:549–56.
398. 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.
399. 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.
400. 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.
401. 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.
402. 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.
403. 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.
404. Moler FW, Silverstein FS, Holubkov R, et al. Therapeutic hypothermia after out-of-hospital cardiac arrest in children. N Engl J Med 2015;372:1898–908.
405. 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.
406. 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.
407. 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.
408. 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.
409. Slonim AD, Patel KM, Ruttimann UE, Pollack MM. Cardiopulmonary resuscitation in pediatric intensive care units. Crit Care Med 1997;25:1951–5.
410. 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.
411. 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.
412. 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.
413. Tibballs J, Kinney S. A prospective study of outcome of in-patient paediatric cardiopulmonary arrest. Resuscitation 2006;71:310–8.
414. Lopez-Herce J, Del Castillo J, Matamoros M, et al. Factors associated with mortality in pediatric in-hospital cardiac arrest: a prospective multicenter multinational observational study. Intensive Care Med 2013;39:309–18.
415. Lopez-Herce J, Garcia C, Dominguez P, et al. Characteristics and outcome of cardiorespiratory arrest in children. Resuscitation 2004;63:311–20.
416. 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.
417. 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.
418. 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:e3.
419. 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.
420. 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.
421. 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.
422. Jones M, Qazi M, Young KD. Ethnic differences in parent preference to be present for painful medical procedures. Pediatrics 2005;116:e191–7.
423. 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.
424. Andrews R, Andrews R. Family presence during a failed major trauma resuscitation attempt of a 15-year-old boy: lessons learned. J Emerg Nurs 2004;30:556–8 [see comment].
425. 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.
426. Gold KJ, Gorenflo DW, Schwenk TL, et al. Physician experience with family presence during cardiopulmonary resuscitation in children. Pediatric Crit Care Med 2006;7:428–33 [see comment].
427. 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.
428. McAlvin SS, Carew-Lyons A. Family presence during resuscitation and invasive procedures in pediatric critical care: a systematic review. Am J Crit Care 2014;23:477–84 (quiz 85).
429. Gaudreault J, Carnevale FA. Should I stay or should I go? Parental struggles when witnessing resuscitative measures on another child in the pediatric intensive care unit. Pediatr Crit Care Med 2012;13:146–51 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
430. Fullbrook S. End-of-life issues: common law and the Mental Capacity Act 2005. Br J Nurs 2007;16:816–8.
431. Giannini A, Miccinesi G. Parental presence and visiting policies in Italian pediatric intensive care units: a national survey. Pediatr Crit Care Med 2011;12:e46–50 (a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies).
432. Perez Alonso V, Gomez Saez F, Gonzalez-Granado LI, Rojo Conejo P. Presence of parents in the emergency room during invasive procedures: do they prefer to be present? An Pediatr (Barc) 2009;70:230–4.
433. Maxton FJ. Parental presence during resuscitation in the PICU: the parents' experience. Sharing and surviving the resuscitation: a phenomenological study. J Clin Nurs 2008;17:3168–76.
434. Dingeman RS, Mitchell EA, Meyer EC, Curley MA. Parent presence during complex invasive procedures and cardiopulmonary resuscitation: a systematic review of the literature. Pediatrics 2007;120:842–54.
435. Meyers TA, Eichhorn DJ, Guzzetta CE, et al. Family presence during invasive procedures and resuscitation. Am J Nurs 2000;100:32–42 (quiz 3).
436. 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.
437. 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.
438. Holzhauser K, Finucane J, De Vries S. Family presence during resuscitation: a randomised controlled trial of the impact of family presence. Aust Emerg Nurs J 2005;8:139–47.
439. 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.
440. Curley MA, Meyer EC, Scoppettuolo LA, et al. Parent presence during invasive procedures and resuscitation: evaluating a clinical practice change. Am J Respir Crit Care Med 2012;186:1133–9.
441. Carroll DL. The effect of intensive care unit environments on nurse perceptions of family presence during resuscitation and invasive procedures. Dimens Crit Care Nurs 2014;33:34–9.
442. Meyers TA, Eichhorn DJ, Guzzetta CE. Do families want to be present during CPR? A retrospective survey. J Emerg Nurs 1998;24:400–5.
443. Hanson C, Strawser D. Family presence during cardiopulmonary resuscitation: Foote Hospital emergency department's nine-year perspective. J Emerg Nurs 1992;18:104–6.
444. 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.
445. Compton S, Madgy A, Goldstein M, et al. Emergency medical service providers' experience with family presence during cardiopulmonary resuscitation. Resuscitation 2006;70:223–8.
446. Vavarouta A, Xanthos T, Papadimitriou L, Kouskouni E, Iacovidou N. Family presence during resuscitation and invasive procedures: physicians' and nurses' attitudes working in pediatric departments in Greece. Resuscitation 2011;82:713–6.
447. Corniero P, Gamell A, Parra Cotanda C, Trenchs V, Cubells CL. Family presence during invasive procedures at the emergency department: what is the opinion of Spanish medical staff? Pediatr Emerg Care 2011;27:86–91.
448. 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 (official journal of the Society for Academic Emergency Medicine).
449. Eppich WJ, Arnold LD. Family member presence in the pediatric emergency department. Curr Opin Pediatr 2003;15:294–8.
450. Eichhorn DJ, Meyers TA, Mitchell TG, Guzzetta CE. Opening the doors: family presence during resuscitation. J Cardiovasc Nurs 1996;10:59–70.
451. Jarvis AS. Parental presence during resuscitation: attitudes of staff on a paediatric intensive care unit. Intensive Crit Care Nurs 1998;14:3–7.