Part 14: Pediatric Advanced Life Support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

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2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

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In contrast to adults, cardiac arrest in infants and children does not usually result from a primary cardiac cause. More often it is the terminal result of progressive respiratory failure or shock, also called an asphyxial arrest. Asphyxia begins with a variable period of systemic hypoxemia, hypercapnea, and acidosis, progresses to bradycardia and hypotension, and culminates with cardiac arrest.[1]

Another mechanism of cardiac arrest, ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), is the initial cardiac rhythm in approximately 5% to 15% of pediatric in-hospital and out-of-hospital cardiac arrests;[2–9] it is reported in up to 27% of pediatric in-hospital arrests at some point during the resuscitation.[6] The incidence of VF/pulseless VT cardiac arrest rises with age.[2,4] Increasing evidence suggests that sudden unexpected death in young people can be associated with genetic abnormalities in myocyte ion channels resulting in abnormalities in ion flow (see "Sudden Unexplained Deaths," below).

Since 2010 marks the 50th anniversary of the introduction of cardiopulmonary resuscitation (CPR),[10] it seems appropriate to review the progressive improvement in outcome of pediatric resuscitation from cardiac arrest. Survival from in-hospital cardiac arrest in infants and children in the 1980s was around 9%.[11,12] Approximately 20 years later, that figure had increased to 17%,[13,14] and by 2006, to 27%.[15–17] In contrast to those favorable results from in-hospital cardiac arrest, overall survival to discharge from out-of-hospital cardiac arrest in infants and children has not changed substantially in 20 years and remains at about 6% (3% for infants and 9% for children and adolescents).[7,9]

It is unclear why the improvement in outcome from in-hospital cardiac arrest has occurred, although earlier recognition and management of at-risk patients on general inpatient units and more aggressive implementation of evidence-based resuscitation guidelines may have played a role. Implementation of a formal pediatric medical emergency team (MET) or rapid response team (RRT) as part of an emergency response system for a deteriorating inpatient has been shown to significantly decrease the incidence of cardiac and respiratory arrests, as well as hospital mortality rates in some large children's hospitals.[18–21] Such teams, often consisting of providers with expertise in assessment and initial management of acutely ill patients (critical-care nurses, respiratory therapists, and critical-care physicians), decreased the number of cardiac and respiratory arrests by as much as 72%[18] and hospital mortality by as much as 35% in institutions where the effect was studied.[19] Although it is possible that most of the impact is due to a decrease in respiratory arrests, this cannot be confirmed by the available published data. Implementation of a pediatric MET/RRT may be beneficial in facilities where children with high risk illnesses are present on general inpatient units (Class IIa, LOE B).

Despite the improved outcome of in-hospital CPR, a majority of children with in-hospital cardiac arrest and an even larger percentage of children with out-of-hospital cardiac arrest do not survive, or they are severely incapacitated if they do. Several studies, discussed later in this document, showed that the presence of family members during resuscitation has helped them deal with the inevitable trauma and grief following the death of a child. Therefore, whenever possible, provide family members with the option of being present during resuscitation of an infant or child (Class I, LOE B).

BLS Considerations During PALS

Pediatric advanced life support (PALS) usually takes place in the setting of an organized response in an advanced healthcare environment. In these circumstances, multiple responders are rapidly mobilized and are capable of simultaneous coordinated action. Resuscitation teams may also have access to invasive patient monitoring that may provide additional information during the performance of basic life support (BLS).

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Simultaneous Actions

BLS (whether for a child or adult) is presented as a series of sequential events with the assumption that there is only one responder, but PALS usually takes place in an environment where many rescuers are rapidly mobilized and actions are performed simultaneously. The challenge is to organize the rescuers into an efficient team. Important considerations for the greatest chance of a successful resuscitation from cardiac arrest include the following:

- Chest compressions should be immediately started by one rescuer, while a second rescuer prepares to start ventilations with a bag and mask. Ventilation is extremely important in pediatrics because of the large percentage of asphyxial arrests in which best results are obtained by a combination of chest compressions and ventilations.[8] Unfortunately ventilations are sometimes delayed because equipment (bag, mask, oxygen, airway) must be mobilized. Chest compressions require only the hands of a willing rescuer. Therefore, start CPR with chest compressions immediately, while a second rescuer prepares to provide ventilations (Class I, LOE C).
- The effectiveness of PALS is dependent on high-quality CPR, which requires an adequate compression rate (at least 100 compressions/min), an adequate compression depth (at least one third of the AP diameter of the chest or approximately 1 ½ inches [4 cm] in infants and approximately 2 inches [5 cm] in children), allowing complete recoil of the chest after each compression, minimizing interruptions in compressions, and avoiding excessive ventilation. Reasons for not performing high-quality CPR include rescuer inattention to detail, rescuer fatigue, and long or frequent interruptions to secure the airway, check the heart rhythm, and move the patient.[22] Optimal chest compressions are best delivered with the victim on a firm surface.[23,24]
- While one rescuer performs chest compressions and another performs ventilations, other rescuers should obtain a monitor/defibrillator, establish vascular access, and calculate and prepare the anticipated medications.

Monitored Patients

Many in-hospital patients, especially if they are in an ICU, are monitored and some have an advanced airway and are receiving mechanical ventilation. If the patient has an indwelling arterial catheter, use the waveform as feedback to evaluate hand position and chest compression depth. A minor adjustment of hand position or depth of compression can significantly improve the amplitude of the arterial waveform, reflecting better chest compression-induced stroke volume. The arterial waveform may also be useful in identification of return of spontaneous circulation (ROSC). If the patient's end-tidal CO_2 (P_{ETCO_2}) is being monitored, it can be used to evaluate the quality of chest compressions; it can also provide an indication of ROSC (see below).

Respiratory Failure

Respiratory failure is characterized by inadequate ventilation, insufficient oxygenation, or both. Anticipate respiratory failure if any of the following signs is present:

- An increased respiratory rate, particularly with signs of distress (eg, increased respiratory effort including nasal flaring, retractions, seesaw breathing, or grunting)
- An inadequate respiratory rate, effort, or chest excursion (eg, diminished breath sounds or gasping), especially if mental status is depressed
- Cyanosis with abnormal breathing despite supplementary oxygen

Shock

Shock results from inadequate blood flow and oxygen delivery to meet tissue metabolic demands. The most common type of shock in children is hypovolemic, including shock due to hemorrhage. Distributive, cardiogenic, and obstructive shock occur less frequently. Shock progresses over a continuum of severity, from a compensated to a decompensated state. Compensatory mechanisms include tachycardia and increased systemic vascular resistance (vasoconstriction) in an effort to maintain cardiac output and perfusion pressure respectively. Decompensation occurs when compensatory mechanisms fail and results in hypotensive shock.

Typical signs of compensated shock include

- Tachycardia
- Cool and pale distal extremities
- Prolonged (>2 seconds) capillary refill (despite warm ambient temperature)
- Weak peripheral pulses compared with central pulses
- Normal systolic blood pressure

As compensatory mechanisms fail, signs of inadequate end-organ perfusion develop. In addition to the above, these signs include

- Depressed mental status
- Decreased urine output
- Metabolic acidosis
- Tachypnea
- Weak central pulses
- Deterioration in color (eg, mottling, see below)

Decompensated shock is characterized by signs and symptoms consistent with inadequate delivery of oxygen to tissues (pallor, peripheral cyanosis, tachypnea, mottling of the skin, decreased urine output, metabolic acidosis, depressed mental status), weak or absent peripheral pulses, weak central pulses, and hypotension.

Learn to integrate the signs of shock because no single sign confirms the diagnosis. For example:

- Capillary refill time alone is not a good indicator of circulatory volume, but a capillary refill time >2 seconds is a useful indicator of moderate dehydration when combined with decreased urine output, absent tears, dry mucous membranes, and a generally ill appearance. Capillary refill time is influenced by ambient temperature,[25] site, and age and its interpretation can be influenced by lighting.[26]
- Tachycardia is a common sign of shock, but it can also result from other causes, such as pain, anxiety, and fever.

- Pulses are weak in hypovolemic and cardiogenic shock, but may be bounding in anaphylactic, neurogenic, and septic shock.
- Blood pressure may be normal in a child with compensated shock but may decline rapidly when the child decompensates. Like the other signs, hypotension must be interpreted within the context of the entire clinical picture.

There are several sources of data that use large populations to identify the 5th percentile for systolic blood pressure at various ages.[27,28] For purposes of these guidelines, hypotension is defined as a ***systolic*** blood pressure:

- <60 mm Hg in term neonates (0 to 28 days)
- <70 mm Hg in infants (1 month to 12 months)
- <70 mm Hg + (2 × age in years) in children 1 to 10 years
- <90 mm Hg in children ≥10 years of age

Airway

Oropharyngeal and Nasopharyngeal Airways

Oropharyngeal and nasopharyngeal airways help maintain an open airway by displacing the tongue or soft palate from the pharyngeal air passages. Oropharyngeal airways are used in unresponsive victims who do not have a gag reflex. Make sure to select the correct size: an oropharyngeal airway that is too small may push the base of the tongue farther into the airway; one that is too large may obstruct the airway.

Nasopharyngeal airways can be used in children who do have a gag reflex. Pay careful attention to proper diameter and length. A nasopharyngeal airway that is too short may not maintain an open airway, while one that is too long may obstruct it. A small-diameter nasopharyngeal airway may be obstructed easily by secretions. It may therefore require frequent suctioning.

Laryngeal Mask Airway (LMA)

Although several supraglottic devices have been used in children, clinical studies of devices other than the LMA in pediatric patients are limited. When bag-mask ventilation (see "Bag-Mask Ventilation," below) is unsuccessful and when endotracheal intubation is not possible, the LMA is acceptable when used by experienced providers to provide a patent airway and support ventilation (Class IIa, LOE C).[29–37] LMA insertion is associated with a higher incidence of complications in young children compared with older children and adults.[38–43]

Oxygen

It is reasonable to ventilate with 100% oxygen during CPR because there is insufficient information on the optimal inspired oxygen concentration (Class IIa, LOE C). Once the circulation is restored, monitor systemic oxygen saturation. It may be reasonable, when the appropriate equipment is available, to titrate oxygen administration to maintain the oxyhemoglobin saturation ≥94%. Provided appropriate equipment is available, once ROSC is achieved, adjust the F_{IO_2} to the minimum concentration needed to achieve an arterial oxyhemoglobin saturation at least 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery. Since an arterial oxyhemoglobin saturation of 100% may correspond to a PaO_2 anywhere between ~80 and 500 mmHg, in general it is appropriate to wean the F_{IO_2} when saturation is 100%, provided the oxyhemoglobin saturation can be maintained ≥94% (Class IIb, LOE C). Remember that adequate oxygen delivery requires not only adequate arterial oxyhemoglobin saturation but also adequate hemoglobin concentration and cardiac output.

Pulse Oximetry

If the patient has a perfusing rhythm, monitor oxyhemoglobin saturation continuously with a pulse oximeter because clinical recognition of hypoxemia is not reliable.[44] Pulse oximetry may, however, also be unreliable in patients with poor peripheral perfusion, carbon monoxide poisoning, or methemoglobinemia.

Bag-Mask Ventilation

Bag-mask ventilation can be as effective, and may be safer, than endotracheal tube ventilation for short periods during out-of-hospital resuscitation.[45–52] In the prehospital setting it is reasonable to ventilate and oxygenate infants and children with a bag-mask device, especially if transport time is short (Class IIa, LOE B). Bag-mask ventilation requires training and periodic retraining in selecting a correct mask size, maintaining an open airway, providing a tight seal between mask and face, providing ventilation, and assessing effectiveness of ventilation (see Part 13, "Pediatric Basic Life Support").

Precautions

Use only the force and tidal volume needed to just make the chest rise visibly (Class I, LOE C); avoid delivering excessive ventilation during cardiac arrest (Class III, LOE C). Evidence shows that cardiac arrest victims frequently receive excessive ventilation.[22,53–55] Excessive ventilation during cardiac arrest increases intrathoracic pressure, which impedes venous return, thus reducing cardiac output and cerebral and coronary blood flow. These effects will reduce the likelihood of ROSC.[54] In addition, excessive ventilation may cause air trapping and barotrauma in patients with small airway obstruction. It also increases the risk of stomach inflation, regurgitation, and aspiration.

If the infant or child is not intubated, pause after 30 chest compressions (1 rescuer) or after 15 chest compressions (2 rescuers) to give 2 ventilations (mouth-to-mouth, mouth-to-mask, or bag-mask). Deliver each breath with an inspiratory time of approximately 1 second. If the infant or child is intubated, ventilate at a rate of about 1 breath every 6 to 8 seconds (8 to 10 times per minute) without interrupting chest compressions (Class I, LOE C). It may be reasonable to do the same if an LMA is in place (Class IIb, LOE C).

In the victim with a perfusing rhythm but absent or inadequate respiratory effort, give 1 breath every 3 to 5 seconds (12 to 20 breaths per minute), using the higher rate for the younger child (Class I, LOE C). One way to achieve that rate with a ventilating bag is to use the mnemonic "squeeze-release-release" at a normal speaking rate.[45,56]

Two-Person Bag-Mask Ventilation

A 2-person ventilation technique may be preferable when personnel are available and may be more effective than ventilation by a single rescuer if the patient has significant

airway obstruction, poor lung compliance, or the rescuer has difficulty in creating a tight mask-to-face seal.[57,58] One rescuer uses both hands to maintain an open airway with a jaw thrust and a tight mask-to-face seal while the other compresses the ventilation bag. Both rescuers should observe the victim's chest to ensure chest rise.

Gastric Inflation

Gastric inflation may interfere with effective ventilation[59] and cause regurgitation, aspiration of stomach contents, and further ventilatory compromise. The risk of gastric inflation can be decreased by

- Avoiding excessive peak inspiratory pressures by ventilating slowly and giving only enough tidal volume to just achieve visible chest rise.[45]
- Applying cricoid pressure in an unresponsive victim to reduce air entry into the stomach (Class IIa, LOE B).[60–62] This may require a third rescuer if cricoid pressure cannot be applied by the rescuer who is securing the bag to the face. Avoid excessive cricoid pressure so as not to obstruct the trachea (Class III, LOE B).[63]
- Passing a nasogastric or orogastric tube to relieve gastric inflation, especially if oxygenation and ventilation are compromised. Pass the tube after intubation because a gastric tube interferes with gastroesophageal sphincter function, allowing regurgitation during intubation. If a gastrostomy tube is present, vent it during bag-mask ventilation to allow gastric decompression.

Ventilation With an Endotracheal Tube

Endotracheal intubation in infants and children requires special training because the pediatric airway anatomy differs from that of the adult. The likelihood of successful endotracheal tube placement with minimal complications is related to the length of training, supervised experience in the operating room and in the field,[64,65] adequate ongoing experience,[66] and use of rapid sequence intubation (RSI).[67,68]

Rapid Sequence Intubation (RSI)

To facilitate emergency intubation and reduce the incidence of complications, skilled, experienced providers may use sedatives, neuromuscular blocking agents, and other medications to rapidly sedate and neuromuscularly block the pediatric patient.[69]

Use RSI only if you are trained, and have experience using these medications and are proficient in the evaluation and management of the pediatric airway. If you use RSI you must have a secondary plan to manage the airway in the event that you cannot achieve intubation.

Actual body weight, rather than ideal body weight, should be used for some non-resuscitation medications (eg, succinylcholine).[70–85]

Cricoid Pressure During Intubation

There is insufficient evidence to recommend routine cricoid pressure application to prevent aspiration during endotracheal intubation in children. Do not continue cricoid pressure if it interferes with ventilation or the speed or ease of intubation (Class III, LOE C).[86,87]

Cuffed Versus Uncuffed Endotracheal Tubes

Both cuffed and uncuffed endotracheal tubes are acceptable for intubating infants and children (Class IIa, LOE C). In the operating room, cuffed endotracheal tubes are associated with a higher likelihood of correct selection of tube size, thus achieving a lower reintubation rate with no increased risk of perioperative complications.[88–90] In intensive care settings the risk of complications in infants and in children is no greater with cuffed tubes than with noncuffed tubes.[91–93] Cuffed endotracheal tubes may decrease the risk of aspiration.[94] If cuffed endotracheal tubes are used, cuff inflating pressure should be monitored and limited according to manufacturer's instruction (usually less than 20 to 25 cm H_2O).

In certain circumstances (eg, poor lung compliance, high airway resistance, or a large glottic air leak) a cuffed endotracheal tube may be preferable to an uncuffed tube, provided that attention is paid to endotracheal tube size, position, and cuff inflation pressure (Class IIa, LOE B).[88,91,92]

Endotracheal Tube Size

Length-based resuscitation tapes are helpful and more accurate than age-based formula estimates of endotracheal tube size for children up to approximately 35 kg,[77,95,96] even for children with short stature.[97]

In preparation for intubation with either a cuffed or an uncuffed endotracheal tube, confirm that tubes with an internal diameter (ID) 0.5 mm smaller and 0.5 mm larger than the estimated size are available. During intubation, if the endotracheal tube meets resistance, place a tube 0.5 mm smaller instead. Following intubation, if there is a large glottic air leak that interferes with oxygenation or ventilation, consider replacing the tube with one that is 0.5 mm larger, or place a cuffed tube of the same size if an uncuffed tube was used originally. Note that replacement of a functional endotracheal tube is associated with risk; the procedure should be undertaken in an appropriate setting by experienced personnel.

If an uncuffed endotracheal tube is used for emergency intubation, it is reasonable to select a 3.5-mm ID tube for infants up to one year of age and a 4.0-mm ID tube for patients between 1 and 2 years of age. After age 2, uncuffed endotracheal tube size can be estimated by the following formula:

$$\text{Uncuffed endotracheal tube ID (mm)} = 4 + (\text{age}/4)$$

If a cuffed tube is used for emergency intubation of an infant less than 1 year of age, it is reasonable to select a 3.0 mm ID tube. For children between 1 and 2 years of age, it is reasonable to use a cuffed endotracheal tube with an internal diameter of 3.5 mm (Class IIa, LOE B).[89,98–100] After age 2 it is reasonable to estimate tube size with the following formula (Class IIa, LOE B:[89,98–101]):

$$\text{Cuffed endotracheal tube ID (mm)} = 3.5 + (\text{age}/4)$$

Verification of Endotracheal Tube Placement

There is a risk of endotracheal tube misplacement (ie, in the esophagus, the pharynx above the vocal cords, or a mainstem bronchus) and an ongoing risk of displacement or obstruction,[45,102] especially during patient transport.[103] Since no single confirmation technique, including clinical signs[104] or

the presence of water vapor in the tube,[105] is completely reliable, use both clinical assessment and confirmatory devices to verify proper tube placement immediately after intubation, again after securing the endotracheal tube, during transport, and each time the patient is moved (eg, from gurney to bed) (Class I, LOE B).

The following are methods for confirming correct position:

- Look for bilateral chest movement and listen for equal breath sounds over both lung fields, especially over the axillae.
- Listen for gastric insufflation sounds over the stomach. They should *not* be present if the tube is in the trachea.[104]
- Check for exhaled CO_2 (see "Exhaled or End-Tidal CO_2 Monitoring," below).
- If there is a perfusing rhythm, check oxyhemoglobin saturation with a pulse oximeter. Remember that following hyperoxygenation, the oxyhemoglobin saturation detected by pulse oximetry may not decline for as long as 3 minutes even without effective ventilation.[106,107]
- If you are still uncertain, perform direct laryngoscopy and visualize the endotracheal tube to confirm that it lies between the vocal cords.
- In hospital settings, perform a chest x-ray to verify that the tube is not in a bronchus and to identify proper position in the midtrachea.

After intubation, secure the tube; there is insufficient evidence to recommend any single method. After securing the tube, maintain the patient's head in a neutral position; neck flexion may push the tube farther into the airway, and extension may pull the tube out of the airway.[108,109]

If an intubated patient's condition deteriorates, consider the following possibilities (mnemonic DOPE):

- **D**isplacement of the tube
- **O**bstruction of the tube
- **P**neumothorax
- **E**quipment failure

Exhaled or End-Tidal CO_2 Monitoring

When available, exhaled CO_2 detection (capnography or colorimetry) is recommended as confirmation of tracheal tube position for neonates, infants, and children with a perfusing cardiac rhythm in all settings (eg, prehospital, emergency department [ED], ICU, ward, operating room) (Class I, LOE C)[110–114] and during intrahospital or interhospital transport (Class IIb, LOE C).[115,116] Remember that a color change or the presence of a capnography waveform confirms tube position in the airway but does not rule out right mainstem bronchus intubation. During cardiac arrest, if exhaled CO_2 is not detected, confirm tube position with direct laryngoscopy (Class IIa, LOE C)[110,117–120] because the absence of CO_2 may reflect very low pulmonary blood flow rather than tube misplacement.

Confirmation of endotracheal tube position by colorimetric end-tidal CO_2 detector may be altered by the following:

- If the detector is contaminated with gastric contents or acidic drugs (eg, endotracheally administered epinephrine), a consistent color rather than a breath-to-breath color change may be seen.
- An intravenous (IV) bolus of epinephrine[121] may transiently reduce pulmonary blood flow and exhaled CO_2 below the limits of detection.[120]
- Severe airway obstruction (eg, status asthmaticus) and pulmonary edema may impair CO_2 elimination below the limits of detection.[120,122–124]
- A large glottic air leak may reduce exhaled tidal volume through the tube and dilute CO_2 concentration.

Esophageal Detector Device (EDD)

If capnography is not available, an esophageal detector device (EDD) may be considered to confirm endotracheal tube placement in children weighing >20 kg with a perfusing rhythm (Class IIb, LOE B),[125,126] but the data are insufficient to make a recommendation for or against its use in children during cardiac arrest.

Transtracheal Catheter Oxygenation and Ventilation

Transtracheal catheter oxygenation and ventilation may be considered for patients with severe airway obstruction above the level of the cricoid cartilage if standard methods to manage the airway are unsuccessful. Note that transtracheal ventilation primarily supports oxygenation as tidal volumes are usually too small to effectively remove carbon dioxide. This technique is intended for temporary use while a more effective airway is obtained. Attempt this procedure only after proper training and with appropriate equipment (Class IIb, LOE C).[127]

Suction Devices

A properly sized suction device with an adjustable suction regulator should be available. Do not insert the suction catheter beyond the end of the endotracheal tube to avoid injuring the mucosa. Use a maximum suction force of -80 to -120 mm Hg for suctioning the airway via an endotracheal tube. Higher suction pressures applied through large-bore noncollapsible suction tubing and semirigid pharyngeal tips are used to suction the mouth and pharynx.

CPR Guidelines for Newborns With Cardiac Arrest of Cardiac Origin

Recommendations for infants differ from those for the newly born (ie, in the delivery room and during the first hours after birth) and newborns (during their initial hospitalization and in the NICU). The compression-to-ventilation ratio differs (newly born and newborns – 3:1; infant two rescuer - 15:2) and how to provide ventilations in the presence of an advanced airway differs (newly born and newborns – pause after 3 compressions; infants – no pauses for ventilations). This presents a dilemma for healthcare providers who may also care for newborns outside the NICU. Because there are no definitive scientific data to help resolve this dilemma, for ease of training we recommend that newborns (intubated or not) who require CPR in the newborn nursery or NICU receive CPR using the same technique as for the newly born in the delivery room (ie, 3:1 compression-to-ventilation ratio

with a pause for ventilation). Newborns who require CPR in other settings (eg, prehospital, ED, pediatric intensive care unit [PICU], etc.), should receive CPR according to infant guidelines: 2 rescuers provide continuous chest compressions with asynchronous ventilations if an advanced airway is in place and a 15:2 ventilation-to-compression ratio if no advanced airway is in place (Class IIb, LOE C). It is reasonable to resuscitate newborns with a primary cardiac etiology of arrest, regardless of location, according to infant guidelines, with emphasis on chest compressions (Class IIa, LOE C). For further information, please refer to Part 13, "Pediatric Basic Life Support," and Part 15, "Neonatal Resuscitation."

Extracorporeal Life Support (ECLS)

Extracorporeal life support (ECLS) is a modified form of cardiopulmonary bypass used to provide prolonged delivery of oxygen to tissues. Consider early activation of ECLS for a cardiac arrest that occurs in a highly supervised environment, such as an ICU, with the clinical protocols in place and the expertise and equipment available to initiate it rapidly. ECLS should be considered only for children in cardiac arrest refractory to standard resuscitation attempts, with a potentially reversible cause of arrest (Class IIa, LOE C).[128–154] When ECLS is employed during cardiac arrest, outcome for children with underlying cardiac disease is better than the outcome for children with noncardiac disease. With underlying cardiac disease, long-term survival when ECLS is initiated in a critical-care setting has been reported even after >50 minutes of standard CPR.[128,129,139,147]

Monitoring

Electrocardiography

Monitor cardiac rhythm as soon as possible so both normal and abnormal cardiac rhythms are identified and followed. Continuous monitoring is helpful in tracking responses to treatment and changes in clinical condition.

Echocardiography

There is insufficient evidence for or against the routine use of echocardiography in pediatric cardiac arrest. When appropriately trained personnel are available, echocardiography may be considered to identify patients with potentially treatable causes of the arrest, particularly pericardial tamponade and inadequate ventricular filling (Class IIb, LOE C).[155–162] Minimize interruption of CPR while performing echocardiography.

End-Tidal CO_2 (P_{ETCO_2})

Continuous capnography or capnometry monitoring, if available, may be beneficial during CPR, to help guide therapy, especially the effectiveness of chest compressions (Class IIa, LOE C). Animal and adult studies show a strong correlation between P_{ETCO_2} and interventions that increase cardiac output during CPR or shock.[53,163–169] If the P_{ETCO_2} is consistently <10 to 15 mm Hg, focus efforts on improving chest compressions and make sure that the victim does not receive excessive ventilation. An abrupt and sustained rise in P_{ETCO_2} in adults[170,171] and animals[110] is observed just prior to clinical identification of ROSC, so use of P_{ETCO_2} may spare the rescuer from interrupting chest compressions for a pulse check. P_{ETCO_2} must be interpreted with caution for 1 to 2 minutes after administration of epinephrine or other vasoconstrictive medications because these medications may decrease the end-tidal CO_2 level by reducing pulmonary blood flow.

Vascular Access

Vascular access is essential for administering medications and drawing blood samples. Obtaining peripheral venous access can be challenging in infants and children during an emergency; intraosseous (IO) access can be quickly established with minimal complications by providers with varied levels of training.[172–179] Limit the time spent attempting to establish peripheral venous access in a critically ill or injured child.[180]

Intraosseous (IO) Access

IO access is a rapid, safe, effective, and acceptable route for vascular access in children,[172–179,181] and it is useful as the initial vascular access in cases of cardiac arrest (Class I, LOE C). All intravenous medications can be administered intraosseously, including epinephrine, adenosine, fluids, blood products,[182,183] and catecholamines.[184] Onset of action and drug levels for most drugs are comparable to venous administration.[185] IO access can be used to obtain blood samples for analysis including for type and cross match and blood gases during CPR,[186] but acid-base analysis is inaccurate after sodium bicarbonate administration via the IO cannula.[187] Use manual pressure or an infusion pump to administer viscous drugs or rapid fluid boluses;[188,189] follow each medication with a saline flush to promote entry into the central circulation.

Venous Access

Peripheral IV access is acceptable during resuscitation if it can be placed rapidly, but placement may be difficult in a critically ill child. Although a central venous catheter can provide more secure long-term access, its placement requires training and experience, and the procedure can be time-consuming. Therefore central venous access is not recommended as the initial route of vascular access during an emergency. If both central and peripheral accesses are available, administer medications into the central circulation since some medications (eg, adenosine) are more effective when administered closer to the heart, and others (eg, calcium, amiodarone, procainamide, sympathomimetics) may be irritating when infused into a peripheral vein. The length of a central catheter can contribute to increased resistance, making it more difficult to push boluses of fluid rapidly through a multilumen central than a peripheral catheter.

Endotracheal Drug Administration

Vascular access (IO or IV) is the preferred method for drug delivery during CPR, but if it is not possible, lipid-soluble drugs, such as lidocaine, epinephrine, atropine, and naloxone (mnemonic "LEAN")[190,191] can be administered via an endotracheal tube.[192] However, the effects may not be uniform with tracheal as compared with intravenous administration. One study of children in cardiac arrest[193] demonstrated similar ROSC and survival rates regardless of the method of

Table 1. Medications for Pediatric Resuscitation

Medication	Dose	Remarks
Adenosine	0.1 mg/kg (maximum 6 mg) Second dose: 0.2 mg/kg (maximum 12 mg)	Monitor ECG Rapid IV/IO bolus with flush
Amiodarone	5 mg/kg IV/IO; may repeat twice up to 15 mg/kg Maximum single dose 300 mg	Monitor ECG and blood pressure; adjust administration rate to urgency (IV push during cardiac arrest, more slowly–over 20–60 minutes with perfusing rhythm). Expert consultation strongly recommended prior to use when patient has a perfusing rhythm Use caution when administering with other drugs that prolong QT (obtain expert consultation)
Atropine	0.02 mg/kg IV/IO 0.04–0.06 mg/kg ET* Repeat once if needed Minimum dose: 0.1 mg Maximum single dose: 0.5 mg	Higher doses may be used with organophosphate poisoning
Calcium Chloride (10%)	20 mg/kg IV/IO (0.2 mL/kg) Maximum single dose 2 g	Administer slowly
Epinephrine	0.01 mg/kg (0.1 mL/kg 1:10,000) IV/IO 0.1 mg/kg (0.1 mL/kg 1:1000) ET* Maximum dose 1 mg IV/IO; 2.5 mg ET	May repeat every 3–5 minutes
Glucose	0.5–1 g/kg IV/IO	Newborn: 5–10 mL/kg $D_{10}W$ Infants and Children: 2–4 mL/kg $D_{25}W$ Adolescents: 1–2 mL/kg $D_{50}W$
Lidocaine	Bolus: 1 mg/kg IV/IO Infusion: 20–50 mcg/kg/minute	
Magnesium Sulfate	25–50 mg/kg IV/IO over 10–20 minutes, faster in torsades de pointes Maximum dose 2 g	
Naloxone	Full Reversal: <5 y or ≤20 kg: 0.1 mg/kg IV/IO/ET* ≥5y or >20 kg: 2 mg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1–5 mcg/kg titrate to effect)
Procainamide	15 mg/kg IV/IO Adult Dose: 20 mg/min IV infusion to total maximum dose of 17 mg/kg	Monitor ECG and blood pressure; Give slowly–over 30–60 minutes. Use caution when administering with other drugs that prolong QT (obtain expert consultation)
Sodium bicarbonate	1 mEq/kg per dose IV/IO slowly	After adequate ventilation

IV indicates intravenous; IO, intraosseous; and ET, via endotracheal tube.
*Flush with 5 mL of normal saline and follow with 5 ventilations.

drug delivery, while three studies of adults in cardiac arrest[194–196] demonstrated reduced ROSC and survival to hospital discharge with tracheal administration of epinephrine compared to vascular delivery. If CPR is in progress, stop chest compressions briefly, administer the medications, and follow with a flush of at least 5 mL of normal saline and 5 consecutive positive-pressure ventilations.[197] Optimal endotracheal doses of medications are unknown; in general expert consensus recommends doubling or tripling the dose of lidocaine, atropine or naloxone given via the ETT. For epinephrine, a dose ten times the intravenous dose (0.1 mg/kg or 0.1 mL/kg of 1:1000 concentration) is recommended (see Table 1).

The effectiveness of endotracheal epinephrine during cardiac arrest is controversial. Some studies showed it to be as effective as vascular administration[193,198,199] while other studies have not found it to be as effective.[194–196,200] Animal studies[201–206] suggested that a higher dose of epinephrine is required for endotracheal than for intravascular administration because the lower epinephrine concentrations achieved when the drug is delivered by the endotracheal route may produce predominant transient peripheral β_2-adrenergic vasodilating effects. These effects can be detrimental, and cause hypotension, lower coronary artery perfusion pressure and flow, and a reduced potential for ROSC.

Non-lipid-soluble drugs (eg, sodium bicarbonate and calcium) may injure the airway; they should not be administered via the endotracheal route.

Emergency Fluids and Medications

Estimating Weight

In the out-of-hospital setting, a child's weight is often unknown, and even experienced personnel may not be able to estimate it accurately.[74] Tapes with precalculated doses printed at various patient lengths have been clinically validated[74,77,95] and are more accurate than age-based or observer (parent or provider) estimate-based methods in the prediction of body weight.[70–77] Body habitus may also be an important consideration.[70,72,78,79]

Medication Dose Calculation

To calculate the dose of resuscitation medications, use the child's weight if it is known. If the child's weight is unknown, it is reasonable to use a body length tape with precalculated doses (Class IIa, LOE C).[70–77]

It is unclear if an adjustment in the calculation of resuscitation medications is needed in obese children. Use of the actual body weight in calculation of drug doses in obese patients may result in potentially toxic doses. Length-based tapes estimate the 50th percentile weight for length (ie, ideal body weight), which may, theoretically, result in inadequate doses of some medications in obese patients. Despite these theoretical considerations, there are no data regarding the safety or efficacy of adjusting the doses of resuscitation medications in obese patients. Therefore, regardless of the patient's habitus, use the actual body weight for calculating initial resuscitation drug doses or use a body length tape with precalculated doses (Class IIb, LOE C).

For subsequent doses of resuscitation drugs in both nonobese and obese patients, expert providers may consider adjusting doses to achieve the desired therapeutic effect. In general, the dose administered to a child should not exceed the standard dose recommended for adult patients.

Medications (See Table 1)

Adenosine

Adenosine causes a temporary atrioventricular (AV) nodal conduction block and interrupts reentry circuits that involve the AV node. The drug has a wide safety margin because of its short half-life. Adenosine should be given only IV or IO, followed by a rapid saline flush to promote drug delivery to the central circulation. If adenosine is given IV, it should be administered as close to the heart as possible. (See also "Arrhythmia.")

Amiodarone

Amiodarone slows AV conduction, prolongs the AV refractory period and QT interval, and slows ventricular conduction (widens the QRS). Expert consultation is strongly recommended prior to administration of amiodarone to a pediatric patient with a perfusing rhythm. (See also "Arrhythmia.")

Precautions

Monitor blood pressure and electrocardiograph (ECG) during intravenous administration of amiodarone. If the patient has a perfusing rhythm, administer the drug as slowly (over 20 to 60 minutes) as the patient's clinical condition allows; if the patient is in VF/pulseless VT, give the drug as a rapid bolus. Amiodarone causes hypotension through its vasodilatory property, and the severity is related to the infusion rate; hypotension is less common with the aqueous form of amiodarone.[207] Decrease the infusion rate if there is prolongation of the QT interval or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Other potential complications of amiodarone include bradycardia and torsades de pointes ventricular tachycardia. Amiodarone should not be administered together with another drug that causes QT prolongation, such as procainamide, without expert consultation.

Atropine

Atropine sulfate is a parasympatholytic drug that accelerates sinus or atrial pacemakers and increases the speed of AV conduction.

Precautions

Small doses of atropine (<0.1 mg) may produce paradoxical bradycardia because of its central effect.[208] Larger than recommended doses may be required in special circumstances such as organophosphate poisoning[209] or exposure to nerve gas agents.

Calcium

Calcium administration is not recommended for pediatric cardiopulmonary arrest in the absence of documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia (Class III, LOE B). Routine calcium administration in cardiac arrest provides no benefit[210–221] and may be harmful.[210–212]

If calcium administration is indicated during cardiac arrest, either calcium chloride or calcium gluconate may be considered. Hepatic dysfunction does not appear to alter the ability of calcium gluconate to raise serum calcium levels.[222] In critically ill children, calcium chloride may be preferred because it results in a greater increase in ionized calcium during the treatment of hypocalcemia.[222A] In the nonarrest setting, if the only venous access is peripheral, calcium gluconate is recommended because it has a lower osmolality than calcium chloride and is therefore less irritating to the vein.

Epinephrine

The α-adrenergic-mediated vasoconstriction of epinephrine increases aortic diastolic pressure and thus coronary perfusion pressure, a critical determinant of successful resuscitation from cardiac arrest.[223,224] At low doses, the β-adrenergic effects may predominate, leading to decreased systemic vascular resistance; in the doses used during cardiac arrest, the vasoconstrictive α-effects predominate.

Precautions

- Do not administer catecholamines and sodium bicarbonate simultaneously through an IV catheter or tubing because alkaline solutions such as the bicarbonate inactivate the catecholamines.
- In patients with a perfusing rhythm, epinephrine causes tachycardia; it may also cause ventricular ectopy, tachyarrhythmias, vasoconstriction, and hypertension.

Glucose

Because infants have a relatively high glucose requirement and low glycogen stores, they may develop hypoglycemia when energy requirements rise.[225] Check blood glucose concentration during the resuscitation and treat hypoglycemia promptly (Class I, LOE C).[226]

Lidocaine

Lidocaine decreases automaticity and suppresses ventricular arrhythmias,[227] but is not as effective as amiodarone for improving ROSC or survival to hospital admission among

adult patients with VF refractory to shocks and epinephrine.[228] Neither lidocaine nor amiodarone has been shown to improve survival to hospital discharge.

Precautions

Lidocaine toxicity includes myocardial and circulatory depression, drowsiness, disorientation, muscle twitching, and seizures, especially in patients with poor cardiac output and hepatic or renal failure.[229,230]

Magnesium

Magnesium is indicated for the treatment of documented hypomagnesemia or for torsades de pointes (polymorphic VT associated with long QT interval). There is insufficient evidence to recommend for or against the routine administration of magnesium during cardiac arrest.[231–233]

Precautions

Magnesium produces vasodilation and may cause hypotension if administered rapidly.

Procainamide

Procainamide prolongs the refractory period of the atria and ventricles and depresses conduction velocity.

Precautions

There is limited clinical data on using procainamide in infants and children.[234–236] Infuse procainamide very slowly (over 30 to 60 minutes) while monitoring the ECG and blood pressure. Decrease the infusion rate if there is prolongation of the QT interval, or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Do not administer together with another drug causing QT prolongation, such as amiodarone, without expert consultation. Prior to using procainamide for a hemodynamically stable patient, expert consultation is strongly recommended.

Sodium Bicarbonate

Routine administration of sodium bicarbonate is not recommended in cardiac arrest (Class III, LOE B).[212,237,238] Sodium bicarbonate may be administered for treatment of some toxidromes (see "Toxicological Emergencies," below) or special resuscitation situations such as hyperkalemic cardiac arrest.

Precautions

During cardiac arrest or severe shock, arterial blood gas analysis may not accurately reflect tissue and venous acidosis.[239,240] Excessive sodium bicarbonate may impair tissue oxygen delivery;[241] cause hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality;[242,243] decrease the VF threshold;[244] and impair cardiac function.

Vasopressin

There is insufficient evidence to make a recommendation for or against the routine use of vasopressin during cardiac arrest. Pediatric[245–247] and adult[248,249] case series/reports suggested that vasopressin[245] or its long-acting analog, terlipressin,[246,247] may be effective in refractory cardiac arrest when standard therapy fails. A large pediatric NRCPR case series, however, suggested that vasopressin is associated with lower ROSC, and a trend toward lower 24-hour and discharge survival.[250] A preponderance of controlled trials in adults do not demonstrate a benefit.[251–256]

Pulseless Arrest

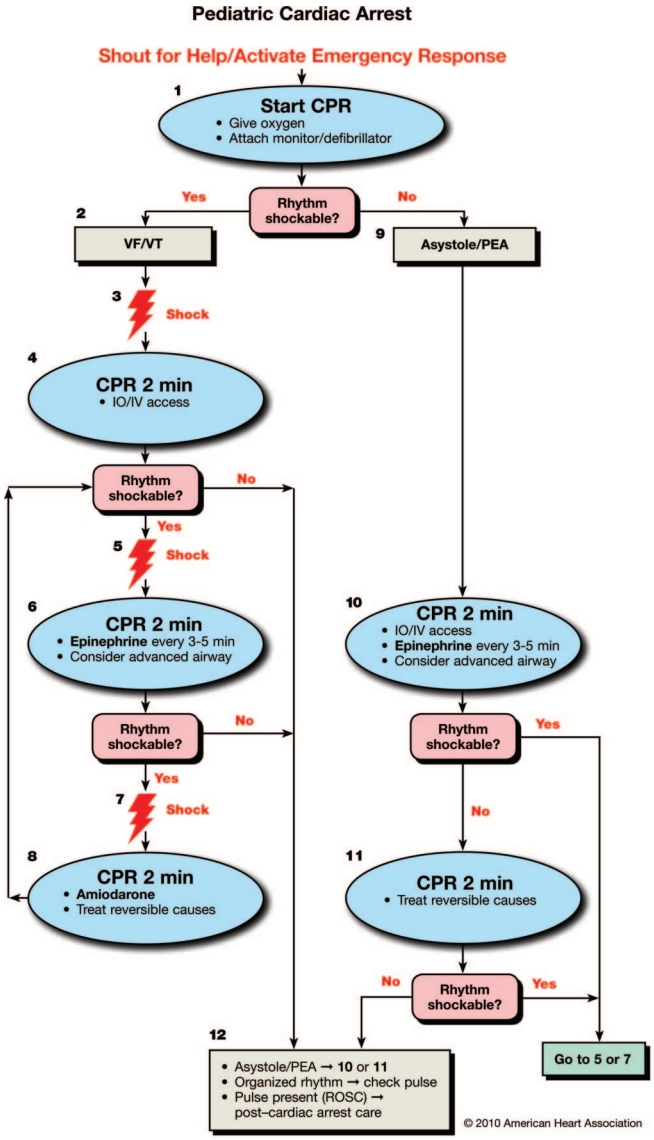
In the text below, box numbers identify the corresponding step in the algorithm (Figure 1).

- (Step 1) As soon as the child is found to be unresponsive with no breathing, call for help, send for a defibrillator (manual or AED), and start CPR (with supplementary oxygen if available). Attach ECG monitor or AED pads as soon as available. Throughout resuscitation, emphasis should be placed on provision of high-quality CPR (providing chest compressions of adequate rate and depth, allowing complete chest recoil after each compression, minimizing interruptions in compressions and avoiding excessive ventilation).
- While CPR is being given, determine the child's cardiac rhythm from the ECG or, if you are using an AED, the device will tell you whether the rhythm is "shockable" (eg, VF or rapid VT) or "not shockable" (eg, asystole or PEA). It may be necessary to temporarily interrupt chest compressions to determine the child's rhythm. Asystole and bradycardia with a wide QRS are most common in asphyxial arrest.[1] VF and PEA are less common[13] but VF is more likely to be present in older children with sudden witnessed arrest.

"Nonshockable Rhythm": Asystole/PEA (Step 9)

PEA is an organized electric activity—most commonly slow, wide QRS complexes—without palpable pulses. Less frequently there is a sudden impairment of cardiac output with an initially normal rhythm but without pulses and with poor perfusion. This subcategory, formerly known as electromechanical dissociation (EMD), may be more reversible than asystole. For asystole and PEA:

- (Step 10) Continue CPR with as few interruptions in chest compressions as possible. A second rescuer obtains vascular access and delivers epinephrine, 0.01 mg/kg (0.1 mL/kg of 1:10 000 solution) maximum of 1 mg (10 mL), while CPR is continued. The same epinephrine dose is repeated every 3 to 5 minutes (Class I, LOE B). There is no survival benefit from high-dose epinephrine, and it may be harmful, particularly in asphyxia (Class III, LOE B).[257–261] High-dose epinephrine may be considered in exceptional circumstances, such as β-blocker overdose (Class IIb, LOE C).
- Once an advanced airway is in place, 1 rescuer should give continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The second rescuer delivers ventilations at a rate of 1 breath every 6 to 8 seconds (about 8 to 10 breaths per minute). Rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions. Check rhythm every 2 minutes with minimal interruptions in chest compressions. If the rhythm is "nonshockable" continue with cycles of CPR and epinephrine administration until there is evidence of ROSC or you decide to terminate the effort. If at any time the rhythm



Doses/Details

CPR Quality
- Push hard (≥1/3 of anterior-posterior diameter of chest) and fast (at least 100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 15:2 compression-ventilation ratio. If advanced airway, 8-10 breaths per minute with continuous chest compressions

Shock Energy for Defibrillation
First shock 2 J/kg, second shock 4 J/kg, subsequent shocks ≥4 J/kg, maximum 10 J/kg or adult dose.

Drug Therapy
- **Epinephrine IO/IV Dose:** 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration). Repeat every 3-5 minutes. If no IO/IV access, may give endotracheal dose: 0.1 mg/kg (0.1 mL/kg of 1:1000 concentration).
- **Amiodarone IO/IV Dose:** 5 mg/kg bolus during cardiac arrest. May repeat up to 2 times for refractory VF/pulseless VT.

Advanced Airway
- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place give 1 breath every 6-8 seconds (8-10 breaths per minute)

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes
- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypoglycemia
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Figure 1. PALS Pulseless Arrest Algorithm.

becomes "shockable," give a shock (Step 7) and immediately resume chest compressions for 2 minutes before rechecking the rhythm. Minimize time between chest compressions and shock delivery (ie, check rhythm and deliver shocks immediately after compressions rather than after rescue breaths, if possible) and between shock delivery and resumption of chest compressions.

- Search for and treat reversible causes.

"Shockable Rhythm": VF/Pulseless VT (Step 2)

Defibrillation is the definitive treatment for VF (Class I, LOE B) with an overall survival rate of 17% to 20%.[4,262,263] Survival is better in primary than in secondary VF.[6] In adults, the probability of survival declines by 7% to 10% for each minute of arrest without CPR and defibrillation.[264] Survival is better if early, high-quality CPR is provided with minimal interruptions. Outcome of shock delivery is best if rescuers minimize the time between last compression and shock delivery, so rescuers should be prepared to coordinate (brief) interruptions in chest compressions to deliver shocks, and should resume compressions immediately after shock delivery.

Defibrillators

Defibrillators are either manual or automated (AED), with monophasic or biphasic waveforms. For further information see Part 6, "Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing."

AEDs in institutions caring for children at risk for arrhythmias and cardiac arrest (eg, hospitals, EDs) must be capable of recognizing pediatric cardiac rhythms and should ideally have a method of adjusting the energy level for children.

The following should be considered when using a manual defibrillator:

Paddle Size

In general, manual defibrillators have two sizes of hand-held paddles: adult and infant. The infant paddles may slide over or be located under the adult paddles. Manual defibrillators can also be used with hands-free pads that are self adhesive.

Use the largest paddles or self-adhering electrodes[265–267] that will fit on the child's chest without touching (when possible, leave about 3 cm between the paddles or electrodes). Paddles and self-adhering pads appear to be equally effective.[268] Self-adhering pads should be pressed firmly on the chest so that the gel on the pad completely touches the child's chest. An appropriate paddle or self-adhesive pad size is

- "Adult" size (8 to 10 cm) for children >10 kg (> approximately 1 year)
- "Infant" size for infants <10 kg

Interface

The electrode–chest wall interface is part of the self-adhesive pad; in contrast, electrode gel must be applied liberally on manually applied paddles. Do not use saline-soaked pads, ultrasound gel, bare paddles, or alcohol pads.

Paddle Position

Follow package directions for placement of self-adhesive AED or monitor/defibrillator pads.

Place manual paddles over the right side of the upper chest and the apex of the heart (to the left of the nipple over the left lower ribs) so the heart is between the two paddles. Apply firm pressure. There is no advantage in an anterior-posterior position of the paddles.[268]

Energy Dose

The lowest energy dose for effective defibrillation and the upper limit for safe defibrillation in infants and children are not known; more data are needed. It has been observed that in children with VF, an initial monophasic dose of 2 J/kg is only effective in terminating ventricular fibrillation 18% to 50% of the time,[269,270] while similar doses of biphasic shocks are effective 48% of the time.[268] Children with out-of-hospital VF cardiac arrest often receive more than 2 J/kg,[271,272] and one in-hospital cardiac arrest study[268] showed that children received doses between 2.5 and 3.2 J/kg to achieve ROSC. Energy doses >4 J/kg (up to 9 J/kg) have effectively defibrillated children[272–274] and pediatric animals[275] with negligible adverse effects. Based on data from adult studies[276,277] and pediatric animal models,[278–280] biphasic shocks appear to be at least as effective as monophasic shocks and less harmful.

It is acceptable to use an initial dose of 2 to 4 J/kg (Class IIa, LOE C), but for ease of teaching an initial dose of 2 J/kg may be considered (Class IIb, LOE C). For refractory VF, it is reasonable to increase the dose to 4 J/kg (Class IIa, LOE C). Subsequent energy levels should be at least 4 J/kg, and higher energy levels may be considered, not to exceed 10 J/kg or the adult maximum dose (Class IIb, LOE C).

AEDs

Many AEDs can accurately detect VF in children of all ages.[271,281–283] They can differentiate "shockable" from "nonshockable" rhythms with a high degree of sensitivity and specificity.[281,282] It is recommended that systems and institutions that have AED programs and that care for children should use AEDs with a high specificity to recognize pediatric shockable rhythms and a pediatric attenuating system that can be used for infants and children up to approximately 25 kg (approximately 8 years of age).[274,284] If an AED with an attenuator is not available, use an AED with standard electrodes (Class IIa, LOE C).

In infants <1 year of age a manual defibrillator is preferred. If a manual defibrillator is not available, an AED with a dose attenuator may be used. An AED without a dose attenuator may be used if neither a manual defibrillator nor one with a dose attenuator is available (Class IIb, LOE C).

Integration of Defibrillation With Resuscitation Sequence

The following are important considerations:

- Provide CPR until the defibrillator is ready to deliver a shock; after shock delivery, resume CPR, beginning with chest compressions. Minimize interruptions of chest compressions. In adults with prolonged arrest[285,286] and in animal models,[287] defibrillation is more likely to be successful after a period of effective chest compressions. Ideally chest compressions should be interrupted only for ventilations (until an advanced airway is in place), rhythm check, and shock delivery. If a "shockable" rhythm is still present, continue chest compressions after a rhythm check (when possible) while the defibrillator is charging (so chest compressions are delivered until shock delivery).
- (Step 3) Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR, beginning with chest compressions. If 1 shock fails to eliminate VF, the incremental benefit of another immediate shock is low, and resumption of CPR is likely to confer a greater value than another shock. CPR may provide coronary perfusion, increasing the likelihood of defibrillation with a subsequent shock. It is important to minimize the time between chest compressions and shock delivery and between shock delivery and resumption of postshock compressions.
- (Step 4) Continue CPR for about 2 minutes. In in-hospital settings with continuous invasive monitoring, this sequence may be modified at the expert provider's discretion (see, also Part 8.2: "Management of Cardiac Arrest"). If sufficient rescuers are present, obtain vascular (IO or IV) access.
- After 2 minutes of CPR, check the rhythm; recharge the defibrillator to a higher dose (4 J/kg).
- (Step 5) If a "shockable" rhythm persists, give another shock (4 J/kg). If rhythm is "nonshockable," continue with the asystole/PEA algorithm (Steps 10 and 11).
- (Step 6) Immediately resume chest compressions. Continue CPR for approximately 2 minutes. During CPR give epinephrine 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration), maximum of 1 mg (Class I, LOE B) **every 3 to 5 minutes.** It is helpful if a third rescuer prepares the drug doses *before* the rhythm is checked so epinephrine can be administered as soon as possible. Epinephrine should be administered during chest compressions, but the timing of drug administration is less important than the need to minimize interruptions in chest compressions. Just prior to the rhythm check, the rescuer operating the defibrillator should prepare to recharge the defibrillator (4 J/kg or more

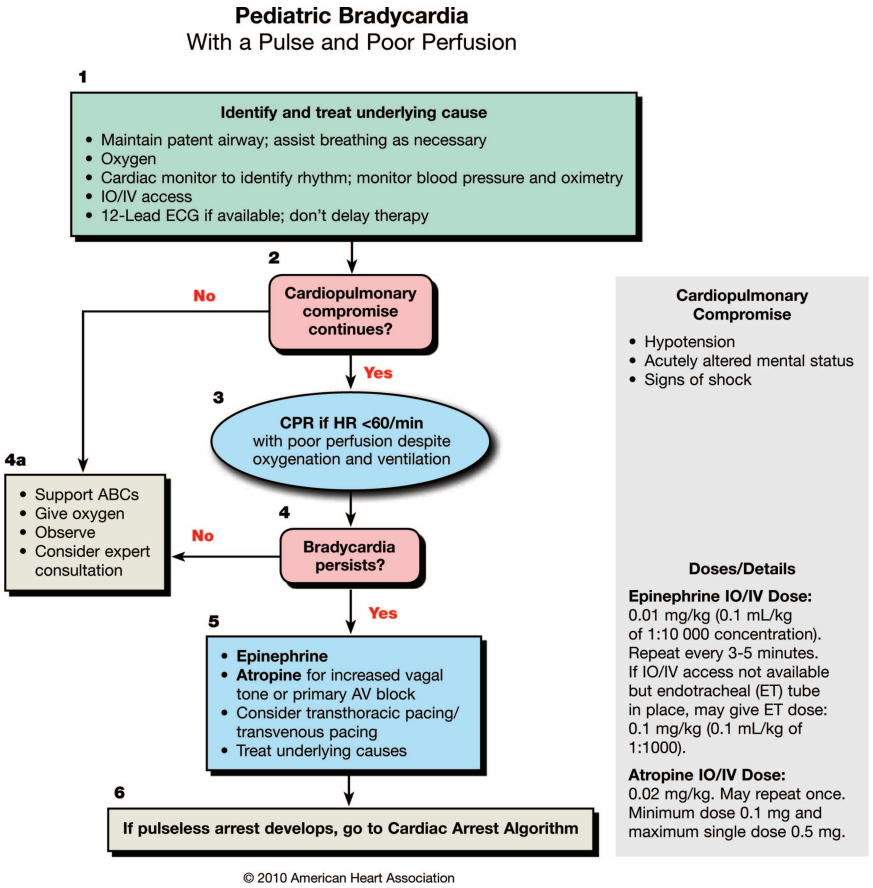


Figure 2. PALS Bradycardia Algorithm.

with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower).

- Check the rhythm
- (Step 7) If the rhythm is "shockable," deliver another shock (4 J/kg or more with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower) and immediately resume CPR (beginning with chest compressions).
- (Step 8) While continuing CPR, give amiodarone (Class IIb, LOE C)[228,288–290] or lidocaine if amiodarone is not available.
- If at any time the rhythm check shows a "nonshockable" rhythm, proceed to the "Pulseless Arrest" sequence (Steps 10 or 11).
- Once an advanced airway is in place, 2 rescuers no longer deliver cycles of CPR (ie, compressions interrupted by pauses for ventilation). Instead, the compressing rescuer gives continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The rescuer delivering ventilation provides about 1 breath every 6 to 8 seconds (8 to 10 breaths per minute). Two or more rescuers should rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions.
- If defibrillation successfully restores an organized rhythm (or there is other evidence of ROSC, such as an abrupt rise in P_{ETCO_2} or visible pulsations on an arterial waveform), check the child's pulse to determine if a perfusing rhythm is present. If a pulse is present, continue with postresuscitation care.
- If defibrillation is successful but VF recurs, resume CPR and give another bolus of amiodarone before trying to defibrillate with the previously successful shock dose.
- Search for and treat reversible causes

Torsades de Pointes

This polymorphic VT is associated with a long QT interval, which may be congenital or may result from toxicity with type IA antiarrhythmics (eg, procainamide, quinidine, and disopyramide) or type III antiarrhythmics (eg, sotalol and amiodarone), tricyclic antidepressants (see below), digitalis, or drug interactions.[291,292]

Treatment

Torsades de pointes VT typically deteriorates rapidly to VF or pulseless VT, so providers should initiate CPR and proceed with defibrillation when pulseless arrest develops (see above). Regardless of the cause, treat torsades de pointes with a rapid (over several minutes) IV infusion of magnesium sulfate (25 to 50 mg/kg; maximum single dose 2 g).

Bradycardia

Box numbers in the text below refer to the corresponding boxes in the PALS Bradycardia Algorithm (see Figure 2). This algorithm applies to the care of the infant or child with bradycardia and cardiorespiratory compromise, but a palpable pulse. If at any time the patient develops pulseless arrest, see the PALS Pulseless Arrest Algorithm.

Emergency treatment of bradycardia is indicated when the rhythm results in hemodynamic compromise.

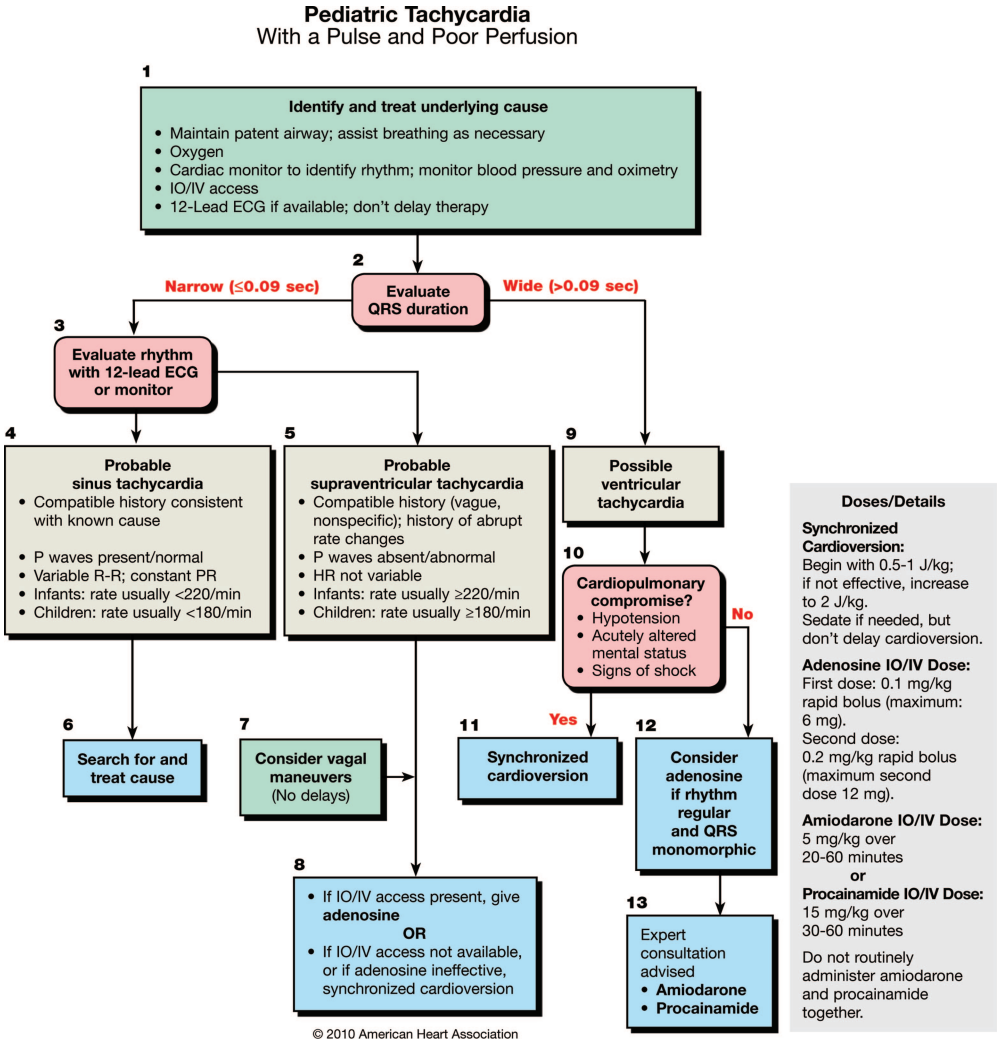


Figure 3. PALS Tachycardia Algorithm.

- (Box 1) Support a patent airway, breathing, and circulation as needed. Administer oxygen, attach an ECG monitor/defibrillator, and obtain vascular access.
- (Box 2) Reassess the patient to determine if bradycardia persists and is still causing cardiorespiratory symptoms despite adequate oxygenation and ventilation.
- (Box 4a) If pulses, perfusion, and respirations are adequate, no emergency treatment is necessary. Monitor and proceed with evaluation.
- (Box 3) If heart rate is <60 beats per minute with poor perfusion despite effective ventilation with oxygen, start CPR.
- (Box 4) After 2 minutes reevaluate the patient to determine if bradycardia and signs of hemodynamic compromise persist. Verify that the support is adequate (eg, check airway, oxygen source, and effectiveness of ventilation).
- (Box 5) Medications and pacing:
  - Continue to support airway, ventilation, oxygenation, and chest compressions (Class I, LOE B). If bradycardia persists or responds only transiently, give epinephrine IV (or IO) 0.01 mg/kg (0.1 mL/kg of 1:10,000 solution) or if IV/IO access not available, give endotracheally 0.1 mg/kg (0.1 mL/kg of 1:1,000 solution) (Class I, LOE B).
  - If bradycardia is due to increased vagal tone or primary AV conduction block (ie, not secondary to factors such as hypoxia), give IV/IO atropine 0.02 mg/kg or an endotracheal dose of 0.04 to 0.06 mg/kg (Class I, LOE C).
  - Emergency transcutaneous pacing may be lifesaving if the bradycardia is due to complete heart block or sinus node dysfunction unresponsive to ventilation, oxygenation, chest compressions, and medications, especially if it is associated with congenital or acquired heart disease (Class IIb, LOE C).[293] Pacing is not useful for asystole[293,294] or bradycardia due to postarrest hypoxic/ischemic myocardial insult or respiratory failure.

Tachycardia

The box numbers in the text below correspond to the numbered boxes in the Tachycardia Algorithm (see Figure 3).

- If there are signs of poor perfusion and pulses are not palpable, proceed with the PALS Pulseless Arrest Algorithm (see Figure 1).
- (Box 1) If pulses are palpable and the patient has adequate perfusion

–Assess and support airway, breathing, and circulation
–Provide oxygen.
–Attach monitor/defibrillator.
–Obtain vascular access.
–Evaluate 12-lead ECG and assess QRS duration (Box 2).

Narrow-Complex (≤0.09 Second) Tachycardia

Evaluation of a 12-lead ECG (Box 3) and the patient's clinical presentation and history (Boxes 4 and 5) should help differentiate sinus tachycardia from supraventricular tachycardia (SVT). If the rhythm is sinus tachycardia, search for and treat reversible causes.

Supraventricular Tachycardia (Box 5)

- Monitor rhythm during therapy to evaluate the effect of interventions. The choice of therapy is determined by the patient's degree of hemodynamic instability.
- Attempt vagal stimulation (Box 7) first, unless the patient is hemodynamically unstable or the procedure will unduly delay chemical or electric cardioversion (Class IIa, LOE C). In infants and young children, apply ice to the face without occluding the airway.[295,296]
- In older children, carotid sinus massage or Valsalva maneuvers are safe.[297–299]
- One method for performing a Valsalva maneuver is to have the child blow through a narrow straw.[298] Do not apply pressure to the eye because this can damage the retina.
- Pharmacologic cardioversion with adenosine (Box 8) is very effective with minimal and transient side effects.[300–304] If IV/IO access is readily available, adenosine is the drug of choice (Class I, LOE C). Side effects are usually transient.[300–304] Administer IV/IO adenosine 0.1 mg/kg using 2 syringes connected to a T-connector or stopcock; give adenosine rapidly with 1 syringe and immediately flush with ≥5 mL of normal saline with the other. An IV/IO dose of Verapamil, 0.1 to 0.3 mg/kg is also effective in terminating SVT in older children,[305,306] but it should not be used in infants without expert consultation (Class III, LOE C) because it may cause potential myocardial depression, hypotension, and cardiac arrest.[306,307]
- If the patient is hemodynamically unstable or if adenosine is ineffective, perform electric synchronized cardioversion (Box 8). Use sedation, if possible. Start with a dose of 0.5 to 1 J/kg. If unsuccessful, increase the dose to 2 J/kg (Class IIb, LOE C). If a second shock is unsuccessful or the tachycardia recurs quickly, consider amiodarone or procainamide before a third shock.
- Consider amiodarone 5 mg/kg IO/IV[308,309] or procainamide 15 mg/kg IO/IV[236] for a patient with SVT unresponsive to vagal maneuvers and adenosine and/or electric cardioversion; for hemodynamically stable patients, expert consultation is strongly recommended prior to administration (Class IIb, LOE C). Both amiodarone and procainamide must be infused slowly (amiodarone over 20 to 60 minutes and procainamide over 30 to 60 minutes), depending on the urgency, while the ECG and blood pressure are monitored. If there is no effect and there are no signs of toxicity, give additional doses (Table 1). Avoid the simultaneous use of amiodarone and procainamide without expert consultation.

Wide-Complex (>0.09 Second) Tachycardia (Box 9)

Wide-complex tachycardia often originates in the ventricles (ventricular tachycardia) but may be supraventricular in origin.[310]

Because all arrhythmia therapies have a potential for serious adverse effects, consultation with an expert in pediatric arrhythmias is strongly recommended before treating children who are hemodynamically stable.

The following are important considerations in treating wide-complex tachycardia in hemodynamically stable patients:

- Adenosine may be useful in differentiating SVT from VT and converting wide-complex tachycardia of supraventricular origin (Box 12). Adenosine should be considered only if the rhythm is regular and the QRS is monomorphic. Do not use adenosine in patients with known Wolff-Parkinson-White syndrome and wide-complex tachycardia.
- Consider electric cardioversion after sedation using a starting energy dose of 0.5 to 1 J/kg. If that fails, increase the dose to 2 J/kg (Class IIb, LOE C) (Box 11).
- Consider pharmacologic conversion with either intravenous amiodarone (5 mg/kg over 20 to 60 minutes) or procainamide (15 mg/kg given over 30 to 60 minutes) while monitoring ECG and blood pressure. Stop or slow the infusion if there is a decline in blood pressure or the QRS widens (Box 13). Expert consultation is strongly recommended prior to administration.

In hemodynamically unstable patients:

- Electric cardioversion is recommended using a starting energy dose of 0.5 to 1 J/kg. If that fails, increase the dose to 2 J/kg (Class 1, LOE C).

Special Resuscitation Situations

Septic Shock

- There appears to be no clinically important difference in survival of children who are treated for septic shock with colloid compared with those who are treated with isotonic crystalloid solutions.[311–314] Although colloid may be beneficial as part of a protocol-driven strategy,[315] it is reasonable to use isotonic crystalloid solution as the initial fluid for the treatment of septic shock (Class IIa, LOE C).
- Monitoring the central venous (superior vena cava) oxygen saturation (ScvO2) may be useful to titrate therapy in infants and children with septic shock. Protocol-driven or "goal-directed" therapy, with a target ScvO2 ≥70% appears to improve patient survival in severe sepsis (Class IIb, LOE B).[316–318]
- Early assisted ventilation may be considered as part of a protocol-driven strategy for septic shock (Class IIb, LOE C).[315,319]
- Etomidate has been shown to facilitate endotracheal intubation in infants and children with minimal hemodynamic effect,[320–322] but do not use it routinely in pediatric patients with evidence of septic shock (Class III, LOE B). Adrenal

suppression is seen after administration of etomidate in children[323] and adults.[324] In children and adults with septic shock, etomidate administration is associated with a higher mortality rate.[323,325]

Hypovolemic Shock

- Use an isotonic crystalloid solution (eg, lactated Ringer's solution or normal saline)[326,327] as the initial fluid for the treatment of shock (Class I, LOE A). There is no added benefit in using colloid (eg, albumin) during the early phase of resuscitation.[328,329]
- Treat signs of shock with a bolus of 20 mL/kg of isotonic crystalloid even if blood pressure is normal (Class IIb, LOE C). Crystalloids may have an associated survival benefit over colloid for children with shock secondary to general trauma, traumatic brain injury, and burns.[329–332] There is no evidence to support the use of a specific isotonic crystalloid. Give additional boluses (20 mL/kg) if systemic perfusion fails to improve. There are insufficient data to make a recommendation for or against use of hypertonic saline for shock associated with head injuries or hypovolemia.[333,334]
- There is insufficient evidence in infants and children to make a recommendation about the best timing or extent of volume resuscitation for children with hemorrhagic shock following trauma.

Trauma

Some aspects of trauma resuscitation require emphasis because improperly performed resuscitation is a major cause of preventable pediatric deaths.[335]

Common errors in pediatric trauma resuscitation include failure to open and maintain the airway, failure to provide appropriate fluid resuscitation, and failure to recognize and treat internal bleeding. Involve a qualified surgeon early and, if possible, transport a child with multisystem trauma to a trauma center with pediatric expertise.

The following are special aspects of trauma resuscitation:

- When the mechanism of injury is compatible with cervical spinal injury, restrict motion of the cervical spine and avoid traction or movement of the head and neck. Open and maintain the airway with a jaw thrust, and do not tilt the head.
- If the airway cannot be opened with a jaw thrust, use a head tilt–chin lift because you must establish a patent airway. Because of the disproportionately large head of infants and young children, optimal positioning may require recessing the occiput[336] or elevating the torso to avoid undesirable backboard-induced cervical flexion.[336,337]
- Do not routinely hyperventilate even in case of head injury (Class III, LOE C).[338,339] Intentional brief hyperventilation may be used as a temporizing rescue therapy if there are signs of impending brain herniation (eg, sudden rise in measured intracranial pressure, dilation of one or both pupils with decreased response to light, bradycardia, and hypertension).
- Suspect thoracic injury in all thoraco-abdominal trauma, even in the absence of external injuries. Tension pneumothorax, hemothorax, or pulmonary contusion may impair oxygenation and ventilation.
- If the patient has maxillofacial trauma or if you suspect a basilar skull fracture, insert an orogastric rather than a nasogastric tube (Class IIa, LOE C).[340]
- In the very select circumstances of children with cardiac arrest from penetrating trauma with short transport times, consider performing resuscitative thoracotomy (Class IIb, LOE C).[341,342]
- Consider intra-abdominal hemorrhage, tension pneumothorax, pericardial tamponade, and spinal cord injury in infants and children, and intracranial hemorrhage in infants, as causes of shock.[343,344]

Single Ventricle

Standard prearrest and arrest resuscitation procedures should be followed for infants and children with single ventricle anatomy following Stage I palliation or in the infant or neonate with a univentricular heart and a shunt to augment pulmonary blood flow. Heparin may be considered for infants with a systemic-pulmonary artery shunt or right ventricular-pulmonary artery shunt. Following resuscitation from cardiac arrest, oxygen administration should be adjusted to balance systemic and pulmonary blood flow, targeting an oxyhemoglobin saturation (Spo_2) of approximately 80%. End-tidal CO_2 ($PETCO_2$) in the single-ventricle patient during cardiac arrest may not be a reliable indicator of CPR quality because pulmonary blood flow changes rapidly and does not necessarily reflect cardiac output during CPR.[345]

Neonates in a prearrest state due to elevated pulmonary-to-systemic flow ratio prior to Stage I repair might benefit from a $Paco_2$ of 50 to 60 mm Hg, which can be achieved during mechanical ventilation by reducing minute ventilation, increasing the inspired fraction of CO_2, or administering opioids with or without chemical paralysis (Class IIb, LOE B).[346,347] Neonates in a low cardiac output state following stage I repair may benefit from systemic vasodilators such as α-adrenergic antagonists (eg, phenoxybenzamine) to treat or ameliorate increased systemic vascular resistance, improve systemic oxygen delivery, and reduce the likelihood of cardiac arrest (Class IIa, LOE B).[348–350] Other drugs that reduce systemic vascular resistance (eg, milrinone or nipride)[351] may also be considered for patients with excessive Qp:Qs (Class IIa, LOE B).[352] Following Stage I repair, evaluation of oxygen delivery and extraction (eg, using central venous oxygen saturation [$ScvO_2$] and near-infrared spectroscopy) may help identify evolving changes in hemodynamics that may herald impending cardiac arrest.[353–355] During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with single ventricle anatomy who have undergone Stage I procedure (Class IIa, LOE B).[129,132,152,356,357]

Hypoventilation may improve oxygen delivery in patients in a prearrest state with Fontan or hemi-Fontan/bidirectional Glenn (BDG) physiology (Class IIa, LOE B).[358–361] Negative-pressure ventilation may improve cardiac output (Class IIa, LOE C).[362,363] During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with Fontan physiology (Class IIa, LOE C).[364] It is

unclear at this time whether patients with hemi-Fontan/BDG physiology in cardiac arrest might benefit from ECMO.

Pulmonary Hypertension

Standard PALS, including oxygenation and ventilation, should be provided to patients with pulmonary hypertension and a cardiopulmonary arrest. It may be beneficial to attempt to correct hypercarbia. Administration of a bolus of isotonic fluid may be useful to maintain preload to the systemic ventricle. If intravenous or inhaled therapy to decrease pulmonary hypertension has been interrupted, reinstitute it (Class IIa, LOE C). Consider administering inhaled nitric oxide (iNO) or aerosolized prostacyclin or analogue to reduce pulmonary vascular resistance (Class IIa, LOE C). If iNO is not available, consider giving an intravenous bolus of prostacyclin (Class IIa, LOE C).[365–367] ECMO may be beneficial if instituted early in the resuscitation (Class IIa, LOE C).[368]

Children With Special Healthcare Needs

Children with special healthcare needs[369] may require emergency care for chronic conditions (eg, obstruction of a tracheostomy), failure of support technology (eg, ventilator failure), progression of their underlying disease, or events unrelated to those special needs.[370]

For additional information about CPR see Part 13: "Pediatric Basic Life Support."

Ventilation With a Tracheostomy or Stoma

Parents, school nurses, and home healthcare providers should know how to assess patency of the airway, clear the airway, replace the tracheostomy tube, and perform CPR using the artificial airway in a child with a tracheostomy.

Parents and providers should be able to ventilate via a tracheostomy tube and verify effectiveness by assessing chest expansion. If, after suctioning, the chest does not expand with ventilation, remove the tracheostomy tube and replace it or insert a same-sized endotracheal tube, if available, into the tracheal stoma. If a clean tube is unavailable, perform mouth-to-stoma or mask-to-stoma ventilations. If the upper airway is patent, bag-mask ventilation via the nose and mouth may be effective if the tracheal stoma is manually occluded.

Toxicological Emergencies

Overdose with local anesthetics, cocaine, narcotics, tricyclic antidepressants, calcium channel blockers, and β-adrenergic blockers may require specific treatment modalities in addition to the usual resuscitative measures.

Local Anesthetic

Local anesthetics are used topically, intravenously, subcutaneously, and in epidural or other catheters for delivery of regional analgesia. The toxicity of local anesthetics is well recognized in children; they may cause changes in mental status, seizures, arrhythmias, or even cardiac arrest in settings of overdose or inadvertent vascular administration. Multiple case reports, including some pediatric reports, have described successful treatment of local anesthetic toxicity with intravenous lipid emulsion.[371]

Cocaine

Acute coronary syndrome, manifested by chest pain and cardiac rhythm disturbances (including VT and VF), is the most frequent cocaine-related reason for hospitalization in adults.[372,373] Cocaine also may prolong the action potential and QRS duration and impairs myocardial contractility.[374,375]

Treatment

- Hyperthermia, which may result from cocaine-induced hypermetabolism, is associated with an increase in toxicity;[376] therefore treat elevated temperature aggressively.
- For coronary vasospasm consider nitroglycerin (Class IIa, LOE C),[377,378] a benzodiazepine, and phentolamine (an α-adrenergic antagonist) (Class IIb, LOE C).[379,380]
- *Do not* give β-adrenergic blockers (Class III, LOE C),[376]
- For ventricular arrhythmia, consider sodium bicarbonate (1 to 2 mEq/kg) administration (Class IIb, LOE C)[381,382] in addition to standard treatment.
- To prevent arrhythmias secondary to myocardial infarction, consider a lidocaine bolus followed by a lidocaine infusion (Class IIb, LOE C).

Tricyclic Antidepressants and Other Sodium Channel Blockers

Toxic doses cause cardiovascular abnormalities, including intraventricular conduction delays, heart block, bradycardia, prolongation of the QT interval, ventricular arrhythmias (including torsades de pointes, VT, and VF), hypotension, seizures,[375,383] and a depressed level of consciousness.

Treatment

- Give 1 to 2 mEq/kg intravenous boluses of sodium bicarbonate until arterial pH is >7.45; then provide an infusion of 150 mEq NaHCO3 per liter of D5W to maintain alkalosis. In cases of severe intoxication increase the pH to 7.50 to 7.55.[375,384] Do not administer Class IA (quinidine, procainamide), Class IC (flecainide, propafenone), or Class III (amiodarone and sotalol) antiarrhythmics, which may exacerbate cardiac toxicity (Class III, LOE C).[384]
- For hypotension, give boluses (10 mL/kg each) of normal saline. If hypotension persists, epinephrine and norepinephrine are more effective than dopamine in raising blood pressure.[385,386]
- Consider ECMO if high-dose vasopressors do not maintain blood pressure.[387,388]

Calcium Channel Blockers

Manifestations of toxicity include hypotension, ECG changes (prolongation of the QT interval, widening of the QRS, and right bundle branch block), arrhythmias (bradycardia, SVT, VT, torsades de pointes, and VF),[389] seizures, and altered mental status.

Treatment

- Treat mild hypotension with small boluses (5 to 10 mL/kg) of normal saline because myocardial depression may limit the amount of fluid the patient can tolerate.

- The effectiveness of calcium administration is variable (Class IIb, LOE C).[389–393] Infuse 20 mg/kg (0.2 mL/kg) of 10% calcium chloride intravenously over 5 to 10 minutes; if there is a beneficial effect, give an infusion of 20 to 50 mg/kg per hour. Monitor serum ionized calcium concentration to prevent hypercalcemia. It is preferable to administer calcium chloride via a central venous catheter; use caution when infusing into a peripheral IV because infiltration can cause severe tissue injury. If no central venous catheter is available, infuse calcium gluconate through a secure peripheral IV. For bradycardia and hypotension, consider vasopressors and inotropes such as norepinephrine or epinephrine (Class IIb, LOE C).[392]
- There are insufficient data to recommend for or against an infusion of insulin and glucose[394–397] or sodium bicarbonate.

Beta-Adrenergic Blockers

Toxic doses of β-adrenergic blockers cause bradycardia, heart block, and decreased cardiac contractility, and some (eg, propranolol and sotalol) may also prolong the QRS and the QT intervals.[397–400]

Treatment

- High-dose epinephrine infusion may be effective (Class IIb, LOE C).[400,401]
- Consider glucagon (Class IIb, LOE C).[397,400,402,403] In adolescents infuse 5 to 10 mg of glucagon over several minutes followed by an IV infusion of 1 to 5 mg/hour.
- Consider an infusion of glucose and insulin (Class IIb, LOE C).[394]
- There are insufficient data to make a recommendation for or against using calcium (Class IIb, LOE C).[390,404,405]
- Calcium may be considered if glucagon and catecholamines are ineffective (Class IIb, LOE C).

Opioids

Narcotics may cause hypoventilation, apnea, bradycardia, and hypotension in addition to depressed responsiveness.

Treatment

- Support of oxygenation and ventilation is the initial treatment for severe respiratory depression from any cause (Class I).
- Naloxone reverses the respiratory depression of narcotic overdose (Class I, LOE B),[406–410] but in persons with long-term addictions or cardiovascular disease, naloxone may markedly increase heart rate and blood pressure and cause acute pulmonary edema, cardiac arrhythmias (including asystole), and seizures. Ventilation before administration of naloxone appears to reduce these adverse effects.[411] Intramuscular administration of naloxone may lower the risk by slowing the onset of drug effect.

Postresuscitation Stabilization (Post Cardiac Arrest Care)

The goals of postresuscitation care are to preserve neurologic function, prevent secondary organ injury, diagnose and treat the cause of illness, and enable the patient to arrive at a pediatric tertiary-care facility in an optimal physiologic state. Frequent reassessment of the patient is necessary because cardiorespiratory status may deteriorate.

Respiratory System

- Data suggest that hyperoxemia (ie, a high PaO_2) enhances the oxidative injury observed following ischemia-reperfusion. Therefore, one goal of the postresuscitation phase is to reduce the risk of oxidative injury while maintaining adequate oxygen delivery. A practical way to achieve that goal is to reduce the FIO_2 to reduce the PaO_2 while ensuring adequate arterial oxygen content. Specifically, use the lowest inspired oxygen concentration that will maintain the arterial oxyhemoglobin saturation ≥94%. Provided appropriate equipment is available, once ROSC is achieved, adjust the FIO_2 to the minimum concentration needed to achieve transcutaneous or arterial oxygen saturation at least 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery. Since an arterial oxyhemoglobin saturation of 100% may correspond to a PaO_2 anywhere between ~80 and 500 mmHg, in general it is appropriate to wean the FIO_2 for a saturation of 100%, provided the oxyhemoglobin saturation can be maintained ≥94%.
- In addition to the usual clinical signs of adequate perfusion, laboratory parameters of adequate oxygen delivery over time include resolution of metabolic acidosis, reduced lactate concentration, and normalization of venous oxygen saturation.
- Assist ventilation if there is significant respiratory compromise (tachypnea, respiratory distress with agitation or decreased responsiveness, poor air exchange, cyanosis, hypoxemia). If the patient is already intubated, verify tube position, patency, and security. In the hospital setting, consider obtaining arterial blood gases 10 to 15 minutes after establishing the initial mechanical ventilator settings and make appropriate adjustments. Ideally, correlate blood gases with capnographic end-tidal CO_2 concentration ($PETCO_2$) to enable noninvasive monitoring of ventilation.
- Control pain and discomfort with analgesics (eg, fentanyl or morphine) and sedatives (eg, lorazepam or midazolam). Neuromuscular blocking agents (eg, vecuronium or pancuronium) with analgesia or sedation, or both, may improve oxygenation and ventilation in case of patient-ventilator dyssynchrony or severely compromised pulmonary function. Neuromuscular blockers, however, can mask seizures and impede neurologic examinations.
- Monitor exhaled CO_2 ($PETCO_2$), especially during transport and diagnostic procedures (Class IIa, LOE B).[116,412,413]
- Insert a gastric tube to relieve and help prevent gastric inflation.

Cardiovascular System

- Monitor heart rate and blood pressure. Repeat clinical evaluations at frequent intervals until the patient is stable. Consider monitoring urine output with an indwelling catheter. A 12-lead ECG may be helpful in establishing the cause of the cardiac arrest.
- Remove the IO access after alternative (preferably 2) secure venous catheters are placed. Monitor venous or arterial blood gas analysis and serum electrolytes, glucose,

Table 2. Medications to Maintain Cardiac Output and for Postresuscitation Stabilization

Medication	Dose Range	Comment
Inamrinone	0.75–1 mg/kg IV/IO over 5 minutes; may repeat × 2 then: 5–10 mcg/kg per minute	Inodilator
Dobutamine	2–20 mcg/kg per minute IV/IO	Inotrope; vasodilator
Dopamine	2–20 mcg/kg per minute IV/IO	Inotrope; chronotrope; renal and splanchnic vasodilator in low doses; pressor in high doses
Epinephrine	0.1–1 mcg/kg per minute IV/IO	Inotrope; chronotrope; vasodilator in low doses; pressor in higher doses
Milrinone	Loading dose: 50 mcg/kg IV/IO over 10–60 min then 0.25–0.75 mcg/kg per minute	Inodilator
Norepinephrine	0.1–2 mcg/kg per minute	Vasopressor
Sodium nitroprusside	Initial: 0.5–1 mcg/kg per minute; titrate to effect up to 8 mcg/kg per minute	Vasodilator Prepare only in D_5W

IV indicates intravenous; and IO, intraosseous.
Alternative formula for verifying dose during continuous infusion:
Infusion rate

$$(\text{mL/h}) = \frac{[\text{weight (kg)} \times \text{dose (mcg/kg per min)} \times 60\ (\text{min/hour})]}{\text{concentration(mcg/mL)}}.$$

and calcium concentrations. A chest x-ray should be performed to evaluate endotracheal tube position, heart size, and pulmonary status. Consider obtaining arterial lactate and central venous oxygen saturation to assess adequacy of tissue oxygen delivery.

Drugs Used to Maintain Cardiac Output (Table 2)

Myocardial dysfunction and vascular instability are common following resuscitation from cardiac arrest.[414–419] Systemic and pulmonary vascular resistances are often increased initially, except in some cases of septic shock.[420] The postarrest effects on the cardiovascular system may evolve over time, with an initial hyperdynamic state replaced by worsening cardiac function. Therefore in infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest, it is reasonable to administer vasoactive drugs titrated to improve myocardial function and organ perfusion.

There are no studies evaluating the benefit of specific vasoactive agents after ROSC in infants and children. In animal studies after resuscitation from cardiac arrest[418,419,421–424] and post–cardiac surgical experience in children[352] and adults,[425–428] hemodynamic improvement was associated with administration of selected vasoactive agents. Each drug and dose must be tailored to the patient because clinical response is variable. Infuse all vasoactive drugs into a secure IV line. The potential adverse effects of catecholamines include local ischemia and ulceration, tachycardia, atrial and ventricular tachyarrhythmias, hypertension, and metabolic changes (hyperglycemia, increased lactate concentration,[429] and hypokalemia).

Epinephrine

Low-dose infusions (<0.3 mcg/kg per minute) generally produce β-adrenergic actions (tachycardia, potent inotropy, and decreased systemic vascular resistance). Higher-dose infusions (>0.3 mcg/kg per minute) cause α-adrenergic vasoconstriction.[430,431] Because there is great interpatient variability in response,[432,433] titrate the drug to the desired effect. Epinephrine or norepinephrine may be preferable to dopamine in patients (especially infants) with marked circulatory instability and decompensated shock.[434]

Dopamine

Dopamine can produce direct dopaminergic effects and indirect β- and α-adrenergic effects through stimulation of norepinephrine release. Titrate dopamine to treat shock that is unresponsive to fluids and when systemic vascular resistance is low (Class IIb, LOE C).[420,435] Typically a dose of 2 to 20 mcg/kg per minute is used. Although low-dose dopamine infusion has been frequently recommended to maintain renal blood flow or improve renal function, data do not show benefit from such therapy.[436,437] At higher doses (>5 mcg/kg per minute), dopamine stimulates cardiac β-adrenergic receptors, but this effect may be reduced in infants and in patients with chronic congestive heart failure. Infusion rates >20 mcg/kg per minute may result in excessive vasoconstriction.[430,431] In one study in single ventricle postoperative cardiac patients, dopamine increased oxygen consumption while not improving blood pressure or cardiac output.[438]

Dobutamine Hydrochloride

Dobutamine has a relatively selective effect on β1- and β2-adrenergic receptors due to effects of the two isomers; one is an α-adrenergic agonist, and the other is an α-adrenergic antagonist.[439] Dobutamine increases myocardial contractility and can decrease peripheral vascular resistance. Titrate the infusion[432,440,441] to improve cardiac output and blood pressure due to poor myocardial function.[441]

Norepinephrine

Norepinephrine is a potent vasopressor promoting peripheral vasoconstriction. Titrate the infusion to treat shock with low systemic vascular resistance (septic, anaphylactic, spinal, or vasodilatory) unresponsive to fluid.

Sodium Nitroprusside

Sodium nitroprusside increases cardiac output by decreasing vascular resistance (afterload). If hypotension is related to poor myocardial function, consider using a combination of sodium nitroprusside to reduce afterload and an inotrope to improve contractility. Fluid administration may be required secondary to vasodilatory effects.

Inodilators

Inodilators (inamrinone and milrinone) augment cardiac output with little effect on myocardial oxygen demand. It is reasonable to use an inodilator in a highly monitored setting for treatment of myocardial dysfunction with increased systemic or pulmonary vascular resistance (Class IIa, LOE B[352,442–444]). Administration of fluids may be required secondary to vasodilatory effects.

Inodilators have a long half-life with a delay in reaching a steady-state hemodynamic effect after the infusion rate is changed (18 hours with inamrinone and 4.5 hours with

milrinone). In cases of toxicity the cardiovascular effects may persist for several hours even after the infusion is discontinued.

Neurologic System

A primary goal of resuscitation is to preserve brain function. Limit the risk of secondary neuronal injury by adhering to the following precautions:

- Do not routinely provide excessive ventilation or hyperventilation. Hyperventilation has no benefit and may impair neurologic outcome by adversely affecting cardiac output and cerebral perfusion.[445] Intentional brief hyperventilation may be used as temporizing rescue therapy in response to signs of impending cerebral herniation (eg, sudden rise in measured intracranial pressure, dilated pupil[s] not responsive to light, bradycardia, hypertension).
- Therapeutic hypothermia (32°C to 34°C) may be considered for children who remain comatose after resuscitation from cardiac arrest (Class IIb, LOE C).[446,447] It is reasonable for adolescents resuscitated from sudden, witnessed, out-of-hospital VF cardiac arrest (Class IIa, LOE C). Although there are no randomized studies in the pediatric population on the effect of therapeutic hypothermia, it is of benefit in adults following witnessed out-of-hospital VF arrest[448,449] and in asphyxiated newborns.[450,451]
- The ideal method and duration of cooling and rewarming are not known. Prevent shivering by providing sedation and, if needed, neuromuscular blockade, recognizing that this can mask seizure activity. Closely watch for signs of infection. Other potential complications of hypothermia include diminished cardiac output, arrhythmia, pancreatitis, coagulopathy, thrombocytopenia, hypophosphatemia, hypovolemia from cold diuresis, hypokalemia, and hypomagnesemia.
- Monitor temperature continuously, if possible, and treat fever (>38°C) aggressively with antipyretics and cooling devices because fever adversely influences recovery from ischemic brain injury (Class IIa, LOE C).[452–458]
- Treat postischemic seizures aggressively; search for a correctable metabolic cause such as hypoglycemia or electrolyte imbalance.
- Avoid rewarming from 32 to 34°C faster than 0.5°C per 2 hours unless the patient requires rapid rewarming for clinical reasons.

Renal System

Decreased urine output (<1 mL/kg per hour in infants and children or <30 mL/hour in adolescents) may be caused by prerenal conditions (eg, dehydration, inadequate systemic perfusion), renal ischemic damage, or a combination of factors. Avoid nephrotoxic medications and adjust the dose of medications excreted by the kidneys until you have checked renal function.

Interhospital Transport

Ideally postresuscitation care should be provided by a trained team from a pediatric tertiary care facility. Contact such a team as early as possible during the resuscitation attempt and coordinate transportation with the receiving unit.[459] Transport team members should be trained and experienced in the care of critically ill and injured children[103,460] and supervised by a pediatric emergency medicine or pediatric critical care physician. The mode of transport and composition of the team should be established for each system based on the care required by each patient.[461] Monitor exhaled CO2 (qualitative colorimetric detector or capnography) during interhospital or intrahospital transport of intubated patients (Class IIa, LOE B).[116,413]

Family Presence During Resuscitation

Family presence during CPR is increasingly common, and most parents would like to be given the opportunity to be present during resuscitation of their child.[462–471] Studies show that family members who are present at a resuscitation would recommend it to others.[462,463,465,471,472] Parents of chronically ill children are comfortable with medical equipment and emergency procedures, but even family members with no medical background who were at the side of a loved one to say goodbye during the final moments of life believe that their presence was beneficial to the patient,[462–464,466,471–476] comforting for them,[462–465,468–471,476] and helpful in their adjustment[463–465,472,473,476,477] and grieving process.[477] Standardized psychological examinations suggest that, compared with those not present, family members present during attempted resuscitations have less anxiety and depression and more constructive grieving behavior.[477] Parents or family members often fail to ask, but healthcare providers should offer the opportunity in most situations.[474,478,479] Whenever possible, provide family members with the option of being present during resuscitation of an infant or child (Class I, LOE B).[474,478,479]

Family presence during resuscitation, in general, is not disruptive,[464,472,475,476,480,481] and does not create stress among staff or negatively affect their performance.[462,464,480,482] If the presence of family members creates undue staff stress or is considered detrimental to the resuscitation,[483] then family members should be respectfully asked to leave (Class IIa, LOE C). Members of the resuscitation team must be sensitive to the presence of family members, and one person should be assigned to remain with the family to comfort, answer questions, and support the family.[484]

Termination of Resuscitative Efforts

There are no reliable predictors of outcome to guide when to terminate resuscitative efforts in children.

Clinical variables associated with survival include length of CPR, number of doses of epinephrine, age, witnessed versus unwitnessed cardiac arrest, and the first and subsequent rhythm.[6,7,11–13,15,16,151,485–489] None of these associations, however, predict outcome. Witnessed collapse, bystander CPR, and a short interval from collapse to arrival of professionals improve the chances of a successful resuscitation. Intact survival has been documented after unusually prolonged in-hospital resuscitation.[13,133,134,151,490,491]

Sudden Unexplained Deaths

Increasing evidence demonstrates that some cases of sudden infant death syndrome (SIDS) and sudden death in older children and young adults may be associated with genetic mutations causing cardiac ion channelopathies. Channelopathies are dysfunctional myocyte ion channels that result in abnormal movement of electrolytes into and/or out of the cell and predispose the

heart to arrhythmia.[492–501] Mutations causing cardiac ion channelopathies are found in 2% to 10% of victims[492–498] and in 14% to 20% of young adults with sudden death in whom the cause of death is not evident in a routine autopsy.[499–501] Clinical and laboratory (eg, ECG, molecular-genetic screening) investigations of first- and second-degree relatives of patients with sudden unexplained death reported inherited, arrhythmogenic disease in 22% to 53% of families.[502–505]

Therefore when sudden unexplained cardiac arrest occurs in children and young adults, obtain a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents or drownings, or sudden unexpected death at <50 years old) and review previous ECGs.

All infants, children, and young adults with sudden unexpected death should, where resources allow, have an unrestricted, complete autopsy, preferably performed by a pathologist with training and experience in cardiovascular pathology. Consider appropriate preservation and genetic analysis of tissue to determine the presence of a channelopathy. Refer families of patients that do not have a cause of death found on autopsy to a healthcare provider or center with expertise in arrhythmias (Class I, LOE C).

Disclosures

Guidelines Part 14: PALS Writing Group Disclosures

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Monica E. Kleinman	Children's Hospital Anesthesia Foundation–Senior Associate in Critical Care Medicine	None	None	None	None	None	None
Leon Chameides	Emeritus Director Pediatric Cardiology, Clinical Professor, University of Connecticut	None	None	None	None	None	None
Stephen M. Schexnayder	University of Arkansas for Medical Sciences—Professor/ Division Chief; †AHA Compensated Consultant as Associate Senior Science Editor	*Pharmacokinetics of Proton Pumps inhibitors in Critically Ill patients	None	None	None	None	*Expert witness in several cases involving pediatric critical care & emergency medicine
Ricardo A. Samson	The University of Arizona: clinical care, teaching and research related to the field of Pediatric Cardiology in academic setting-Professor	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing—Professor; AHA ECC Product Development—Senior Science Editor- †Significant compensation as a paid AHA consultant to help develop and edit the 2010 AHA Guidelines for CPR and ECC.	None	None	None	None	None	None
Dianne L. Atkins	University of Iowa—Professor *Compensated worksheet editor for the 2010 AHA Guidelines. Money is divided 2/3 to my institution and 1/3 to me.	None	None	None	None	None	*Defense expert witness for episode of ventricular fibrillation in a 2 year old child. Attorney are Buckley and Thereoux of Princeton, New Jersey
Marc D. Berg	University of Arizona - Staff Intensivist; Asso. Prof. Clinical Pediatrics, Attending Intensivist, Pediatric Critical Care Medicine	None	None	Travel expenses defrayed with an honorarium of $4000 for speaking at 13th Asian Australasian Congress of Anesthesiologists, Fukuoka, Japan 6/2010	None	None	None
Allan R. de Caen	Self employed, pediatric intensivist	None	None	None	None	None	*Medical expert for Canadian Medical ProtectiveAssoc

(*Continued*)

Guidelines Part 14: PALS Writing Group Disclosures, *Continued*

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Ericka L. Fink	Children's Hospital of Pittsburgh of UPMC–Assistant Professor	†National Institutes of Health, NINDS K23, Laerdal Foundation, and Children's Hospital of Pittsburgh Clinical and Translational Science Institute grants to study duration of hypothermia after pediatric cardiac arrest.	None	None	None	None	None
Eugene B. Freid	Nemours Childrens Clinics–Anesthesiologist and Intensivist	None	None	*$1500.00 from University of North Carolina to Nemours Childrens Clinics for 3 lectures at annual anesthesiology conference-lectures related to anesthesia management of patients with cancer, operating room ventilators & postoperative nausea/vomiting. No direct conflicts with Pediatric Life support topics	None	None	None
Robert W. Hickey	University of Pittsburgh–Pediatric Emergency Medicine Physician	†NIH sponsored research on the effect of cyclopentenone prostaglandins upon post-ischemic brain.	None	None	None	None	*Occasional expert witness (1–2 times per year)
Bradley S. Marino	Cincinnati Children's Hospital Medical Center–Associate Professor of Pediatrics	None	None	None	None	None	None
Vinay M. Nadkarni	University of Pennsylvania, Children's Hospital of Philadelphia–Attending Physician, Pediatric Critical Care	†NIH RO1: Coinvestigator, Therapeutic Hypothermia After Pediatric Cardiac Arrest Center of Excellence Grant, PI, Laerdal Foundation for Acute Care Medicine AHRQ: Agency for Healthcare Research and Quality: PI, Tracheal Intubation Safety in Pediatric ICUs *NHTSA: Coinvestigator, Chest compression characteristics in children	None	None	None	None	*Volunteer (no salary or remuneration), World Federation of Pediatric Intensive and Critical Care Societies Volunteer (no salary), Data Safety and Monitoring Board, CIRC study
Lester T. Proctor	University of Wisconsin-Madison College of Medicine and Public Health–Professor	None	None	None	None	None	None
Faiqa A. Qureshi	Children's Specialty Group—Partner	None	None	None	None	None	None
Kennith Sartorelli	University of Vermont–Associate Professor of Surgery	None	None	None	None	None	None
Alexis Topjian	University of Pennsylvania–Assistant Professor	*Site principal investigator at the Children's hospital of Philadelphia for the "Therpaeutic Hypothermia after Pediatric Cardiac Arrest" funded via an NIH U01	None	None	None	None	None
Elise W. van der Jagt	University of Rochester–Professor of Pediatrics and Critical Care	None	None	None	None	None	None
Arno L. Zaritsky	Childen's Hospital of The King's Daughters-Sr. VP for Clinical Services	None	None	None	None	*Data Safety Monitoring Board for NIH-funded pediatric hypothermia after cardiac arrest research project	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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