

**CME** **Pediatric Perioperative Life Support**

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Pediatric advanced life support training and guidelines are typically designed for first-responders and out-of-hospital resuscitation. Guidelines and scenarios that are more applicable to the perioperative environment would be beneficial for anesthesiologists. The goal of this article is to review resuscitation of pediatric patients during the perioperative period. We use a format that focuses on preresuscitation preparation, resuscitation techniques, and postresuscitation management in the perioperative period. In an effort to provide information of maximum benefit to anesthesiologists, we include common pediatric perioperative arrest scenarios with detailed description of their management. We also provide a section on postresuscitation management and review the techniques for maintaining the child's hemodynamic and metabolic stability. Finally, 3 appendices are included: an example of an intraoperative arrest record that provides feedback for interventions; a table of key medications for pediatric perioperative resuscitation; and a review of defibrillator use and simulation exercises to promote effective defibrillation. (Anesth Analg 2013;117:960–79)

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**1. INTRODUCTION**

Cardiac arrest is associated with a “no-flow” interval between arrest and the start of cardiopulmonary resuscitation (CPR) and a “low-flow” interval between the start of CPR and the return of spontaneous circulation (ROSC). The presence of medically trained personnel at a cardiac arrest increases the likelihood that CPR and advanced life support (ALS) will be provided early, decreasing both the no-flow and low-flow intervals. A cardiac arrest in an operating room (OR) staffed with anesthesia personnel who are monitoring the patient should minimize both of these intervals and provide the best chance of resuscitation and recovery. Indeed, intraoperative cardiac arrest outcomes are among the best reported, with high incidences of ROSC, survival to discharge, and return to baseline, and a low incidence of a new neurologic deficit.<sup>1–3</sup>

Historically, anesthesiologists have been leaders in resuscitation education and research. Over the years, advances in anesthetic drugs and improvements in cardiorespiratory monitoring technology have improved the safety of

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anesthesia. Ironically, these advances that help to reduce the incidence of cardiac arrest in the OR may also reduce anesthesiologists' experience and proficiency in CPR and ALS. Additionally, advances in resuscitation science and frequent updates to guidelines contribute to the difficulty of maintaining proficiency in CPR and ALS, resulting in the potential for a gap in resuscitation knowledge and up-to-date skills for anesthesiologists.

The programs to maintain skills for ALS available from the American Heart Association (AHA) are intended for a broad range of health care providers in a broad range of settings. Anesthesiologists already have expertise in the airway skills portion taught in these training programs and are familiar with most of the medications used in ALS. However, not all anesthesiologists regularly encounter the need to administer cardiac medications and electrical interventions (e.g., pacing and defibrillation). Pediatric anesthesiologists, in particular, need applicable guidelines that emphasize circulatory resuscitation, especially as it pertains to children in the perioperative environment. Modifying the standard pediatric ALS (PALS) guidelines for pediatric anesthesiologists by reducing emphasis on airway management and out-of-hospital resuscitation and increasing emphasis on perioperative resuscitation and cardiovascular therapies should improve applicability, help engage and prepare anesthesiologists and benefit patients in the perioperative setting.

Here, we review advances in resuscitation and current guidelines in the context of the perioperative setting. The scope of this review differs from that of typical PALS by including intraoperative techniques such as the use of end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>), arterial line, or central line monitoring to measure the effectiveness of CPR, prone CPR and defibrillation, open-chest CPR, and extracorporeal life support (ECLS). The scenarios are based on intraoperative events seen in children, including anesthetic overdose, laryngospasm, local anesthetic toxicity, venous air embolism, hyperkalemia, arrest with a malfunctioned ventriculoperitoneal

(VP) shunt, and anaphylaxis. The review is designed for anesthesiologists who deliver care to children and emphasizes techniques that are less frequently required for pediatrics, such as defibrillation skills and resuscitation medication administration.

## 2. PRERESUSCITATION PREPARATION/PREVENTION

### Common Etiologies of Pediatric Perioperative Arrest

Pediatric perioperative arrest prevention and treatment require an understanding of risk factors and etiologies. The risk of intraoperative cardiac arrest for young children is inversely proportional to age, with the greatest risk in neonates (younger than 1 month of age), followed by infants younger than 1 year, then children <2 years old. The risk approaches that of adult rates by the time the patient reaches teenage years.<sup>1,3,4</sup> ASA physical status is another variable that has been associated with risk of intraoperative arrest for children. As in adults, the risk of cardiac arrest is least for those who are ASA I and increases linearly to ASA V (ASA V patients often are excluded from anesthesia-related cardiac arrest studies because they are likely to arrest even without an anesthetic).<sup>1,3-7</sup>

Studies that describe common etiologies of cardiac arrest in the pediatric population include a national perioperative cardiac arrest database that covers 1998 to 2004<sup>1</sup> and a review of 1 institution's cardiac arrests from 1998 to 2005.<sup>3</sup> The most common etiologies of pediatric perioperative arrest include hypovolemia, hyperkalemia, laryngospasm, inhaled induction, central line complications, venous air embolism, and hypoxia from various causes. Local anesthetic overdose, relative anesthetic overdose, malignant hyperthermia, and anaphylaxis are other important but less frequent causes of pediatric perioperative arrest (Table 1).

Hypovolemia is a common etiology of pediatric perioperative arrest and has been described most often with

**Table 1. Categorization of Causes of Pediatric Perioperative Circulatory Failure**

Finding	Causes
Intravascular volume status	
Low preload	Inability to keep up with hemorrhage, inaccurate assessment of volume loss, inadequate IV access for replacement of fluid loss
Volume redistribution	Vasodilation (relative anesthetic overdose, anaphylaxis, sepsis, neurogenic, hypoxia), capillary leak (anaphylaxis, sepsis)
Obstructed venous return	Tamponade, pneumothorax, venous air embolism
Myocardial contractility	
Dysfunction	Anesthetic overdose, metabolic, sepsis, hypoxia
Myopathy	Infectious, idiopathic, chemotherapy
Vascular resistance	
Low vascular resistance	Vasodilation (relative anesthetic overdose, anaphylaxis, sepsis, neurogenic, hypoxia)
High vascular resistance	Pulmonary hypertension, catecholamine excess, medication related
Rate/rhythm	
Metabolic	Hyperkalemia (transfusion, hyperalimentation, iatrogenic, renal failure), malignant hyperthermia, hypocalcemia (transfusion, renal, DiGeorge syndrome)
Hypoxia	Respiratory failure
Ischemia	Williams syndrome (supravalvular aortic stenosis)
Congenital	Prolonged QT (Jervell and Lange-Nielsen, Romano Ward)
Mechanical	Central line related
Pharmacologic	Succinylcholine, neostigmine
Pacemaker failure	Device dysfunction, acidosis, hypoxia

procedures involving posterior spinal fusion, craniotomy, craniofacial reconstruction, or major abdominal surgery.<sup>1</sup> Ongoing hemorrhage is the most common cause of intraoperative hypovolemia.<sup>1</sup> Preexisting hypovolemia and inadequate IV access to replace intravascular volume loss are contributing risk factors.<sup>1,3,4</sup> The reviews suggest that careful estimation of intravascular volume status is important, and that having central venous access may help the physician to monitor volume status and replace volume losses.<sup>1</sup> A recent report of large-blood-loss surgery (e.g., craniofacial reconstruction) showed a lack of response to hypotension with an increase in heart rate in children younger than 24 months. Thus, the anesthesia team may not be able to rely on the changes in vital signs for young children as they do in older children.<sup>8</sup>

Hyperkalemia leading to perioperative arrest has multiple causes. The rapid transfusion of large volumes of red blood cells (RBCs), particularly if they have been stored for >2 weeks or irradiated, is associated with a high potassium load. Lower potassium levels are found in RBC products that have been stored for <1 week.<sup>9</sup> Use of succinylcholine can increase the serum potassium by 0.5 mEq/L or more, which can be significant if the patient already has an elevated serum potassium level (such as from potassium-sparing diuretics or renal failure). Additionally, patients with burns, direct muscle trauma, neurologic disorders involving motor muscle defects, or myopathies that weaken skeletal muscle membranes are at risk of releasing a large load of potassium from rhabdomyolysis after receiving succinylcholine.<sup>10</sup> Intraoperative hyperkalemia can also result from reperfusion of ischemic organs or limbs. Hyperkalemia should be suspected when T waves increase in height or appear "peaked." If not treated immediately, hyperkalemia can lead to wide-complex ventricular arrhythmias. Treatment includes alkalinization (hyperventilation and bicarbonate administration), calcium, and a combination of glucose and insulin.

In addition to hyperkalemia, rapid transfusion of blood products can be associated with sudden hypotension as a result of hypocalcemia, hemolysis, allergic reaction, bacterial contamination, or excessive intravascular volume leading

to cardiac distension and heart failure. Hypocalcemia during blood product administration results when the liver is unable to metabolize the sudden volume of citrate (used as an anticoagulant) that binds calcium and leads to myocardial depression and hypotension. Citrate is contained in the plasma of whole blood, fresh frozen plasma, or platelets and is less of a problem with the transfusion of packed RBCs. To avoid citrate toxicity, a maximal transfusion rate of 1.33 mL/min times the weight in kilograms is recommended.<sup>11</sup>

Well known to those who care for anesthetized children is loss of the airway secondary to laryngospasm or other upper airway obstructions during anesthetic induction or emergence, or after tracheal extubation (Table 2). The risk of laryngospasm is increased if the airway is irritated from a respiratory infection.<sup>12</sup> Hypoxemia and cardiac arrest usually can be prevented by early recognition and treatment of the obstructed airway. Bag-mask ventilation with 100% oxygen, positive pressure, and an appropriate-sized oral airway will often break laryngospasm. If the loss of airway is from soft tissue obstruction, insertion of a laryngeal mask airway (LMA) may successfully overcome the obstruction, particularly if bag-mask ventilation is ineffective and tracheal intubation is not immediately possible.<sup>13,14</sup>

A relative anesthetic overdose may result in bradycardia or hypotension that leads to cardiac arrest. A relative anesthetic overdose is defined as the use of an appropriate dose of an inhaled or IV anesthetic that has an unexpected hemodynamic effect. Although dosing of IV anesthetics for children is usually weight based, a child with hypovolemia and compensated shock may decompensate and suffer arrest when given an appropriate weight-based dose of an anesthetic that causes vasodilation or decreases cardiac contractility. Recognizing children at risk and titrating anesthetics during administration are the best ways to prevent a relative anesthetic overdose.

Inhaled induction with halothane was a well-known cause of bradycardia and hypotension that, when unrecognized, could lead to cardiac arrest in children, particularly in infants who were not pretreated with atropine or glycopyrrolate. Since sevoflurane has replaced halothane, the incidence of cardiac arrest on induction solely from volatile

**Table 2. Categorization of Causes of Pediatric Perioperative Respiratory Failure**

Finding	Causes (specific examples in parentheses)
<b>Upper airway</b>	
Difficult intubation	Micrognathia (Pierre Robin anomalad), macroglossia (Beckwith-Wiedemann syndrome), mucopolysaccharide deposition (Hunter/Hurler syndromes), blood (bleeding tonsil, trauma)
Obstruction	Tonsil hypertrophy, foreign body, epiglottitis
Laryngospasm	URI, gastric reflux
Laryngeal swelling	Postextubation croup
Equipment problems	Obstruction or leaks in the anesthesia machine, the breathing circuit, or the ETT or tracheostomy tube (kink, plug from mucous or blood), malposition of ETT, leak around tracheostomy tube or ETT, cuff leak
<b>Lower airway</b>	
Bronchospasm	Reactive airway disease
Compression	Mediastinal mass
<b>Parenchymal disease</b>	
BPD/CLD	Prematurity-associated chronic lung disease
Pulmonary edema	Postobstructive, cardiac dysfunction, excessive intravascular volume, capillary dysfunction
Pneumonia	Aspiration, infection
Disordered control	Prematurity, central hypoventilation, recurarization, opioid respiratory depression, ventilator failure
Other	Narcotic-induced rigidity, change in compliance of chest during thoracic surgery

URI = upper respiratory infection; ETT = endotracheal tube; BPD = bronchopulmonary dysplasia; CLD = chronic lung disease.

anesthetics has become rare. This reduced incidence may be related to different cardiovascular profiles of the drugs. Alternatively, the halothane vaporizer may have allowed delivery of a higher multiple of the minimum alveolar concentration (MAC = 0.8%; maximum vaporizer setting 5%) of anesthetic than is allowed by the sevoflurane vaporizer (MAC = 3.3%; maximum vaporize setting 8%). Additionally, more liberal fasting guidelines that allow clear liquids closer to the administration of the anesthetic (usually up to 2 hours before induction) help prevent hypovolemia that may be associated with hypotension on induction in children. The combination of hypovolemia and hypotension during inhaled induction increases the difficulty of obtaining IV access so that resuscitation in this situation may require the use of endotracheal or intraosseous (IO) medications.

Other causes of perioperative bradycardic arrest in children are the administration of succinylcholine or neostigmine; procedural traction or pressure on neck, eyes, or heart; and laparoscopic insufflation of the abdomen. Bradycardia in infants is particularly ominous because the cardiac output is primarily rate dependent, and CPR may be necessary when heart rate decreases <60 beats per minute.

Having central venous access is beneficial for cases with anticipated large intravascular volume losses or hemodynamic instability because it enables monitoring of volume status and provides access for fluid and vasoactive medication administration. However, catheter insertion is associated with complications (arrhythmias, hemothorax, and pneumothorax) that can lead to cardiac arrest. The use of ultrasound for catheter insertion may aid in placement and in reducing complications. A chest radiograph taken after insertion helps to confirm line placement, reveals the presence of air or fluid in the pleural space, and may detect a catheter that has migrated outside the vascular space.

Regional blocks in children are often performed under general anesthesia because children are usually uncooperative with these procedures. However, general anesthesia can mask the early symptoms that would indicate inadvertent IV administration of the local anesthetic, including circumoral tingling, dizziness, tinnitus, and loss of consciousness. Local anesthetic-induced arrhythmias are reported most often with inadvertent IV administration. Using local anesthetic that contains epinephrine improves the ability to recognize intravascular injection in children because intravascular epinephrine produces T-wave changes, tachycardia, and hypertension.<sup>15</sup> The occurrence of these signs should prompt the discontinuation of local anesthetic administration and investigation of the catheter or needle tip location.

Anaphylaxis-induced cardiac arrest in children has been reported with exposure to latex or the injection of contrast dye, nondepolarizing neuromuscular blocking drugs, antibiotics, and dextran solutions. Anaphylaxis usually presents as hypotension, erythema, and bronchospasm with associated increases in peak inspiratory pressure requirements.

Venous air embolism has been associated with fatalities during orthopedic and neurosurgical procedures that involve exposure of open blood vessels positioned above the level of the heart. Such embolisms can be prevented by maintaining the operative site at or below heart level

and maintaining intravascular volume to avoid a decrease in central venous pressure (CVP). Sensitive monitors of venous air embolism include continuous end-tidal nitrogen detection, transesophageal echocardiography, or precordial Doppler. Immediate treatment includes lowering the operative site to below heart level if possible and flooding the operative field to prevent further air entry. Positioning the patient to prevent entry of air into the pulmonary artery helps prevent obstruction of flow to the left heart (left side down and Trendelenburg). Aspiration of air from a central line may be possible if a large amount of air is present; multi-orifice catheters perform only slightly better than single lumen, but both are better than balloon-directed catheters for aspiration of air.<sup>16</sup>

### **Categorization of Risks and Causes for Pediatric Perioperative Arrest**

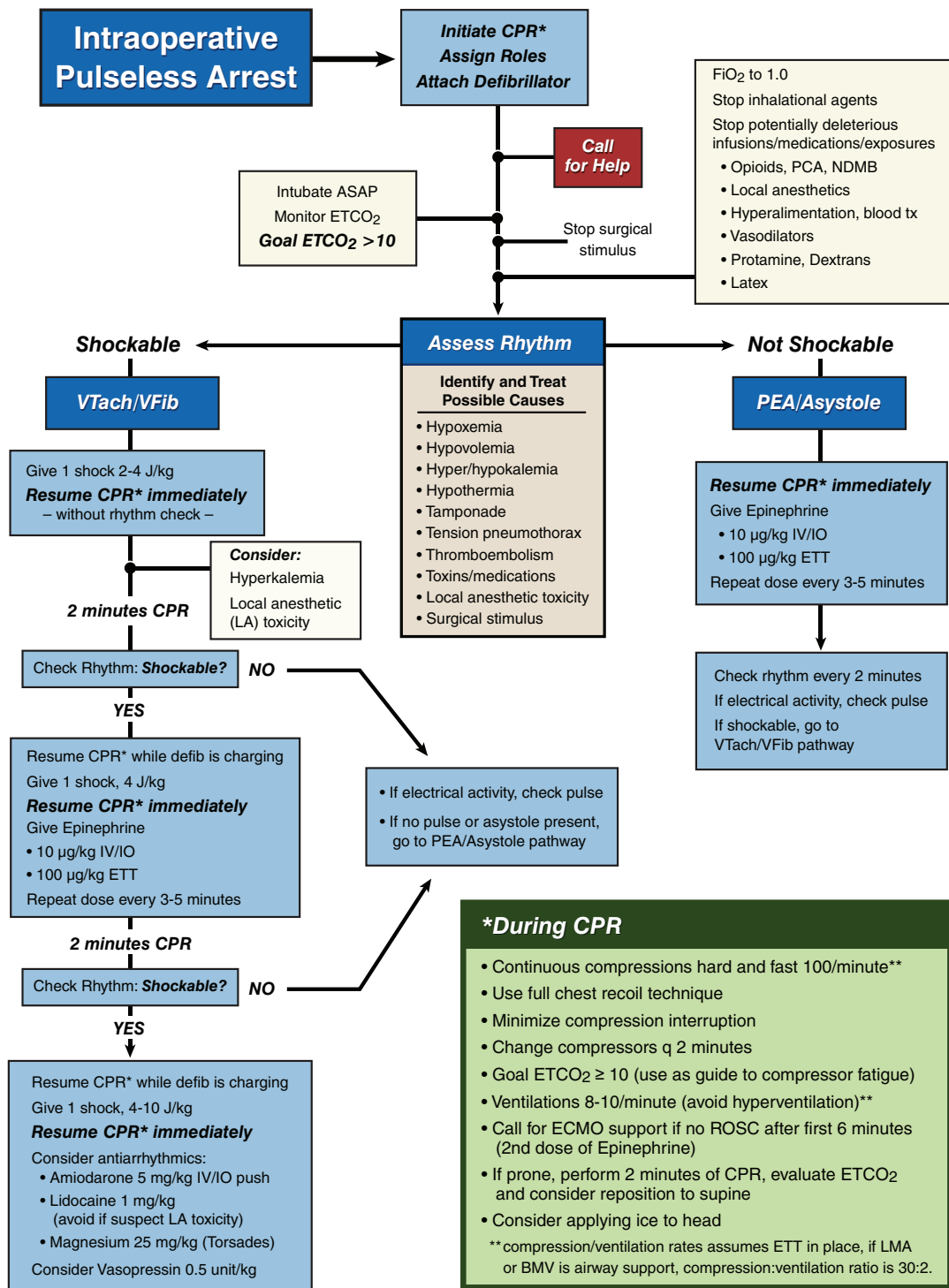
The initial and most crucial response to perioperative cardiac arrest involves the restoration of oxygenated blood flow to the vital organs with CPR. While CPR is ongoing, attempts are made to eliminate factors that could contribute to the patient's deterioration. Examples of factors that should be eliminated include anesthetics that depress cardiovascular function, potassium-containing blood products or hyperalimentation infusions, and exposure to potential allergens. These actions should be taken whether or not the cause of cardiac arrest is known. Simultaneously, efforts should be made to determine the cause of the arrest and administer specific therapy. The cause of the arrest is not always determined or it may not be determined until a later time. When the cause of arrest is unknown, it is useful to have a systematic approach to avoid missing treatable conditions.

AHA PALS categorizes conditions associated with cardiovascular collapse into *respiratory failure* (upper airway obstruction, lower airway obstruction, parenchymal disease, and disordered control of breathing), *circulatory failure* (hypovolemic shock, distributive shock, cardiogenic shock, obstructive shock), and a category called *sudden cardiac*. For anesthesiologists, the PALS organization of respiratory causes into upper airway, lower airway, parenchymal, and control of breathing is applicable. Circulatory causes could be organized for anesthesiologists into the 4 components of cardiac output: heart rate (dysrhythmias), preload (intravascular volume status), contractility, and afterload (vascular resistance). This type of categorization allows treatment options to be addressed systematically (i.e., use of chemical or electrical measures to treat heart rate or rhythm causes, volume or diuresis for preload causes, inotropes or negative inotropes for contractility causes, and vasoconstrictors or dilators for afterload causes).

### **Familiarization with Resuscitation Algorithms, Skills, and Equipment**

The AHA PALS algorithms used for pediatric resuscitation are also useful for perioperative arrest management and can be modified to include perioperative concerns. A modification of the AHA PALS pulseless arrest algorithm for the intraoperative environment is shown in Figure 1. An algorithm for the recognition and management of causes of





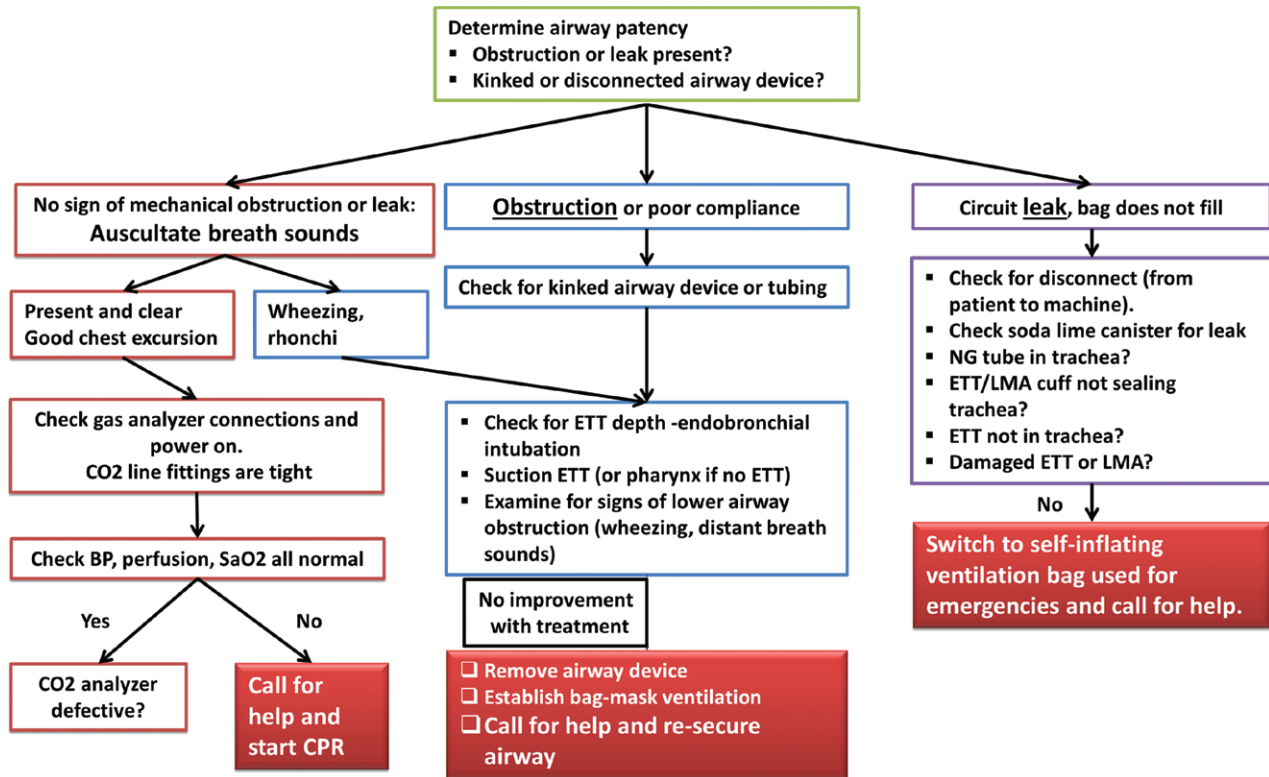
**Figure 1.** The American Heart Association pediatric advanced life support algorithm for pulseless arrest/cardiopulmonary resuscitation (CPR) modified for intraoperative events. ETCO<sub>2</sub> = end-tidal CO<sub>2</sub>; ETT = endotracheal tube; IO = intraosseous; NDMB = nondepolarizing muscular blocker; PCA = patient-controlled analgesia; PEA = pulseless electrical activity; ROSC = return of spontaneous circulation; LMA = laryngeal mask airway; BMV = bag-mask ventilation. Adapted from Schwartz et al.<sup>64</sup>

hypoxia or loss of ETCO<sub>2</sub> is shown in Figure 2. Cognitive aids with algorithms for specific types of intraoperative arrests have been developed by the Society for Pediatric Anesthesia and may be available in some ORs. These algorithms, also known as Pediatric Critical Event Checklists, are available on the Society for Pediatric Anesthesia website (www.

pedsanesthesia.org) or at the following URL: [http://www.pedsanesthesia.org/newnews/Critical\\_Event\\_Checklists.pdf](http://www.pedsanesthesia.org/newnews/Critical_Event_Checklists.pdf). These checklists are intended for use by those who care for children in the perioperative period because certain crises arise infrequently, and individual practitioners may experience them personally only a few times within their

## Loss of End Tidal CO<sub>2</sub> and/or Hypoxia in the Anesthetized Patient

- Give 100% O<sub>2</sub>
- If on ventilator, switch to hand-bag ventilation
- Ensure O<sub>2</sub> delivery from wall or tank
- If patient hypoxic, call for help and declare an emergency



**Figure 2.** Algorithm for management of the loss of end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) and/or hypoxia in the anesthetized patient. BP = arterial blood pressure; CPR = cardiopulmonary resuscitation; ETT = endotracheal tube; LMA = laryngeal mask airway; NG = nasogastric; SaO<sub>2</sub> = arterial oxygen saturation.

professional careers. Having checklists available for such circumstances can aid in recalling necessary facts, decision points, and treatment management. Another cognitive aid is shown in Appendix 1; this is an example of a combined perioperative arrest record/cognitive aid that prompts the recorder when certain events are due to be performed (change compressors every 2 minutes, give epinephrine every 4 minutes, activate extracorporeal membrane oxygenation [ECMO] after 6 minutes or second epinephrine dose).

Several of the specific pediatric situations and their intraoperative management are presented below in Section 4. The common medications used for perioperative arrest that staff should be familiar with are listed in Appendix 2. In addition to the usual resuscitation skills, pediatric perioperative personnel should be familiar with the performance of CPR when the patient is in the prone position. Children undergoing spinal fusion or craniofacial reconstruction surgery are at high risk of cardiac arrest and may be in the prone position when arrest occurs. Initiating CPR when the patient is in the prone position may minimize the no-flow interval. Anesthesiologists should be familiar with delivering (or instructing surgeons to deliver) chest compressions to patients in the prone position (Fig. 3).<sup>17,18</sup> Prone compressions can be delivered in the midline over the spine or, if

there is a midline incision, as in spinal surgery, bilaterally over the scapulae. The production of adequate levels of ETCO<sub>2</sub> should help the team determine if they are providing effective compressions. A sternal counter pressure can be tried by placing a fist or sandbag under the sternum if compressions do not appear to be adequate. By applying compressions in the prone position, the team can improve cardiac output while waiting for the patient to be turned supine. Six adults who received CPR in an intensive care unit setting exhibited better hemodynamics when they were in the prone position than when they were supine.<sup>19</sup> Successful defibrillation of patients in the prone position also has been reported; gel pads are placed away from the incision and over as much of the heart as possible.<sup>20</sup>

Caregivers for children in the perioperative environment should be familiar with the equipment used to prevent, detect, or treat pediatric perioperative cardiac arrest (Table 3). Familiarity with the defibrillator available on the code cart will help minimize the interval to defibrillation. Other timesavers include preoperative placement of defibrillator pads on the patient under the surgical drapes when there is concern for intraoperative arrhythmia, knowing when and how to use pediatric-sized paddles, and being familiar with the use of the available model of defibrillator.

**Table 3. Equipment and Supplies Needed for Pediatric Perioperative Arrest Management**

Equipment/ supplies	Comments
Monitoring	Use for early recognition of need for resuscitation Invasive monitoring when indicated ETCO <sub>2</sub> (quantitative) for effectiveness of compressions, maintenance of airway, and return of spontaneous circulation
Blood products	Availability of adequate blood product replacement (cross matched when possible, in room if necessary)
IV access	Adequate access for monitoring and delivery of fluids and medications IO available (useful when problems on induction without IV access or when unable to obtain IV access)
Crash carts	Medications and equipment not available in anesthesia carts Dosing guides (calculated for patient or length based, color coded) Cognitive aids or copies of algorithms Lipid for treatment of local anesthetic toxicity Malignant hyperthermia kit
Defibrillator	For treatment of dysrhythmias requiring cardioversion or defibrillation Familiarity with model of defibrillator Know when to use pediatric-sized paddles Place pads on before drapes when concern for intraoperative arrhythmia
ECMO/CPS/CPB	For reversible causes of in-hospital arrest that is unresponsive to CPR Activate process early (blood available, circuit primed, cannula available, surgeon available)

ETCO<sub>2</sub> = end-tidal CO<sub>2</sub>; IO = intraosseous; CPR = cardiopulmonary resuscitation; ECMO = extracorporeal membrane oxygenation; CPS = cardiopulmonary support; CPB = cardiopulmonary bypass.

Hunt et al.<sup>21</sup> showed that involving the trainee in hands-on defibrillator practice resulted in discharging the defibrillator 87% faster than those without hands-on practice in all the steps. Appendix 3 lists defibrillator key points and exercises useful for pediatric OR personnel.

Availability of and familiarity with the equipment for IO access can minimize the time to vascular access during an arrest. Arrest during inhaled induction, especially in fast-ing infants who are hypovolemic and chubby, contributes to difficult IV access and may require immediate IO access. Less than one-third of attendees surveyed at a pediatric anesthesia conference had IO needles stocked in their OR.<sup>22</sup>

Cognitive aids (code cards) containing resuscitation algorithms should be attached to the “code” or “crash” cart. If weight-based medication cards for the patient are unavailable, alternatives include length-based systems (Broselow, Armstrong Medical Industries, Lincolnshire, IL) or use of a cart color-coded by age range and containing appropriate medications and equipment for ease of dosing by individuals less familiar with pediatric requirements. Code carts in the OR should contain resuscitation medications that are unavailable, or available in limited amounts, in the anesthesia carts. Examples of medications that should be available in pediatric ORs are intralipid and dantrolene. Intralipid (20% emulsion) and instructions for its use should be available for treatment of toxicity from inadvertent IV administration of local anesthetic. Failure of intralipid administration to reverse toxicity may necessitate the use of ECMO or cardiopulmonary bypass (CPB) support. Dantrolene and a malignant hyperthermia kit with cool fluids and instructions for their use should be available to minimize the risk of arrhythmia in patients who suffer a malignant hyperthermia reaction to anesthesia.

The impedance threshold device (ITD) is used to improve venous blood return to the thorax during relaxation of chest compression. As the chest reexpands, this device prevents air entry through the endotracheal tube (ETT) unless a 20 cm H<sub>2</sub>O or greater airway pressure gradient is generated. This pressure gradient helps to maintain a negative intrathoracic pressure during the relaxation phase of chest compressions, thereby helping to pull venous blood return into the thorax.

Assisted ventilation overcomes the device’s negative 20 cm H<sub>2</sub>O “cracking pressure” on the 1:10 ventilation during compression cycles, but the negative intrathoracic pressure is maintained during relaxation in the other 9:10 nonventilation compression cycles. The goal is to increase venous return, or “pump prime,” before the next chest compression. Use of this device in children has not been reported, but studies are ongoing in adults. ITDs may be stocked in hospital code carts or used in prehospital jurisdictions. It is helpful for the anesthesiologist to recognize these devices and how they function if assuming care of a patient with an ITD in use.

If it is available, ECMO is a resource that may be helpful to provide life support in situations of reversible toxicities. ECMO, cardiopulmonary support (CPS), and CPB, collectively known as ECLS or extracorporeal CPR, can provide both hemodynamic and respiratory support. The ECLS systems require specialized equipment and often blood priming when used for children, surgical insertion of cannulae, and specially trained personnel. They can be used to support circulation and respiration while determining whether native circulation will recover. Additionally, they can provide the hemodynamic support needed to accomplish dialysis for metabolic or toxic events. Placing a child on ECLS often takes time (to activate, prime, and obtain access) and therefore needs to be considered early and activated quickly to minimize the low-flow interval.

### **Familiarization with Resuscitation Team, Roles, and Communication**

Before a perioperative arrest occurs, the care teams should understand their roles and how to optimize communication. The teams must have a clear understanding of what calling for help does, who should do it, when they should do it, and who responds. They should know if a code cart and defibrillator are available or must be sent for and what is included in the code cart.

Calling for help (“any anesthesiologist to OR 1”) may provide no further assistance or may produce a myriad of personnel who do not have a role in the resuscitation of the patient. *Incremental help* is the concept that only necessary,

additional resources are provided, and avoids the activation of unneeded staff who contribute to a noisy and crowded environment. The method of calling for help, the team member roles needed, and the personnel who will provide them should be practiced before an event to increase effectiveness of the response. The nurses, surgeons, and anesthesiologists caring for the patient should all have defined roles and be included in the resuscitation. The typical roles as outlined by AHA PALS are listed in Table 4 and include: Leader, Airway, Compressor, Lines/meds, Defibrillator/monitor, and Recorder. These roles and the communication between them in the OR are discussed more in the next section; they should be clarified for the pre- and postanesthetic care areas as well.

Training should include decisions about when to perform and who will perform procedures like tracheostomy, thoracentesis, or pericardiocentesis; when to consult a cardiologist or intensivist; and when to activate ECMO or transfer the patient to an ECMO center.

### 3. PERIOPERATIVE RESUSCITATION

#### Recognizing the Need for Resuscitation

Recognition of the need for resuscitative efforts and immediate start of chest compressions are key to minimizing the no-flow period and allowing the best chance for ROSC. Typical intraoperative indications for resuscitative efforts include: inadequate heart rate or arterial blood pressure for age; apnea or agonal respiratory efforts; cyanosis or dark blood in wound; failure of pulse oximetry; failure of noninvasive blood pressure determination; loss of arterial line waveform; absent or abnormal heart tones; and abrupt decrease in  $\text{ETCO}_2$ . Early resuscitation responses are as follows:

1. Inform the surgical and nursing teams.
2. Stop surgical stimulation.
3. Place the patient on 100% oxygen (unless fire in airway).
4. Consider Trendelenburg position and wide-open (isotonic) fluids if the patient is hypotensive.
5. Start chest compressions if vital organ blood flow is compromised.
6. Call for help and a crash cart.

7. Stop the administration of potentially deleterious substances (including inhalational agents, sedative/opioid infusions, local anesthetic infusions, patient-controlled anesthesia pumps, hyperalimentation with significant potassium or glucose, blood products with significant potassium or calcium chelators, vasodilators or negative inotropes, medications associated with anaphylaxis [antibiotics, dextrans, nondepolarizing paralytics, protamine, latex-containing substances]).
8. Apply bags of ice to the child's head during chest compressions or low-flow states as a possible means of neuroprotection.
9. Assign leader and roles and start resuscitation record.

These early responses can be performed quickly, often concurrently. A checklist of these items that the recorder completes can be used to verify with the team that these responses are considered. Note, there is no evidence regarding the effectiveness of placing ice around the child's head during CPR (it is often attempted in cardiac ORs during resuscitative efforts). Using ice may be considered if it is readily available, but care should be taken to avoid frostbite damage.

#### Airway Management

An early consideration in perioperative resuscitation is intubation of the airway. Many patients may already be intubated at the time of their perioperative arrest. Anesthesiologists have the skill to provide rapid tracheal intubation with minimal interruption of resuscitative efforts in patients who are not already intubated. It is unknown whether resuscitation with intubation is superior to resuscitation with a mask or a supraglottic airway such as the LMA. If a patient undergoing anesthesia is unintubated (no airway or with an LMA) and suffers a cardiac arrest, securing the airway by tracheal intubation during CPR has several advantages. It (1) prevents the need to interrupt compressions to deliver ventilations, (2) allows use of an easy-to-remember 100:10 ratio of compressions/min to ventilations/min, (3) obviates the need for a team member to hold continuous cricoid pressure during ventilation if potential aspiration is a concern, (4) reduces the risk of stomach overdistension leading to aspiration or to a reduction of functional residual capacity, and

**Table 4. Roles and Responsibilities During an Intraoperative Arrest**

Role	Responsibilities	Team member
Leader	Assigns roles. Directs resuscitation. Monitors performance.	Anesthesiologist most familiar with patient and course
Airway	Prepares equipment and $\text{O}_2$ . Performs airway and gastric intubation. Ventilates patient.	Second anesthesiologist
Compressor	Delivers chest compressions. Need at least 2 to rotate every 2 min.	Surgeon or scrub nurse if sterility needed
Access	Obtains intravascular or intraosseous access. Administers fluid and medications.	Surgeon or anesthesiologist (access) Nurse or anesthesiologist (medications)
Monitor	Operates monitors and defibrillator. Performs pulse checks. Performs rhythm analysis.	Surgeon or scrub nurse if sterility needed
Recorder	Records resuscitative efforts. Compares efforts to goals on resuscitation list. Reviews record as needed by Leader and team.	Circulating nurse



(5) provides access for medication delivery when no other access is available. However, patients with a difficult airway may be better served by an LMA for airway management rather than having ventilation interrupted for long periods while the anesthesiologist tries to insert an ETT. In bystander CPR, compressions are interposed with ventilation delivered by mouth to patient. The delays for interposed compressions are detrimental to maintaining vital organ blood flow by chest compression. After tracheal intubation, airway ET $\text{CO}_2$  can be monitored continuously during CPR to confirm tracheal intubation, maintenance of tracheal intubation, and the effectiveness of CPR (see below).

It is also unknown whether the use of a cuff on the ETT makes a difference during resuscitation. The most recent version of the AHA PALS guidelines indicates that either cuffed or uncuffed ETTs may be used safely as long as size, position, and cuff pressure are appropriate.<sup>23</sup> They suggest that a cuffed tube may be preferable if the anesthesiologist has concerns regarding poor lung compliance, high airway resistance, or a large glottic gas leak. Cuffed ETTs are associated with a lower rate of reintubation for problems with excessive gas leak and therefore may save time in an emergency.<sup>24,25</sup>

Another consideration during perioperative resuscitation is whether to use an inspired oxygen concentration of 100%, 21%, or a value in between. Some physicians are concerned that high levels of oxygen administered during or after ischemia may contribute to oxygen free radical production and lead to increased cellular damage. Animal data indicate that despite the use of 100% inspired oxygen during resuscitation, the brain tissue remains hypoxic.<sup>26</sup> The brains of such animals then become hyperoxic after ROSC. Hence, the administration of 100% oxygen is needed during the low-flow state of chest compressions. The concern for hyperoxia-induced injury should be focused on the postresuscitation period and weaning down the inspired oxygen from levels that produce a 100% saturation to those that produce a 94% to 99% saturation.<sup>27</sup>

Other factors involved in intraoperative airway management must be considered. Overventilation should be avoided to prevent increased intrathoracic pressure (which could decrease venous return) or unwanted alkalosis (which could interfere with cerebral blood flow during the CPR low-flow state). No data are available regarding whether hand ventilation is preferable to mechanical ventilation during CPR. The decision of which to use may depend on availability of help and effectiveness of ventilation efforts. In addition to the usual assessment of ventilatory efforts during CPR, the intraoperative environment may provide opportunities to monitor airway pressures or tidal volumes through ventilation monitors. Lung expansion or recruitment may be observed directly when the child is undergoing thoracotomy or median sternotomy. Radiographs, fluoroscopy, and bronchoscopy are other adjuncts available in the intraoperative setting that can be used to help assess adequacy of ventilation.

### Circulation

Strong evidence supports the need for emphasis on effective chest compressions during CPR for adults and children. The recommendations are to push hard, push fast, minimize

interruptions, and use a ratio of 100 compressions and 8 to 10 ventilations per minute without the interposition of ventilation when the patient is intubated.<sup>28</sup> Interruptions cause prolonged deteriorations in the vital organ blood flow and should only occur at 2-minute intervals for pulse/rhythm checks. Rescuers who are performing the compressions should be switched during these 2-minute intervals to prevent fatigue from decreasing the effectiveness of the chest compressions. The recorder keeps track of the switch in compressors and can include the information on the record (a prompt can appear on the record at 2-minute intervals to remind the recorder—Appendix 1). To minimize the interruption of compressions, the PALS guidelines recommend a single biphasic shock and immediate resumption of compressions. One should not wait for the rhythm to appear on the monitor before resuming compressions. A sudden increase in the ET $\text{CO}_2$  level will reveal when adequate ROSC has occurred.<sup>29,30</sup>

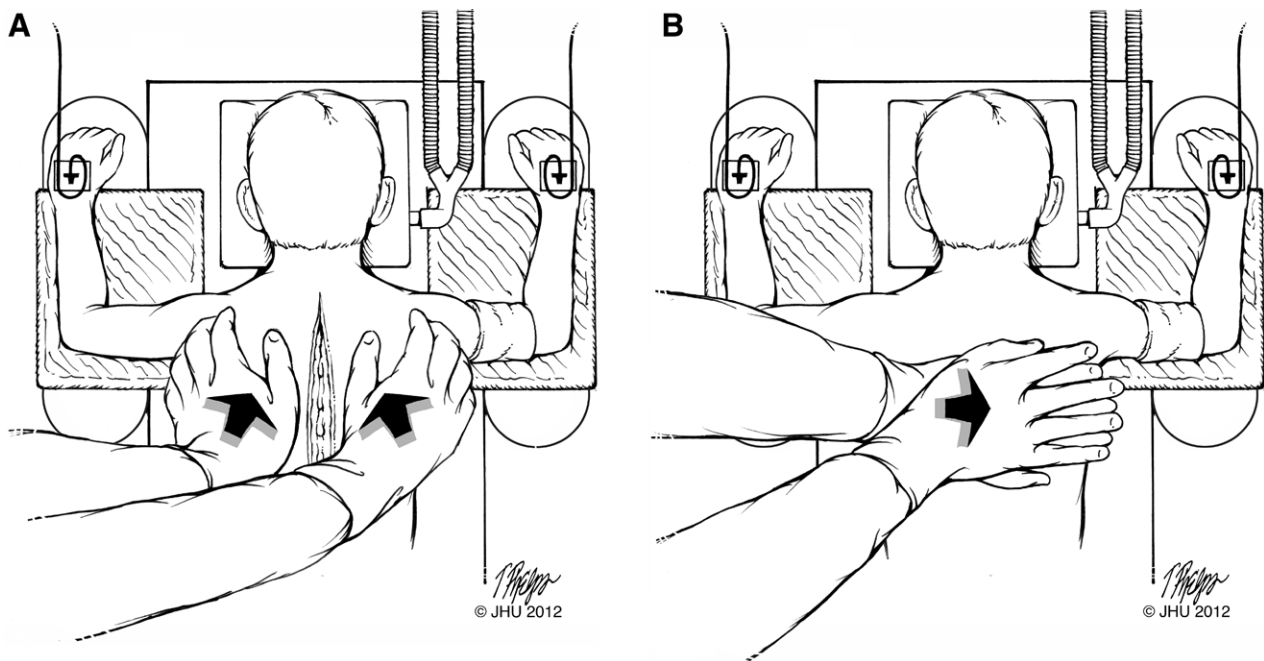
The encircling, 2 hands/2 thumbs method is believed to be the most effective way to deliver compressions for infants. Children have very compliant chests, and rescuers must allow full chest recoil. Therefore, the compressor must avoid leaning on the child's chest during the relaxation phase. Continued pressure on the chest or leaning during relaxation may cause continued increase of intrathoracic pressure and decrease venous return before the next compression. Similarly, overventilation may increase intrathoracic pressure and reduce venous return of blood to chest before compression.

### Medication Administration

Venous access is needed for medication administration; the ETT is a less effective route than IV or IO access for medication administration during resuscitation. When given in the ETT, medications must be delivered at 2.5 to 10 times the dose needed for IV or IO. The ETT route is used when IV or IO access is not available; delivery should switch to IV or IO when they become available. Only half of the responding attendees at a pediatric anesthesiology conference reported having attempted IO access.<sup>22</sup> However, IO access is as effective as IV access and may be easier to obtain during circulatory collapse. The dosing of medications in the IO line is the same as for IV dosing.

Peripheral IV administration of medications during arrest is complicated by the low-flow state, and the likelihood that medication will reach the central circulation is decreased. Central lines with the tip below the diaphragm, IO lines, and peripheral IV lines require an appropriate flush because of the low-flow state in these areas during CPR. Flushing the line in these low-flow areas after administration of a medication during CPR is believed to push the medication from these sites into the central circulation. Animal studies indicate that a flush of at least 0.25 mL/kg is adequate.<sup>31</sup> This volume translates to 20 mL of normal saline for an adult-size (80 kg) adolescent, 10 mL for a child, and 5 mL for an infant. This saline flush should be performed after every dose of medication administered peripherally during CPR.

The use of individual medications in perioperative arrest is similar to that recommended by AHA PALS (see Appendix 2 for individual medication indications and



**Figure 3.** A, Prone chest compressions with a midline posterior incision. B, Prone chest compressions with no midline incision.

dosing). Epinephrine is the most effective medication during CPR in children. The usual epinephrine dose is 10  $\mu\text{g}/\text{kg}$  every 3 to 5 minutes. Administering epinephrine every 4 minutes allows coordination with every other chest compressor change. Subsequent epinephrine doses should not be increased, as larger doses are not helpful and may be harmful. Epinephrine delivered by IO access requires the same dose as IV delivery. Epinephrine given by the ETT route should be dosed at 10 times the IV dose. Use of the ETT route may result in poor absorption, and low dosing of epinephrine has a risk of decreasing vascular tone and coronary perfusion.

Vasopressin has no clear indication in children in the perioperative setting. If desired, vasopressin may be given at a dose of 0.5 units/kg. The literature contains no descriptions of repeat dosage or timing for vasopressin administration in children. Amiodarone is a useful antiarrhythmic during resuscitation. The dosing is 5 mg/kg, IV push if the patient is hemodynamically unstable or IV over 20 to 30 minutes if the patient is hemodynamically stable. No information is available about the usefulness of amiodarone (which has sodium channel blocking properties) in the resuscitation of children from arrhythmias caused by local anesthetics (which also block sodium channels).

### Defibrillation and Cardioversion

Defibrillation of a child can be performed by applying 1 shock with a biphasic defibrillator at an initial dose of 2 to 4 joules/kg. Older defibrillators deliver a monophasic shock, which is described as current passing in 1 direction (1 paddle to the other), generating a waveform that becomes positive and then returns to zero. The newer model defibrillators deliver a biphasic shock, which is described as current passing in 2 directions (1 paddle to the next then back to the first), generating a waveform that becomes first positive and then negative before returning to zero. The biphasic

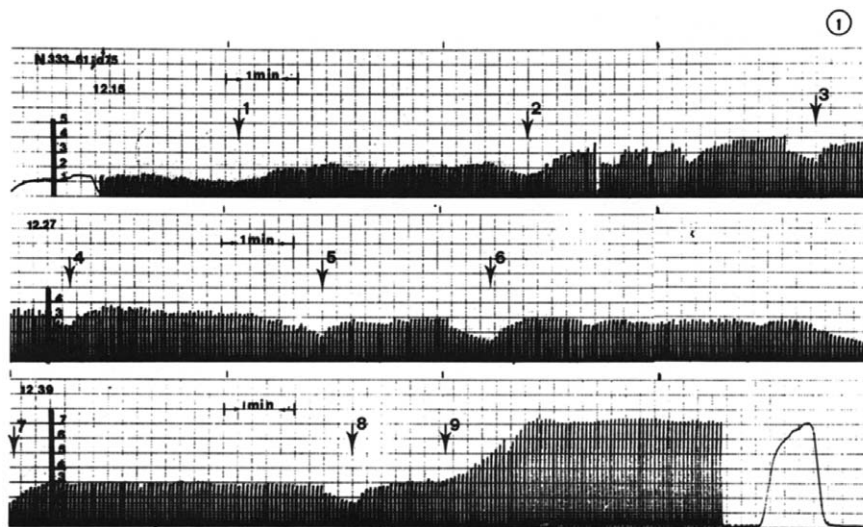
shock waveform is more effective and requires less energy to terminate fibrillation in the heart. The previous 3-shock resuscitation algorithm with a monophasic defibrillator interrupted chest compressions for a substantial length of time. The use of biphasic energy has resulted in increased success with fewer complications and has led to the need for only 1 shock at a time, thus reducing the interruption of chest compressions and reducing no-flow time during defibrillation attempts. The second attempt at defibrillation can be performed with 4 joules/kg, and subsequent shock doses may be increased to a maximum of 10 joules/kg or an adult dose (200 joules).

Cardioversion is used when cardiac rhythm will allow synchronization that prevents delivery of the energy pulse at a part of the electrical cycle that will result in a more chaotic rhythm. Cardioversion is usually attempted for arrhythmias in the presence of a pulse and requires less energy delivery than does defibrillation. The starting dose is 0.5 to 1.0 joule/kg. If it is ineffective, the dose can be increased to 2 joules/kg.<sup>32</sup> See Appendix 3 for defibrillator key points and hands-on exercises.

Automated external defibrillators (AEDs) are being modified for safer use in children. Anesthesiologists comfortable with rhythm analysis and defibrillator use may be able to analyze and deliver shock with a manual defibrillator in less time than an AED takes to evaluate a rhythm and deliver a shock. Not all perioperative providers are facile with the use of defibrillators, and an AED, especially one with pediatric settings, may be useful and lifesaving in situations of perioperative cardiac arrest.

### Monitoring the Effectiveness of CPR

According to the American Society of Anesthesiologists standards, quantitative  $\text{ETCO}_2$  monitoring should be readily available in settings where ETTs or LMAs are inserted. Quantitative  $\text{ETCO}_2$  monitoring can be used as a surrogate



**Figure 4.** Tracing of end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) during chest compressions, showing decreasing ETCO<sub>2</sub> levels as rescuer fatigues (arrows 1–8) and sudden increase in ETCO<sub>2</sub> during compression when spontaneous circulation returns (9). Adapted from Kalenda.<sup>38</sup>

marker of effective blood flow through the lungs during chest compressions. ETCO<sub>2</sub> levels are used to confirm that compressions are generating blood flow and can be measured with any airway: mask, LMA, or ETT. ETCO<sub>2</sub> levels >10 mm Hg are associated with higher likelihood of ROSC.<sup>33–35</sup> If the ETCO<sub>2</sub> is <10 mm Hg, efforts should be made to improve compressions and increase preload by administering a vasopressor to tighten the vasculature and improve blood return to the thorax and by administering fluid to increase the circulating blood volume.

Other methods of monitoring the effectiveness of CPR may be available in the perioperative setting. An arterial catheter can be monitored to determine the diastolic blood pressure, which, during chest compressions, has been shown to correlate with myocardial blood flow and likelihood of ROSC. Diastolic pressure resulting in coronary perfusion pressure (relaxation intraaortic pressure – relaxation right atrial pressure) <15 mm Hg in adults during chest compressions was associated with no ROSC.<sup>36</sup> In that same study, the maximal aortic relaxation pressure (the equivalent of diastolic blood pressure) was significantly higher in patients with ROSC (35 mm Hg) than in those without ROSC (24 mm Hg). A central venous catheter can be used to determine the central venous saturation. In adult patients, a central venous saturation of <30% was associated with no ROSC.<sup>37</sup> Although these invasive monitors may have some utility if they are available, it is not recommended to interfere with resuscitation efforts to insert an arterial or central catheter solely to measure effectiveness of CPR. It is also important to note that the palpation of pulses during CPR, a method commonly believed to represent effective compressions, may lead to the misinterpretation of retrograde venous pulsations as arterial flow because of adjacent vascular structures. Overall, the ETCO<sub>2</sub> may be the most readily available measure of the effectiveness of chest compressions to produce blood flow.

Anesthesiologists are well aware that ETCO<sub>2</sub> monitoring is also useful for confirming airway and ventilation. The continued presence of ETCO<sub>2</sub> during resuscitation efforts indicates that the ETT remains in the trachea. Absent ETCO<sub>2</sub>

during resuscitation should raise the suspicion of inappropriate placement of the ETT or lack of pulmonary blood flow (pulmonary embolism, ineffective compressions, or prolonged arrest).

The ETCO<sub>2</sub> level should be used to determine if the compressor is becoming fatigued, because the ETCO<sub>2</sub> level decreases as the effectiveness of compression decreases (Fig. 4). Likewise, it can be used to compare the effectiveness of a new compressor with that of the previous compressor at the 2-minute switch interval.<sup>38</sup> The next rescuer to deliver compressions should try to maintain or exceed the ETCO<sub>2</sub> achieved by the previous compressor. Anesthesiologists also can use ETCO<sub>2</sub> level during CPR to determine whether ROSC occurs without interrupting compressions.<sup>30,39,40</sup> A sudden increase in ETCO<sub>2</sub> during compressions indicates that native circulation has returned.

### Open-Chest CPR

Open-chest CPR may be indicated during situations that independently require the chest to be opened, for example, penetrating trauma. It may also be indicated if closed-chest CPR is unlikely to be successful, such as in patients with significant aortic stenosis. Depending on the patient and surgeons involved, the application of open-chest CPR may be more appropriate in the OR. There are no specific indications for open-chest CPR in pediatric patients.

### Extracorporeal Cardiac Life Support

ECLS requires ECMO, CPB, or CPS equipment and may be helpful when resuscitation efforts are promising but likely to be prolonged. Unfortunately, these modalities often require 30 to 45 minutes to initiate because they require mobilizing a team and priming equipment. Facilities with the capability to use these modalities should consider activation early, such as when a second dose of epinephrine is required for lack of ROSC (approximately 5 minutes of resuscitation). It is better to resuscitate a patient and send the team away than to call the ECMO team late and have the added mobilization time contribute to the low-flow period that the patient experiences during CPR. Arrests in the OR



are good opportunities to use this technology because early recognition of arrest combined with rapidly initiated, high-quality resuscitation efforts leads to better flow for longer periods and allows for better outcome for a patient going onto extracorporeal support. Extracorporeal CPR may be especially helpful when either hyperkalemia or local anesthetic toxicity is involved. Extracorporeal CPR can support the circulation for a prolonged period while the toxic agents are metabolized or eliminated. Patients receiving chest compressions cannot undergo dialysis, but if a patient is on ECMO support, a dialysis cartridge can be added to the circuit and used for the rapid elimination of potassium. Children with local anesthetic toxicity or hyperkalemia often have prolonged or repeated arrests until the toxins are eliminated from the body.

### Applying PALS Assignments of Team Roles and Responsibilities in the OR

Resuscitation is a complex skill performed under stressful conditions. The complexity and stress encountered during a pediatric resuscitation can contribute to both a decrease in effectiveness and an increase in errors. To address these concerns, one can develop and train a team to have designated roles and responsibilities and improved communication skills.

The usual resuscitation begins with a call for help in the form of "Any anesthesiologist to operating room...." Ideally, after this call for help, the appropriate additional staff would arrive and be assigned roles for resuscitation. Often, however, more personnel arrive than are needed, and neither a team leader nor team member roles are assigned. The noisy chaos that follows can make an already difficult situation worse. Practicing who should respond to a call for help, designating a team leader, and assigning roles for the resuscitation team can help prevent this chaotic situation.

The suggested resuscitation roles from AHA PALS training can be modified to make the 6 roles and responsibilities appropriate for OR staff.

The *Leader* has responsibility for overall management and also for determining an etiology of the arrest. This position may be most appropriate for the anesthesiologist who was monitoring the patient's physiology before the arrest. The assignment of this role is maintained for the entire resuscitation. The leader should only oversee management and not assume any of the other roles.

The *Airway* role may be assigned to a second anesthesiologist, if one is available, who can decide whether the airway is adequate and whether hand ventilation is preferable to mechanical ventilation. The team member in this role should also monitor ET $\text{CO}_2$  to establish that the airway placement continues to be appropriate and report the saturation and ET $\text{CO}_2$  levels to the leader and recorder every 2 minutes or after a sudden change. The person in this role may also be in the ideal position to apply ice to the child's head if doing so is deemed appropriate.

The *Access/Meds* role may be assigned to a surgeon, anesthesiologist, or nurse who will obtain any additional access, IV or IO, and administer medications and fluids as directed by the team leader. The individual in the access role should report each medication and postmedication flush to both the recorder and leader at the time of administration.

Closed-loop communication should be used (recipient repeats the message in a way that shows the sender it was received and understood).

The *Monitor/Defibrillator* role may be assigned to a surgeon, anesthesiologist, or nurse who notes the child's rhythm during pauses in compression and administers shocks as needed. Depending on the area involved in the surgery and whether the chest needs to remain sterile, a surgeon who is already scrubbed and remaining sterile may be preferred.

The *Compressor* role may preferably be assigned to a surgeon who is scrubbed and sterile because he/she may need to maintain a sterile field. As with this role outside the OR, fatigue is a concern, and rotation should occur every 2 minutes, if possible, to maintain the quality of the chest compressions. The compressor needs to be familiar with the 2-thumb encircling technique for infants, prone compressions for prone children, and the use of ET $\text{CO}_2$  to monitor the effectiveness of the CPR.

The *Recorder* role may be assigned to a nurse or anesthesiologist. A more specific recording tool than the anesthesia record would be helpful to track interventions delivered for review by the team and to prompt future interventions at the appropriate time to reduce the chance that they are missed or delayed (Appendix 1). The recorder should track and announce the 2-minute compressor change and rhythm check, the medications administered and when they are due again, the timing of shocks delivered, and when ECMO should be activated.

Additional roles that are common to arrest situations include a *Gofer*, who is responsible for delivery of necessary items, *Security*, who is responsible for controlling the crowd and noise and asking unwanted participants to leave, and a *Parental Advocate*, who accompanies the child's parent (if present), explains the team's efforts, and monitors the parent's response to the situation.

The 8 communication skills described in the AHA PALS training also should be incorporated into the OR environment for pediatric resuscitations. These skills include: clear roles and responsibilities, mutual respect, clear messages, closed-loop communication, knowing (and sharing) one's limitations, knowledge sharing, constructive intervention, and reevaluation and summarizing.

## 4. TREATMENT FOR SPECIFIC PEDIATRIC SITUATIONS

### Arrest During Inhaled Induction

Children often receive general anesthesia by breathing anesthetic gas through a mask to avoid the stress of IV access while awake. This type of inhaled induction can lead to a cardiac arrest in the event of laryngospasm or relative overdose of anesthetic gas.

Laryngospasm during an inhaled induction should be treated by discontinuing nitrous oxide administration and ventilating with 100% oxygen. Inhaled drug may be continued if one is attempting to deepen anesthetic and the patient is not pulseless. The administration of continuous positive airway pressure and the use of an oral airway may be helpful during ventilation attempts. If the patient is flaccid, then intubation should be attempted; if the patient's tone prevents an intubation attempt and there is no IV



access, the anesthesiologist should consider IM or submental atropine 0.02 mg/kg (0.1 mg minimum dose) and IM or submental succinylcholine 4 mg/kg (maximum dose 150 mg). If the patient has IV or IO access, then a lower dose of succinylcholine should be used to break laryngospasm (0.3–1.0 mg/kg). Additionally, if the child has IV access, IV anesthetic drugs can be tried to break laryngospasm in a hemodynamically stable patient. Nondepolarizing muscle relaxants may also be used, but time to onset and duration of action will be longer. Chest compressions may be needed to circulate the medications if cardiac output is low. If IV or ETT access is unavailable and the child continues in arrest, then IO access should be established immediately and the appropriate resuscitation algorithm followed.

Cardiac arrest secondary to inhaled drug relative overdose may occur because of unrecognized hypovolemia (prolonged fast or bowel prep) or if the anesthesiologist fails to appreciate the depth and duration of administration. Hypothermia, opioid or clonidine treatment, acute alcohol ingestion, and age younger than 1 month are factors that cause anesthetic drug potency to be increased and may contribute to overdose and arrest. Treatment includes discontinuation of nitrous oxide and the inhaled drug and supporting ventilation with 100% oxygen. If the patient is pulseless, the provider should start chest compressions and intubate the patient. Intubation of the trachea may stimulate a sympathetic response that improves hemodynamics. Placing an ETT also will allow access for endotracheal epinephrine (100 µg/kg) if IV or IO access is not available. Atropine may be administered IM or submental before intubation if the patient is bradycardic or for bradycardia prophylaxis if the patient is <1 year old. The provider should obtain IV or IO access and follow the appropriate resuscitation algorithm.

### Arrest in a child with a VP Shunt Malfunction

A common pediatric surgical emergency is VP shunt malfunction with increased intracranial pressure (ICP). If a child with increased ICP suffers a cardiac arrest, the neurosurgeon should immediately tap the VP shunt to remove cerebrospinal fluid (CSF) and reduce ICP. Because increased ICP limits brain perfusion during resuscitation, it is important to tap the VP shunt at the beginning of CPR. During chest compressions (without increased ICP) one-third of the intrathoracic pressure generated may be transmitted to the ICP via the vertebral veins and CSF.<sup>41</sup> This transmission of intrathoracic pressure to ICP is increased when intracranial compliance is reduced and ICP is already increased, thus greatly diminishing cerebral perfusion during CPR.<sup>42</sup> In patients with increased ICP, resuscitation efforts may succeed in perfusing the heart but may significantly underperfuse the brain, resulting in potentially devastating results when ROSC finally occurs (emphasizing the need to have CSF removed during resuscitation).

### Posterior Spinal Fusion and Craniofacial Reconstruction Surgeries

Cardiac arrest during spine and cranial surgeries is often associated with hypovolemia or venous air embolism. The hypovolemia may result from unrecognized blood loss or inadequate access for intravascular volume replacement. Central venous access enables the provider to

monitor preload and provides access for intravascular volume replacement. Tachycardia may not be a reliable indicator of hypovolemia for young children under general anesthesia. Tachycardia was not associated with hypotension thought to be secondary to hypovolemia during craniofacial surgery in children younger than 24 months.<sup>8</sup>

Differentiating hypovolemia from venous air embolism may be difficult. Either may cause a rapid decrease in arterial blood pressure that results in a pulseless electrical activity (PEA; no pulse detectable and no pressure by arterial blood pressure monitoring) or a pseudo-PEA (no peripheral pulses detectable but still some central pressure generated, usually inadequate). In the case of PEA, no ET<sub>CO</sub><sub>2</sub> is detected, and with pseudo-PEA, ET<sub>CO</sub><sub>2</sub> is greatly reduced. Detection of end-tidal nitrogen or of bubbles with Doppler indicates venous air embolism. Increased CVP would more likely indicate an embolism in the pulmonary vasculature, whereas decreased CVP is associated with hypovolemia. Another condition associated with increasing CVP and PEA-like physiology is tamponade from central line complication.

The treatment for venous air embolism includes informing the surgeons, administering 100% inspired oxygen, discontinuing nitrous oxide and inhaled drugs, stopping air entry (lowering surgical site below the level of the heart, irrigating/sealing wounds, increasing intravascular pressure with Trendelenburg/fluid administration), reducing air entry into the pulmonary circulation (left side down to trap air in the right side of the heart), and aspirating air from the central line. CPR and vasopressor administration may be needed.

When children undergoing surgery experience cardiac arrest in the prone position, the delivery of compressions with the patient prone should be considered as an option until the patient can be turned supine. Posterior compressions may be effective if the patient has support under the sternum. In the case of a midline posterior incision, a hand may be placed on the ribs on either side of the incision (Fig. 3A). Otherwise, compressions may be applied with 1 hand over the spine (Fig. 3B).<sup>17,18</sup> A fist or a sandbag placed under the sternum has been described, but it is not clear that an object under the sternum increases effectiveness.<sup>19,43</sup>

### Transfusion-Related Hyperkalemia

Pediatric patients, particularly infants, are at risk for perioperative cardiac events caused by hyperkalemia (potassium >6 mEq/L or electrocardiogram changes) associated with RBC transfusion, especially with large-volume transfusions. Numerous case reports<sup>44–48</sup> and case series studies<sup>49,50</sup> describe fatal and nonfatal transfusion-related hyperkalemia in children. In 1 study, hyperkalemia secondary to transfusion of stored blood was the second leading cardiovascular cause of intraoperative cardiac arrest.<sup>1</sup> Furthermore, cardiac arrests during noncardiac operations have been reported to be related to hyperkalemia in 19% of pediatric patients.<sup>50</sup> Transfusion-related hyperkalemia appears to be associated primarily with rapid or large-volume RBC transfusion. Routine transfusions in critically ill children are not associated with changes in potassium concentration.<sup>51</sup> The risks of transfusion-related hyperkalemia are increased when the RBCs administered have an older shelf life or have been irradiated to prevent a graft-versus-host reaction. Extracellular

potassium concentration in stored RBC products increases with duration of storage at the rate of approximately 1 mmol/d to a mean of 38 mmol/L after 30 days and to as high as 78.5 mmol/L in RBCs stored in CPDA-1 preservative for 35 to 42 days.<sup>46</sup> Additional increases can occur in RBC products after irradiation, which can cause potassium levels to increase to >20 mEq/L after 1 day.

Hyperkalemia may result in ventricular arrhythmias and ventricular fibrillation or asystole, particularly if the potassium increases very quickly. The treatment of hyperkalemia includes driving K<sup>+</sup> into cells and removing the potassium from the body. When the burden of potassium in the body is high, rearrest is common as K<sup>+</sup> reexits cells. Removal of potassium from the body is the better option but requires adequate perfusion of the kidney for diuretics to work or of the intestine for sodium polystyrene (Kayexalate, Sanofi-Aventis, Malvern, PA) to be effective. The use of ECMO is an alternative, and when it is equipped with a dialysis cartridge, ECMO can be used to remove K<sup>+</sup> from the body in the absence of native cardiac output.

The acute resuscitation to drive potassium into cells and reduce cardiotoxicity includes:

Alkalosis	Hyperventilation (can see immediate decrease in T waves) NaHCO <sub>3</sub> 1–2 mEq/kg IV or IO
Calcium	CaCl <sub>2</sub> 20 mg/kg or Calcium Gluconate 60 mg/kg IV or IO
Glucose/insulin	D <sub>25</sub> W 2 mL/kg and regular insulin 0.1 U/kg

Additional methods to treat hyperkalemia in stable patients include the use of an inhaled β-agonist to drive K<sup>+</sup> into cells (nebulized albuterol) and forced diuresis with IV administration of furosemide and saline.

### Local Anesthetic Toxicity

Local anesthetic toxicity may be more difficult to recognize in children than in adults because, unlike adults, children are usually under general anesthesia during placement of the local anesthetic and are unable to report the symptoms associated with increasing systemic levels. The central nervous system changes (agitation, confusion, twitching, and seizures) would likely be masked under general anesthesia, particularly if the patient has received muscle relaxants. The first signs of toxicity may be electrocardiogram changes with prolonged PR interval, progressive bradycardia, and cardiac conduction block leading to hypotension, decreased contractility, and asystole. If seizures are evident, immediate treatment with benzodiazepines is recommended.<sup>52</sup> If cardiac arrest occurs, chest compressions should be started and epinephrine should be given, preferably at low initial doses (1 μg/kg based on adult recommendations).<sup>52</sup> Standard doses of epinephrine and vasopressin are not recommended initially as they decreased the efficacy of lipid emulsions in animal studies.<sup>52</sup> For arrhythmias, the recommended antiarrhythmic drug is amiodarone; lidocaine and procainamide are not recommended because these antiarrhythmics are also local anesthetics that block sodium channels, and their administration may cause additive toxicity.

Based primarily on animal studies and human case reports, administration of IV lipid emulsion (ILE) is 1 of

the recommended treatments for bupivacaine overdose.<sup>53,54</sup> There are also case reports of ILE use in children for treatment of toxicity to bupivacaine<sup>55,56</sup> and ropivacaine.<sup>57</sup> ILE rescue is not only of possible benefit for potentially life-threatening cardiotoxicity from amide anesthetics (bupivacaine, mepivacaine, ropivacaine) but also from other medications (haloperidol, tricyclic antidepressants, β-blockers, and calcium channel blockers).<sup>58</sup> The lipid treatment protocol includes administering a 1.5 mL/kg bolus of 20% intralipid over 1 minute and then immediately beginning intralipid infusion at 0.25 mL/kg/min for 10 minutes after hemodynamic stability is reached. If hemodynamic stability is not achieved, then an additional 1.5 mL/kg bolus should be administered, and the infusion rate should be increased to 0.5 mL/kg/min. A total dose of 10 mL/kg of lipid emulsion over 30 minutes is recommended as the maximum for initial management.<sup>52</sup> CPS, ECMO, or CPB should be considered to support patients, because they frequently rearrest until the local anesthetic is metabolized. Patients have survived and returned to baseline when CPR was continued during transport to a facility that provides ECMO services. Propofol is not a proper substitute for lipid emulsion.

### Anaphylaxis

Anaphylaxis may present as sudden cardiovascular collapse in the anesthetized child. Mortality of patients under anesthesia from immediate-hypersensitivity reactions is in the range of 3% to 9%.<sup>59</sup> Manifestations of anaphylaxis include hypotension, rash, bronchospasm, pulmonary edema, pulmonary hypertension, arrhythmias, increased peak inspiratory pressures, hypoxemia, stridor, hives, and angioedema. The common causes of anaphylaxis in the OR are similar in adults and children<sup>60</sup> and include neuromuscular blocking drugs (63%), latex (14%), hypnotics (7%), antibiotics (6%), plasma substitutes (3%), and morphine-like substances (2%).<sup>41</sup> Treatment of anaphylaxis involves stopping or removing the likely allergens, preparing to stop surgery, reducing or discontinuing potent anesthetics, administering 100% oxygen, using epinephrine (the most important intervention, which can be given as 10 μg/kg/dose IM [assumes adequate perfusion] up to 0.5 mg/dose q 20 minutes or an IV infusion of 0.1 to 1 μg/kg/min), IV fluids (boluses of 20 mL/kg), Trendelenburg positioning, histamine blockers (H1 antagonist diphenhydramine as 1–2 mg/kg, 50 mg maximum, and the H2 antagonist ranitidine, also as 1–2 mg/kg with 50 mg maximum), albuterol for wheezing, and corticosteroids (methylprednisolone 2 mg/kg, maximum 60 mg, or hydrocortisone 2 mg/kg, maximum 100 mg). Serum tryptase level is a useful indicator that mast cell degranulation occurred. The test for serum tryptase is time sensitive and needs to be obtained within 6 hours. When available, a plasma histamine level may increase diagnostic accuracy, but it has a narrower window and should be drawn within 30 minutes if possible, 2 hours at most.

## 5. POSTRESUSCITATION MANAGEMENT

### Maintain Normotension

In the postresuscitation period, patients typically have a hyperadrenergic response as endogenous and exogenous catecholamines are still surging. This brief hyperdynamic state is often followed by a hypotensive slump that may

last for several hours and may require hemodynamic support. It is unclear whether the hyperdynamic phase has as deleterious an effect in children as it would in adults with ischemic heart disease. While pain management needs to be considered, the provider should anticipate the subsequent hemodynamic instability and use caution with treatment administered during the usually transient hyperdynamic phase. The subsequent slump with hemodynamic instability often requires treatment with fluids, vasopressors, and metabolic stabilization.

### Temperature Maintenance

Children often become hypothermic from fluid administration and exposure during resuscitation efforts. Postresuscitation hypothermia may have protective effects and has been shown to be associated with improved outcome in comatose adults who experience ventricular fibrillation arrest.<sup>61,62</sup> Studies are underway to investigate whether hypothermia is also protective in children with cardiac arrest. Active rewarming is likely to be associated with overshoot, causing hyperthermia. Passive rewarming is less likely to result in hyperthermia and should be allowed if during an arrest the child is hypothermic but at a temperature above 32°C. Hyperthermia is problematic because it may increase brain metabolic needs during a period when the brain is at risk for additional ischemic injury. For children whose temperature decreases <32°C, the risk of arrhythmias may warrant active rewarming to 32°C, a temperature less associated with arrhythmias in children.

### Oxygen Saturation Maintenance

During the postresuscitation period, as the brain and heart recover from ischemia, additional hypoxia may be poorly tolerated. It is prudent to monitor the patient closely to ensure that additional hypoxia is avoided. Hyperoxia is another theoretical concern during the postresuscitation period because of the potential for oxygen radical production with reperfusion. If arterial oxygen levels can be measured easily, then hyperoxia should also be avoided in the postresuscitation period by maintaining arterial saturation in the 94% to 99% range.<sup>27</sup>

### Avoid Hyperventilation

Intrinsic positive end-expiratory pressure and hypocarbia can be caused by hyperventilation during postresuscitation management. Increased intrathoracic pressure may limit venous return, and hypocarbia and alkalosis may reduce brain perfusion in this vulnerable period. Hyperventilation should be avoided unless there is concern for cerebral herniation.

### Maintain Normoglycemia

It is unclear whether hyperglycemia in children in the postresuscitation period has an effect on outcome. It is not unusual for endogenous or exogenous catecholamines to cause transient hyperglycemia after a cardiac arrest. When deciding to treat hyperglycemia, care should be taken to avoid overtreatment that leads to hypoglycemia. Frequent monitoring is recommended.

### Breaking Bad News to Parents/Families

Outside of the OR, in areas such as emergency departments and intensive care units, parents may be present and

supervised during resuscitation efforts for their children. It is unclear whether this practice is applicable to the OR and whether parents would benefit from being present at a child's arrest in the OR. Anesthesiologists generally have little experience with breaking bad news to parents because pediatric perioperative arrest is such an infrequent occurrence. It is unknown whether parents would benefit from updates when their child's condition deteriorates and whether such updates lessen their response to subsequent bad news delivered by the OR team. It may be helpful for the team to have a debriefing session to discuss how events will be presented to the parents. The surgical team may have more of a relationship with the parents than do the anesthesiologists or nurses, but the cause of the arrest may be related to the responsibilities of any team member. Being clear on how the information is presented to the family may prevent later confusion and anger.

### Team Debriefing

Debriefing is another important postresuscitation activity. Team members should discuss the sequence of the arrest and resuscitation, ways to prevent such events, and how to improve the team's response to future resuscitations. Another important function of debriefing is allowing individuals to express feelings regarding such events. The emotional impact on the team may be eased if feelings are expressed and dealt with at this meeting. At this time or at a later meeting, a review of the knowledge and skills used during the arrest can serve as a learning opportunity to improve team response in future resuscitations. Reviewing actual resuscitation data improves subsequent performance and outcomes.<sup>63</sup>

### SUMMARY

Pediatric perioperative life support requires teamwork from nurses, surgeons, and anesthesiologists who may work together infrequently and may not have the opportunity to practice or engage in resuscitation efforts together. It is important in this setting to understand the likely causes of perioperative arrest in children, how to prepare for them, what skills are needed, what resources are available, and how to practice and maintain these infrequently used skills. Typical PALS courses may emphasize prehospital management without covering information that pertains to perioperative resuscitation. Anesthesiologists should take the responsibility to understand and share the knowledge needed to maximize pediatric perioperative resuscitation, such as effective monitoring of CPR efforts and management of anesthetic overdose, laryngospasm, hyperkalemia, local anesthetic toxicity, VP shunt complications, Williams syndrome, prolonged QT syndrome, arrest in the prone position, and the institution of ECLS. The anesthesiologist is in an optimum position to incorporate (1) the typical PALS training, including universal resuscitation techniques and medication administration; (2) expertise in airway management, pharmacology, physiology, and technology; and (3) the knowledge presented here, in specific perioperative resuscitation situations. By incorporating all 3 aspects, anesthesiologists can best help direct perioperative resuscitation efforts when 1 of these infrequent and terrifying scenarios occurs during the care of a child. ■

**APPENDIX 1: PERIOPERATIVE ARREST RECORD/COGNITIVE AID (PARCA)**

Included is an example of a perioperative arrest record (designed by authors) that also serves as a cognitive aid. The recorder enters data from the arrest into the open boxes in the record at the designated times. These open boxes also

indicate when actions are recommended, such as changing compressors, administering epinephrine, calling for ECMO, etc. Closed boxes indicate that no action is required at that time interval. The top half of the record is a checklist that includes the early resuscitation response to arrest actions listed in the section "Recognizing the need for resuscitation."

The Perioperative Arrest Record/Cognitive Aid is shown below.

**Perioperative Arrest Record / Cognitive Aid (PARCA): Demo**

**Time CPR starts (computer screen time):** \_\_\_\_\_ **Pt Name:** \_\_\_\_\_ **Weight:** \_\_\_\_\_ kg

**Date:** \_\_\_\_\_ **Allergies:** \_\_\_\_\_

Stop surgery; cover incision	100% O2	Wound lower than heart	Consider head cooling
Call 'Arrest Team'	Inhalational agents off	Flood field if above heart	Call 5-5260 for ECMO/PICU
Code Cart in room	Anesthetic infusions off	PCA, TPN stopped	Call 3-8594 for OLHN
Backboard under pt	Open IV fluids	Catheters out of heart	Call 3-3081 for Cardiology
Defibrillator pads on pt	Check ETT	Consider chest Xray	

**Name of Person Recording Data:** \_\_\_\_\_ **Name of Arrest Leader:** \_\_\_\_\_

Minute	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Compressor initials																				
EtCO2 mmHg																				
Compressions/min																				
Ventilations/min																				
Rhythm																				
Pt HR if present																				
Pt BP if present																				
Temp (site: )																				
Defibrillate (2-4 J/kg)																				
Epi (10 µg/kg IV/IO)																				
Call ECMO																				
PICU consult																				
Review with Leader																				
Other meds:																				

ETT = endotracheal tube; PCA = patient-controlled analgesia; TPN = total parenteral nutrition; ECMO = extracorporeal membrane oxygenation; OLHN = otolaryngology head and neck surgery; EtCO2 = end-tidal CO<sub>2</sub>; Pt HR = patient (native, not compression) heart rate; Pt BP = patient (native, not compression) blood pressure; Epi = epinephrine; IO = intraosseous; PICU = pediatric intensive care unit.



**APPENDIX 2: MEDICATIONS FOR PERIOPERATIVE ARREST: PEDIATRIC DOSING**

Remember to use a “flush” of normal saline when administering a medication by peripheral venous or intraosseous access during a low-flow state such as CPR. A flush should also be used when administering medications in a femorally inserted central venous catheter whose tip is below the diaphragm, as there is very little subdiaphragmatic blood

flow when adrenergic tone is high and blood flow is low. A normal saline bolus should follow each medication dose administered. The normal saline bolus should be either a minimum of 0.25 mL/kg,<sup>31</sup> or 5 mL for an infant, 10 mL for a child, and 20 mL for an adolescent.

Also, intraosseous medication administration is not limited and all medications, including epinephrine, adenosine and blood products, can be administered by this route.

Medication	Dosing suggestions
Adenosine	Initial, 0.1 mg/kg IV or IO, rapid push (maximum dose 6 mg) Repeat, 0.2 mg/kg IV or IO, rapid push (maximum dose 12 mg) Repeat, 0.3 mg/kg IV or IO, rapid push (maximum dose 12 mg)
Amiodarone	Initial, 5 mg/kg IV or IO (maximum 300 mg) Repeat, same dose (maximum total dose 15 mg/kg) Push if pulseless; if patient has pulse give over 20–60 min to prevent collapse Caution with other drugs that prolong QT interval (procainamide)
Atropine	Initial, 0.02 mg/kg IV or IO or 0.04–0.06 mg/kg in ETT Minimum dose 0.1 mg Maximum single dose 0.5 mg child, 1 mg adolescent Repeat, same dose (maximum total dose 1 mg child, 3 mg adolescent)
Calcium chloride	20 mg/kg IV or IO (maximum 2 g) Administer slowly, through central access if available May need to push if pulseless from hyperkalemia
Dantrolene	2.5 mg/kg rapidly through large-bore IV Repeat q 5 min until response 10 mg/kg is usual limit but more may be necessary May be at risk for recrudescence for 12 h Use for malignant hyperthermia
Diphenhydramine	1–2 mg/kg IV, IO, or IM (maximum 50 mg) Repeat q 4–6 h Use for anaphylactic shock
Epinephrine	10 µg/kg IV or IO (max 1 mg) or 100 µg/kg in ETT (maximum 2.5 mg) Repeat q 3–5 min
Glucose (dextrose)	0.5–1 g/kg IV or IO Newborn use D10W 5–10 mL/kg Infants and children use D25W 2–4 mL/kg Adolescents use D50W 1–2 mL/kg Use for hypoglycemia
Hydrocortisone	2 mg/kg IV (maximum 100 mg) Use for adrenal insufficiency
Insulin (regular)	0.1 unit/kg IV with D25W 2 mL/kg IV over 30 min Repeat in 30 min to 1 h Use for hyperkalemia
Intralipid 20%	1.5 mL/kg bolus IV over 1 min and infusion at 0.25 mL/kg/min Continue infusion for 10 min of hemodynamic stability If no hemodynamic stability after 3–5 min, repeat 1.5 mL/kg bolus Increase infusion to 0.5 mL/kg Maximum dose is 10 mL/kg in 30-min period Use for local anesthetic toxicity
Lidocaine	1 mg/kg IV or IO (maximum 100 mg) or 2–3 mg/kg ETT Infusion 20–50 µg/kg/min IV or IO
Magnesium sulfate	25–50 mg/kg IV or IO (maximum 2 g) Push if pulseless or Torsades de Pointes, if pulse give over 10–20 min
Naloxone	0.1 mg/kg IV, IO, ETT, or IM (maximum 20 mg) for toxicity from overdose 0.001–0.005 mg/kg IV, IO, or IM if reversing therapeutic opioid dose
Procainamide	0.25 mg/kg/min IV or IO over 30–60 min (maximum 15 mg/kg) Use with caution with other drugs that prolong QT interval Do not use routinely with amiodarone
Sodium bicarbonate	1 mEq/kg IV or IO slowly May need to push if pulseless from hyperkalemia Ensure adequate ventilation to prevent paradoxical acidosis
Vasopressin	0.5 unit/kg IV/IO (maximum 40 units) for cardiac arrest 0.0003 to 0.002 units/kg/min infusion for catecholamine-resistant shock

Adapted from Kleinman et al.<sup>65</sup>

IO = intraosseous; ETT = endotracheal tube.

**APPENDIX 3: DEFIBRILLATOR KEY POINTS AND HANDS-ON EXERCISES**

External defibrillation	2–4 joules/kg initial dose, 4 joules/kg second dose, 4-10 joules/kg subsequent doses. Used for pulseless arrests with <i>shockable</i> rhythms: V fib and V tach. Biphasic preferred—more effective, less energy needed therefore less injury likely. One shock preferred to stacked, multiple shocks to limit no-flow periods.
External synchronized cardioversion	0.5–1 joule/kg initial dose, 2 joules/kg subsequent doses. Used for SVT (including A fib and A flutter) and V tach when a pulse is present.
Internal defibrillation	2–3 joules as initial dose (start with lowest dose the defibrillator delivers). Use with open chest. Need sterile paddles.
Exercise 1	Defibrillation: Practice with the controls on unit base and then with controls on paddles for units that have controls at both locations. Should be practiced as hands-on to invoke motor skill memory (ideally with manikin that allows safe defibrillator discharge). <ol style="list-style-type: none"> <li>1. Analyze rhythm (if have to stop compressions to analyze rhythm and analysis reveals a shockable pulseless rhythm then restart compressions while setting up and charging defibrillator).</li> <li>2. Apply conductive gel to paddles.</li> <li>3. Turn on defibrillator.</li> <li>4. Select defibrillation (for cardioversion finding the <i>synch</i> button can be difficult, should try turning <i>synch</i> on and off several times).</li> <li>5. Select desired energy dose.</li> <li>6. Charge unit.</li> <li>7. Stop compressions and clear proximity of personnel and oxygen at risk of exposure to shock.</li> <li>8. Discharge unit (if have capability to do so in a safe manner).</li> <li>9. Restart compressions until an ETCO<sub>2</sub> increase indicates the return of a native circulation that allows compressions to be held.</li> </ol>
Exercise 2	Practice switching between adult and pediatric paddles and applying to manikin. Know that 10 kg is limit for most adult paddles and switch to pediatric paddles for patients <10 kg. Know that paddles that are big for the patient and touch will decrease energy delivered to myocardium and increase likelihood of cutaneous burns.
Exercise 3	Practice switching between paddles and gel pads and applying to manikin. Know where gel pads are kept on code cart or how to obtain them. Know where paddles disconnect and gel pads connect to unit, often not well marked. Gel pads can be placed under sterile field in anticipation of need and they facilitate delivery when frequent shocks are anticipated. Gel pads increase ability to have personnel clear.
Exercise 4	Practice switching between external paddles and internal paddles. Know where internal paddles are kept on code cart or how to obtain them. Internal paddles are needed when chest is open and the anatomy is distorted or air spaces disrupt delivery of electricity to myocardium.
Exercise 5	Practice maintaining sterility of paddles when attaching to unit and passing to surgeon. Synchronized cardioversion: <ol style="list-style-type: none"> <li>1. Attach pads and limb leads to patient, if using paddles apply conductive gel.</li> <li>2. Turn defibrillator on.</li> <li>3. Analyze rhythm.</li> <li>4. Select synchronized mode (finding <i>synch</i> button can be difficult, should try turning <i>synch</i> on and off several times).</li> <li>5. Look for markers on R waves.</li> <li>6. Select desired energy dose.</li> <li>7. Charge unit.</li> <li>8. Clear proximity of personnel and oxygen at risk of exposure to shock.</li> <li>9. Discharge unit (if have capability to do so in a safe manner).</li> </ol>
Exercise 6	External (transcutaneous) pacing: <ol style="list-style-type: none"> <li>1. Attach pads and limb leads to patient, if using paddles apply conductive gel.</li> <li>2. Turn defibrillator on.</li> <li>3. Analyze rhythm.</li> <li>4. Select pacer mode.</li> <li>5. Select desired rate.</li> <li>6. Increase energy output (mA) until see consistent capture.</li> <li>7. Validate mechanical capture with blood pressure, pulses and pulse ox response.</li> </ol>

## DISCLOSURES

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**Contribution:** This author helped in study design, data collection, and manuscript preparation.

**Attestation:** Donald H. Shaffner prepared and approved the final manuscript.

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## REFERENCES

- Bhananker SM, Ramamoorthy C, Geiduschek JM, Posner KL, Domino KB, Haberkern CM, Campos JS, Morray JP. Anesthesia-related cardiac arrest in children: update from the Pediatric Perioperative Cardiac Arrest Registry. *Anesth Analg* 2007;105:344–50
- Bharti N, Batra YK, Kaur H. Paediatric perioperative cardiac arrest and its mortality: database of a 60-month period from a tertiary care paediatric centre. *Eur J Anaesthesiol* 2009;26:490–5
- Flick RP, Sprung J, Harrison TE, Gleich SJ, Schroeder DR, Hanson AC, Buenvenida SL, Warner DO. Perioperative cardiac arrests in children between 1988 and 2005 at a tertiary referral center: a study of 92,881 patients. *Anesthesiology* 2007;106:226–37; quiz 413–4
- Gobbo Braz L, Braz JR, Módolo NS, do Nascimento P, Brushi BA, Raquel de Carvalho L. Perioperative cardiac arrest and its mortality in children. A 9-year survey in a Brazilian tertiary teaching hospital. *Paediatr Anaesth* 2006;16:860–6
- Morray JP, Geiduschek JM, Ramamoorthy C, Haberkern CM, Hackel A, Caplan RA, Domino KB, Posner K, Cheney FW. Anesthesia-related cardiac arrest in children: initial findings of the Pediatric Perioperative Cardiac Arrest (POCA) Registry. *Anesthesiology* 2000;93:6–14
- Murat I, Constant I, Maud'huy H. Perioperative anaesthetic morbidity in children: a database of 24,165 anaesthetics over a 30-month period. *Paediatr Anaesth* 2004;14:158–66
- Newland MC, Ellis SJ, Lydiatt CA, Peters KR, Tinker JH, Romberger DJ, Ullrich FA, Anderson JR. Anesthetic-related cardiac arrest and its mortality: a report covering 72,959 anesthetics over 10 years from a US teaching hospital. *Anesthesiology* 2002;97:108–15
- Stricker PA, Lin EE, Fiadjoe JE, Sussman EM, Jobs DR. Absence of tachycardia during hypotension in children undergoing craniofacial reconstruction surgery. *Anesth Analg* 2012;115:139–46
- Barcelona SL, Thompson AA, Coté CJ. Intraoperative pediatric blood transfusion therapy: a review of common issues. Part I: hematologic and physiologic differences from adults; metabolic and infectious risks. *Paediatr Anaesth* 2005;15:716–26
- Gronert GA. Succinylcholine-induced hyperkalemia and beyond. 1975. *Anesthesiology* 2009;111:1372–7
- Rudolph R, Boyd CR. Massive transfusion: complications and their management. *South Med J* 1990;83:1065–70
- von Ungern-Sternberg BS, Boda K, Chambers NA, Rebmann C, Johnson C, Sly PD, Habre W. Risk assessment for respiratory complications in paediatric anaesthesia: a prospective cohort study. *Lancet* 2010;376:773–83
- Apfelbaum JL, Hagberg CA, Caplan RA, Blitt CD, Connis RT, Nickinovich DG, Hagberg CA, Caplan RA, Benumof JL, Berry FA, Blitt CD, Bode RH, Cheney FW, Connis RT, Guidry OF, Nickinovich DG, Ovassapian A; American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology* 2013;118:251–70
- Cavallone LF, Vannucci A. Extubation of the difficult airway and extubation failure. *Anesth Analg* 2013;116:368–83
- Fisher QA, Shaffner DH, Yaster M. Detection of intravascular injection of regional anaesthetics in children. *Can J Anaesth* 1997;44:592–8
- Hanna PG, Gravenstein N, Pashayan AG. *In vitro* comparison of central venous catheters for aspiration of venous air embolism: effect of catheter type, catheter tip position, and cardiac inclination. *J Clin Anesth* 1991;3:290–4
- Dequin PF, Hazouard E, Legras A, Lanotte R, Perrotin D. Cardiopulmonary resuscitation in the prone position: Kouwenhoven revisited. *Intensive Care Med* 1996;22:1272
- Tobias JD, Mencia GA, Atwood R, Gurwitz GS. Intraoperative cardiopulmonary resuscitation in the prone position. *J Pediatr Surg* 1994;29:1537–8
- Mazer SP, Weisfeldt M, Bai D, Cardinale C, Arora R, Ma C, Sciacca RR, Chong D, Rabbani LE. Reverse CPR: a pilot study of CPR in the prone position. *Resuscitation* 2003;57:279–85
- Miranda CC, Newton MC. Successful defibrillation in the prone position. *Br J Anaesth* 2001;87:937–8
- Hunt EA, Vera K, Diener-West M, Haggerty JA, Nelson KL, Shaffner DH, Pronovost PJ. Delays and errors in cardiopulmonary resuscitation and defibrillation by pediatric residents during simulated cardiopulmonary arrests. *Resuscitation* 2009;80:819–25
- Heitmiller ES, Nelson KL, Hunt EA, Schwartz JM, Yaster M, Shaffner DH. A survey of anesthesiologists' knowledge of American Heart Association Pediatric Advanced Life Support Resuscitation Guidelines. *Resuscitation* 2008;79:499–505
- Kleinman ME, Chameides L, Schexnayder SM, Samson RA, Hazinski MF, Atkins DL, Berg MD, de Caen AR, Fink EL, Freid EB, Hickey RW, Marino BS, Nadkarni VM, Proctor LT, Qureshi FA, Sartorelli K, Topjian A, van der Jagt EW, Zaritsky AL. Part 14: pediatric advanced life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 2010;122:S876–908
- Khine HH, Corrdry DH, Ketrlick RG, Martin TM, McCloskey JJ, Rose JB, Theroux MC, Zagnoev M. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. *Anesthesiology* 1997;86:627–31
- Weiss M, Dullenkopf A, Fischer JE, Keller C, Gerber AC; European Paediatric Endotracheal Intubation Study Group. Prospective randomized controlled multi-centre trial of cuffed or uncuffed endotracheal tubes in small children. *Br J Anaesth* 2009;103:867–73
- Cavus E, Bein B, Dörger V, Stadlbauer KH, Wenzel V, Steinfath M, Hanss R, Scholz J. Brain tissue oxygen pressure and cerebral metabolism in an animal model of cardiac arrest and cardiopulmonary resuscitation. *Resuscitation* 2006;71:97–106
- Peberdy MA, Callaway CW, Neumar RW, Geocadin RG, Zimmerman JL, Donnino M, Gabrielli A, Silvers SM, Zaritsky AL, Merchant R, Vanden Hoek TL, Kronick SL. Part 9: post-cardiac arrest care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation* 2010;122(suppl 3):S768–86
- American Heart Association. Summary of key BLS components for adults, children, and infants. In: Chameides L, Samson RA, Schexnayder SM, Hazinski MF, eds. American Heart Association. Pediatric Advanced Life Support: Provider Manual. 2011:149
- Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;318:607–11

30. Garnett AR, Ornato JP, Gonzalez ER, Johnson EB. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. *JAMA* 1987;257:512–5
31. Orlowski JP, Porembka DT, Gallagher JM, Lockrem JD, VanLente F. Comparison study of intraosseous, central intravenous, and peripheral intravenous infusions of emergency drugs. *Am J Dis Child* 1990;144:112–7
32. American Heart Association. Cardioversion (for unstable SVT or VT with a pulse). In: Chameides L, Samson RA, Schexnayder SM, Hazinski MF, eds. American Heart Association. *Pediatric Advanced Life Support: Provider Manual*. 2011:128
33. Callahan M, Barton C. Prediction of outcome of cardiopulmonary resuscitation from end-tidal carbon dioxide concentration. *Crit Care Med* 1990;18:358–62
34. Wayne MA, Levine RL, Miller CC. Use of end-tidal carbon dioxide to predict outcome in prehospital cardiac arrest. *Ann Emerg Med* 1995;25:762–7
35. Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med* 1997;337:301–6
36. Paradis NA, Martin GB, Rivers EP, Goetting MG, Appleton TJ, Feingold M, Nowak RM. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. *JAMA* 1990;263:1106–13
37. Rivers EP, Martin GB, Smithline H, Rady MY, Schultz CH, Goetting MG, Appleton TJ, Nowak RM. The clinical implications of continuous central venous oxygen saturation during human CPR. *Ann Emerg Med* 1992;21:1094–101
38. Kalenda Z. The capnogram as a guide to the efficacy of cardiac massage. *Resuscitation* 1978;6:259–63
39. Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med* 1988;318:607–11
40. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. *Am J Emerg Med* 1996;14:349–50
41. Guerci AD, Shi AY, Levin H, Tsitlik J, Weisfeldt ML, Chandra N. Transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation in dogs. *Circ Res* 1985;56:20–30
42. Halperin HR, Tsitlik JE, Guerci AD, Mellits ED, Levin HR, Shi AY, Chandra N, Weisfeldt ML. Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. *Circulation* 1986;73:539–50
43. Sun WZ, Huang FY, Kung KL, Fan SZ, Chen TL. Successful cardiopulmonary resuscitation of two patients in the prone position using reversed precordial compression. *Anesthesiology* 1992;77:202–4
44. Buntain SG, Pabari M. Massive transfusion and hyperkalaemic cardiac arrest in craniofacial surgery in a child. *Anaesth Intensive Care* 1999;27:530–3
45. Chen CH, Hong CL, Kau YC, Lee HL, Chen CK, Shyr MH. Fatal hyperkalemia during rapid and massive blood transfusion in a child undergoing hip surgery—a case report. *Acta Anaesthesiol Sin* 1999;37:163–6
46. Parshuram CS, Cox PN. Neonatal hyperkalemic-hypocalcemic cardiac arrest associated with initiation of blood-primed continuous venovenous hemofiltration. *Pediatr Crit Care Med* 2002;3:67–9
47. Tsukamoto S, Maruyama K, Nakagawa H, Iwase Y, Kitamura A, Hayashida M. Fatal hyperkalemia due to rapid red cell transfusion in a critically ill patient. *J Nippon Med Sch* 2009;76:258–64
48. Woodforth IJ. Resuscitation from transfusion-associated hyperkalaemic ventricular fibrillation. *Anaesth Intensive Care* 2007;35:110–3
49. Brown KA, Bissonnette B, MacDonald M, Poon AO. Hyperkalaemia during massive blood transfusion in paediatric craniofacial surgery. *Can J Anaesth* 1990;37:401–8
50. Smith HM, Farrow SJ, Ackerman JD, Stubbs JR, Sprung J. Cardiac arrests associated with hyperkalemia during red blood cell transfusion: a case series. *Anesth Analg* 2008;106:1062–9
51. Parshuram CS, Joffe AR. Prospective study of potassium-associated acute transfusion events in pediatric intensive care. *Pediatr Crit Care Med* 2003;4:65–8
52. Neal JM, Mulroy MF, Weinberg GL; American Society of Regional Anesthesia and Pain Medicine. American Society of Regional Anesthesia and Pain Medicine checklist for managing local anesthetic systemic toxicity: 2012 version. *Reg Anesth Pain Med* 2012;37:16–8
53. Weinberg G, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. *Reg Anesth Pain Med* 2003;28:198–202
54. Weinberg G. Lipid infusion resuscitation for local anesthetic toxicity: proof of clinical efficacy. *Anesthesiology* 2006;105:7–8
55. Markowitz S, Neal JM. Immediate lipid emulsion therapy in the successful treatment of bupivacaine systemic toxicity. *Reg Anesth Pain Med* 2009;34:276
56. Wong GK, Joo DT, McDonnell C. Lipid resuscitation in a carnitine deficient child following intravascular migration of an epidural catheter. *Anaesthesia* 2010;65:192–5
57. Ludot H, Tharin J, Belouadah M, Mazoit J, Malinovsky J. Successful resuscitation after ropivacaine and lidocaine induced ventricular arrhythmia following posterior lumbar plexus block in a child. *Anesth Analg* 2008;106:1572–4
58. Cave G, Harvey M, Graudins A. Intravenous lipid emulsion as antidote: a summary of published human experience. *Emerg Med Australas* 2011;23:123–41
59. Mertes PM, Malinovsky JM, Jouffroy L, Aberer W, Terreehorst I, Brockow K, Demoly P; Working Group of the SFAR and SFA; ENDA; EAACI Interest Group on Drug Allergy. Reducing the risk of anaphylaxis during anesthesia: 2011 updated guidelines for clinical practice. *J Investig Allergol Clin Immunol* 2011;21:442–53
60. Karila C, Brunet-Langot D, Labbez F, Jacqmarcq O, Ponvert C, Paupe J, Scheinmann P, de Blic J. Anaphylaxis during anesthesia: results of a 12-year survey at a French pediatric center. *Allergy* 2005;60:828–34
61. The Hypothermia After Cardiac Arrest (HACA) Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346:549–56
62. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Tertridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med* 2002;346:557–63
63. Edelson DP, Litzinger B, Arora V, Walsh D, Kim S, Lauderdale DS, Vanden Hoek TL, Becker LB, Abella BS. Improving in-hospital cardiac arrest process and outcomes with performance debriefing. *Arch Intern Med* 2008;168:1063–9
64. Schwartz JM, Hunt EA, Heitmiller ES, Shaffner DH. Chapter 38: Cardiopulmonary resuscitation. In: Davis PJ, Cladis FP, Motoyama EK, eds. *Smith's Textbook of Anesthesia for Infants and Children*. Philadelphia, PA: Elsevier Mosby, 2011:1200–49
65. Kleinman ME, Chameides L, Schexnayder SM, Samson RA, Hazinski MF, Atkins DL, Berg MD, de Caen AR, Fink EL, Freid EB, Hickey RW, Marino BS, Nadkarni VM, Proctor LT, Qureshi FA, Sartorelli K, Topjian A, van der Jagt EW, Zaritsky AL; American Heart Association. *Pediatric advanced life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care*. *Pediatrics* 2010;126:e1361–99