Pediatric Cardiac Arrest Resuscitation

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KEYWORDS

• Pediatric • Cardiac arrest • Epidemiology • Treatment • Prognosis

KEY POINTS

- Cardiac arrest in children is a rare but devastating presentation that is almost always due to a primary respiratory event that progresses to cardiac failure.
- Early recognition and resuscitation of patients in the "prearrest" states of shock or respiratory failure are critical to successful outcomes.
- As respiratory illness is frequently a precursor to cardiac arrest, meticulous support of oxygenation and ventilation is essential in the critically ill infant or child.
- Both targeted temperature management and extracorporeal membrane oxygenation are not associated with marked improvement in outcome in out-of-hospital cardiac arrest in the pediatric population.

INTRODUCTION

Pediatric cardiac arrest is a rare clinical scenario and has profound impacts on both families and clinical care teams. Current evidence-based resuscitation recommendations and research are focused on intervening in the pre–cardiac arrest phase of critical illness, because of the unique causes that occur in the pediatric population. Knowledge of the anatomic and physiologic differences across the pediatric age spectrum is critical to successful management of these challenging situations. This article focuses on the initial assessment of pediatric patients in the prearrest states of respiratory distress or shock, cardiac arrest management, treatment of special circumstances that lead to cardiac arrest, post–cardiac arrest care, and treatment of select underlying causes of cardiac arrest in the pediatric patient.

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EPIDEMIOLOGY

The incidence of pediatric cardiac arrest follows a bimodal distribution, with the first peak in infancy (2.1 cases per 100,000 person-years) and the second peak in adolescence (1.44 cases per 100,000 person-years).[1] Mortality for out-of-hospital cardiac arrest (OHCA) in the pediatric population is 90% or greater and likely reflects the differences in pathologic condition compared with adults that lead to the arrest.[2]

CAUSE

In contrast to adults, whereby cardiac arrest is typically due to a primary arrhythmia, pediatric patients more commonly experience a respiratory event that leads to hypoxia, acidosis, bradycardia, and arrest. Pulmonary causes of respiratory decompensation that may lead to cardiac arrest are numerous and include acquired conditions (eg, bronchiolitis, pertussis, sepsis, pneumonia, hypoventilation from seizures/status epilepticus) and respiratory failure owing to ingested toxins or nonaccidental trauma. Because of the frequency of respiratory causes of pediatric cardiac arrest, guidelines from the American Heart Association (AHA) and the Pediatric Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) continue to emphasize support of oxygenation and ventilation.[3]

Primary arrhythmias do occur in infants and children, particularly those with underlying congenital or structural heart disease, or those with acquired heart conditions (eg, myocarditis). Channelopathies, congenital prolonged QT syndrome, Wolff-Parkinson-White syndrome, Brugada syndrome, and complex congenital heart disease (eg, Tetralogy of Fallot, transposition of the great arteries, hypoplastic left heart syndrome) have all been implicated in cases of pediatric sudden cardiac death. In up to 50% of cases of arrhythmogenic cardiac arrest, patients report warning or prodromal symptoms that include syncope, presyncope, chest pain, or exercise fatigue.[4–6] Patients also may report a family history of early, unexpected, or unexplained death.

INITIAL ASSESSMENT

The goal of the initial assessment of a critically ill child is to quickly identify life-threatening respiratory failure and shock to prevent the onset of cardiac arrest. Current AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care recommend a systematic approach to assessing the ill or injured child using the Pediatric Advanced Life Support (PALS) algorithm.[7] This systematic approach consists of the initial impression, primary assessment, and secondary assessment. If cardiac arrest is identified at any point during this initial assessment, immediately start cardiopulmonary resuscitation (CPR) with high-quality chest compressions and proceed with the Pediatric Cardiac Arrest Algorithm.[8] Chest compressions in children should occur at a rate of 100 to 120 per minute, compress at least one-third of the anterior-posterior chest diameter, and interruptions should be avoided.

If CPR is not immediately required, a primary assessment consisting of a hands-on evaluation of the patient's cardiopulmonary and neurologic function using the "ABCDE" model should be performed (**Table 1**).[9] Importantly, vital signs, such as heart rate, blood pressure, and respiratory rate, vary by age (**Table 2**).[10] Any life threat identified during the primary assessment (ie, impending respiratory failure) should be addressed immediately.

After a primary assessment, a secondary assessment consisting of a focused history and thorough physical examination should be performed. Diagnostic laboratory

Table 1
"ABCDE" primary assessment model

	Primary Assessment	Signs of a Life-Threatening Condition
A: Airway	Is the airway patent? Is there movement of the chest and abdomen? Are there breath sounds?	Increased inspiratory effort with retractions Snoring or stridor Lack of airway sounds or phonation
B: Breathing	What is the respiratory rate and pulse oximetry? Is there increased work of breathing? Is there chest expansion and air movement? Are there breath sounds? Are they abnormal?	Apnea or bradypnea Significant increased work of breathing (tachypnea, retractions, nasal flaring, head bobbing or seesaw respirations, grunting) Abnormal or absent breath sounds Hypoxia
C: Circulation	What is the heart rate and rhythm? Are peripheral and central pulses present? What is the capillary refill time? What is the skin color and temperature? What is the blood pressure?	Cardiac arrest Poor perfusion (delayed capillary refill, cyanosis, cool skin, mottling) Hypotension Arrhythmia
D: Disability	What is the patient's Glasgow Coma Scale (GCS)? What is the patient's level of consciousness? What is the pupillary response to light?	Unresponsive or depressed GCS Seizures Abnormal pupils (unequal size or poor response to light)
E: Exposure	Is there fever or hypothermia? Are there skin findings to suggest trauma? Is there significant bleeding? Is there a suggestive rash?	Hypothermia Hemorrhage/significant bruising Petechia/purpura

Data from Thim T, Krarup NH, Grove EL, Rohde CV, Løfgren B. Initial assessment and treatment with the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach. Int J Gen Med. 2012;5:117-121. https://doi.org/10.2147/IJGM.S28478.

studies (ie, chemistries, complete blood count, cultures), an electrocardiogram, point-of-care ultrasound, and diagnostic imaging studies (eg, radiograph, computed tomography) may be warranted to further evaluate and identify life-threatening conditions. Some laboratory and imaging studies may need to be repeated to assess the clinical response to interventions (eg, preintubation and postintubation arterial blood gases).

PRECARDIAC ARREST STATES

Respiratory Distress

Basic airway maneuvers for the patient with a patent airway include supplemental oxygen (nasal cannula, oxygen masks), suctioning, and noninvasive positive pressure ventilation. These techniques may be used in spontaneously breathing patients with hypoxemia or respiratory distress.[11] If these prove inadequate, then assisted

Table 2
Normal vital signs by age

Age	Respiratory Rate (10th–90th Percentile)	Heart Rate (10th–90th Percentile)
0–3 mo	34–57	123–164
3–6 mo	33–55	120–159
6–9 mo	31–52	114–152
9–12 mo	30–50	109–145
12–18 mo	28–46	103–140
18–24 mo	25–40	98–135
2–3 y	22–34	92–128
3–4 y	21–29	86–123
4–6 y	20–27	81–117
6–8 y	18–24	74–111
8–12 y	16–22	67–103
12–15 y	15–21	62–96
15–18 y	13–19	58–92

Data from Fleming S, Thompson M, Stevens R, et al. Normal ranges of heart rate and respiratory rate in children from birth to 18 years of age: a systematic review of observational studies. Lancet 2011; 377:1011.

respirations with bag-valve-mask (BVM), insertion of supraglottic airway devices, or intubation may be required. Indications for assisted respirations include severe respiratory distress, depressed mental status, upper-airway obstruction (eg, oropharyngeal burns, anaphylaxis, epiglottitis), or refractory hypoxemia.[12]

Shock

Shock in pediatric patients can have many causes (Table 3). Shock can be characterized as compensated or uncompensated with uncompensated typically defined as the presence of poor perfusion with hypotension.

Compensated shock

Patients with inadequate organ perfusion who maintain adequate blood pressure are said to have "compensated" shock. Early physical examination and laboratory signs of compensated shock include the following:

- Tachycardia
- Tachypnea
- Delayed capillary refill
- Cool extremities
- Orthostatic vital signs
- Decreased urine output
- Depressed mental status (eg, sleepiness or irritability)
- Elevated blood lactate level, acidosis, or increased base deficit

Uncompensated shock

Hypotensive or "uncompensated" shock occurs when compensatory mechanisms fail. In addition to hypotension, patients with uncompensated shock may exhibit worsening tachycardia, mottled extremities, markedly delayed capillary refill, worsening tachypnea (compensatory respiratory alkalosis), and altered mental status. Laboratory

Table 3
Mechanism, cause, features, and treatments of shock

Type of Shock	Mechanism	Common Causes	Features	Treatment
Cardiogenic	Decreased cardiac contractility leading to decreased cardiac output	Arrhythmia Congenital heart disease (ALCAPA) Myocarditis Drug ingestions (eg, cocaine) Metabolic derangements (eg, hypoglycemia)	Elevated HR Decreased BP Delayed capillary refill with cool extremities	Inotropes Cautious use of fluid (functional rather than absolute circulating volume deficit) ECMO
Dissociative	Impaired cellular oxygen delivery or utilization due to presence of a toxic metabolite or drug	Carbon monoxide poisoning Cyanide poisoning Methemoglobinemia	Elevated HR Normal or elevated BP Normal capillary refill	Specific antidotes (call local poison control center) Hyperbaric oxygen therapy
Distributive	Inappropriate vasodilation and peripheral pooling of blood	Sepsis Anaphylaxis Drug ingestion (eg, atypical antipsychotics)	Elevated HR Decreased BP Capillary refill may be flash or delayed Extremities may be warm or cool	Fluid resuscitation Antibiotics if infection suspected Vasopressors Epinephrine for anaphylaxis
Hypovolemic (most common cause of shock in children)	Decreased circulating blood volume	Dehydration (diarrhea, vomiting) Osmotic diuresis (hyperglycemia/DKA) Plasma losses (burns) Hemorrhage	Elevated HR Decreased BP Delayed capillary refill with cool extremities	Fluid resuscitation or transfusion Cessation of bleeding (may require operating room)
Neurogenic	Severely reduced systemic vascular resistance from disrupted sympathetic nerve stimulation	Spinal cord injury Traumatic brain injury	Depressed HR and BP Warm extremities	Fluid resuscitation Vasopressors
Obstructive	Mechanical obstruction to ventricular outflow	Cardiac tamponade Tension pneumothorax Massive pulmonary embolism Left-sided congenital heart disease (HLHS, aortic coarctation, critical aortic valve stenosis)	Elevated HR Decreased BP Delayed capillary refill with cool extremities	Pericardiocentesis for tamponade Needle decompression and chest tube for pneumothorax Prostaglandins (for ductal-dependent congenital cardiac lesions)

Abbreviations: ALCAPA, anomalous left coronary artery arising from the pulmonary artery; BP, blood pressure; DKA, diabetic ketoacidosis; HLHS, hypoplastic left heart syndrome; HR, heart rate.

studies may show signs of progressive end-organ failure, including worsening metabolic acidosis, renal failure, coagulopathy, thrombocytopenia, hyperbilirubinemia, or transaminitis. If uncompensated shock is not addressed, it may rapidly progress into cardiopulmonary failure and arrest. Treatment is specific to the cause of the shock and reversal of the underlying cause (see **Table 3**).

Rapid sequence intubation

Intubation and mechanical ventilation may be necessary in shock states for airway protection and to reduce work of breathing. Caution must be taken in the patient who requires mechanical ventilation in the setting of shock, because positive pressure ventilation may lead to depressed venous return and cardiovascular collapse. The patient may require intravenous (IV) fluid resuscitation or vasopressor administration to maintain cardiac output. These therapies should ideally be administered before rapid sequence intubation (RSI) if time allows. Aggressive fluid resuscitation with balanced crystalloid in 20 mL/kg aliquots before intubation may help mitigate the physiologic perturbations that occur with the transition from negative to positive pressure ventilation. In the absence of contraindications, ketamine is the preferred sedative for RSI in pediatric patients with shock.[13]

CARDIAC ARREST

If a critically ill pediatric patient is noted to be unresponsive with apnea or gasping respirations, feel a pulse for no more than 10 seconds. If no pulse is detected, immediately begin chest compression and proceed with the PALS Cardiac Arrest Algorithm.[14] Refer to the AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care for the complete Pediatric Advanced Life Support Algorithm. A link to the current guideline is provided here: https://www.ahajournals.org/doi/pdf/10.1161/CIRCULATIONAHA.110.971101.

Airway Management

In the pediatric patient with OHCA, BVM followed by intubation conferred no survival benefit when compared with BVM alone.[15] Decreased survival to hospital discharge has also been shown in cases of in-hospital cardiac arrest that are intubated compared with those that receive only BVM.[16] It is therefore reasonable to continue BVM through the duration of the resuscitation if effective oxygenation and ventilation are achieved. If BVM ventilation is ineffective, advanced airway interventions, such as supraglottic airway devices or intubation, should be considered. While managing the airway during cardiac arrest, it is important to avoid excessive ventilation because this may lead to decreased cardiac output and decreased coronary perfusion pressure. Excessive ventilation via BVM may also cause gastric distension, which can impede diaphragmatic excursion and impair ventilation. Gastric decompression with an orogastric or nasogastric tube may be necessary if BVM ventilation is prolonged. When ventilating with a mask, it is important to maintain the recommended ratio of 2 breaths for every 15 compressions, and to give each breath more than 1 second with just enough volume to make the chest rise.[14]

Rhythm Analysis

After the initiation of chest compressions, attach defibrillator pads or a cardiac monitor to analyze the rhythm. Ventricular tachycardia (VT) and ventricular fibrillation (VF) are considered “shockable” rhythms.[14,17]

Nonshockable rhythms, such as asystole and pulseless electrical activity (PEA), are the most common presenting rhythms in both in-hospital and out-of-hospital pediatric

cardiac arrest.[12,14] Rhythms in PEA may have a fast, slow, or normal rate and may be associated with other abnormalities, including conduction delays, prolonged PR or QT intervals, and a widened QRS complex. Potentially reversible causes of cardiac arrest to consider in patients with PEA are known as the "H's and T's" (**Box 1**).

Primary VF is rare in pediatric patients and may quickly degenerate into asystole. VF may be secondary to a cardiac abnormality, channelopathy, sudden impact to the chest, or the "H's and T's."

Patients with VT may present with or without a pulse. Pulseless VT often degenerates into VF and then ultimately asystole. Patients presenting with cardiac arrest whose initial rhythm is shockable (VF or VT) have improved survival and outcome compared with their "nonshockable" counterparts.[13]

Defibrillation

When the presenting rhythm is VF or pulseless VT, early defibrillation is critical to survival. Outcomes are improved when defibrillation is coupled with early, high-quality CPR. An initial defibrillation dose of 2 to 4 J/kg should be administered as soon as the rhythm is deemed shockable. Resume chest compressions immediately after the shock is delivered. If VF or pulseless VT persists at the next rhythm check, the second shock should be given at a dose of 4 J/kg, with increasing doses as needed for refractory shockable rhythms up to a maximum of 10 J/kg and not to exceed the maximum adult dose (200 J biphasic, 360 J monophasic).[18,19]

Pharmacology

CPR should not be interrupted for the administration of medications during cardiac arrest resuscitation. Current AHA guidelines recommend delivery of resuscitation drugs during chest compressions immediately before, or after, shock delivery to allow these agents to circulate before the next rhythm check. Resuscitation medications are calculated based on the child's weight (if known), or with the use of a length-based tape with precalculated dosages. Length-based systems have been shown to reduce the cognitive burden of pediatric resuscitation.[20] Epinephrine increases aortic diastolic pressure and coronary perfusion pressure and is the main pharmacologic agent used to treat cardiac arrest. Epinephrine can be given via the IV or intraosseous route

Box 1
Reversible cause of cardiac arrest (H's and T's)

- Hypovolemia
- Hypoxia
- Hydrogen ion excess (metabolic acidosis)
- Hypoglycemia
- Hypokalemia or hyperkalemia
- Hypothermia or hyperthermia
- Tension pneumothorax
- Tamponade (cardiac)
- Toxin
- Thrombosis (pulmonary or coronary)
- Trauma

at a dose of 0.01 mg/kg or via the endotracheal tube at a dose of 0.1 mg/kg. In patients with a nonshockable rhythm, epinephrine should be given as soon as PEA or asystole is identified (see PALS algorithm for Pediatric Cardiac Arrest step 10). In patients with a shockable rhythm, the same dose of epinephrine is used and should be administered after the delivery of 2 shocks and CPR (see PALS algorithm for Pediatric Cardiac Arrest step 6). Regardless of the rhythm, epinephrine can be repeated every 3 to 5 minutes. **Table 4** provides a summary of additional resuscitation medications that can be used in the treatment of pediatric cardiac arrest and dysrhythmias.[21]

SPECIAL CIRCUMSTANCES

Hypothermic Arrest

Patients with severe hypothermia may be unresponsive to resuscitative efforts until the core body temperature is rewarmed to at least 30°C. CPR should continue until the body is rewarmed to at least this temperature before terminating resuscitation. Active external rewarming techniques include electric blankets, hot water bottles, warm IV fluids, and overhead warmers. Although simple to deploy, these methods may not have a large or rapid effect on rewarming.[22] Body cavity irrigation with warm fluids is a more invasive, yet more effective, technique for rewarming. Extracorporeal circulation is the most rapid and effective technique for rewarming severely hypothermic patients with cardiac arrest. Transfer to a facility with pediatric extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass capabilities should be considered.[22] Survival has been described in patients with prolonged cold-water submersion and resuscitation times up to 40 and 120 minutes, respectively.[14]

Extracorporeal Membrane Oxygenation and Extracorporeal Cardiopulmonary Resuscitation

Beyond its use in hypothermic cardiac arrest, there has been growing interest in ECMO as a method of circulatory rescue in cases of cardiac arrest from other causes and is commonly used in the pediatric population after surgery for congenital heart disease.[23,24] Extracorporeal cardiopulmonary resuscitation (eCPR) is the rapid deployment of venoarterial ECMO during active CPR or for patients with intermittent return of spontaneous circulation (ROSC). In patients who experience an in-hospital arrest, there is an association with favorable neurologic outcomes in the group who received eCPR versus those who were treated with conventional PALS protocols.[25] The ILCOR states that eCPR can be considered for "pediatric patients with cardiac diagnosis who have in-hospital cardiac arrest in settings with existing ECMO protocols, expertise and equipment (Class 2b; Level of evidence C-LD)."[3] Patients who present with OHCA, or those in whom a noncardiac cause for arrest is suspected, should not be considered candidates for eCPR based on the current body of literature.

POSTRESUSCITATION MANAGEMENT

In the early postresuscitation period, the patient should be monitored closely for hypotension and/or arrhythmias that may be a harbinger of recurrent cardiopulmonary collapse. Other goals of postresuscitation care are to preserve neurologic function, prevent secondary end-organ damage, and stabilize the patient for transport to a pediatric tertiary-care facility. Postresuscitation care includes airway management, treatment of shock, glucose management, electroencephalogram (EEG) monitoring, and targeted temperature management (TTM). During this time, the clinician should

Table 4
Drugs of pediatric resuscitation

Medication	Cardiovascular Effects	Indication	Dose	Additional Information
Adenosine	AV nodal conduction block	SupraVT	First dose: 0.1 mg/kg IV or IO (maximum dose 6 mg) Second dose: 0.2 m/kg IV or IO (maximum dose 12 mg)	Follow dose with rapid saline flush Administer in IV as close to the heart as possible (avoid hand or lower extremity)
Amiodarone	Class III antiarrhythmic Slows AV and ventricular conduction Prolongs AV refractory period and QT interval	Cardiac arrest with refractory VF/pulseless VT Stable supraventricular or VT	5 mg/kg IV or IO (maximum dose 300 mg) May repeat dose twice	IV push during cardiac arrest Given over 20–60 min with perfusing rhythm (expert consultation recommended) Do not give with other QT prolonging agents
Atropine	Accelerates sinus or atrial pacemakers Increases speed of AV conduction	Symptomatic bradycardia	0.02 mg/kg IV or IO OR 0.04–0.06 mg/kg ET Minimum dose: 0.1 mg Maximum single dose: 0.5 mg	May repeat dose once if needed
Calcium chloride 10%	Enhanced cardiac automaticity and contractility	Documented hypocalcemia Calcium channel blocker overdose Hypermagnesemia Hyperkalemia	20 mg/kg IV or IO Maximum single dose: 2 g	Administer slowly Not recommended for routine use in cardiac arrest without clear indication
Epinephrine	A1 receptors: increases peripheral vascular resistance B1 receptors: positive chronotropy and inotropy	Cardiac arrest	0.01 mg/kg (0.1 mL/kg 1:10,000) IV or IO 0.1 mg/kg (0.1 mL/kg 1:1000) ET Maximum dose: 1 mg IV/IO or 2.5 mg ET	Repeat every 3–5 min as needed per PALS algorithm
Lidocaine	Class I antiarrhythmic Decreases automaticity Suppresses ventricular arrhythmia	Cardiac arrest with refractory VF/pulseless VT	1 mg/kg IV/IO bolus 20–50 μg/kg/min infusion	Not shown to improve survival to hospital discharge

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Table 4
(*continued*)

Medication	Cardiovascular Effects	Indication	Dose	Additional Information
Magnesium sulfate	Inhibits calcium channels leading to smooth muscle relaxation	Torsades de pointes Hypomagnesemia	25–50 mg/kg IV or IO over 10–20 min Maximum dose: 2 g	Give faster in torsades de pointes Insufficient evidence to recommend for or against routine administration during cardiac arrest
Procainamide	Prolongs refractory periods Depresses conduction velocity	VT with a pulse Supraventricular tachycardia Atrial fibrillation/flutter	15 mg/kg IV or IO	Expert consultation recommended Do not give with other QT prolonging agents

Abbreviations: ET, endotracheal tube; IO, intraosseous.

also continue to investigate and treat the underlying cause of the patient's cardiac arrest.[26]

Airway Management

Postresuscitation airway management includes continuous monitoring of oxygenation and ventilation, and placement of a gastric tube to reduce distention of the stomach (**Table 5**). Sudden decompensation of the intubated child may be due to a DOPE (Dislodged endotracheal tube, endotracheal tube Obstruction, Pneumothorax, or Equipment failure).[26]

Extrapolating from adult studies, recent pediatric resuscitation guidelines advocate that oxygen saturation should be maintained between 94% and 99% after ROSC, with the goal of maintaining normoxia (defined as Pa_{O_2} between 60 mm Hg and 300 mm Hg).[19,27] Although smaller studies of the pediatric population have failed to demonstrate a connection between arterial oxygenation and mortality, a large retrospective study suggests that both hypoxia, and to a lesser extent, hyperoxia are associated with increased mortality in cardiac arrest patients who survive to pediatric intensive care unit admission.[28–31] Given that an oxygen saturation of 100% may correlate with Pa_{O_2} elevations up to 500 mm Hg, it is reasonable to titrate inspired oxygen concentration to maintain the saturations within the target range.

Overventilation after pediatric intubation is common in the prehospital and hospital setting.[32,33] Limited observational data suggest that both severe postresuscitation hypocapnia and hypercapnia may be associated with higher mortality.[29] Unless contraindicated by the patient's condition, current guidelines recommend that Pa_{CO_2} be closely monitored and kept within the target range of 30 mm Hg to 50 mm Hg once ROSC has been achieved.

Treatment of Shock

After ROSC, recurrent shock owing to myocardial dysfunction and vascular instability is common.[34,35] Early derangements in heart rate, blood pressure, urine output, and cardiac rhythm are associated with increased morbidity and mortality.[36–39] Prompt treatment with IV fluids and vasopressors is critical to improve survival and neurologic outcome (**Table 6**). Close monitoring of vital signs, perfusion, and mental status are used to guide fluid and vasoactive medication administration, with the goal to maintain systolic blood pressure above the fifth percentile for age. Currently, there are no studies comparing specific vasoactive agents after ROSC in the pediatric population.

Table 5
Postresuscitation airway monitoring techniques

Postresuscitation Airway Monitoring	Confirmation
Endotracheal tube position	Bilateral chest wall movement Chest radiograph Auscultation of bilateral breath sounds Absent sounds over the stomach Condensation in endotracheal tube Exhaled Co_2
Oxygenation	Continuous pulse oximetry
Ventilation	Continuous $Etco_2$ monitoring Intermittent blood gas measurements

Table 6
Vasoactive agents for postresuscitaiton management of shock

Vasoactive Agent	Dose Range	Indication
Dopamine	2–20 μg/kg/min IV or IO	First line for fluid refractory shock
Epinephrine	0.05–1 μg/kg/min IV or IO	First line for fluid refractory shock
Norepinephrine	0.05–2 μg/kg/min IV or IO	First line for fluid refractory shock
Vasopressin	0.0002–0.004 units/kg/min IV (maximum rate 0.04 units/min IV)	Catecholamine-resistant shock
Dobutamine	2–20 μg/kg/min IV or IO	Shock with normal BP Second-line agent for hypotensive shock
Milrinone	Load: 50 μg/kg IV or IO over 10–60 min (may cause hypotension) Infusion: 0.25–1 μg/kg/min IV or IO	Shock with normal BP

Glucose Management

Hyperglycemia should be avoided after resuscitation, and studies suggest that both peak blood glucose level and duration of hyperglycemia are predictors of mortality in the critically ill child.[40,41] In addition, hyperglycemia may lead to an osmotic diuresis that can exacerbate hemodynamic instability. Conversely, there is increased mortality after cardiac arrest in nondiabetic patients with hypoglycemia (blood glucose <70 mg/dL). Therefore, low blood glucose should also be corrected.[42]

Electroencephalogram Monitoring

Hypoxic ischemic brain injury secondary to cardiac arrest may lead to seizures, status epilepticus, and even brain death. Seizures and the absence of reactivity on EEG are associated with increased odds of death and unfavorable neurologic outcomes at hospital discharge.[43] To decrease the risk of secondary neurologic injury, EEG should be used in the comatose child to evaluate for seizure activity. Postischemic seizures should be treated aggressively in the same way that seizures are treated in the non-arrest patient.[44–46]

Targeted Temperature Management

TTM refers to the induction of hypothermia in patients after cardiac arrest and has become standard therapy in adults after ROSC and in newborns with hypoxic-ischemic encephalopathy. It is thought that TTM treats reperfusion syndrome that occurs after cardiac arrest by decreasing metabolic demand and free radical production. Current studies have generally focused on 2 temperature ranges: 32°C to 34°C and 36°C to 37.5°C (so-called controlled normothermia). Hypoxic-ischemic injury in the setting of arrest is frequently associated with fever, and elevated body temperature is associated with worse outcomes in most studies. Early research in TTM in children was limited by varied treatment protocols, which made definitive recommendations about efficacy difficult. Two recent pediatric studies (Therapeutic Hypothermia After Pediatric Cardiac Arrest Out-of-Hospital and Therapeutic Hypothermia After Pediatric Cardiac Arrest In-Hospital) examined the efficacy of TTM at 32°C to 34°C and 36°C to 37.5°C for 120 hours (5 days) in both out-of-hospital and in-hospital cardiac

arrest.[47,48] Both studies failed to show a significant difference in the primary endpoint of favorable neurobehavioral outcome at 1 year or a difference in the secondary outcomes of survival at 1 year and change in neurobehavioral outcome. Despite these negative outcomes, the 2019 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations from ILCOR considers TTM at either 32°C to 34°C and 36°C to 37.5°C to be reasonable for infants and children between 24 hours and 18 years of age who remain comatose after out-of-hospital or in-hospital cardiac arrest.[3] Despite clinical equipoise in the pediatric cardiac arrest literature, TTM can be considered, although it is imperative to avoid temperatures greater than 37.5°C in the immediate postarrest period.

TREATMENT OF UNDERLYING CAUSE

Sepsis

Sepsis should be considered in all patients presenting with cardiovascular collapse. Early volume resuscitation is a key component to the treatment of septic shock. Although 0.9% saline (normal saline) has been used for volume resuscitation for years, there is a growing body of literature, both in adults and in pediatric patients, that use of balanced crystalloid fluids may be preferable.[49–51] Patients suspected of septic shock should receive an initial 20 mL/kg fluid bolus, followed by a clinical reassessment to determine if further fluid resuscitation is warranted. The total amount of fluid given to patients in septic shock should be determined on a case-by-case basis, taking into consideration patient status and available critical care resources. The PALS guideline also recommends monitoring of the central venous oxygen saturation ($Scvo_2$), if able, with a target $Scvo_2 \geq 70\%$, and consideration of early assisted ventilation in patients with septic shock and severe sepsis. Etomidate may cause transient adrenal suppression for up to 24 hours after single-dose use when used during RSI in pediatric patients with sepsis.[52] Despite this physiologic fact, there are no well-done studies demonstrating an outcome difference attributable to etomidate usage. Ketamine is a reasonable alternative in pediatric patients with severe sepsis, understanding that reduced dosing may be required to mitigate the hemodynamic effects of induction in shock states regardless of agent chosen.[53]

Structural Heart Disease

Cyanotic congenital heart disease may lead to cardiogenic or obstructive shock and cardiac arrest. As more patients undergo palliative surgical procedures, they may require resuscitation during their preoperative or postoperative course or at times of critical illness. In general, these patients should undergo standard PALS resuscitation practices. Additional prearrest, intraarrest, and postarrest considerations in this special pediatric population include the following:

- Prearrest:
  - Infants with single-ventricle physiology and elevated pulmonary-to-systemic flow ratio before stage I repair may benefit from elevated $Paco_2$ levels of 50 to 60 mm Hg.
  - Infants with single-ventricle physiology who have undergone stage I repair may benefit from systemic vasodilators, such as phenoxybenzamine, milrinone, or nitroprusside.
  - Central venous oxygen saturation ($Scvo_2$) monitoring should be considered to detect hemodynamic changes in the periarrest patient.

- Hypoventilation and negative pressure ventilation may improve oxygen delivery and cardiac output, respectively, in patients with Fontan or hemi-Fontan/bidirectional Glenn physiology.

- Intraarrest:
  - ECMO should be considered for patients with single-ventricle anatomy who have undergone stage I palliation and for patients with Fontan physiology.
  - Occlusion is a known complication of systemic-pulmonary artery or right ventricular-pulmonary artery shunts, and therefore, heparin may be considered adjunctive therapy to the PALS algorithm in these patients.
  - Differences in pulmonary physiology make end-tidal Co_2 is an unreliable indicator of CPR quality in the single-ventricle patient.
- Postarrest:
  - Unlike the previously healthy patient, children with cyanotic congenital heart disease should have a target postresuscitation oxygen saturation of 80%.

A recent AHA scientific statement regarding resuscitation of infants and children with cardiac disease is also available for further review of this complex topic.[54]

Respiratory Causes

Respiratory failure is a leading cause of pediatric cardiac arrest.[55] Upper-airway obstruction, lower-airway obstruction, intrinsic lung disease, and disordered control of breathing can all precipitate respiratory failure (**Table 7**).

Trauma and Abuse

In the patient with undifferentiated shock or cardiac arrest, it is important to consider intentional or accidental traumatic injuries, such as intraabdominal trauma, tension pneumothorax, pericardial tamponade, spinal cord injury, and intracranial hemorrhage, even in the absence of external findings. Proper management of the "ABC's" may prevent or reverse cardiopulmonary collapse in this patient population. Cervical spine precautions may need to be maintained while managing the airway. Consider maneuvers, such as recessing the occiput or elevating the child's torso, to avoid unwanted neck flexion in the younger patient.[56] Trauma to the chest (pneumothorax, hemothorax, pulmonary contusion) can cause respiratory failure. In children with severe head injuries and impending brain herniation, a trial of hyperventilation may be used as a temporizing measure.[57] Resuscitative thoracotomy can be considered in pediatric patients with penetrating trauma and arrest, although outcomes are poor.[58]

Toxic/Metabolic

Accidental or intentional overdose may lead to swift cardiopulmonary collapse and cardiac arrest in pediatric patients. It is important to consider toxic ingestion in the otherwise healthy pediatric patient with cardiac arrest of unknown cause. Treatment of common and/or harmful toxic exposures is provided in **Table 8**.[59–61]

PARENTAL PRESENCE DURING RESUSCITATION

Offering caregivers the option to be present at the bedside is becoming a common practice during pediatric resuscitations and should be encouraged.[62] Research indicates that parents who were present during resuscitation would choose to be present again and would recommend being present to others in a similar situation.[63] Caregivers who were present were also noted to have more constructive grief behaviors and less distress than those who were not present. If family is present, a staff member

Table 7
Cause and management of respiratory failure

Type of Respiratory Distress	Examples	Treatment
Upper-airway obstruction	Infection (croup, epiglottitis) Edema (anaphylaxis) Foreign body	Allowing child to assume position of comfort Jaw thrust or head tilt–chin lift Foreign body removal Suctioning Medications to reduce airway edema (steroids, epinephrine) Minimizing patient agitation Possible disposition to the operating room for advanced airway management
Lower-airway obstruction	Bronchiolitis Asthma	Supplemental oxygen Suctioning Medications (albuterol, ipratropium bromide, steroids, magnesium, ketamine, epinephrine, terbutaline, Heliox)[a] High-flow nasal cannula or BIPAP Endotracheal intubation[b]
Lung tissue disease	Pneumonia (infectious, chemical, aspiration) Cardiogenic pulmonary edema ARDS Traumatic pulmonary contusion	Supplemental oxygen Positive expiratory pressure (noninvasive ventilation such as CPAP or BIPAP, mechanical ventilation with PEEP) Medications as indicated (antibiotics, vasoactive agents, diuretics)
Disordered control of breathing	Increased intracranial pressure Neuromuscular disease Depressed mental status (CNS infection, seizures, metabolic derangement, overdose)	Avoid hypoxemia, hypercarbia, hyperthermia for patients with increased ICP Antidote for overdose Noninvasive or invasive ventilatory support as mental status dictates

Abbreviations: ARDS, acute respiratory distress syndrome; BIPAP, bilevel positive airway pressure; CNS, central nervous system; CPAP, continuous positive airway pressure; ICP, intracranial pressure; PEEP, positive end expiratory pressure.

[a] Medications listed are possible treatments for asthma. Albuterol trial may be indicated in patients with bronchiolitis; however, per American Academy of Pediatrics guidelines no additional medications are shown to be beneficial in patients with bronchiolitis.[42]

[b] If assisted ventilation is required in this population use a low respiratory rate to allow adequate time for exhalation.

should be assigned to convey clinical information and provide comfort, and all team members must be mindful of their presence while remaining focused on the patient.

TERMINATION OF RESUSCITATION

Survival rates for pediatric cardiac arrest remain low.[64,65] The decision to cease resuscitation efforts is multifactorial because no single variable is predictive of

Table 8
Common pediatric toxic exposures, symptoms, and treatments

Overdose	Signs and Symptoms	Treatment
Local anesthetics (topical, IV, epidural)	Altered mental status Seizures Arrhythmia and cardiac arrest	Lipid emulsion therapy: 1.5 mL/kg up to 70 kg 100 mL ≥70 kg[36]
Cocaine	Sympathomimetic toxidrome Acute coronary syndrome Cardiac dysrhythmia	Normalize core temperature Nitroglycerin, benzodiazepines, or phentolamine for coronary vasospasm Sodium bicarbonate 1–2 mEq/kg for ventricular arrhythmia Lidocaine bolus followed by infusion to prevent arrhythmia secondary to myocardial infarction AVOID unopposed α-adrenergic stimulation; do not give β-adrenergic blockers
Tricyclic antidepressants	Hypotension Seizure Altered mental status Cardiovascular effects (IVCD, bradycardia, heart block, prolonged QT interval, ventricular dysrhythmia)	1–2 mEq/kg IV sodium bicarbonate boluses until arterial pH >7.45 followed by sodium bicarbonate infusion to maintain alkalosis 10 mL/kg boluses of normal saline for first-line treatment of hypotension; consider epinephrine or norepinephrine as second-line treatment Consider ECMO if persistent hypotension despite vasopressors AVOID class IA, class IC, and class III antiarrhythmics
Calcium channel blockers[37]	Seizures Altered mental status Hypotension Cardiovascular effects (prolonged QT interval, wide QRS, bradycardia, RBBB, SVT, tornados de pointes, ventricular dysrhythmia)	5–10 mL/kg IV boluses of normal saline for hypotension IV CaCl 10% 20 mg/kg over 10–15 min, may repeat every 10–15 min or follow with infusion of 20–50 mg/kg/h High-dose IV insulin bolus of 0.5–1 unit/kg, followed with 0.5–1 unit/kg/h (titrate every 15–20 min as needed for hemodynamic stability) IV dextrose infusion to maintain euglycemia Potassium repletion as needed Norepinephrine and/or epinephrine as first-line vasopressors. Atropine 0.02 mg/kg IV/IO (maximum dose 0.5 mg) for bradycardia If refractory to above measures consider: • Lipid emulsion therapy • Pacemaker • ECMO

(*continued on next page*)

Table 8 (*continued*)

Overdose	Signs and Symptoms	Treatment
β-adrenergic blockers	Cardiovascular effects (bradycardia, heart block, decreased cardiac contractility)	Epinephrine infusion Consider glucagon in adolescents Consider infusion of glucose and insulin Consider IV calcium administration if cardiovascular effects are refractory to glucagon and catecholamines
Opioids	Hypoventilation/apnea Bradycardia Hypotension Depressed mental status	Support oxygenation/ventilation with BVM and/or intubation Naloxone • 0.1 mg/kg/dose IV/IM/SC/IO (IV preferred), maximum dose 2 mg; repeat every 2–3 min as needed[38] • Alternate dosing for intranasal or endotracheal routes available

Abbreviations: IM, intramuscular; IVCD, interventricular conduction delay; RBBB, right bundle branch block; SC, subcutaneous; SVT, supraventricular tachycardia.

outcome after pediatric cardiac arrest. Duration of CPR, number of doses of epinephrine, age of the patient, presenting cardiac rhythm, cause of arrest, and the likelihood of reversible causes are all factors. Meaningful survival after prolonged resuscitation has been described in the setting of poisoning, hypothermia, recurring or refractory shockable rhythm, and patients with isolated heart disease resuscitated with ECMO.

SUMMARY

Pediatric cardiac arrest is most commonly caused by an initial respiratory insult that progresses to cardiovascular collapse as hypoventilation and hypoxia lead to acidosis. Although relatively rare, it is accompanied by a high degree of stress for caregivers because of the myriad of conditions that must be considered as well as the unique anatomy and physiology of pediatric patients. The range of drug doses and equipment sizes can be daunting, and this cognitive burden can be lessened by length-based resuscitation aids. Aggressive treatment of respiratory distress and shock before arrest is essential given the poor outcome associated with cardiac arrest.

DISCLOSURE

The authors have nothing to disclose.

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