In absolute terms, cardiac arrest is not rare in pediatric patients; it occurs in 2 to 6% of children admitted to a pediatric intensive care unit (PICU) and about 16,000 children per year out of hospital in the United States (i.e., 8-20 per 100,000 children per year). From the perspective of the individual care provider in the prehospital or inpatient medical setting outside the intensive care unit (ICU), however, pediatric resuscitations constitute an infrequent occurrence. Significant disparity in favorable outcomes continues to exist between out-of-hospital and in-hospital pediatric cardiac arrest.

Epidemiology

Data from the National Heart, Lung, and Blood Institute (NHLBI)–funded Resuscitation Outcomes Consortium demonstrated an overall population-based incidence of nontraumatic pediatric out-of-hospital cardiac arrests (OOHCAs) of 8 per 100,000 pediatric person-years compared with 125 per 100,000 adult person-years; however, the rate among infants (73 per 100,000) was on the same order of magnitude as in adults. More important, pediatric patients were more likely to survive to discharge than adults (6.4% vs. 4.5%). Specifically, children and adolescents were twice as likely to survive to hospital discharge as infants and adults. The number needed to treat (NNT) of 13 to save a pediatric life compares favorably with NNT for other aggressive interventions considered quite effective, such as implantation of a cardioverter-defibrillator in patients with ventricular arrhythmias (NNT of 8) and immediate revascularization in patients with cardiogenic shock (NNT of 13). Furthermore, the potential years of life gained for pediatric survivors are much greater than for adult survivors.

Among published studies of out-of-hospital pediatric cardiac arrests, the most common causes are sudden infant death syndrome (SIDS; 20-23%), trauma (15-20%), and submersion injury (8-12%). Prognosis after cardiac arrest varies considerably based on the underlying cause, with outcomes after SIDS and trauma being particularly dismal and outcomes after submersion including a higher prevalence of survival. Table 10-1 shows the prevalence of survival to discharge after out-of-hospital and in-hospital pediatric cardiac arrest as reported by multiple studies over the past three decades. It is noteworthy that survival after in-hospital cardiac arrest has improved substantially from less than 10% in the 1980s to greater than 25% in the first decade of the 21st century. By contrast, survival after OOHCA has changed very little in 30 years, with survival less than 10% in virtually all studies over that time period. The American Heart Association (AHA) National Registry of Cardiopulmonary Resuscitation (Get With The Guidelines—Resuscitation [GWTG-R]), formerly known as NRCPR, database has yielded analyses demonstrating specific epidemiologic features of in-hospital pediatric arrest associated with improved survival, including young age and events occurring in hospitals with higher levels of pediatric staffing.

Cardiac arrest occurring in the emergency department (ED) (as distinct from out-of-hospital arrest with continued resuscitation in the ED) accounts for a minority (approximately 11-13%) of in-hospital events for children and adults. Historical outcomes for children who arrest while in the ED suggest that survival outcomes are less prevalent than in cases of in-hospital arrest but better than in out-of-hospital arrest. More recent controlled analyses from the GWTG-R demonstrate a significant discrepancy between outcomes among children who arrest in the ED, with an adjusted odds ratio of 0.39 for survival compared with other in-hospital locations, highlighting differences in pathophysiology of ED versus in-hospital cardiac arrest in children. These data stand in stark contrast to data in adults, in whom arrests in the ED were associated with significantly improved survival of 22% compared with 10-15% for events in the ICU or wards.

Pathophysiologic Principles

Physiology of Cardiac Arrest

Three common pathways to arrest have been identified: asphyxial, ischemic, and arrhythmogenic. Asphyxial cardiac arrests are precipitated by acute hypoxia or hypercarbia and are the most common in children. Ischemic arrests are precipitated by inadequate myocardial blood flow, in children most commonly as a result of shock from hypovolemia, sepsis, or myocardial dysfunction. Arrhythmogenic arrests are most often sudden, precipitated by ventricular fibrillation (VF) or ventricular tachycardia (VT). The immediate causes of arrest in two recent in-hospital studies were arrhythmogenic in 10%, asphyxial in 67%, and ischemic in 61% (many patients had both asphyxia and ischemia). The vast majority of out-of-hospital arrests are also either asphyxial or ischemic, and 5 to 20% are arrhythmogenic.

Pediatric Anatomy Relevant to Cardiopulmonary Resuscitation

Appropriate pediatric cardiopulmonary resuscitation (CPR) differs from that for adults owing to children’s differences in
anatomy and physiology, as well as the differences in the pathogenesis of cardiac arrest and the common rhythm disturbances in children. Prolonged hypoxia and acidosis impair cardiac function and ultimately lead to cardiac arrest. The use of closed-chest cardiac massage to provide adequate circulation during cardiac arrest was initially demonstrated in small dogs with compliant chest walls. Therefore children were among the first patients successfully treated with this technique. The presumed mechanism of blood flow was direct compression of the heart between the sternum and the spine in children with compliant chest walls. Later investigations indicated that blood flow could also be circulated during CPR by the thoracic pump mechanism. That is, chest compression-induced increases in intrathoracic pressure can generate a gradient for blood to flow from the pulmonary vasculature, through the heart, and into the peripheral circulation.

**Clinical Features**

The triad of pulselessness, apnea, and unresponsiveness defines the clinical state of cardiac arrest. For decades, published guidelines by the International Liaison Committee on Resuscitation (ILCOR) on the assessment of patients suspected to be in cardiac arrest used the mnemonic ABC (airway, breathing, circulation) for the stepwise assessment and intervention sequence. This sequence included opening the airway, assessing respirations with the “look-listen-feel” technique for up to 10 seconds, providing two rescue breaths, and checking for a central pulse (brachial or femoral in children, carotid in adults). For the first time, in 2005, treatment recommendations published by ILCOR removed the pulse check as a necessary step for lay rescuers, based on data demonstrating poor specificity and sensitivity of a 10-second carotid pulse check by health care providers in anesthetized adults undergoing aortic cross-clamping. The implication for rescuers is that any patient who is unresponsive and apneic and appears hypoperfused by gross appearance (i.e., “appears dead”) should have chest compressions initiated immediately without a check for a pulse. This recommendation may have particular pertinence for children, given the high prevalence of bradycardia and hypoperfusion in the prearrest phase and the potential improvement in outcomes when CPR is provided for bradycardia (see later).

In 2010 the AHA made the recommendation to change the algorithmic sequence of rescue interventions for the arrested patient from ABC to CAB (compressions, airway, breathing) because blood flow during cardiac arrest depends on chest compressions, and efforts to address A and B first delay the time to reestablishment of blood flow. The value of this approach is most dramatically demonstrated by the success of compression-only CPR (i.e., without rescue breathing) in adults. Therefore CAB is the adult recommendation, and it is reasonable to use the same approach in children to simplify training for the lay rescuer. In addition, starting resuscitation for an arrested child with compressions instead of ventilations will result in only a brief delay before the first rescue breath (estimated 18 seconds for the lone rescuer, 9 seconds for multiple rescuers). Finally, in a health care setting where a team of providers responds to an arrest, multiple tasks are undertaken simultaneously by task-specific personnel; given that chest compressions can be applied instantaneously and positive-pressure ventilation requires several seconds of equipment preparation and application to the patient’s face, CAB is unlikely to produce different results in in-hospital events.

Studies on the pulse check in pediatrics have predominantly focused on healthy children, where the data on the accuracy of brachial and femoral pulse checks are varied. A recent pediatric ICU study examined the accuracy of the pulse check by health care providers on children receiving extracorporeal circulatory support (i.e., either extracorporeal membrane oxygenation [ECMO] or a ventricular assist device [VAD]) in whom native pulsatile cardiac activity was variably diminished or absent; it was found that a 10-second femoral or brachial pulse check had a sensitivity of 86% and a specificity of 64%. In other words, making a decision to provide CPR based on the pulse check alone in this patient set would have resulted in chest compressions being given to 36% of patients in whom they were not indicated and compressions being withheld from 14% of children who were either pulseless or critically hypoperfused enough to require them. Based largely on this study, the 2010 ILCOR guidelines for pediatric resuscitation have removed the pulse check for lay rescuers. Health care providers may spend up to 10 seconds assessing a central pulse, but the empirical provision of chest compressions without a pulse check for the child who appears dead is appropriate.

**Four Phases of Cardiac Arrest**

Cardiac arrest may be categorized into four phases, each with unique physiology and treatment strategies: (1) prearrest, (2) no flow (untreated cardiac arrest), (3) low flow (CPR), and (4) postresuscitation.

**Prearrest Phase.** Because most out-of-hospital pediatric cardiac arrests are caused by progressive asphyxia or ischemia, they can often be prevented by avoiding the precipitating insult. For example, infant and child car seats and seat belts for older children can prevent cardiac arrests resulting from motor vehicle collisions. Similarly, fences around swimming pools with self-closing gates can prevent drowning. Both the BRESUS study in the United Kingdom and the AHA’s GWTG-R national registry data clearly demonstrate that most in-hospital cardiac arrests are asphyxial or ischemic rather than sudden arrhythmia-induced events. Most important, many of these arrests could be prevented by early recognition and treatment of respiratory failure and shock. These issues were appreciated by the founders of the Pediatric Advanced Life Support (PALS) course, which was therefore designed to prevent cardiac arrests by early recognition and treatment of respiratory failure and shock in children.

**No-Flow Phase (Untreated Cardiac Arrest).** Interventions during the no-flow phase of untreated pulseless cardiac arrest focus on early recognition of cardiac arrest and initiation of basic and

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*Table 10-1: Outcomes after Pediatric Cardiac Arrest*

<table>
<thead>
<tr>
<th>AUTHOR</th>
<th>DATE</th>
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<td>Reis</td>
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<td>64%</td>
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<tr>
<td>Nadkarni</td>
<td>2006</td>
<td>52%</td>
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<table>
<thead>
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<tr>
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<tr>
<td>Young</td>
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<tr>
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<td>NR</td>
<td>6.4%</td>
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NR, data not reported; ROSC, return of spontaneous circulation.
advanced life support. Yet only a third of children with OOHCA are provided with bystander CPR.

Low-Flow Phase (Resuscitation). During untreated cardiac arrest, circulation has stopped (i.e., the no-flow phase). The low-flow phase begins once resuscitation measures (chest compressions) are implemented to generate blood flow. Specific considerations related to chest compressions and advanced life support medications are discussed later.

Postresuscitation Phase. The postarrest syndrome is a unique combination of pathophysiologic processes that occur after successful resuscitation. This postarrest syndrome includes (1) postarrest brain injury, (2) postarrest myocardial dysfunction, (3) systemic ischemia-reperfusion response, and (4) the unresolved pathologic process that caused the cardiac arrest.

Clinical manifestations of postarrest brain injury include coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction (ranging from memory deficits to persistent vegetative state), and brain death. Mild induced hypothermia is the best-established postresuscitation therapy for adult postarrest brain injury. Two seminal articles established that induced hypothermia (32-34°C) could improve outcome for comatose adults after resuscitation from VF cardiac arrest. In both randomized controlled trials, the inclusion criteria included patients older than 18 years who were persistently comatose after successful resuscitation from nontraumatic VF. Interpretation and extrapolation of these studies to children are difficult. Hyperthermia after resuscitation from cardiac arrest is common in children because of inflammatory mediators. It is reasonable to believe that mild induced systemic hypothermia may benefit children resuscitated from nontraumatic cardiac arrest. However, benefit from this treatment has not been rigorously studied and reported in children or in any patients with non-VF arrests. Multicenter trials of induced hypothermia in children after both in-hospital cardiac arrest and traumatic arrest are ongoing. Emerging neonatal trials of selective brain cooling and whole-body cooling show improved outcomes for this therapy in neonatal hypoxic-ischemic encephalopathy.

Postarrest myocardial dysfunction and hypertensive shock are very common among pediatric survivors of cardiac arrest. It is interesting to note that postarrest myocardial dysfunction appears to be pathophysiologically similar to sepsis-related myocardial dysfunction, including increases in inflammatory mediator and nitric oxide production. Although the optimal management of postarrest hypotension and myocardial dysfunction have not been defined, data suggest that aggressive hemodynamic support may improve outcomes. Controlled trials in animal models have shown that dobutamine, milrinone, or levosimendan can effectively ameliorate postarrest myocardial dysfunction. In clinical observational studies, fluid resuscitation has been provided for pediatric patients with hypotension and concomitant low central venous pressure, and various vasoactive infusions, including epinephrine, dobutamine, and dopamine, have been provided for the myocardial dysfunction.*

Management

Chest Compressions

Blood flow during CPR is generated by chest compressions. For children the main mechanism of blood flow is from cardiac compression. The cardiac output (CO) depends on the product of the stroke volume and heart rate. The force of compressions is a major determinant of stroke volume, and the rate of compressions is the sole determinant of heart rate. Stroke volume also depends on preload. Therefore patients with cardiac arrests precipitated by circulatory shock (e.g., hypovolemic or septic shock) may need additional intravascular volume to generate an adequate stroke volume with chest compressions. Notably, excellent CPR can result in a CO 10 to 25% of that in normal sinus rhythm.

Adequate myocardial blood flow is necessary for return of spontaneous circulation. During CPR, myocardial blood flow depends on coronary perfusion pressure or the “driving pressure” of blood into the coronary arteries from the aorta (i.e., the difference between the aortic and right atrial pressures during the relaxation phase). If the coronary perfusion pressure falls below 15 mm Hg during CPR in adults, the likelihood for a return of spontaneous circulation is substantially decreased. Animal data suggest that outcomes improve as coronary perfusion pressure increases above 25 mm Hg. Moreover, even relatively brief interruptions to chest compressions (e.g., 4-second pauses for two rescue breaths) lead to substantial decreases in the aortic relaxation pressure and coronary perfusion pressure, thereby resulting in inadequate myocardial perfusion.

Circumferential versus Focal Sternal Compressions

In animal models of cardiac arrest, circumferential CPR (e.g., vest or load-distributing band CPR, encircling hands and compression with two thumbs) provides better CPR hemodynamics than focal compressions (e.g., hands on chest, two-finger technique). In smaller infants, the recommended CPR technique for two rescuers is to encircle the chest with both hands and depress the sternum with the thumbs while compressing the thorax circumferentially (when the rescuer’s hands are relatively large enough to do so) (Fig. 10-2). This “two-thumb” circumferential compression technique results in higher systolic and diastolic blood pressures and a higher pulse pressure than traditional two-finger compression of the sternum.

Chest Compression Rate

Although the optimal chest compression rate is unknown, data from large studies of animals have shown that coronary perfusion pressure, CO, and survival are substantially superior with chest compression rates of 100 per minute compared with rates of less than 80 per minute. In addition, clinical studies in adults have shown that end-tidal carbon dioxide levels, a marker of cardiac output during CPR, were significantly higher with chest compressions at 120 versus 80 per minute.

Although restoration of coronary perfusion during CPR is critical for successful return of spontaneous circulation, adequate cerebral perfusion is important for mitigating the injurious effects of cerebral anoxia during cardiac arrest. Unlike myocardial blood flow, cerebral blood flow is generated during the compression phase of CPR. Forceful, uninterrupted chest compressions at a rate of 120 per minute compared with 60 per minute improves both cerebral and coronary perfusion pressures compared with less forceful compressions or compressions at a lower rate. In addition, vasoconstrictors, such as epinephrine or vasopressin, preferentially direct the CO during CPR to the coronary and cerebral circulations.

During this phase, the only source of coronary and cerebral perfusion comes from the blood pressure generated by good chest compressions. Any interruption in chest compressions, whether to perform procedures, analyze rhythms, check for pulses, or change compressors, is potentially harmful. For VF and pulseless VT, rapid determination of electrocardiographic rhythm and prompt defibrillation when appropriate are important. For cardiac arrests resulting from asphyxia or ischemia, adequate myocardial perfusion and myocardial oxygen delivery with ventilation to match blood flow is important.

Despite evidence-based guidelines, extensive provider training, and provider credentialing in resuscitation medicine, the quality of

*References 41, 42, 45, 46, 52, 53.
CPR is typically poor. Slow compression rates, inadequate depth of compression, and substantial pauses are the norm. Moreover, observed ventilation rates during professional rescuer CPR are often too high, potentially leading to deleterious effects on venous return and outcome. The resuscitation mantra is Push hard, push fast, minimize interruptions, allow full chest recoil, and do not overventilate. This approach can markedly improve myocardial, cerebral, and systemic perfusion and will likely improve outcomes.

Chest Compression–Ventilation Ratios (Table 10-2)

Ideal compression-to-ventilation ratios for pediatric patients are unknown. Physiologic estimates suggest that the amount of ventilation needed during CPR is much less than the amount needed during a normal perfusing rhythm because the CO (and therefore pulmonary blood flow) during CPR is only 10 to 25% of that during normal sinus rhythm. The best ratio of compressions to ventilations depends on many factors, including the compression rate, the tidal volume, the blood flow generated by compressions, and the time that compressions are interrupted to perform ventilations. In a manikin model of pediatric CPR, a chest compression-to-ventilation (CC:V) ratio of 15:2 delivered the same minute ventilation as CPR with a CC:V ratio of 5:1, but the number of chest compressions delivered was 48% higher with the 15:2 ratio. The benefits of positive pressure ventilation (increased arterial oxygen content and carbon dioxide elimination) are balanced against the adverse consequence of impeding circulation because of increases in intrathoracic pressure and venous return to the heart.

For adults, mathematical models of oxygen delivery during CPR suggest that the optimal CC:V ratio is approximately 30:2 for two-rescuer health care provider CPR and is closer to 50:2 for single-lay rescuers. Similar mathematical models adjusted to the known physiologic variables in children indicate that CC:V ratios from 10:2 to 30:2 would be reasonable to optimize tissue oxygen delivery during CPR.

The present recommendations for CC:V ratios during CPR are based on rational conjecture from animal, manikin, and mathematical models, as well as educational theory on the retention of skills in adult learners. In the 2010 AHA guidelines, a universal CC:V ratio of 30:2 is recommended for single-person bystander CPR. For two-rescuer CPR, a 15:2 ratio is recommended for neonates, infants, and children beyond the newly born (infant at the time of birth) period. For the newly born, a ratio of 3:1 is recommended, resulting in a greater number of ventilations...
per minute, but nearly the same number of compressions (100 vs. 90 per minute). This recommended ratio was arrived at by consensus to balance educational issues (i.e., the benefit to single-rescuer bystanders of remembering only one compression-to-ventilation ratio of 30:2) with what is known about the physiology of the cardiac and pulmonary circulations of children during cardiac arrest.

Leaning

In a large observational study of OOHCA, incomplete release or “leaning” occurred in more than 10% of compressions and was observed in 16 of 173 (9%) CPR episodes. Observations during in-hospital pediatric CPR indicate that leaning is a common phenomenon, occurring in 23% of chest compressions. Leaning pressures of approximately 15% of body weight may affect intrathoracic pressure and impede the hemodynamics of CPR.

Real-Time Cardiopulmonary Resuscitation Feedback

In an effort to optimize CPR quality, new technology has been developed that monitors CPR through a force sensor and accelerometer on the chest. This information is transmitted to a defibrillator monitor to provide quantitative verbal feedback to the rescuer on the rate and force of compressions as well as the frequency and volume of ventilations. Recent studies document that poor-quality CPR, as analyzed by a feedback device, reduces the likelihood of defibrillation success, and rescuers can use this type of automated feedback to improve CPR quality and compliance with current guidelines. The optimal goals for aortic pressures during pediatric CPR are unknown. Animal data and adult data suggest that a reasonable goal for the aortic diastolic (or relaxation) pressure is greater than 20 to 30 mm Hg. Similarly, a reasonable goal for the aortic systolic (or compression) pressure is greater than 50 mm Hg for a newborn, 60 mm Hg for an infant, 70 to 80 mm Hg for a child, and 80 to 90 mm Hg for an adolescent.

Compression-Ventilation (Standard) versus Compression-Only (“Hands-Only”) Cardiopulmonary Resuscitation

Foregoing ventilation in the pediatric patient during CPR provided by trained providers is not prudent because respiratory arrest and asphyxia generally precede pediatric cardiac arrest. Multiple studies in adults with OOHCA have demonstrated that chest compressions alone (“hands-only” CPR) yielded survival outcomes that were as good if not better than standard CPR with ventilations included. Not surprising, animal studies of bystander CPR for asphyxia-precipitated cardiac arrests demonstrate that the addition of rescue breathing to compressions results in much better outcomes than chest compressions alone. Chest compressions alone, however, were superior to no CPR at all, even with hypoxia-induced cardiac arrest. These studies support the need for rescue breathing as a critical component of CPR for pediatric asphyxia-precipitated cardiac arrests. A large population-based study in Japan found that children with OOHCA of noncardiac origin (i.e., asphyxial or ischemic) had significantly improved survival when standard bystander CPR (with ventilation included) was provided when compared with compression-only CPR. This survival benefit, however, was not found to be present among children whose OOHCA was of primary cardiac origin. Thus, compression-only CPR is not recommended for the majority of pediatric cardiac arrests. For an older child with a sudden collapse from cardiac arrest (i.e., presumed VF or VT), compression-only CPR is a reasonable choice for bystander CPR.

Advanced Life Support Medications during the Low-Flow Phase of Cardiopulmonary Resuscitation

Figure 10-3 demonstrates a simplified algorithm for pediatric pulseless cardiac arrest. Table 10-3 lists medications commonly used during pediatric resuscitation including dosages and indications.

Although animal studies indicate that epinephrine can improve initial resuscitation success after both asphyxial and VF cardiac arrests, no single medication has been shown to improve survival to hospital discharge outcome after pediatric cardiac arrests. Medications commonly used for CPR in children are vasopressors (epinephrine or vasopressin), calcium salts, sodium bicarbonate, and antiarrhythmics (amiodarone or lidocaine). During CPR, epinephrine’s alpha-adrenergic effect increases systemic vascular resistance (SVR), increasing diastolic blood pressure, which in turn increases coronary perfusion pressure and blood flow and increases the likelihood of the return of spontaneous circulation (ROSC). Epinephrine also increases cerebral blood flow during CPR because peripheral vasoconstriction directs a greater proportion of flow to the cerebral circulation. The beta-adrenergic effect increases myocardial contractility and heart rate and relaxes smooth muscle in the skeletal muscle vascular bed and bronchi, although this effect is of less importance. Epinephrine also changes the character of VF (i.e., higher amplitude, more “coarse”), increasing the likelihood of successful defibrillation.

Prospective and retrospective studies indicate that use of high-dose epinephrine in adults or children (0.05-0.2 mg/kg) does not improve survival and may be associated with a worse neurologic outcome. A randomized, blinded, controlled trial of high-dose epinephrine versus standard-dose epinephrine after failed initial standard-dose epinephrine for pediatric in-hospital cardiac arrest demonstrated a worse 24-hour survival rate in the high-dose epinephrine group. High-dose epinephrine cannot be recommended for routine use during CPR.

Calcium salts (calcium gluconate, calcium chloride) are commonly used in pediatric resuscitation for sepsis, transfusion-associated ionized hypocalcemia, specific toxicidromes, and in newborns after cardiac surgery. Data on calcium salts in cardiac arrest, however, do not support its routine use. A controlled analysis of the GWTG-R database demonstrated significantly decreased survival to hospital discharge among children receiving calcium salts during CPR. Current recommendations for calcium salts during CPR are limited to cases of documented hypocalcemia, hyperkalemia, hypermagnesemia, or known or suspected intoxication with calcium channel blockers.
Figure 10-3. Management algorithm for infants and children with cardiopulmonary arrest. AED, automated external defibrillator; CPR, cardiopulmonary resuscitation; IO, intraosseous; IV, intravenous; PEA, pulseless electrical activity; VF, ventricular fibrillation; VT, ventricular tachycardia. (Adapted from Pediatric Advanced Life Support Course Guide. Copyright © 2010, American Heart Association.)
asystole and PEA. However, GWTG-R data establish that survival to discharge was more common among children with initial VF/VT than among children with subsequent VF/VT (35% vs. 11%; odds ratio 2.6, 95% confidence interval [CI] 1.2-5.8). Surprisingly, the subsequent VF/VT group had worse outcomes than children with asystole/PEA (11% vs. 27% survival). These data suggest that outcomes after initial VF/VT in children (an arrhythmogenic arrest) are “good,” but outcomes after subsequent VF/VT (i.e., VF/VT in the setting of an asphyxial or ischemic arrest) are worse, even compared with initial asystole/PEA without subsequent VF/VT.

Defibrillation

Defibrillation is necessary for successful resuscitation from VF cardiac arrest. When prompt defibrillation is provided soon after the induction of VF in a cardiac catheterization laboratory, the rates of successful defibrillation and survival approach 100%. In general, the mortality rate increases by 5 to 10% per minute of delay to defibrillation. Because pediatric cardiac arrests are commonly a result of progressive asphyxia or shock (or both), the initial treatment of choice is prompt CPR, not defibrillation. This emphasis is balanced against the increasing evidence that VF in children is not rare, that outcomes after arrhythmogenic VF arrests

### Table 10-3 Medications Used in Pediatric Resuscitation

<table>
<thead>
<tr>
<th>DRUG</th>
<th>INDICATIONS AND DOSAGE</th>
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| Adenosine            | SVT
0.1 mg/kg IV/IO rapid push (max 6 mg), second dose 0.2 mg/kg IV/IO rapid push (max 12 mg)                                                        |
| Alprostadil (PGE1)   | Ductal-dependent congenital heart disease (all forms)
0.05-0.1 µg/kg/min IV/IO infusion initially, then 0.01-0.05 µg/kg/min IV/IO                                                                             |
| Amiodarone           | SVC VT (with pulses)
5 mg/kg IV/IObolus load over 20-60 min (max 300 mg), repeat to daily max 15 mg/kg (or 2.2 g)
Pulessense arrest (i.e., VF/pulseless VT)
5 mg/kg IV/IO bolus (max 300 mg), repeat to daily max 15 mg/kg (or 2.2 g)                                                                                 |
| Atropine sulfate     | Bradycardia (symptomatic)
0.02 mg/kg IV/IO (min dose 0.1 mg, max single dose child 0.5 mg, max single dose adolescent 1 mg), may repeat dose once, max total dose child 1 mg, max total dose adolescent 2 mg
0.04-0.06 mg/kg ETT                                                                                                                                      |
| Calcium chloride 10% | Hypocalcemia, hyperkalemia, hypermagnesemia, calcium channel blocker overdose
20 mg/kg (0.2 mL/kg) IV/IO slow push during arrest or if severe hypotension, repeat PRN                                                                    |
| Dextrose (glucose)   | Hypoglycemia
0.5-1 g/kg IV/IO (D25W 2-4 mL/kg; D3W 5-10 mL/kg)                                                                                                       |
| Dobutamine           | Congestive heart failure, cardiogenic shock
2-20 µg/kg/min IV/IO infusion; titrate to desired effect                                                                                                |
| Dopamine             | Cardiogenic shock, distributive shock
2-20 µg/kg/min IV/IO infusion; titrate to desired effect                                                                                                |
| Epinephrine          | Pulseless arrest, bradycardia (symptomatic)
0.01 mg/kg (0.1 mL/kg) 1:10,000 IV/IO q3-5 min (max 1 mg; 1 mL)
0.1 mg/kg (0.1 mL/kg) 1:1000 ETT q3-5 min
Hypotensive shock
0.1-1 µg/kg/min IV/IO infusion (consider higher doses if needed)                                                                                       |
| Hydrocortisone       | Adrenal insufficiency
2 mg/kg IV bolus (max 100 mg)                                                                                                                        |
| Lidocaine            | VF/pulseless VT, wide-complex tachycardia (with pulses)
1 mg/kg IV/IO bolus
Maintenance: 20-50 µg/kg/min IV/IO infusion (repeat bolus dose if infusion initiated >15 min after initial bolus)
2-3 mg/kg ETT                                                                                                                                         |
| Milrinone            | Myocardial dysfunction and increased SVR, PVR
Loading dose: 50-75 µg/kg IV/IO over 10-60 min followed by 0.5-0.75 µg/kg/min IV/IO infusion                                                         |
| Norepinephrine       | Hypotensive (usually distributive) shock (i.e., low SVR and fluid refractory)
0.1-2 µg/kg/min IV/IO infusion; titrate to desired effect                                                                                             |
| Oxygen               | Hypoxia, hypoxemia, shock, trauma, cardiopulmonary failure, cardiac arrest
Administer 100% O2 via high-flow O2 delivery system (if spontaneous ventilations) or ETT (if intubated); titrate to desired effect                   |
| Procainamide         | SVT, atrial flutter, VT (with pulses)
15 mg/kg IV/IO load over 30-60 min (do not use routinely with amiodarone)                                                                                  |
| Sodium bicarbonate (NaHCO3) | Metabolic acidosis (severe), hyperkalemia
1 mEq/kg IV/IO slow bolus
Sodium channel blocker overdose (e.g., tricyclic antidepressant)
1-2 mEq/kg IV/IO bolus until serum pH > 7.45 (7.50-7.55 for severe overdose) followed by IV/IO infusion of 150 mEq NaHCO3/L solution to maintain alkalosis |

D3W, dextrose 10% in water; D25W, dextrose 25% in water; ETT, endotracheal tube; IV, intravenously; IO, intrasosseously; O2, molecular oxygen; PRN, as needed; PVR, pulmonary vascular resistance; SVR, systemic vascular resistance; SVT, supraventricular tachycardia; VT, ventricular tachycardia.
are superior to those after other types of cardiac arrests, and that early rhythm recognition is necessary for optimal care.

Because of the increasing awareness that “shockable” rhythms are not uncommon in children, greater attention has been focused on the dose for pediatric defibrillation. The recommended shock dose is 2 to 4 joules (J)/kg, which is based on animal studies of short-duration VF and a single retrospective study of in-hospital (short-duration) VF with 91% (52 of 57) defibrillation success. More recent animal and pediatric data indicate that 2 J/kg is often ineffective at terminating fibrillation, and higher doses up to 10 J/kg may be needed to terminate VF.99,94 Thus recent recommendations suggest an initial dose of 2 to 4 J/kg followed by 4 J/kg if VF is not terminated; if VF continues, consider increasing the defibrillation dose up to 10 J/kg, not to exceed adult maximum doses.

**Cardiopulmonary Resuscitation in Pediatric Bradycardia**

Neonates, infants, and children are primarily dependent on heart rate for maintenance of CO. Their ability to augment stroke volume to increase CO is limited, and physiologic or pathophysiologic states leading to an increase in CO are hallmarked by tachycardia. Conversely, illnesses or injuries resulting in negative chronotropy (e.g., heart block, toxicity from beta-blockers or calcium channel blockers) tend to result in more profound shock and hypoperfusion in children than in adults with similar processes.

Bradycardia with hypoperfusion (without pulselessness) is a very common hemodynamic state for critically ill children during the prearrest phase. Early clinical studies of terminally ill children demonstrated almost ubiquitous prevalence of bradycardia before the onset of cardiac arrest.93 Animal models of asphyxia have demonstrated a predictable hemodynamic progression from tachycardia to bradycardia with hypotension, followed by PEA and asystole,94 and that CPR earlier in this continuum is associated with more favorable outcomes.92,95 Given that the majority of children with cardiac arrest experience respiratory insufficiency or circulatory insufficiency or both before the onset of pulselessness, a bradycardic child in shock should be considered to be in a prearrest state. Multiple reversible causes need to be considered, but immediate support of cardiovascular status is essential.

Neonatal resuscitation algorithms have recommended escalation of respiratory and cardiac support for the neonate whose heart rate is less than 60 beats/min, including the provision of chest compressions if bradycardia does not resolve with effective ventilation and oxygenation. Multiple studies of pediatric patients from the GWTG-R database have shown that 18 to 54% of patients younger than age 18 who receive chest compressions in the hospital are in a state of bradycardia and hypoperfusion, as opposed to pulselessness, when CPR is initiated.19,23,36 A multivariate analysis from the NRCPR demonstrated an association between CPR for bradycardia (as opposed to pulseless arrest) and survival to discharge, even when controlling for age, diagnosis, and event characteristics.96 Current AHA guidelines recommend the consideration of immediate chest compressions for a child with a heart rate of less than 60 with obvious hypoperfusion.86

**Extracorporeal Circulatory Support during Cardiac Arrest**

The use of extracorporeal cardiovascular support for cardiac arrest depends on the rapid availability of the resources, equipment, and personnel to establish mechanical circulatory support, most typically ECMO or, less commonly, VADs. Multiple case series have reported favorable outcomes in children placed on ECMO during cardiac arrest, with some demonstrating a relationship between shorter duration of CPR before ECMO support and survival.97–99 One retrospective series of 66 children placed on ECMO for cardiac arrest demonstrated an overall survival to hospital discharge of 45%; the median time of CPR before established ECMO circulation was 46 minutes.100 Another single-center study demonstrated 7 of 31 patients (22.5%) surviving to discharge after extracorporeal cardiopulmonary resuscitation (ECPR).101 Current consensus statements from the AHA state that there is insufficient evidence for a time-based threshold within which ECPR may be beneficial.86 Centers with resources for ECPR may consider its use for patients with other epidemiologic features known to be favorable (e.g., witnessed arrests, short CPR times).

**SEPTIC SHOCK IN THE PEDIATRIC PATIENT**

**Perspective**

Worldwide, sepsis is the leading cause of death in children. In the United States the annual incidence of severe sepsis (defined as sepsis with organ system dysfunction) is 0.56 cases per 1000; mortality for severe sepsis in the pediatric population is 10% overall, with a higher mortality (14%) among children with chronic medical conditions.102 With historical cohorts exhibiting mortality above 90%, these figures represent a substantial decline in mortality from pediatric sepsis in the past 30 years.103 In the past decade, both the American College of Critical Care Medicine (ACCM) and the International Consensus Conference on Pediatric Sepsis have put forth recommendations for rigorous definitions and treatment recommendations104–105; these efforts have effectively summarized the existing data on pediatric sepsis but are also illustrative of the need for rigorous clinical trials to improve outcomes further.

**Pathophysiologic Principles**

By definition, shock represents the physiologic state in which cellular oxygen demand for adenosine triphosphate (ATP) production is in excess of oxygen supply. Disruptions in oxygen supply (hypoxia), oxygenation of pulmonary capillary beds (respiratory illness), oxygen content (e.g., anemia or altered oxyhemoglobin binding such as methemoglobinemia), oxygen delivery (hypoperfusion), or oxygen usage at the tissue or cellular level (mitochondrial dysfunction, as in cyanide poisoning) can result in shock. The International Consensus Conference on Pediatric Sepsis defines septic shock as the simultaneous presence of (1) systemic inflammatory response, as evidenced by elevation or depression of temperature, heart rate, respiratory rate, and white blood cell count, (2) a documented or suspected infection, and (3) cardiovascular dysfunction, defined as hypotension or need for vasoactive medications despite more than 40 mL/kg crystalloid resuscitation.105

The developmental progression of cardiovascular physiology from neonate to adult has important bearings on shock physiology. Adults with sepsis most commonly exhibit a hemodynamic profile consistent with vasomotor paralysis, marked by vasodilatation and increased CO (“warm” shock). By contrast, infants and children with sepsis exhibit greater variability in their hemodynamic profile in sepsis and are most frequently in “cold” shock, marked by relative hypovolemia, vasoconstriction, and myocardial failure. One ICU-based study of children with sepsis with thermodilution catheters found that 58% of patients had low CO and increased SVR as opposed to 20% having low SVR and increased or normal CO.106 A more recent study of 30 children in an ICU with fluid-resistant shock showed that the majority (94%) of children with catheter-associated bloodstream infections demonstrated low SVR–high CO (warm) shock, whereas children with community-acquired infections were predominantly (86%) in high SVR–low CO (cold) shock.107 Neonates have a particular sensitivity to acidosis with respect to increased pulmonary vascular resistance and decreased
myocardial function. The syndrome of persistent pulmonary hypertension of the newborn (PPHN) is among the prevalent manifestations of sepsis in the newly born; therapy in these children frequently includes a hemodynamic therapeutic regimen targeted toward right ventricular failure.

**Clinical Features**

Table 10-4 demonstrates the physical signs of compensated, advanced compensated, and decompensated shock as summarized in PALS. Given the dependency of the pediatric patient on heart rate for CO, it can be intuited that tachycardia is the most sensitive sign of shock in children. It is important to note that hypotension is a very late sign in children with shock, and its absence should not be construed as a reassuring sign; patients may have decompensated shock with existing end-organ hypoperfusion and still have a measured blood pressure within normal limits.

Current guidelines from the ACCM recommend that the combined clinical signs of tachycardia, fever, and altered perfusion and altered mental status be used to diagnose sepsis in children. Unfortunately, this clinical triad is lacking in specificity and positive predictive value. Fever is among the most common chief complaints for children presented to an ED, occurring in more than 25% of all patients; discerning what degree of alteration in behavior in a febrile child constitutes altered mentation can be very challenging. A study from a pediatric ED examining children with fever, tachycardia, and tachypnea found that altered mental status (as judged by attending physicians) was present in 19% of children, and its presence had a specificity of 84% and positive predictive value of 24% in predicting sepsis with clinically significant organ system dysfunction. Conversely, data on prehospital care of children in shock have demonstrated that shock may be just as easily underdiagnosed clinically. A study of children referred to a tertiary PICU found that among 1803 children transported to a tertiary care center with shock, only 335 (19%) were referred specifically for shock, suggesting that signs of hypoperfusion were underappreciated in a substantial subset of patients. Despite these shortcomings of relying on the physical signs alone, the consensus of the ACCM is to recommend “early recognition of pediatric septic shock by clinical examination, not biochemical tests.”

Given the difficulty with lack of precision of the physical examination as discussed previously, laboratory and radiologic studies with greater precision for identifying sepsis are desirable. In adult patients, data supporting the use of serum lactate in initial diagnosis as well as in ongoing sepsis management have been repeatedly demonstrated in both ED and ICU studies. Additional markers used in adults include procalcitonin, B-natriuretic peptide, and markers of coagulopathy or disseminated intravascular coagulation; reports examining these laboratory markers in pediatric patients have demonstrated some promise, but robust clinical trials have not yet been performed.

Multiple noninvasive techniques for directly assessing hemodynamic states have been examined in pediatric patients; the use of focused bedside ultrasound examining cardiac function, inferior vena cava diameter, aortic pulse wave contour analysis, and electrical velocimetry to measure preload status as well as cardiac index have been documented in small studies. These techniques may hold promise for a more objective hemodynamic assessment in the febrile child to enable an emergency physician to distinguish a child with a harmless febrile illness from one with early septic shock.

**Management**

Figure 10-4 is an algorithm of the recommended sequence of care in the pediatric patient with sepsis. The pathway is designed to encompass the continuum of care from initial presentation in the ED through definitive management in the ICU setting. The remainder of this chapter focuses on the management elements pertinent to the ED.

**Substrate Correction: Hypoglycemia and Hypocalcemia**

Hypoglycemia is common among children with sepsis, especially the very young child with poor glycogen stores and underdeveloped capacity for gluconeogenesis. Documented hypoglycemia should be immediately corrected with an IV bolus of dextrose from 0.5 to 1 g/kg. The “rule of 50s” (5 mL/kg of 10% dextrose, 2 mL/kg of 25% dextrose, or 1 mL/kg of 50% dextrose) can be used to rapidly calculate the weight-based dose of dextrose. Given the association of hyperglycemia and poor outcomes among critically ill children and adults, empirical use of glucose (in the absence of documented hypoglycemia) should be avoided.

Developmental cellular physiology is pertinent to calcium metabolism in children; neonates and young infants have poorer capacity for calcium storage in the sarcoplasmic reticulum and underdeveloped T-tubule systems for propagation of calcium channel–associated cardiac myocyte depolarization and maintenance of tetanic contraction. Ionized hypocalcemia has been shown to be prevalent in critically ill children, particularly those having cardiac arrest in the context of septic shock.

**Fluid Resuscitation**

Studies documenting the relationship between amount of initial crystalloid resuscitation and survival in children with sepsis have existed for decades. Current AHA recommendations state that isotonic crystalloid fluids (normal saline, lactated Ringer’s) are the initial resuscitation fluid of choice for pediatric patients in shock, irrespective of cause or physiology. Septic shock in children is most frequently marked by relative or absolute hypovolemia; outcomes from shock in children have been shown to be substantially improved when the shock state is reversed as early as possible. Volumes of up to and above 60 mL/kg of crystalloid given intravenously (IV) or intraosseously (IO) are frequently necessary for children in shock.

The administration of fluids to the child in shock should be regarded differently than fluid administration in the child who is hypovolemic but not in shock. Current guidelines from the ACCM recommend the administration of fluid in a rapid fashion, ideally with 20 mL/kg being given in a 5-minute period. This rate of fluid administration would permit 60 mL/kg of saline to be given to a septic patient in 15 minutes. A recent study in children in an ED examined multiple methods of fluid administration, showing that 58% of patients received 20 mL/kg in 5 minutes when a pressure bag was used, 68% of patients when a manual push-pull system was used, and none when fluid was administered by gravity

| Table 10-4 Signs of Shock in Children |
|-------------------------------|-------------------------------|-------------------------------|
| COMPENSATED | ADVANCED COMPENSATED | DECOMPENSATED |
| Tachycardia | Depressed mental status | Any of the signs of compensated shock plus: |
| Cool extremities | Tachypnea | Hypotension |
| Prolonged capillary refill | Decreased urine output | Nondetectable distal pulses |
| Weak peripheral pulses (compared with central pulses) | Weak central pulses | |
| | Acidosis | |
| | Narrowed or widened pulse pressure (with normal systolic blood pressure) | |

**PART I: Fundamental Clinical Concepts / Section One • Critical Management Principles**
First, the hemodynamic profile of a child in septic shock is variable and often changes over time. Second, obtaining central venous access in an ill child is an uncommon procedure in most EDs and can be challenging in a hypovolemic patient. Among available agents, dopamine may have the greatest usefulness as a first-line agent given its safety in peripheral venous or intraosseous administration and the ability to titrate the dose to target dopaminergic, beta, and alpha receptors. Dopamine has been associated with leukopenia and a possible risk of infectious complications based on its downregulation of pituitary prolactin and hypocalcemia. Begin antibiotics.

Vasoactive Medications

The choice to use vasoactive infusions in children with fluid-refractory septic shock takes multiple considerations into account. First, the hemodynamic profile of a child in septic shock is variable and often changes over time. Second, obtaining central venous access in an ill child is an uncommon procedure in most EDs and can be challenging in a hypovolemic patient. Among available agents, dopamine may have the greatest usefulness as a first-line agent given its safety in peripheral venous or intraosseous administration and the ability to titrate the dose to target dopaminergic, beta, and alpha receptors. Dopamine has been associated with leukopenia and a possible risk of infectious complications based on its downregulation of pituitary prolactin and hypocalcemia. Begin antibiotics.

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thyrotropin-releasing hormone secretion; adult ICU data on dopamine use for shock have shown an association between dopamine use and adverse outcomes. Epinephrine and dobutamine may both be prepared in concentrations that are safe for temporary peripheral administration and, depending on the patient’s hemodynamic profile, may be appropriate first choices as well.

Corticosteroids

Adrenal insufficiency in sepsis has been reported as a moderately prevalent phenomenon in children and adults. Studies on the use of hydrocortisone replacement for sepsis showed early promise in adults, but the most robust randomized trial to date (the CORTICUS trial) showed no significant survival benefit conferred by hydrocortisone compared with placebo. Studies in pediatric patients have yielded inconclusive results. Current treatment recommendations by the AHA state that there is insufficient evidence to support or refute the use of exogenous corticosteroids in children with sepsis, nor is there a clear diagnostic modality for adrenal insufficiency that is of optimal reliability. In cases where corticosteroid supplementation is deemed to be warranted, an initial dose of 100 mg/m² of hydrocortisone is recommended.

RESPIRATORY FAILURE

Perspective and Epidemiology

Some of the earliest studies in pediatric resuscitation clearly demonstrated improved outcomes among children experiencing respiratory arrest alone compared with patients with cardiac arrest. The recognition and management of respiratory failure in children is a focal point of the AHA PALS curriculum; this is based on the fact that effective management of respiratory failure is, in effect, preventative of cardiac arrest in many children.

Pathophysiologic Principles

Anatomic Features Pertinent to Laryngoscopy and Intubation in Children

- **Size**—airway structures are smaller, and the field of vision is more narrow.
- **Adenoidal hypertrophy**—common in young children.
- **Developing teeth**—young infants are edentulous, but the underlying alveolar ridge contains developing tooth buds that are susceptible to disruption.
- **Primary teeth**—in young children, primary teeth can be easily avulsed or aspirated.
- **Tongue**—large relative to size of oropharynx.
- **Superior larynx**—often referred to as anterior, the laryngeal opening in infants and young children is actually located in a superior position (in infants, the larynx is opposite C₇-C₈ as opposed to C₇-C₈ in adults). This makes the angle of the laryngeal opening with respect to the base of the tongue more acute and visualization more difficult.
- **Hyoepliglottic ligament** (connects base of tongue to epiglottis)—has less strength in young children; thus a laryngoscope blade in the vallecula will not elevate the epiglottis as efficiently as in an adult.
- **Epiglottis**—in children, the epiglottis is narrow and angled acutely with respect to the tracheal axis; thus the epiglottis covers the tracheal opening to a greater extent and can be more difficult to mobilize.
- **Narrowest point** of the young child’s airway—occurs at the level of the cricoid cartilage instead of at the level of the glottic opening itself.

Clinical Features

Pediatric Respiratory Physiology

**Lung.** Infants have fewer and smaller alveoli than young children, and their overall gas exchange surface area is disproportionately small. Surface area reaches proportions similar to that in adulthood by 8 years of age. Channels for collateral ventilation (pores of Kuhn and Lambert’s channels) are absent in infancy. The overall effect of these phenomena is a greater tendency for alveolar hypoventilation and for the development of atelectasis during a respiratory illness.

**Respiratory Mechanics.** The pediatric thoracic skeleton is largely cartilaginous and much more compliant than the adult skeleton. Elastic recoil of the chest wall in the young child is essentially absent. A given change in thoracic pressure will result in a larger change in lung volume, similar to the physiology seen in an adult with emphysema. A given change in volume is associated with little or no change in pressure, so a greater amount of work is required to generate a tidal breath.

The high compliance of the pediatric chest wall results in a closing volume (CV; volume at which terminal bronchioles collapse because they are no longer supported by elastic recoil) that can be elevated with respect to functional residual capacity (FRC). If the already diminished elastic recoil is impaired—for example, by supine positioning—CV can exceed FRC to an even greater extent, resulting in the absence of ventilation of some lung segments during normal tidal breathing. Young patients therefore have a greater tendency for intrapulmonary shunting and hypoxemia with the positioning required for airway management.

Accessory respiratory muscles in young children are composed of a lower percentage of slow-twitch muscle fibers and are more susceptible to fatigue compared with the diaphragm. Also, the architecture of the pediatric thorax (horizontal rib orientation with extensive cartilage composition) is such that intercostal and suprasternal muscles are poorly recruited to assist in respiratory effort.

**Airway.** Airway diameter and length increase with age. The distal airway (bronchioles) lags in growth behind the proximal airway during the first few years of life. Poiseuille’s law states that airflow resistance is inversely proportional to the fourth power of the radius of the airway. Thus young children have higher resistance to airflow at baseline in their lower airways, and a change in airway diameter of a given dimension will have a much more profound effect on airway resistance in a small child than in an older child or adult. Illnesses that affect the caliber of small airways (such as asthma and viral bronchiolitis) produce a disproportionate increase in work of breathing in infants and children.

**Cellular Oxygenation.** Resting oxygen consumption in the newborn is twice that of an adult (6 mL/kg/min vs. 3 mL/kg/min). Oxygen consumption in infants is extremely sensitive to physiologic derangement such as fever and hypothermia. The oxyhemoglobin dissociation curve for young infants is shifted to the left (greater affinity for oxygen and poorer tissue oxygen delivery) by the presence of elevated amounts of fetal hemoglobin (HbF). HbF is the main oxygen transport protein in the fetus during the last seven months of development in the uterus and in the newborn until roughly 6 months of age. Functionally, HbF differs most from adult hemoglobin in that it is able to bind oxygen with greater affinity than the adult form, giving the developing fetus better access to oxygen from the mother’s bloodstream. In newborns, HbF is nearly completely replaced by adult hemoglobin by approximately the 12th week of postnatal life.

In a clinical study of elective surgery patients, the mean time to desaturation to 90% was 118 seconds in infants, 168 seconds in toddlers, and 248 seconds in children older than 3 years. The time...
required for the saturation to fall from 95% to 90% was significantly shorter in infants than in older children as well (8 seconds compared with 16 seconds). These findings occurred after a 2-minute period of ventilation with 100% oxygen before neuromuscular blockade, a preoxygenation time determined by some of the same authors in a separate clinical study to be optimal for minimizing risk of early desaturation. It is logical to conclude that these times to critical desaturation occur even more rapidly in ill children.

Management Considerations Specific to Pediatrics

Effectiveness of Tracheal Intubation versus Bag-Valve Mask Ventilation in Prehospital Care

Pediatric patients constitute a small minority of patients cared for by prehospital personnel, reported frequency of tracheal intubation for individual prehospital care communities is generally very low, with incidence for an individual provider being as infrequent as once every 3 years. Optimal methods of maintaining competency for prehospital care providers have not been established.

Previous studies of prehospital airway management in children with cardiac arrest found some associations between successful intubations and improved short-term outcomes, although no strong association with survival. The most robust trial to date examining prehospital management of pediatric respiratory failure involved a controlled trial of bag-valve-mask ventilation versus tracheal intubation (performed on alternate days) by prehospital care providers in California. In this trial, 820 patients were enrolled; no significant difference in survival or neurologically intact survival was demonstrated between the two groups. Limitations ascribed to this landmark study include relatively short prehospital care times (median 20-23 minutes) and the fact that intubation was at that time a new skill for the provider community in question.

Equipment Considerations for Pediatric Intubation

Endotracheal Tubes. The two most commonly applied rules of thumb for sizing of endotracheal tubes (ETTs) are the age-based rule and selection based on body length (the Broselow-Luten tape). The age-based rule is as follows:

\[
\text{[Age in years/4]} + 4 = \text{ETT size}
\]

King and colleagues found this rule to correctly predict ETT size within a range of 1 mm in 97.5% of patients. The Broselow-Luten tape selects size of ETT based on the length of the patient. The initial study of the validity of the Broselow-Luten tape found it to be more accurate than age-based selection criteria. A more recent study by Hofer and colleagues also found that the Broselow-Luten tape was more accurate (correct in 55% of patients) than the age-based rule (correct in 41%), but also found that the Broselow-Luten tape was prone to underestimating ETT size (in 39% of patients) whereas the age-based rule tended to overestimate (in 57% of patients). Another recent study found that there was no difference between the accuracy rates of the two methods, and that Broselow-Luten tape measurement and the age-based rule predicted the same size of ETT in 66% of patients. Either method may be used to determine ETT size in children, but the advantage of the Broselow-Luten tape is that mathematical calculations do not need to be used and therefore this method is preferred in most settings.

Cuffed ETTs exist in sizes as small as 2.5 mm inner diameter (ID). Multiple studies in the pediatric operating room (OR) and ICU have demonstrated no significant difference in the incidence of postextubation stridor or extubation failure when comparing children intubated with cuffed versus uncuffed ETTs. Current AHA recommendations state that either cuffed or uncuffed tubes may be considered for emergency intubation for children but that cuffed tubes may be more appropriate when high inflating pressures are anticipated. If a cuffed tube is to be used, select a tube that is one half size smaller than the size determined by standard calculations for an uncuffed ETT tube.

Medications

Atropine. Premedication with atropine is recommended by the American College of Emergency Physicians for children younger than 1 year and those undergoing intubation with succinylcholine (SCh). Atropine premedication is believed to minimize bradycardia during laryngoscopy and ETT placement, given the increased vagal tone in the pediatric patient. Clinical data supporting the efficacy of this recommendation are lacking; a study of pediatric ED intubations showed a very low incidence of bradycardia during intubation and no significant difference in its occurrence between patients premedicated with atropine and those who were not.

Succinylcholine. The U.S. Food and Drug Administration (FDA) Advisory Committee for Anesthetic and Life Support Drugs recommended a labeling revision for SCh at its meeting in 1992. The new label states that routine use of SCh in children should be avoided and alternative agents (nondepolarizing neuromuscular blockers [NMBs]) used except in specific circumstances, including laryngospasm, full stomach, or intramuscular use when intravenous access is difficult or absent. Succinylcholine, however, remains in widespread use in children for rapid sequence intubation (RSI) despite the labeling change. In one large multihospital database, SCh was the NMB used in 91% of patients who underwent RSI. Succinylcholine may still be considered a first-line NMB for RSI in children; initial administration of SCh in children should be at a higher dose of 2 mg/kg.

Rescue Devices

The laryngeal mask airway (LMA) is the airway rescue device of choice in children. Studies have documented ease of placement and use by multiple classes of care providers with a minimal incidence of complications. No consistent rule of sizing exists for LMAs in children; all LMAs are labeled with parameters of the appropriate weight of patients in whom the device can be used, and the Broselow-Luten resuscitation tape has LMA sizes based on length. As with ETTs, a range of sizes should be available for use in the event of an initial device performing suboptimally. One OR-based study found that an LMA can be accurately sized in the majority of children by comparing the width of the cuff with the width of the patient’s second, third, and fourth digits; however, this method is not routinely used by emergency providers.

ACUTE LIFE-THREATENING EVENTS

Acute life-threatening event (ALTE) is a description of a characteristic clinical presentation; therefore the pathophysiology of an ALTE has not been clearly defined. Children with ALTE pose a diagnostic and therapeutic challenge to emergency physicians because the cause of such events is diverse and ranges from minor to life-threatening. In addition, studies of children with ALTE reveal that in 50% of the cases, a definitive diagnosis will not be made. The etiology of ALTE is extensive and includes infection (sepsis; respiratory syncytial virus or other respiratory viruses; pertussis; or central nervous system infection), gastroesophageal reflux disease (GERD) with or without obstructive apnea, congenital
The infant with a history of an ALTE may look well and act normally at the time of the evaluation by the nonhospital medical provider or the emergency physician. However, infants who are judged by nonhospital personnel to have choked, who have turned blue, or who are showing other signs suggestive of a possible ALTE should be considered seriously ill and transported to the ED for evaluation.\(^{162}\) Fifty percent of children brought to the ED after an ALTE have an entirely normal clinical examination, and the final diagnosis often correlates poorly with the presenting signs and symptoms, which often include cyanosis, breathing difficulties, abnormal movements, loss of consciousness, vomiting, pallor, and choking.\(^{162}\)

The ED evaluation of children with ALTE is tailored to the child’s history and physical examination findings but often includes laboratory and radiographic studies. Laboratory evaluation may include complete blood count, serum glucose, electrolytes, blood and urine cultures if the child is younger than 1 month or febrile, a toxicology screen, and an electrocardiogram.\(^{164}\) The infant may also undergo a screen for inborn errors of metabolism, chest radiograph, computed tomographic scan of the head, and lumbar puncture depending on signs and symptoms at presentation, although few diagnostic test results are positive.\(^{165}\) Several studies have evaluated the risk of bacteremia in infants with an ALTE. Essentially, the risk of serious bacterial infections in this population is similar to age-matched cohorts without ALTE.\(^{166,167}\) However, in a study by Zuckerbraun of 182 well-appearing infants less than 60 days of age with ALTE, prematurity increased the risk of bacterial infection significantly (6.7% vs. 0.8%, RR = 2.8, 95% CI 1.5-5.1); absence of chocking (RR 2.6, 95% CI 1.4-4.7), color change to blue (RR 2.1, 95% CI 1.2-3.8), and abnormal examination findings in ED (RR 2.8, 95% CI 1.6-4.9) were significantly associated with the need for admission.\(^{168}\) Fu and Moon suggest other conditions under which it may be safe to discharge a patient with an ALTE, and these include the following: (1) the episode is brief, nonsevere, and self-resolving; (2) the cause is probably a nonprogressive condition such as GERD; and (3) the infant has no comorbidities and appears well.\(^{170}\) Kaji and others\(^{170}\) developed and validated a clinical decision rule for ALTE in 832 infants. In addition to patients in whom it is obvious that admission is necessary at the end of the ED stay, other predictors of those warranting hospitalization include a significant medical history (OR = 2.1, 95% CI 1.1-4.3) and greater than one ALTE in 24 hours (OR = 2.1, 95% CI 1.2-4.6). Any infant not appearing well at the time of ED evaluation should be admitted for monitoring and further evaluation, but this rule allows for the option to discharge with close follow-up.

### Sudden Infant Death Syndrome

The National Center for Health Statistics reports that SIDS is the third leading cause of death in infants, accounting for 8% of deaths in children younger than 1 year.\(^{171}\) SIDS may occur at any time during the first 2 years of life, but it is rare (1%) in children younger than 1 month and in those older than 1 year (2%). Ninety-five percent of SIDS infants die before 6 to 8 months, with a peak occurring at 2 to 4 months of age.\(^{171,172}\) Some epidemiologic variation occurs among different racial and ethnic groups, with black, Native American, and Alaskan Native infants having rates two to three times higher than the national average.\(^{172}\) Other epidemiologic risk factors include male sex and multiple births.\(^{173,175}\)

The most important modifiable risk factor for SIDS is prone sleeping. In 1992 the American Academy of Pediatrics (AAP) recommended that infants be placed to sleep in a nonprone position to reduce the risk of SIDS.\(^{176}\) The “Back to Sleep” (BTS) campaign was initiated in 1994 under the leadership of the National Institute of Child Health and Human Development (NICHD), as a collaborative effort of the U.S. Public Health Service, the AAP, the SIDS Alliance, and the Association of SIDS and Infant Mortality Programs. Since then, the frequency of prone sleeping has decreased 50 to 90% worldwide, as has the rate of SIDS.\(^{174,177,178}\)

Because CPR is unsuccessful in the majority of cases, the emergency physician should provide supportive care for the family. When the cause of death is unknown, appropriate samples (blood, urine) should be obtained. Recent data have shown that 10 to 20% of SIDS cases are caused by genetic variants in either ion channel or ion channel–associated proteins.\(^{179,180}\) Therefore an autopsy and postmortem genetic testing should be performed by a competent and experienced pathologist on all infants who have died from SIDS so that families can be appropriately counseled on risk of sudden death in other family members.\(^{180}\)

#### Psychosocial Considerations for Sudden Infant Death Syndrome

The emergency physician and pediatrician address psychosocial considerations in any SIDS case. The physician should be direct when informing parents that their child has died. The word dead or died should be used instead of confusing euphemisms such as passed on.\(^{182,183}\) Parents universally experience intense guilt, and siblings may also have guilt over the loss of their brother or sister. In addition, parents may further intensify the guilt by accusing each other of not taking adequate care of the infant. The police investigation may arouse suspicion in neighbors and friends and leave the parents and caretakers socially alienated. The overall toll of guilt and social alienation is enormous, and the effects are manifested in increased rates of miscarriage, divorce, and infertility after a SIDS death. The outcome of SIDS for the family depends on the support they receive. Thus, the team approach, which includes the nurse, social worker, chaplain, emergency physician, and pediatrician, may provide comfort and information to the grieving family. The emergency physician and the pediatrician...
should recognize that they can play a pivotal role in helping the family to adjust to their loss, initiate the process of grieving, and educate the family about SIDS prevention.152,153 The AAP and the American College of Emergency Physicians have outlined recommendations for emergency physicians in a joint policy statement entitled “Death of a Child in the Emergency Department.”

KEY CONCEPTS

- Excellent CPR is the foundation for successful resuscitation from cardiac arrest. The mantra for excellent CPR is “Push hard, push fast, allow full chest recoil, and minimize interruptions.”
- Despite evidence supporting the efficacy of chest compression–only CPR in adults, children in cardiac arrest should receive ventilation plus chest compressions.
- Bradycardia with hypoperfusion should be considered a prearrest state in children, and chest compressions for bradycardia may improve survival.
- Septic shock in children is most commonly associated with hypovolemia and myocardial failure as opposed to vasomotor paralysis. Rapid administration of isotonic fluids is the initial step in resuscitation for all forms of pediatric shock.
- The diagnosis of septic shock in children is primarily clinical, and distinguishing the child with an innocent febrile illness from one with early sepsis is difficult. The role of ancillary biochemical and radiologic testing remains unclear.

- Respiratory failure in children is more common than cardiac arrest and is the most important pathophysiologic cause of cardiac arrest. Pediatric anatomy, physiology, and pathophysiology are important considerations in managing respiratory failure and performing tracheal intubation.
- The availability of a range of equipment for airway management is essential. Cuffed endotracheal tubes can be safely and effectively used throughout the pediatric age range. Laryngeal mask airways (LMAs) are the airway rescue device of choice in pediatric patients.
- Acute life-threatening events constitute a range of clinical phenomena with an incidence of serious bacterial infection that is no different from that in the overall pediatric population; however, in young infants with a history of prematurity, microbiologic testing may be warranted to search for a potential infectious source. There is no association between ALTE and SIDS; workup in the ED should be driven by the clinical presentation.
- Sudden infant death syndrome remains the most common cause of death among patients younger than 1 year. The supine sleeping position for infants is believed to be the major contributor to the decreasing incidence over time. Channelopathies may be an important cause and warrant postmortem genetic testing. Psychological considerations for both families and care team members should be borne in mind when dealing with infants who die in the ED as a result of SIDS.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.
References


90. Reference deleted in proofs.


142. Reference deleted in proofs.

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