Part 10: Pediatric Advanced Life Support

Major Guidelines Changes

International Terminology
In the preparation of these guidelines, we recognized that certain terms that are commonplace in the United States are uncommon internationally and vice versa. Because these are international guidelines, efforts were made to use terms consistently throughout. To avoid confusion, the reader should note the use of the following terms:

- **Tracheal tube**—commonly called an endotracheal tube. Note that a tracheal tube may be incorrectly placed in the esophagus, so the term does not mean a correctly positioned tube in the trachea. Moreover, a tracheostomy tube is not the same as a tracheal tube as used in these guidelines, even though both tubes are placed in the trachea. The procedure of placing a tracheal tube is still called endotracheal intubation.

- **Manual resuscitator**—refers to a bag-valve device used to provide mask, tracheal tube, or tracheostomy tube ventilation to a victim. A manual resuscitator may be self-inflating or flow-inflating (i.e., an anesthesia manual resuscitator).

- **Exhaled CO\(_2\) detection**—refers to detection of carbon dioxide in exhaled gas. End-tidal CO\(_2\) monitors are a subset of exhaled CO\(_2\) detectors, but they specifically detect and measure the quantity of CO\(_2\) at the end of exhalation. Capnography graphically displays the change in exhaled CO\(_2\) over time, whereas exhaled CO\(_2\) detectors often are colorimetric systems designed to detect any CO\(_2\) during exhalation and not just at the end of expiration.

- **Defibrillation**—although commonly used interchangeably with “shocks,” defibrillation is the untimed (asynchronous) depolarization of the myocardium that successfully terminates ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). Thus, shocks are administered to victims in an attempt to achieve defibrillation.

Epidemiology and Recognition of Shock and Respiratory Failure

- We emphasize the need for better data regarding the epidemiology and treatment of pediatric cardiopulmonary arrest. There is a critical need for identification, tracking, and reporting of key resuscitation interventions and their relationship to various outcome measures, such as return of spontaneous circulation, survival, and neurological outcome. Published reports of resuscitation outcome are essential to provide data in future guideline reviews. Data collection efforts should use consistent terminology and record important time intervals. Critical elements for data collection have been described by an international consensus process called the Pediatric Utstein Guidelines for Reporting Outcome of Pediatric Cardiopulmonary Arrest.\(^1\)

- An age-defined sequence of “phone fast” resuscitation is still appropriate for treatment of out-of-hospital arrest in infants and children, but a “phone first” approach to resuscitation from sudden collapse should be used for children at high risk for arrhythmias.

Support of Ventilation

- The method of advanced airway support (endotracheal intubation versus laryngeal mask versus bag-mask) provided to the patient should be selected on the basis of the training and skill level of providers in a given advanced life support (ALS) system and on the arrest characteristics and circumstances (e.g., transport time and perhaps the cause of the arrest).

- Proficiency in the skill of bag-mask ventilation is mandatory for anyone providing ALS in prehospital and in-hospital settings (Class IIa).

- Secondary confirmation of proper tracheal tube placement is required for patients with a perfusing rhythm by capnography or exhaled CO\(_2\) detection immediately after intubation and during transport (Class IIa). We strongly encourage the use of exhaled or end-tidal CO\(_2\) detection. It is extremely reliable in a spontaneously perfusing victim (Class IIa), although it has lower specificity in the cardiac arrest victim (Class IIb). Adequate oxygenation should also be confirmed in a victim with a perfusing rhythm using pulse oximetry.

Fluid Therapy

- Rescuers should increase attention to early vascular access, including immediate intraosseous access for victims of cardiac arrest, and extend the use of intraosseous techniques to victims >6 years old.

Medications

- There is renewed emphasis on the need to identify and treat reversible causes of cardiac arrest and symptomatic arrhythmias, such as toxic drug overdose or electrolyte abnormalities.

- For cardiac arrest victims, we provide specific drug selection and dose recommendations but acknowledge the lack of adequate data to make such recommendations on the basis of firm evidence. For example, data supporting the use of high-dose epinephrine and the use of vasopressin in...
cardiac arrest is inadequate to allow firm recommendations (for further details, see the following section, “Drugs Used for Cardiac Arrest and Resuscitation”).

**Treatment of Arrhythmias**

- We introduce vagal maneuvers into the treatment algorithm for supraventricular tachycardia.
- We introduce the drug amiodarone into the treatment algorithms for pediatric VT and shock-refractory VF.
- Automated external defibrillators (AEDs) may be used in the treatment of children ≥8 years of age (approximately >25 kg body weight) in cardiac arrest in the prehospital setting.

**Postarrest Stabilization**

- We place increased emphasis on postresuscitation interventions that may influence neurological survival, which include maintenance of normal ventilation rather than hyperventilation (Class IIa) in most victims, control of temperature (avoid hyperthermia), management of postischemic myocardial dysfunction, and glucose control.

**Education and Training**

- Simplification of education and reinforcement of skill acquisition and core competencies are essential in all American Heart Association courses. See also, in “Part 9: Pediatric Basic Life Support,” Education and Training and Introduction.

**Introduction**

In contrast to cardiac arrest in adults, cardiopulmonary arrest in infants and children is rarely a sudden event and does not often result from a primary cardiac cause.2 In adults, cardiopulmonary arrest is usually sudden and is primarily cardiac in origin; approximately 250,000 adults die annually of sudden cardiac arrest in the United States alone. Consequently, much of the research and training in adult cardiac resuscitation focuses on the identification and treatment of VF in the out-of-hospital setting, since this rhythm is the most amenable to effective therapy. Factors associated with increased survival after adult cardiopulmonary arrest include bystander CPR (relative odds of survival, 2.6; 95% confidence interval, 2.0 to 3.4)3,4 and short interval to defibrillation.5,6

Cardiopulmonary (ie, cardiac) arrest in children is much less common than cardiac arrest in adults. When it does occur, pediatric cardiac arrest frequently represents the terminal event of progressive shock or respiratory failure. Causes of pediatric cardiac arrest are heterogeneous, including sudden infant death syndrome (SIDS), submersion/near-drowning, trauma, and sepsis. The progression from shock or respiratory failure to cardiac arrest associated with each of these causes may vary, making research or outcome reporting difficult, since there is not a “typical” type of cardiac arrest.

The cause of cardiac arrest also varies with age, the underlying health of the child, and the location of the event. In the out-of-hospital location, conditions such as trauma, SIDS, drowning, poisoning, choking, severe asthma, and pneumonia represent the most common causes of arrest. In the hospital, common causes of cardiac arrest include sepsis, respiratory failure, drug toxicity, metabolic disorders, and arrhythmias. These in-hospital causes often complicate an underlying condition. The Emergency Department represents a transition from the out-of-hospital to the hospital location. In the Emergency Department, cardiac arrest may be seen in children with underlying conditions typical for the hospital setting and in children with conditions seen more often in the out-of-hospital setting.

Throughout infancy and childhood, most out-of-hospital cardiac arrest occurs in or around the home. Beyond 6 months of age, trauma is the predominant cause of death.

Pediatric advanced life support (PALS) refers to the assessment and support of pulmonary and circulatory function in the period before an arrest and during and after an arrest. Consistent with the Chain of Survival (Figure 1), PALS should focus on prevention of the causes of arrest (SIDS, injury, and choking) and on early detection and rapid treatment of cardiopulmonary compromise and arrest in the critically ill or injured child. The components of PALS are similar in many respects to those of adult ACLS and include

- Basic life support
- Use of adjunctive equipment and special techniques to establish and maintain effective oxygenation, ventilation and perfusion
- Clinical and ECG monitoring and arrhythmia detection
- Establishment and maintenance of vascular access
- Identification and treatment of reversible causes of cardiopulmonary arrest
• Therapies for emergency treatment of patients with cardiac and respiratory arrest
• Treatment of patients with trauma, shock, respiratory failure, or other preexisting conditions

Because the etiology of cardiopulmonary emergencies and the available treatments and approaches may not be the same in out-of-hospital and hospital settings, these guidelines will highlight evaluation and treatment approaches that are recommended for each setting when appropriate.

These guidelines are based on clinical and experimental evidence of varying quality and quantity. Information on the strength of the scientific data leading to each new recommendation is provided. (For more information on the evidence evaluation process, see Reference 7.) Classes are defined fully in “Part 1: Introduction.”

Ideally, treatments of choice are supported by excellent evidence and are Class I recommendations. Unfortunately the quality of published data on cardiac arrest and resuscitation, especially for children, usually dictates that consensus treatments included in the guidelines are Class IIa or IIb.

**PALS for Children With Special Needs**

Children with special healthcare needs have chronic physical, developmental, behavioral, or emotional conditions and also require health and related services of a type or amount not usually required by other children.8–10 These children may require emergency care for acute, life-threatening complications that are unique to their chronic conditions, such as obstruction of a tracheostomy, failure of support technology (eg, ventilator failure), or progression of underlying respiratory failure or neurological disease. Approximately half of the EMS responses for children with special healthcare needs, however, are unrelated to those special needs.11 Many involve traditional causes of EMS calls, such as trauma,11 that require no treatment beyond the normal EMS standard of care.

Emergency care of children with special healthcare needs can be complicated by lack of specific medical information about the child’s baseline condition, plan of medical care, current medications, and any “Do Not Attempt Resuscitation” orders. Certainly the best source of information about a chronically ill child is a concerned and compassionate person who cares for the child on a daily basis. If that person is unavailable or incapacitated (eg, after an automobile crash), some means is needed to access important information. A wide variety of methods have been developed to make this information immediately accessible, including the use of standard forms, containers kept in a standard place in the home (eg, the refrigerator), window stickers for the home, wallet cards, and medical alert bracelets. No one method of information communication has yet proved to be superior. A standardized form, the Emergency Information Form (EIF), was developed by the American Academy of Pediatrics and the American College of Emergency Physicians,10 to be completed by the child’s primary physician for use by EMS personnel and hospitals. This form is available electronically (http://www.pediatrics.org/cgi/content/full/104/4/e53). Parents and child-care providers should be encouraged to keep copies of essential medical information at home, with the child, and at the child’s school or child-care facility. School nurses should have copies of these forms and should be familiar with signs of deterioration in the child and any existing “Do Not Attempt Resuscitation” orders.11,12

If decisions are made by the physician, parents, and child (as appropriate) to limit resuscitative efforts or to withhold attempts at resuscitation, a physician order indicating the limits of resuscitative efforts must be written for use in the in-hospital setting, and in most countries a separate order must be written for the out-of-hospital setting. Legal issues and regulations vary from country to country and within the United States from state to state regarding requirements for these out-of-hospital “No CPR Directives.” It is always important for a family to inform their local EMS system when such directives are established for out-of-hospital care. For further information about ethical issues of resuscitation, see also “Part 2: Ethical Aspects of CPR and ECC.”

Whenever a child with a chronic or life-threatening condition is discharged from the hospital, parents, school nurses, and any home healthcare providers should be informed about possible causes of deterioration or complications that the child may experience and anticipated signs of deterioration. They should receive specific instructions about CPR and other interventions the child may require and instructions about whom to contact and why.12

If the child has a tracheostomy, anyone responsible for the child’s care (including parents, school nurses, and home healthcare providers) should be taught to assess that the airway is patent, how to clear the airway, and how to provide CPR using the artificial airway. If CPR is required, rescue breathing and bag-mask ventilation are performed through the tracheostomy tube. As with any form of rescue breathing, the key sign of effective ventilation is adequate bilateral chest expansion. If the tracheostomy tube becomes obstructed and it is impossible to provide ventilation through it even after attempts to clear the tube with suctioning, remove and replace the tube. If a clean tube is unavailable, ventilation can be provided using mouth-to-stoma ventilation until an artificial airway can be placed through the stoma. Alternatively, if the upper airway is patent, it may be possible to provide effective conventional bag-mask ventilation through the nose and mouth while occluding the superficial tracheal stoma site.

**International PALS Guidelines**

Following the implementation of the 1992 guidelines,13 the major international resuscitation councils (International Liaison Committee on Resuscitation [ILCOR]) participated in the development of advisory statements reflecting consensus recommendations based on existing resuscitation guidelines, practical experience, and informal interpretation and debate of an international resuscitation database.14,15 A high degree of uniformity exists in current guidelines created by the major resuscitation councils for resuscitation of the newly born, neonates, infants, and young children. Controversies arise mostly from local and regional preferences or customs, training networks, and differences in availability of equipment and medication rather than from differences in interpretation of scientific evidence.
To develop this International Guidelines 2000 document on PALS, the Subcommittee on Pediatric Resuscitation of the AHA and other members of ILCOR identified issues or new developments worthy of further in-depth evaluation. From this list, areas of active research and evolving controversy were identified; evidence-based evaluation of each of these areas was conducted and debated, culminating in assignment of consensus-defined “levels of evidence” for specific guidelines questions. After identification and careful review of this evidence, the Pediatric Working Group of ILCOR updated the PALS guidelines, assigned classes of recommendations where possible, and objectively attempted to link the class of recommendation to the identified level of evidence. During these discussions the authors recognized the need to make recommendations for important interventions and treatment even when the only level of evidence was poor or absent. In the absence of specific pediatric data (outcome validity), recommendations were made or supported on the basis of common sense (face validity) or ease of teaching or skill retention (construct validity).

To reduce confusion and simplify education, whenever possible and appropriate, PALS recommendations are consistent with the adult BLS and ACLS algorithms and guidelines. Areas of departure from the adult algorithms and interventions are noted, and the rationale is explained in the text. Ultimately the practicality of implementing recommendations must be considered in the context of local resources (technology and personnel) and customs. No resuscitation protocol or guideline can be expected to appropriately anticipate all potential scenarios. Rather, these guidelines and treatment algorithms serve as a guiding template that will provide most critically ill children with appropriate support while thoughtful and appropriate etiology-based interventions are assembled and implemented.

Age Definitions: What Defines an Infant, Child, and Adult?

**Definition of Newly Born, Neonate, Infant, and Child**

The term “neonate” refers to infants in the first 28 days (month) of life.16 In AHA ECC and ILCOR publications, the term “newly born” refers specifically to the *neonate* in the first minutes to hours following birth. This term is used to focus resuscitation knowledge and training on the time immediately after birth and during the first hours of life. *Newly born* is designed to emphasize those first hours of life, separate from the first month of life. The term “infant” includes the neonatal period and extends to the age of 1 year (12 months). For the purposes of these guidelines, the term “child” refers to the age group from 1 year to 8 years.

Pediatric BLS and ALS interventions tend to blur at the margins of age because there is no single anatomic, physiological, or management characteristic that is consistently different in the infant versus the child versus the adult victim of cardiac arrest. Furthermore, new technologies such as AEDs and the availability of airway and vascular access adjuncts that can be implemented with a minimum of advanced training create the need to reexamine previous recommendations for therapies based on age.

**Anatomy**

By consensus, the age cutoff for infants is 1 year. Note, however, that this definition is not based on specific anatomic or physiological differences between infants and children. For example, the differences between an 11-month-old “infant” and an 18-month-old “child” are smaller than the differences in anatomy and physiology between an 11-month-old and a 1-week-old infant. Historically the use of the term *child* was limited to ages 1 to 8 years for purposes of BLS education; cardiac compression can be done with 1 hand for victims up to the age of approximately 8 years. However, variability in the size of the victim or the size and strength of the rescuer can require use of the 2-handed adult compression technique for cardiac compression in younger children. For example, a chronically ill 11-month-old infant may be sufficiently small to enable compression using the 2 thumb–encircling hands technique, and a 6- or 7-year-old may be too large for the 1-hand compression technique.

Further anatomic differences are noted in the airway of the child versus the adult. The narrowest portion of the airway in the child is at the level of the cricoid cartilage; in older children and adults the narrowest portion is at the level of the glottic opening. Moreover, the loose areolar tissue in the subglottic space allows for a natural seal without a cuffed tube in most children. Finally, attempting to squeeze a tube through the narrowed area of the cricoid cartilage increases the risk of subglottic stenosis. These anatomic differences and risk of complications led to the recommendation to use uncuffed tracheal tubes in children <8 years of age.13

**Physiology**

Respiratory and cardiac physiology evolves throughout infancy and childhood. In the newly born, for example, fluid-filled alveoli may require higher initial ventilation pressures than subsequent rescue breathing. In infants and children, lung inspiratory and expiratory time constants for alveolar filling and emptying may need to be adjusted according to both anatomic and physiological development. For example, the child with respiratory failure secondary to asthma clearly will require a different approach for mechanical ventilation support than a neonate with alveolar collapse caused by respiratory distress syndrome.

**Epidemiology**

Ideally the sequence of resuscitation should be tailored to the most likely cause of the arrest, but this increases the complexity of BLS and ALS education. For lay rescuers, CPR instruction must remain simple. Retention of current CPR skills and knowledge is now suboptimal, and more complex instruction is more difficult to teach, learn, remember, and perform. In the newly born infant, respiratory failure is the most common cause of cardiopulmonary deterioration and arrest. In the older infant and child, arrest may be related to progression of respiratory failure, shock, or neurological dysfunction. In general, pediatric out-of-hospital arrest is characterized by a progression from hypoxia and hypercarbia to respiratory arrest and bradycardia and then to asystolic cardiac arrest.2,17,18 Therefore, a focus on immediate ventilation and compressions, rather than the “adult” approach of immediate EMS activation or defibrillation, appears to be...
warranted. In this age group, early effective ventilation and oxygenation must be established as quickly as possible.

In some circumstances primary arrhythmic cardiac arrest is more likely than respiratory arrest, and the lay rescuer may be instructed to activate the EMS system first (eg, children with underlying cardiac disease or a history of arrhythmias). If a previously well child experiences a sudden witnessed collapse, this suggests a previously undetected cardiac disorder, and immediate activation of the EMS system may be beneficial. Children with sudden collapse may have a prolonged-QT syndrome, hypertrophic cardiomyopathy, or drug-induced cardiac arrest; the latter is more likely in the adolescent age group, related to a drug overdose.

For optimal patient outcomes, all of the links of the Chain of Survival must be strong. Unfortunately the rate of bystander CPR is disappointing: bystander CPR is provided for only approximately 30% of out-of-hospital pediatric arrests. A low rate of bystander CPR may mask improvements in the structure and function of the EMS system, since data in adults suggests a much worse outcome when bystander CPR is not provided. Because all the links are connected, it is difficult to evaluate components of single links such as the optimal method of EMS system activation or the effect of specific EMS interventions.

In addition, local EMS response intervals, dispatcher training, and EMS protocols may dictate the most appropriate sequence of EMS activation and early life support interventions. For example, providing 1 minute of CPR is recommended in pediatric out-of-hospital arrest before activation of the EMS system. Rather than using a uniform approach, however, perhaps the activation of the EMS system and the sequence of BLS support for out-of-hospital arrest should be based on the cause of arrest (ie, the cause of arrest could be separated into cardiac versus respiratory origin by lay rescuers). The increased educational complexity limits this approach, however. As noted above, if a cardiac cause is suspected on the basis of event circumstances, then immediate EMS activation may be more important than providing 1 minute of CPR. Once EMS providers arrive, early use of AEDs may be beneficial.

Although recommending an etiology-based resuscitation sequence for lay rescuers may be more medically appropriate in certain circumstances, it is more complex and therefore harder to teach, learn, and remember. Consequently, after much deliberation and debate, we continue to recommend the same approach as stated in the 1992 guidelines: phone first for adults and phone fast for children. Nevertheless, it is the responsibility of the healthcare provider to identify and train caretakers to call first when a high risk of a primary cardiac event is identified. It is also appropriate to teach more knowledgeable providers to “call first” for a likely arrhythmic cardiac arrest (eg, sudden collapse at any age) and to “call fast” in other circumstances (eg, trauma, a submersion event, or an apparent choking event).

**Recognition of Respiratory Failure and Shock**

Survival after cardiac arrest in children averages 7% to 11%, with most survivors neurologically impaired. For this reason we emphasize early recognition and treatment of respiratory failure and shock to prevent an arrest from occurring. To clarify terminology we use the following Pediatric Utstein Style definitions: “respiratory arrest” is defined as the absence of respirations (ie, apnea) with detectable cardiac activity. This should be distinguished from respiratory compromise leading to assisted ventilation. In the latter, the patient may have respiratory distress with increased effort or inadequate respiratory effort with no distress. Cardiac arrest is the cessation of cardiac mechanical activity, determined by the inability to palpate a central pulse, unresponsiveness, and apnea (ie, no signs of circulation or life).

Deterioration in respiratory function or imminent respiratory arrest should be anticipated in infants or children who demonstrate any of the following signs: an increased respiratory rate, particularly if accompanied by signs of distress and increased respiratory effort; inadequate respiratory rate, effort, or chest excursion; diminished peripheral breath sounds; gasping or grunting respirations; decreased level of consciousness or response to pain; poor skeletal muscle tone; or cyanosis.

“Respiratory failure” is a clinical state characterized by inadequate oxygenation, ventilation, or both. Strict criteria for respiratory failure are difficult to define because the baseline oxygenation or ventilation of an individual infant or child may be abnormal. For example, an infant with cyanotic congenital heart disease would not be in respiratory failure on the basis of an oxygen saturation of 60%, whereas that would be an appropriate criterion in a child with normal cardiopulmonary physiology. Respiratory failure may be functionally characterized as a clinical state that requires intervention to prevent respiratory or cardiac arrest.

“Shock” is a clinical state in which blood flow and delivery of tissue nutrients do not meet tissue metabolic demand. Shock may occur with increased, normal, or decreased cardiac output or blood pressure. Since shock represents a continuum of severity, it is further characterized as being compensated or decompensated. “ Decompensated shock” is defined as a clinical state of tissue perfusion that is inadequate to meet metabolic demand and hypotension (ie, a systolic blood pressure [SBP] less than the 5th percentile for age). The definition of hypotension in preterm neonates depends on the newborn’s weight and gestational age.

For the PALS guidelines, hypotension is characterized by the following:

- For term neonates (0 to 28 days of age), SBP < 60 mm Hg
- For infants from 1 month to 12 months, SBP < 70 mm Hg
- For children > 1 year to 10 years, SBP < 70+(2 x age in years)
- Beyond 10 years, hypotension is defined as an SBP < 90 mm Hg

Note that these blood pressure thresholds will overlap with normal values, including the 5% of normal children who have an SBP lower than the 5th percentile for age.

Early (ie, compensated) shock is shock without hypotension (ie, shock with a “normal” blood pressure). Compensated shock is detected by evaluation of heart rate, presence
and volume (strength) of peripheral pulses, and adequacy of end-organ perfusion. The latter includes assessment of mental status, capillary refill, skin temperature, and when available, monitoring urine output and determining the presence and magnitude of metabolic acidosis on laboratory evaluation.

Cardiac output is the product of heart rate and stroke volume. If stroke volume is compromised for any reason, tachycardia is a common physiological response in an attempt to maintain cardiac output. Therefore, sustained sinus tachycardia (ST) in the absence of known causes such as fever or pain may be an early sign of cardiovascular compromise. Bradycardia, on the other hand, may be a preterminal cardiac rhythm indicative of advanced shock, and it is often associated with hypotension. When cardiac output and systemic perfusion are compromised, the volume (strength or quality) of peripheral pulses is decreased, capillary refill time may be prolonged, and skin temperature is often cool despite a warm ambient temperature. In some children with shock, however, the pulses may be readily palpable and the skin temperature may be warm. The latter clinical picture, for example, is seen in children with early septic shock and represents inappropriate vasodilation of blood vessels in the skin and skeletal muscle.

**Adjuncts for Airway and Ventilation**

**Standard Precautions**

All fluids from patients should be treated as potentially infectious. Personnel should wear gloves and protective shields during procedures that are likely to expose them to droplets of blood, saliva, or other body fluids. Local precaution standards should be developed in the context of individual circumstances and available resources.

**Out-of-Hospital Considerations**

In the out-of-hospital setting, there is often a need to open the airway and to provide oxygen with or without ventilatory support. This requires the availability of a selection of face masks and a pediatric manual resuscitator (ventilation bag). The manual resuscitator may be used safely in infants and newborns by persons properly trained to avoid excess tidal volumes and pressure that can result in gastric inflation or overinflation of the lungs. Ventilation via a properly placed tracheal tube is the most effective and reliable method of assisted ventilation. However, this “gold standard” method requires mastery of the technical skill to successfully and safely place a tube in the trachea, and it may not always be appropriate in the out-of-hospital setting, depending on factors such as the experience and training of the healthcare provider and the transport time interval. In addition to the patient’s condition, a wide variety of EMS system factors must be evaluated to identify the best method of securing the airway in a given setting. These factors include EMS provider training, the requirement for ongoing provider experience, the EMS indications for and techniques of pediatric endotracheal intubation, and the methods used to evaluate tube placement. In retrospective studies, increased accuracy and reduced complication rate are associated with increased training (including supervised time spent in the operating room as well as in the field), the use of minimal requirements ensuring adequate ongoing experience, and use of paralytic agents.

In some EMS systems the success rate for pediatric intubation is relatively low and the complication rate is high. This probably reflects the infrequent use of intubation skills by paramedics in a single-tiered system. In tiered EMS systems, the second tier of prehospital providers may have sufficient training and ongoing experience to perform intubation safely and effectively. Dedicated critical care or interhospital transport personnel (including helicopter transport personnel) also may have a high success rate with endotracheal intubation. Conversely, in the only prospective pediatric randomized, controlled trial comparing bag-mask ventilation with endotracheal intubation in the prehospital setting, bag-mask ventilation was generally as effective as endotracheal intubation; for the subgroup with respiratory failure, bag-mask ventilation was associated with improved survival. It is important to note that the transport times were short for this EMS system, all providers received detailed training in bag-mask ventilation and endotracheal intubation, and individual ALS providers had infrequent opportunities to perform pediatric intubation. In summary, this study suggests that endotracheal intubation may not improve survival over bag-mask ventilation in all EMS systems, and endotracheal intubation appears to result in increased airway complications.

On the basis of this data, anyone providing prehospital BLS care for infants and children should be trained to deliver effective oxygenation and ventilation using the bag-mask technique as the primary method of ventilatory support, particularly if transport time is short (Class IIa; level of evidence [LOE] 1, 2). Intubation of the seriously ill or injured pediatric patient in the out-of-hospital setting is a skill that requires both adequate initial training and ongoing experience plus outcome monitoring. If an EMS system chooses to provide out-of-hospital endotracheal intubation, the system should ensure proper initial training, monitoring of skill retention, and ongoing monitoring of the safety and effectiveness of this intervention.

When used by properly trained providers, medications can increase the success rate of endotracheal intubation but may introduce additional risks. Because the risk from a misplaced tube is unacceptably high and clinical signs confirming tube placement in the trachea are not completely reliable, use of a device to confirm tracheal tube placement in the field, in the transport vehicle, and on arrival to the hospital is desirable and strongly encouraged. Use of a device to confirm tube placement on arrival at the hospital is especially important because displacement of the tube is most likely to occur when the patient is moved into and out of the transport vehicle, and animal data shows that detection of a displaced or obstructed tube using pulse oximetry or changes in heart rate or blood pressure may be delayed more than 3 minutes. Secondary confirmation of tracheal tube position by use of exhaled CO₂ detection is strongly recommended in infants and children with a perfusing rhythm (Class IIa; LOE 3, 5, 7) and is recommended in patients in cardiac arrest (Class IIb; LOE 5, 7). Unfortunately these devices have been inadequately studied in children for use outside of the
operating room (see “Noninvasive Respiratory Monitoring” later in this part), so additional data is needed before the use of these devices is made a Class I recommendation.

**Oxygen Administration**

Administer oxygen to all seriously ill or injured patients with respiratory insufficiency, shock, or trauma. In these patients, inadequate pulmonary gas exchange and inadequate cardiac output resulting from conditions such as a low circulatory blood volume or disturbed cardiac function limit tissue oxygen delivery.

During cardiac arrest a number of factors contribute to severe progressive tissue hypoxia and the need for supplemental oxygen administration. At best, mouth-to-mouth ventilation provides 16% to 17% oxygen with a maximal alveolar oxygen tension of 80 mm Hg. Even optimal external chest compressions provide only a fraction of the normal cardiac output, so that blood flow and therefore delivery of oxygen to tissues are markedly diminished. In addition, CPR is associated with right-to-left pulmonary shunting caused by ventilation-perfusion mismatch, and respiratory conditions may further compromise oxygenation of the blood. The combination of low blood flow and usually low oxygenation leads to metabolic acidosis and organ failure. Oxygen should be administered to children demonstrating cardiopulmonary arrest or compromise to maximize arterial oxygen content even if measured arterial oxygen tension is high, because oxygen delivery to tissues may still be compromised by a low cardiac output. Whenever possible, humidify administered oxygen to prevent drying and thickening of pulmonary secretions; dried secretions may contribute to obstruction of natural or artificial airways.

Administer oxygen by nasal cannula, simple face masks, and nonrebreathing masks. The concentration of oxygen delivered depends on the oxygen flow rate and the patient’s minute ventilation. As long as the flow of oxygen exceeds the maximal inspiratory flow rate, the prescribed concentration of oxygen will be delivered. If the inspiratory flow rate exceeds the oxygen flow rate, air is entrained, reducing the oxygen concentration delivered.

**Masks**

If the patient demonstrates effective spontaneous ventilation, use a simple face mask to provide oxygen at a concentration of 30% to 50%. If a higher concentration of oxygen is desired, it may be administered through a nonrebreathing mask, typically at a flow of 15 L/min. Masks should be available in a selection of sizes. To provide a consistent concentration of oxygen, the mask of appropriate size should provide an airtight seal without pressure on the eyes. A small undermask volume is desirable to minimize rebreathing of exhaled gases. If the mask has an inflatable rim, the rim can mold to the contours of the child’s face to minimize air leak.

**Nasal Cannulas**

A nasal cannula is used to provide supplemental oxygen to a child who is breathing spontaneously. This low-flow device delivers varying inspired oxygen concentrations, depending on the child’s respiratory rate and effort and the size of the child. In young infants, nasal oxygen at 2 L/min can provide an inspired oxygen concentration >50%. Nasal cannulas are often better tolerated than a face mask and are suitable to use in children who require modest oxygen supplementation. Nasal cannula flow rates >4 L/min for prolonged periods are often poorly tolerated because of the drying effect on the nasal mucosa.

**Oropharyngeal and Nasopharyngeal Airways**

An oropharyngeal airway is indicated for the unconscious infant or child if procedures to open the airway (eg, head tilt–chin lift or jaw thrust) fail to provide a clear, unobstructed airway. Do not use an oropharyngeal airway in the conscious child because it may induce vomiting.

Oropharyngeal airways are available for pediatric patients of all ages. Appropriate selection of airway size requires training and experience. An improperly sized oropharyngeal airway may fail to keep the tongue separated from the back of the pharynx or may actually cause airway obstruction. To select the proper size (length) of oropharyngeal airway from flange to distal tip, choose one equal to the distance from the central incisors to the angle of the jaw. To evaluate the size, place the airway next to the face.

Nasopharyngeal airways are soft rubber or plastic tubes that may be used in conscious patients requiring relief of upper airway obstruction. They may be useful in children with a diminished level of consciousness or in neurologically impaired children who have poor pharyngeal tone leading to upper airway obstruction. They are available in a selection of pediatric sizes. In very young patients, airway secretions and debris readily obstruct small nasopharyngeal airways, making them unreliable. Moreover, children may have large adenoids, which can lead to difficulty in placing the airway; trauma and bleeding may occur during placement. Large adenoids also may compress the nasopharyngeal airway after placement, leading to increased airway resistance and an ineffective airway.

**Laryngeal Mask Airway**

The laryngeal mask airway (LMA) is a device used to secure the airway in an unconscious patient. The LMA consists of a tube with a cuffed mask-like projection at the distal end. The LMA is introduced into the pharynx and advanced until resistance is felt as the tube locates in the hypopharynx. The balloon cuff is then inflated, which seals the hypopharynx, leaving the distal opening of the tube just above the glottic opening and providing a clear, secure airway. (See Figure 3 in “Part 6, Section 3: Adjuncts for Oxygenation, Ventilation, and Airway Control.”)

LMAs are widely used in the operating room and provide an effective means of ventilation and oxygenation, but LMAs are contraindicated in an infant or child with an intact gag reflex. They may be useful in patients with difficult airways, and they have been used successfully in emergency airway control of adults in hospital and out-of-hospital settings. They can be placed safely and reliably in infants and children, although data suggests that proper training and supervision are needed to master the technique. Data also suggests that mastering LMA insertion may be easier than mastering endotracheal intubation. Indeed, nurses have been successfully trained to perform LMA insertion in adults.
in cardiac arrest, and paramedics have been trained to insert an LMA with a higher success rate than endotracheal intubation.

Although LMAs do not protect the airway from aspiration of refluxed gastric contents, a meta-analysis showed that aspiration is uncommon with LMA use in the operating room and was less common than with bag-mask ventilation in adults undergoing in-hospital CPR. Therefore, in the setting of cardiac or respiratory arrest, LMAs may be an effective alternative for establishing the airway when inserted by properly trained healthcare providers, but limited data comparing LMAs to bag-mask ventilation or endotracheal intubation in emergency pediatric resuscitation precludes a confident recommendation (Class Indeterminate; LOE 5, 7). Training for healthcare providers in the use of the LMA should not replace training to use bag-mask ventilation effectively.

An LMA may be more difficult to maintain during patient movement than a tracheal tube, making it problematic to use during transport. Careful attention is needed to ensure that the LMA position is maintained if the LMA is used in the out-of-hospital setting. Furthermore, the LMA is relatively expensive, and a number of sizes are needed to provide airway support to any child at risk. The cost of equipping out-of-hospital providers with LMA devices must be considered.

**Ventilation Bags and Masks**

Ventilation with a bag-mask device requires more skill than mouth-to-mouth or mouth-to-mask ventilation. A bag-mask device should be used only by personnel with proper training. Training should focus on selecting an appropriately sized mask and bag, opening the airway and securing the mask to the face, delivering adequate ventilation, and assessing the effectiveness of ventilation. We recommend periodic demonstration of proficiency.

**Types of Ventilation Bags (Manual Resuscitators)**

There are 2 basic types of manual resuscitators: self-inflating and flow-inflating. Ventilation bags used for resuscitation should be self-inflating and should be available in child and adult sizes, suitable for the entire pediatric age range.

Neonatal-size (250 mL) ventilation bags may be inadequate to support effective tidal volume and the longer inspiratory times required by full-term neonates and infants. For this reason resuscitation bags used for ventilation of full-term newborns, infants, and children should have a minimum volume of 450 to 500 mL. Studies using infant manikins showed that effective infant ventilation can be accomplished using pediatric (and larger) resuscitation bags. Regardless of the size of the manual resuscitator, take care to use only that force and tidal volume necessary to cause the chest to visibly rise. Excessive ventilation volumes and airway pressures may compromise cardiac output by raising the intrathoracic pressure and by distending alveoli, increasing afterload on the right heart. In addition, excessive volumes may distend the stomach, impeding ventilation and increasing the risk of regurgitation and aspiration. In patients with small-airway obstruction (eg, asthma and bronchiolitis), excessive tidal volumes and rate can result in air trapping, barotrauma, air leak, and severe compromise to cardiac output. In head-injured and postarrest patients, excessive ventilation volumes and rate may result in hyperventilation, with potentially adverse effects on neurological outcome. Therefore, the routine target in postarrest and head-injured patients should be physiological oxygenation and ventilation (Class IIa; LOE 5, 6; see “Postresuscitation Stabilization”). Ideally, manual resuscitators used for resuscitation should have either no pressure-relief valve or a pressure-relief valve with an override feature to permit use of high pressures to achieve visible chest expansion if necessary. High pressures may be required during bag-mask ventilation of patients with upper or lower airway obstruction or poor lung compliance. In these patients a pressure-relief valve may prevent delivery of sufficient tidal volume.

Self-Inflating Bags

The self-inflating bag delivers only room air (21% oxygen) unless supplemental oxygen is provided. At an oxygen inflow of 10 L/min, pediatric manual resuscitator devices without oxygen reservoirs deliver from 30% to 80% oxygen to the patient. The actual concentration of oxygen delivered is unpredictable because entrainment of variable quantities of room air occurs, depending on the tidal volume and peak inspiratory flow rate used. To deliver consistently higher oxygen concentrations (60% to 95%), all manual resuscitators used for resuscitation should be equipped with an oxygen reservoir. An oxygen flow of at least 10 to 15 L/min is necessary to maintain an adequate oxygen volume in the reservoir of a pediatric manual resuscitator; this should be considered the minimum flow rate. The larger adult manual resuscitators require at least 15 L/min of oxygen to deliver high oxygen concentrations reliably.

To provide bag-mask ventilation, open the airway, seal the mask to the face, and deliver an adequate tidal volume. To open the airway and seal the mask to the face in the absence of suspected neck trauma, tilt the head back while 2 or 3 fingers are positioned under the angle of the mandible to lift it up and forward, moving the tongue off the posterior pharynx. Place the thumb and forefinger in a “C” shape over the mask and exert downward pressure on the mask while the other fingers maintain the jaw thrust to create a tight seal (Figure 2). This technique of opening the airway and sealing the mask to the face is called the “E-C clamp” technique. The third, fourth, and fifth fingers (forming an E) are positioned under the jaw to lift it forward; then the thumb and index finger (forming a C) hold the mask on the child’s face. Determine appropriate mask size by the ability to seal it around the mouth and nose without covering the eyes or overlapping the chin. Once the mask is properly sealed, the other hand compresses the ventilation bag until the chest visibly rises.

Self-inflating bag-mask systems that contain a fish-mouth or leaf-flap outlet valve cannot be used to provide continuous supplemental oxygen to the child with spontaneous respirations. The valve in the self-inflating bag opens only if the bag is squeezed or the child’s inspiratory effort is significant. If the bag is not squeezed, the valve usually remains closed, so
the child receives only a negligible amount of escaped oxygen and rebreathes the exhaled gases contained within the mask itself.

**Flow-Inflating Bags**

Flow-inflating bags (also called “anesthesia bags”) refill only with oxygen inflow, and the inflow must be individually regulated. Since flow-inflating manual resuscitators are more difficult to use, they should be used by trained personnel only. Flow-inflating bags permit the delivery of supplemental oxygen to a spontaneously breathing victim.

**Two-Person Bag-Mask Ventilation**

Superior bag-mask ventilation can be achieved with 2 persons, and this technique may be necessary when there is significant airway obstruction or poor lung compliance. One rescuer uses both hands to open the airway and maintain a tight mask-to-face seal while the other rescuer compresses the ventilation bag (Figure 3). Both rescuers should observe the chest to ensure chest rise with each breath.

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**Gastric Inflation and Cricoid Pressure**

Gastric inflation in unconscious or obtunded patients can be minimized by increasing inspiratory time to deliver the necessary tidal volume at low peak inspiratory pressures. The rescuer must properly pace the rate of ventilation and ensure adequate time for exhalation. To reduce gastric inflation, a second rescuer can apply cricoid pressure, but use this procedure only with an unconscious victim. Cricoid pressure may also prevent regurgitation (and possible aspiration) of gastric contents. Avoid excessive cricoid pressure because it may produce tracheal compression and obstruction or distortion of the upper airway anatomy. Gastric inflation after prolonged bag-mask ventilation can limit effective ventilation; inflation can be relieved by placement of a nasogastric or orogastric tube. If endotracheal intubation is performed, insertion of the gastric tube should follow the insertion of the tracheal tube.

**Endotracheal Intubation**

When used by properly trained providers, ventilation via a tracheal tube is the most effective and reliable method of assisted ventilation. Advantages of endotracheal intubation include the following:

- The airway is isolated to ensure adequate ventilation and delivery of oxygen without inflating the stomach.
- The risk of pulmonary aspiration of gastric contents is minimized.
- Inspiratory time and peak inspiratory pressures can be controlled.
- Secretions and other debris can be suctioned from the airways.
- Positive end-expiratory pressure can be delivered, if needed, through use of a positive end-expiratory pressure device on the exhalation port.
**TABLE 1. Pediatric Tracheal Tube and Suction Catheter Sizes***

<table>
<thead>
<tr>
<th>Approximate Age/Size (Weight)</th>
<th>Internal Diameter of Tracheal Tube, mm</th>
<th>Suction Catheter Size, F</th>
</tr>
</thead>
<tbody>
<tr>
<td>Premature infant (&lt; 1 kg)</td>
<td>2.5</td>
<td>5</td>
</tr>
<tr>
<td>Premature infant (1–2 kg)</td>
<td>3.0</td>
<td>5 or 6</td>
</tr>
<tr>
<td>Premature infant (2–3 kg)</td>
<td>3.0 to 3.5</td>
<td>6 or 8</td>
</tr>
<tr>
<td>0 month to 1 year/infant (3–10 kg)</td>
<td>3.5 to 4.0</td>
<td>8</td>
</tr>
<tr>
<td>1 year/small child (10–13 kg)</td>
<td>4.0</td>
<td>8</td>
</tr>
<tr>
<td>3 years/child (14–16 kg)</td>
<td>4.5</td>
<td>8 or 10</td>
</tr>
<tr>
<td>5 years/child (16–20 kg)</td>
<td>5.0</td>
<td>10</td>
</tr>
<tr>
<td>6 years/child (18–25 kg)</td>
<td>5.5</td>
<td>10</td>
</tr>
<tr>
<td>8 years/child to small adult (24–32 kg)</td>
<td>6.0 cuffed</td>
<td>10 or 12</td>
</tr>
<tr>
<td>12 years/adolescent (32–54 kg)</td>
<td>6.5 cuffed</td>
<td>12</td>
</tr>
<tr>
<td>16 years/adult (50+ kg)</td>
<td>7.0 cuffed</td>
<td>12</td>
</tr>
<tr>
<td>Adult female</td>
<td>7.0–8.0 cuffed</td>
<td>12 or 14</td>
</tr>
<tr>
<td>Adult male</td>
<td>7.0–8.0 cuffed</td>
<td>14</td>
</tr>
</tbody>
</table>

*These are approximations and should be adjusted on the basis of clinical experience. Tracheal tube selection for a child should be based on the child’s size or age. One size larger and one size smaller should be allowed for individual variation. Color-coding based on length or size of the child may facilitate approximation of correct tracheal tube size.

Indications for endotracheal intubation include:

- Inadequate central nervous system control of ventilation resulting in apnea or inadequate respiratory effort
- Functional or anatomic airway obstruction
- Excessive work of breathing leading to fatigue
- Need for high peak inspiratory pressures or positive end-expiratory pressures to maintain effective alveolar gas exchange
- Lack of airway protective reflexes
- Permitting paralysis or sedation for diagnostic studies while ensuring protection of the airway and control of ventilation

The airway of the child differs from that of the adult. The child’s airway is more compliant, the tongue is relatively larger, the glottic opening is higher and more anterior in the neck, and the airway is proportionally smaller than in the adult. For these reasons, only highly trained medical providers who maintain their skill through experience or frequent retraining should attempt endotracheal intubation. If the provider lacks adequate training or experience, continued ventilation with a manual resuscitator and mask or an LMA may be appropriate until a more skilled provider is available.

The narrowest diameter of the child’s airway is located below the vocal cords at the level of the cricoid cartilage. Since obstruction to passage of a tracheal tube may occur at a point just below the level of the glottic opening, uncuffed tubes typically are used for children < 8 years old. However, cuffed tracheal tubes sized for younger children are available and may be appropriate under circumstances in which high inspiratory pressure is expected. For example, a child in respiratory failure from status asthmaticus or acute respiratory distress syndrome (ARDS) may benefit from a cuffed tracheal tube to permit use of higher ventilatory pressures.

Data suggests that using cuffed tracheal tubes in critically ill children results in complication rates that are no different from those for uncuffed tubes, provided that there is appropriate attention to monitoring cuff pressure.\(^ {53,54}\)

Suggested tracheal tubes and suction catheters for different ages (based on the average sizes of children at different ages) are listed in Table 1. For children older than 1 year, an estimate of tracheal tube size may also be made by use of the following equation:

\[
\text{Tracheal tube size (mm)} = (\text{age in years}/4) + 4.
\]

If a cuffed tracheal tube is needed, a slight modification of this formula works well to predict the tracheal tube size\(^ {54}\):

\[
\text{Tracheal tube size (mm)} = (\text{age in years}/4) + 3.
\]

In general, tubes that are 0.5 mm smaller and 0.5 mm larger than estimated should be available. Because of the normal variation of body and airway size for a given age, appropriate tracheal tube selection is based more reliably on patient size than age.\(^ {55}\) Although the internal diameter of the tracheal tube may appear to be roughly equivalent to the size of the victim’s little finger, estimation of tube size by this method may be difficult and unreliable.\(^ {56,57}\) An alternative method of tube size selection is based on a multicenter study that showed that a child’s body length can predict correct tracheal tube size more accurately than the child’s age.\(^ {55}\) Length-based resuscitation tapes may be helpful in identifying the correct tracheal tube size for children up to approximately 35 kg.\(^ {55}\)

Before attempting intubation, assemble the following equipment:

- A tonsil-tipped suction device or a large-bore suction catheter
- A suction catheter of appropriate size to fit into the tracheal tube
- A properly functioning manual resuscitator, oxygen source, and a face mask of appropriate size
- A stylet to provide rigidity to the tracheal tube and help guide it through and beyond the vocal cords. If a stylet is used, it is important to place the stylet tip 1 to 2 cm proximal to the distal end of the tracheal tube to prevent trauma to the trachea from the stylet. A sterile, water-soluble lubricant or sterile water may be helpful to moisten the stylet and aid its removal from the tracheal tube after successful placement.
- Three tracheal tubes, 1 tube of the estimated required size and tubes 0.5 mm smaller and 0.5 mm larger
- A laryngoscope blade and handle with a functioning bright light (and spare bulb and batteries if possible)
- An exhaled CO₂ detector (capnography or colorimetric) or, in older children and adolescents, an esophageal tube detector
- Tape to secure the tube and gauze to dry the face. An adhesive solution may also be used on the tube and face, or a tracheal tube holder may be considered. Assemble equipment to immobilize the child’s head and shoulders, if appropriate.
The Intubation Procedure

In a child with a perfusing rhythm, endotracheal intubation should always be preceded by the administration of supplemental oxygen. Assist ventilation only if the patient’s effort is inadequate. If a rapid sequence intubation (RSI) procedure is anticipated (see below), avoid assisted ventilation if possible because it often inflates the stomach and increases the risk of vomiting and aspiration. If trauma to the head and neck or multiple trauma is present, the cervical spine should be immobilized during intubation.

Since morbidity can occur from an improperly placed tracheal tube or from hypoxia created during prolonged intubation attempts, attempts should not exceed approximately 30 seconds, and the heart rate and pulse oximetry should be continuously monitored. Interrupt the intubation attempt for any of the following conditions: if bradycardia develops (ie, the heart rate drops precipitously or is <60 beats per minute [bpm]), the child’s color or perfusion deteriorates, or the oxygen saturation by pulse oximetry falls to an unacceptable level. If any of these conditions develops, the intubation attempt generally should be interrupted and assisted ventilation provided with a ventilation bag-mask device and supplemental oxygen until the child’s condition improves.

In some circumstances, such as in a child with ARDS, adequate oxygenation cannot be achieved with bag-mask ventilation. In this setting endotracheal intubation should be strongly considered despite the presence of cyanosis or bradycardia. Intubation is probably best performed by the most skilled provider present. In a child in cardiac arrest, do not delay intubation to apply a device to continuously monitor the rhythm. Furthermore, pulse oximetry will not function if the patient does not have detectable pulsatile perfusion.

Either a straight or a curved laryngoscope blade may be used. When a straight blade is used, the blade tip is usually passed over the epiglottis to rest above the glottic opening. Use the blade traction to lift the base of the tongue and directly elevate the epiglottis anteriorly, exposing the glottis (Figure 4). When using a curved blade, insert the tip of the blade into the vallecula (the space between the base of the tongue and the epiglottis) to displace the base of the tongue anteriorly. Do not use the laryngoscope blade and handle in a prying or levering motion, and do not place pressure directly on the teeth, lips, or gums (Figure 5).

Endotracheal intubation ideally should proceed when the glottic opening is visualized. Glottic visualization in infants and children requires that the head and neck be tipped (or angled) forward and the chin lifted into the “sniffing” position. Place the child’s head on a small pillow (this flexes the neck slightly) to bring the larynx into optimal alignment for intubation.58 In infants and children <2 years of age, use of a pillow to flex the neck is not necessary for oral intubation, and the head should be on a flat surface; often a small shoulder roll is used to elevate the shoulders.58 As noted previously, if trauma to the head and neck or multiple trauma is present, attempt to immobilize the cervical spine during intubation.

The appropriate depth of insertion of a tracheal tube can be estimated from the following formula: Depth of insertion (cm)=internal tube diameter (in mm)×3. An alternative formula to estimate appropriate depth of insertion in children >2 years of age is this: Depth of insertion (cm)=(age in years/2)+12.

Verification of Proper Tube Placement

Once the tracheal tube is positioned, provide positive-pressure ventilation, observe chest wall movement, and listen for breath sounds over the peripheral lung fields. If the tube
is properly positioned, there should be symmetrical, bilateral chest rise during positive-pressure ventilation, and breath sounds should be easily auscultated over both lung fields, especially in the axillary areas. Breath sounds should be absent over the upper abdomen. 28 The presence of water vapor in the tube is not a reliable indicator of proper tracheal tube position. 59 Tracheal tube placement should be confirmed by monitoring exhaled CO₂, especially in children with a perfusing rhythm (see “Noninvasive Respiratory Monitoring”). If there is any doubt about tracheal position of the tube, use the laryngoscope to verify tube position by seeing the tube pass through the glottic opening. In a patient monitored by continuous pulse oximetry, the oxygen saturation typically increases after successful intubation unless the child has severe alteration of oxygen diffusion across the alveolus or severe ventilation-perfusion mismatch (eg, ARDS or severe pneumonia).

After the tube is taped into place, confirm its position within the trachea clinically and by chest x-ray because transmitted breath sounds may be heard over the left hemithorax despite a right main bronchus intubation. In addition, the chest x-ray helps to identify and correct the position of a tube located high in the trachea, which is at high risk of displacement during movement.

Once the tracheal tube is placed and secured, maintain the head in a neutral position. Excessive movement of the head may displace the tracheal tube. Flexion of the head on the neck moves the tube farther into the airway, and extension of the head displaces the tube farther out of the airway. 50, 61 In a responsive patient, consider placement of an oral airway adjacent to the tracheal tube, but not deeply enough into the oropharynx to stimulate a gag reflex, to prevent the child from biting down on the tube and obstructing the airway.

**Rapid Sequence Intubation**

RSI uses pharmacological agents to facilitate emergent endotracheal intubation while reducing adverse effects in responsive patients, including pain, arrhythmias, rise in systemic and intracranial pressures, airway trauma, gastric regurgitation and aspiration, hypoxemia, psychological trauma, and death. The term rapid sequence intubation is preferred over rapid sequence induction because the latter denotes the technique used by anesthesiologists for rapid airway control coincident with the intubation of anesthesia. In emergency settings, RSI should be seen not “as the initiation of anesthe-sia but rather as the use of deep sedation and paralysis to facilitate endotracheal intubation.” 62

In the United States, RSI is used frequently in Emergency Departments and intensive care units and to a lesser extent in the out-of-hospital setting. In many other countries, RSI is limited to trained anesthesiologists to minimize risks from the use of potent drugs to facilitate intubation. Regardless of where RSI is performed, only properly trained persons familiar with its indications and contraindications should use RSI. These persons must be proficient in the evaluation and management of the pediatric airway and must understand the medications (sedatives, neuromuscular blocking agents, and adjunctive agents) used during this procedure. The indications for RSI are the same as outlined above for endotracheal intubation. RSI is not indicated for patients in cardiac arrest or for those who are deeply comatose and require immediate intubation without delay. Relative contraindications to RSI include provider concern that intubation or mask ventilation may be unsuccessful; significant facial or laryngeal edema, trauma, or distortion; or a spontaneously breathing, adequately ventilated patient whose airway maintenance depends on his own upper airway muscle tone and positioning (eg, upper-airway obstruction or epiglottitis). 62

An evidence-based analysis of RSI agents and procedures was not conducted at the evidence evaluation conferences leading to these guidelines. In addition, different pharmacological agents are used by protocol in different hospital and out-of-hospital settings. For these reasons, we cannot recommend uniform guidelines for RSI at this time. The inclusion of this information as an optional module in the PALS course is not an endorsement of RSI. To provide objective information on the value of RSI in various settings for future guidelines, healthcare systems using RSI should monitor the success rate and occurrence of complications.

**Noninvasive Respiratory Monitoring**

**Pulse Oximetry**

Pulse oximetry is an important noninvasive monitor of the child with respiratory insufficiency because it enables continuous evaluation of the arterial oxygen saturation. This monitoring technique is useful in both out-of-hospital and in-hospital settings. 63, 64 It may provide early indication of respiratory deterioration causing hypoxemia (eg, from the loss of an artificial airway, disconnection of the oxygen supply, or impending or actual respiratory failure) and ideally should be used during stabilization and transport, because clinical recognition of hypoxemia is not reliable. 65 If peripheral perfusion is inadequate (eg, shock is present or the child is in cardiac arrest), pulse oximetry is unreliable and often unobtainable because accurate readings require the presence of pulsatile blood flow. In addition, if a patient is hyperoxegenated before intubation, incorrect tube position may not be recognized by pulse oximetry for a variable period depending on the rate of oxygen consumption. 50, 66

**Exhaled or End-Tidal CO₂ Monitoring**

Because clinical confirmation of tracheal tube placement may be unreliable, exhaled CO₂ detection using a colorimetric device or continuous capnography is recommended to confirm tube placement in infants (≥2 kg) and in children (Class IIa; LOE 5, 6, 7). A positive color change or the presence of a capnography waveform showing exhaled CO₂ confirms tube position in the trachea when assessed after 6 ventilations. 67, 68 Six ventilations are recommended to wash out CO₂ that may be present in the stomach and esophagus after bag-mask ventilation. After 6 ventilations, detected CO₂ can be presumed to be from the trachea rather than from a misplaced tube in the esophagus. Note that exhaled CO₂ may be detected with right main bronchus intubation, so exhaled CO₂ detection does not replace the need to document proper tube position in the trachea by chest x-ray and clinical examination.
Although detection of exhaled CO\textsubscript{2} in patients with a perfusing rhythm is both specific and sensitive for tube placement in the trachea, exhaled CO\textsubscript{2} detection is not as useful for patients in cardiac arrest. The presence of a color change or an exhaled CO\textsubscript{2} waveform reliably confirms tracheal tube placement, but the absence of detectable CO\textsubscript{2} does not confirm esophageal tube placement in the cardiac arrest patient. Infants, children, and adolescents in cardiac arrest may have limited pulmonary blood flow and therefore undetectable exhaled CO\textsubscript{2} despite proper placement of the tube in the trachea.\textsuperscript{67,69} The low specificity of exhaled CO\textsubscript{2} monitoring in cardiac arrest limits the strength of recommendation of this test following intubation of a patient in cardiac arrest (Class IIb; LOE 3, 5, 6, 7).\textsuperscript{69,70} In cardiac arrest the absence of a color change or detectable exhaled CO\textsubscript{2} by capnography may indicate either esophageal or tracheal tube placement.\textsuperscript{69–71} If placement is uncertain, tube position must be confirmed by clinical examination and direct laryngeal examination.

In addition to cardiac arrest, other conditions leading to very low exhaled CO\textsubscript{2} may also produce misleading results. Clinical experience in adults, for example, suggests that severe airway obstruction (eg, status asthmaticus) and pulmonary edema may impair CO\textsubscript{2} elimination sufficiently to produce a false-negative test result.\textsuperscript{70,72} If the detector is contaminated with acidic gastric contents or acidic drugs, such as tracheally administered epinephrine, the colorimetric detector may not be reliable. These problems cause a color change consistent with exhaled CO\textsubscript{2}, but the detector remains a constant color throughout the respiratory cycle. Finally, intravenous bolus epinephrine administration may transiently reduce pulmonary blood flow and thus reduce the exhaled CO\textsubscript{2} below the limits of detection in cardiac arrest patients.\textsuperscript{73}

Even though correct tracheal tube placement may not be confirmed by exhaled CO\textsubscript{2} detection in cardiac arrest, the absence of exhaled CO\textsubscript{2} may provide prognostic information in this setting. When correct tracheal tube position is confirmed, experience in animals\textsuperscript{74} and adults\textsuperscript{75–77} shows that absent or low detectable exhaled CO\textsubscript{2} correlates with poor outcome. In addition, efforts that improve closed-chest compression produce increases in exhaled CO\textsubscript{2}\textsuperscript{78,79} This is consistent with data correlating cardiac output to exhaled CO\textsubscript{2} concentration.\textsuperscript{80,81} There is only limited data relating exhaled CO\textsubscript{2} to outcome in pediatric cardiac arrest,\textsuperscript{69} and animal data emphasizes the need to evaluate the exhaled CO\textsubscript{2} after providing several minutes of adequate ventilation in asphyxial arrests, since the initial values will be elevated.\textsuperscript{82,83} On the basis of the limited data, no definite recommendation can be made about the use of exhaled CO\textsubscript{2} to predict outcome in children with cardiac arrest (Class Indeterminate; LOE 5, 6, 7), but we encourage the collection of outcome data correlated with exhaled CO\textsubscript{2} measurement.

**Esophageal Detector Devices**

Esophageal detector devices are based on the ability to readily aspirate air from the cartilage-supported trachea by drawing from gas in the lower airways. If the tracheal tube is placed in the esophagus, the walls of the esophagus collapse when aspiration is attempted by an esophageal detector device, preventing filling of a syringe or self-inflating rubber bulb.\textsuperscript{71} In adults the esophageal device is very sensitive in identifying an esophageal tube placement when used in emergency intubations in patients with a perfusing rhythm.\textsuperscript{84,85} In adults in cardiac arrest the esophageal detector device is useful to identify esophageal intubation, and it therefore can be used to supplement the potentially misleading information from exhaled CO\textsubscript{2} detection to confirm tracheal placement.\textsuperscript{86} Although an esophageal detector device has been used successfully in children,\textsuperscript{87} it appears to be unreliable in children <1 year of age,\textsuperscript{88} in morbidly obese patients,\textsuperscript{89} and in patients in late pregnancy.\textsuperscript{90} In summary, there is insufficient data in emergency intubations in infants and children to recommend the routine use of an esophageal detector device (Class Indeterminate; LOE 5, 6, 7).

**Verification of Tracheal Tube Position**

Several points about the use of supplemental respiratory monitoring devices after intubation deserve emphasis:\textsuperscript{91}

- No single confirmation technique is 100% reliable under all circumstances.
- Devices to confirm tracheal tube placement should always be used in the perfusing patient and are highly recommended in the cardiac arrest patient to supplement the physical examination because physical examination alone is unreliable.
- If the infant or child has a perfusing rhythm, exhaled CO\textsubscript{2} detection is the best method (most sensitive and specific) for verification of tube placement.
- Once placement is confirmed, the tube (and head, if appropriate) should be secured and the tube position at the level of the lip or teeth recorded.
- Repeated confirmation or continuous monitoring of tracheal tube position is highly recommended during stabilization and transport in the out-of-hospital or in-hospital setting.

If the condition of an intubated patient deteriorates, consider several possibilities that can be recalled by the mnemonic **DOPE**: Displacement of the tube from the trachea, Obstruction of the tube, Pneumothorax, and Equipment failure.

**Miscellaneous Adjuncts to Airway and Ventilation**

Suction devices (either portable or installed) should be available for emergency resuscitation. The portable unit should provide sufficient vacuum and flow for pharyngeal and tracheal suctioning. The installed unit should provide an airflow of >30 L/min at the end of the delivery tube and a vacuum of >300 mm Hg when the tube is clamped at full suction. Each device should have an adjustable suction regulator for use in children and intubated patients. Generally a maximum suction force of 80 to 120 mm Hg is used for suctioning the airway of the infant or child.\textsuperscript{92} Large-bore, non collapsible suction tubing should always be joined to the suction units, and semirigid pharyngeal tips (tonsil suction tips) and appropriate sizes of catheters should be available.

Pharyngeal and sterile tracheal suction catheters should be available in a variety of sizes (Table 1) and should be readily available. Part 10: Pediatric Advanced Life Support I-303

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**Table 1**

<table>
<thead>
<tr>
<th>Size (mm)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>Pediatric intubation catheter</td>
</tr>
<tr>
<td>4</td>
<td>Pediatric suction catheter</td>
</tr>
<tr>
<td>5</td>
<td>Pediatric suction catheter</td>
</tr>
<tr>
<td>6</td>
<td>Pediatric suction catheter</td>
</tr>
<tr>
<td>7</td>
<td>Pediatric suction catheter</td>
</tr>
</tbody>
</table>

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accessible. Tracheal suction catheters should have a Y-piece, T-piece, or lateral opening between the suction tube and the suction power control to regulate when suction is applied. The suction apparatus must be designed for easy cleaning and decontamination.

When it is impossible to oxygenate or ventilate the victim with a manual resuscitator or when intubation cannot be accomplished (eg, following severe facial trauma) and standard resuscitative measures to clear the airway fail, tracheal catheter ventilation may be attempted.93 Percutaneous needle cricothyrotomy provides effective ventilation and oxygenation in children during anesthesia if a jet ventilator is used.94,95 although there is a risk of barotrauma.94 There are only anecdotal reports of emergency oxygenation and ventilation using a transtracheal catheter in children, so further evaluation is required. Performance of needle cricothyrotomy requires specialized training. A large-bore (eg, 14-gauge) over-the-needle catheter is used to puncture the cricothyroid membrane percutaneously. The needle is then removed, and the catheter is joined with a standard (3-mm) tracheal tube adapter to an oxygen source and hand resuscitator bag or a high-pressure oxygen source.96 This technique allows effective support of oxygenation, although CO₂ elimination may be suboptimal. Alternatively, emergency cricothyroidotomy may be performed using a modified Seldinger technique, whereby a small-bore needle is used to puncture the cricothyroid membrane.97 A flexible wire is then inserted, followed by a dilator and finally a tracheostomy-like tube, permitting adequate oxygenation and ventilation. In an infant the small size of the cricothyroid membrane limits the feasibility of both techniques.

Circulatory Adjuncts

Bedboard
CPR should be performed where the victim is found. If cardiac arrest occurs in a hospital bed, place a firm support beneath the patient’s back. A bedboard that extends from the shoulders to the waist and across the full width of the bed provides optimal support. The width of the board is especially important in larger children to avoid losing the force of compression by the mattress sinking down when the chest is compressed. Spine boards, preferably with head wells, should be used in ambulances and mobile life support units.98,99 They provide a firm surface for CPR in the emergency vehicle or on a wheeled stretcher and also may be useful for extricating and immobilizing victims. In infants a firm surface should also be used under the back. The 2 thumb–encircling hands technique provides support by positioning the fingers behind the infant’s back (see “Part 9: Pediatric Basic Life Support”).

Mechanical Devices for Chest Compression
Mechanical devices to compress the sternum are not recommended for pediatric patients because they were designed and tested for use in adults, and data on pediatric safety and effectiveness is absent. Active compression-decompression CPR increases cardiac output compared with standard CPR in various animal models,100,101 maintains coronary perfusion during compression and decompression CPR in humans,102 and provides ventilation if the airway is open.102 Clinical trials report variable results with some benefit on short-term outcome measures (eg, return of spontaneous circulation and survival for 24 hours)103–105 but no long-term survival benefits in most trials. On the basis of these variable clinical results, active compression-decompression CPR is considered an optional technique in adults (Class IIb; LOE 2, 5, 7). No recommendation can be made for children given the absence of clinical data (Class Indeterminate; LOE 7).

Interposed Abdominal Compression CPR
The technique of interposed abdominal compression CPR (IAC-CPR) does not use an adjunct piece of equipment but does require a third rescuer. This form of chest compression has been shown to increase blood flow in laboratory and computer models of adult CPR and in some in-hospital clinical settings. IAC-CPR has been recommended as an alternative technique (Class IIb) for in-hospital CPR in adult victims, but it cannot be recommended for use in children at this time.106

Medical Antishock Trousers
The effects of medical antishock trousers (MAST) during resuscitation of pediatric cardiac arrest are unknown, and the use of MAST cannot be recommended (Class III). The efficacy of MAST in the treatment of pediatric circulatory failure is controversial. Although MAST therapy was thought to be helpful in the treatment of hemorrhagic shock, randomized trials show either no benefit of MAST107 or an increased mortality with their use.108 One case series suggests that MAST may be useful in children with pelvic hemorrhage.109 Potential complications of MAST include lower-extremity compartment syndrome and ischemia110 and compromised ventilation.111 If MAST are used, healthcare providers must be familiar with the proper indications, hazards, and complications of this therapy.

Open-Chest Cardiac Compression
Internal (open-chest) cardiac compression generates better cardiac output and cerebral and myocardial blood flow in animals112 and adults113 than closed-chest compressions, but comparable improvement in cardiac output may not be observed in infants and children because the chest wall is extremely compliant in this age group.114,115 The use of open thoracotomy and direct cardiac compression does not appear to be beneficial in the treatment of blunt traumatic pediatric arrest and may increase the cost for short-term survivors,116 although it is usually attempted relatively late in the course. Limited data suggests that early open-chest CPR may be useful in adults with nontraumatic arrest,117 but this technique has not been evaluated in nontraumatic pediatric arrest. In the absence of adequate clinical data showing a beneficial effect, internal cardiac compression for children in cardiac arrest cannot be routinely recommended at this time (Class Indeterminate).

Extracorporeal Membrane Oxygenation
There is limited clinical experience with the use of extracorporeal membrane oxygenation (ECMO) to support the circulation after cardiac arrest. Most of the reported experience is...
in children after cardiac surgery or in the cardiac catheterization laboratory.118–120 Even with standard CPR for >50 minutes, long-term survival is possible with the use of ECMO in selected pediatric cardiac surgical patients,118–120 although application of this technique requires specialized expensive equipment and a readily available experienced team. Emergency cardiopulmonary bypass also has been used, but it is difficult to achieve rapidly and may be associated with significant complications.121 Nevertheless, occasional patients have attained neurologically intact survival despite intervals from arrest to cardiopulmonary bypass longer than 30 minutes.122 Late application of cardiopulmonary bypass, however, was uniformly unsuccessful for 10 adults in an Emergency Department after prolonged arrest before bypass.123 ECMO and emergency cardiopulmonary bypass should be considered optional techniques for selected patients when used by properly trained personnel in experienced specialty centers (Class IIb; LOE 5).

Establishing and Maintaining Vascular Access

**Selection of Site and Priorities of Vascular Access**

Vascular access is vital for drug and fluid administration but may be difficult to achieve in the pediatric patient.124 During CPR the preferred access site is the largest, most accessible vein that does not require interruption of resuscitation.

Although central venous drug administration results in more rapid onset of action and higher peak drug levels than peripheral venous administration in adult resuscitation models,125 these differences were not shown in a pediatric resuscitation model126 and may not be important during pediatric CPR. Central venous lines provide more secure access to the circulation and permit administration of agents that might cause tissue injury if they infiltrate peripheral sites, such as vasopressors, hypertonic sodium bicarbonate, and calcium. For this reason, if a central venous catheter is in place at the time of arrest, it should be used (Class IIa; LOE 6, 7). Experienced providers may attempt central venous access, using the femoral, internal jugular, external jugular, or (in older children) subclavian vein. The femoral vein is probably the safest and easiest to cannulate. For rapid fluid resuscitation, a single-lumen, wide-bore, relatively short catheter is preferred because this results in lower resistance to flow. Catheter lengths of 5 cm in an infant, 8 cm in a young child, and 12 cm in an older child are usually suitable. If central venous pressure monitoring is desired from a femoral catheter, the catheter tip does not need to be inserted to a point above the diaphragm, provided that there is an unobstructed vena cava.127,128

Peripheral venous access provides a satisfactory route for administration of drugs or fluid if it can be achieved rapidly. Peripheral venipuncture can be performed in the veins of the arm, hand, leg, or foot. Drugs administered via peripheral vein during CPR should be followed by a rapid isotonic crystalloid flush (5 to 10 mL) to move the drugs into the central circulation.

The resuscitation team should use a protocol to establish vascular access during CPR. Such a protocol limits the time devoted to attempts at peripheral and central venous cathe-
blood gas analysis through type and crossmatch, even during cardiac arrest. Administration of sodium bicarbonate through an intraosseous cannula, however, eliminates the close correlation of intraosseous blood losses with mixed venous blood gases. Complications were reported in <1% of patients after intraosseous infusion. Complications include tibial fracture, lower-extremity compartment syndrome or severe extravasation of drugs, and osteomyelitis. Some of these complications may be avoided by careful technique. Animal data and one human follow-up study showed that local effects of intraosseous infusion on the bone marrow and bone growth are minimal. Although microscopic pulmonary fat and bone marrow emboli have been reported, they have never been reported clinically and appear to occur just as frequently during cardiac arrest without use of intraosseous drug administration.

Tracheal Drug Administration

Until vascular access is obtained, the tracheal route may be used for administration of lipid-soluble drugs, including lidocaine, epinephrine, atropine, and naloxone (remembered with the mnemonic “LEAN”). Drugs that are not lipid soluble (eg, sodium bicarbonate and calcium) should not be administered by this route because they will injure the airways. Optimal drug dosages for administration by the tracheal route are unknown because drug absorption across the alveolar and bronchiolar epithelium during cardiac arrest may vary widely. Data from animal models including a neonatal piglet model and one adult human study suggests, however, that a standard intravenous dose of epinephrine administered via the tracheal route produces serum concentrations that are only approximately 10% or less than those of an equivalent dose administered by the intravenous route. For this reason the recommended tracheal dose of epinephrine during pediatric resuscitation is approximately 10 times the dose given via an intravenous route (Class I; LOE 5, 6). It is logical to assume that doses of other resuscitation drugs administered tracheally should be increased compared with the intravenous dose.

When drugs are administered by the tracheal route, animal data suggests that dilution of the drug in up to 5 mL of normal saline followed by 5 manual ventilations results in equivalent absorption and pharmacological effect compared with administration through a catheter or feeding tube inserted into the tracheal tube. Therefore, administration of drugs by the tracheal route is preferred, because administration via catheter or feeding tube is often cumbersome and depends on finding the correct-size catheter to place through the tracheal tube.

Fluid and Drug Therapy

Estimating Patient Weight in an Emergency

Pharmacotherapy in children is complicated by the need to adjust dosages to a wide variety of body weights. Unfortunately, during an emergency, particularly in the out-of-hospital and Emergency Department settings, the child’s weight often is unknown. Skilled personnel may not accurately estimate a child’s weight on the basis of appearance. Use of a growth chart to estimate weight from age is also impractical because a growth chart may not be readily available and the child’s age may not be known. Moreover, there is a wide distribution of normal weight for a given age.

Length is easily measured and enables reliable calculation of emergency medication dosages. Tapes to determine weight from length are available with precalculated doses printed at various lengths. These tapes, based on normative data relating body length to weight, have been clinically validated. Such tapes may be extremely helpful during management of pediatric emergencies. For hospitalized children, weight should be recorded and emergency drug doses precalculated, and this information should be easy to locate in case of an emergency.

Intravascular Fluids

Expansion of circulating blood volume is a critical component of PALS in children who have sustained trauma with acute blood loss. It may also be lifesaving in the treatment of nontraumatic shock, such as severe dehydration or septic shock. Early restitution of circulating blood volume is important to prevent progression to refractory shock or cardiac arrest. Volume expansion is best achieved with isotonic crystalloid solutions, such as Ringer’s lactate or normal saline. Meta-analyses of studies comparing crystalloid to colloid administration in various types of shock or hypalbuminemia suggests that albumin administration may be associated with increased mortality, but few children were included in these studies and no firm recommendation can be made against the use of colloid solutions (eg, 5% albumin) in fluid resuscitation of infants and children.

Infusion of hypertonic saline solutions appears to be beneficial in studies of head-injured adult patients and hypovolemic shock, but there is insufficient data in children to recommend the widespread use of these solutions at this time. Consistent with adult trauma life support guidelines, blood replacement is indicated in children with severe acute hemorrhage if the child remains in shock after infusion of 40 to 60 mL/kg of crystalloid.

Dextrose solutions (ie, 5% dextrose in water) should not be used for initial fluid resuscitation of children (Class III; LOE 6) because large volumes of glucose-containing intravenous solutions do not effectively expand the intravascular compartment and may result in hyperglycemia and a secondary osmotic diuresis. Hyperglycemia before cerebral ischemia worsens neurological outcome. Hyperglycemia detected after traumatic or nontraumatic cardiac arrest is also associated with worse neurological outcome. This data suggests that the presence of postarrest or postresuscitation hyperglycemia may reflect multiorgan system injury with impaired use of glucose (ie, postischemic hyperglycemia may be an epiphenomenon and not a cause of the poor neurological outcome).

If hypoglycemia is suspected or confirmed, it is readily treated with intravenous glucose (see “Glucose,” below). During cardiac arrest, intravenous fluids are used to keep an intravenous line patent for drug administration and to flush drugs from the catheter toward the central venous circulation. In general, for children in cardiac arrest or receiving PALS,
Ringer’s lactate or normal saline should be used because some drugs are incompatible in dextrose. Moreover, if the patient requires subsequent fluid resuscitation, use of these isotonic fluids avoids inadvertent bolus administration of dextrose-containing solutions.

**Drugs Used for Cardiac Arrest and Resuscitation**

**Epinephrine**

Epinephrine is an endogenous catecholamine with potent α- and β-adrenergic-stimulating properties. In cardiac arrest, α-adrenergic–mediated vasoconstriction is the most important pharmacological action; vasoconstriction increases aortic diastolic pressure and thus the coronary perfusion pressure, which is a critical determinant of success or failure of resuscitation.172,173 Epinephrine-induced elevation of coronary perfusion pressure during chest compression enhances delivery of oxygen to the heart. Epinephrine also enhances the contractile state of the heart, stimulates spontaneous contractions, and increases the vigor and intensity of VF, increasing the success of defibrillation.174

The most commonly observed rhythms in the pediatric patient with cardiac arrest are asystole and bradyarrhythmia2,175,176; epinephrine may generate a perfusing rhythm in children with these rhythms. In a child with symptomatic bradycardia that is unresponsive to effective assisted ventilation and supplemental oxygenation, epinephrine may be given in a dose of 0.01 mg/kg (0.1 mL/kg of 1:10 000 solution) by the intravenous or intraosseous route or 0.1 mg/kg (0.1 mL/mg of 1:1000 solution) by the tracheal route. Because the action of catecholamines may be depressed by acidosis and hypoxemia,177,178 attention to ventilation, oxygenation, and circulation is essential. Continuous epinephrine infusion (0.1 to 0.2 μg/kg per minute, titrated to effect) may be considered for refractory bradycardia.

High doses of epinephrine (10 to 20 times the routine dose) improve myocardial and cerebral blood flow in animals with cardiac arrest.179,180 High rescue doses of epinephrine (0.2 mg/kg) were associated with improved survival and neurological outcome compared with that in a historic cohort in a single, nonblinded clinical trial of 20 children with witnessed cardiac arrest.181 Enthusiasm was replaced by disappointment, however, after large multi-institutional adult studies,182–186 well-controlled animal outcome studies,187,188 and uncontrolled retrospective pediatric data189,190 failed to show any benefit from high-dose epinephrine. Moreover, high-dose epinephrine can have adverse effects, including increased myocardial oxygen consumption during CPR, a postarrest hyperadrenergic state with tachycardia, hypertension and myocardial dysfunction.187,188,191,192 Finally, since great interpatient variability in catecholamine response is well established in the nonarrest state,193,194 it is possible that a dangerous dose in one patient may be lifesaving in another.

The recommended initial resuscitation dose of epinephrine for cardiac arrest is 0.01 mg/kg (0.1 mL/kg of 1:10 000 solution) given by the intravenous or intraosseous route or 0.1 mg/kg (0.1 mL/kg of 1:1000 solution) by the tracheal route (Table 2 and Figure 2); repeated doses are recommended every 3 to 5 minutes for ongoing arrest. The same dose of epinephrine is recommended for second and subsequent doses for unresponsive asystolic and pulseless arrest, but higher doses of epinephrine (0.1 to 0.2 mg/kg; 0.1 to 0.2 mL/kg of 1:1000 solution) by any intravascular route may be considered (Class IIb; LOE 6). If the initial dose of epinephrine is not effective, administer subsequent doses within 3 to 5 minutes and repeat every 3 to 5 minutes during resuscitation. If high-dose epinephrine is used, note that 2 different dilutions of epinephrine are needed; take care to avoid errors in selecting the correct concentration and dose. If the patient has continuous intra-arterial pressure monitoring during CPR, subsequent epinephrine doses can be titrated to effect. For example, standard epinephrine doses are rational if the aortic diastolic pressure is greater than approximately 20 mm Hg, whereas higher epinephrine doses are rational if the diastolic pressure is lower.

Epinephrine is absorbed when administered by the tracheal route, although absorption and resulting plasma concentrations are unpredictable.185,186 The recommended tracheal dose is 0.1 mg/kg (0.1 mL/kg of a 1:1000 solution) (Class IIb; LOE 6). Once vascular access is obtained, administer epinephrine intravascularly, beginning with a dose of 0.01 mg/kg, if the victim remains in cardiac arrest.

A continuous infusion of epinephrine may be useful once spontaneous circulation is restored. The hemodynamic effects are dose related: low-dose infusions (<0.3 μg/kg per minute) generally produce prominent β-adrenergic action, and higher-dose infusions (>0.3 μg/kg per minute) result in β- and α-adrenergic–mediated vasoconstriction.196 Since there is great interpatient variability in catecholamine pharmacology,194,197 the infused dose should be titrated to the desired effect.

Administer epinephrine through a secure intravascular line, preferably into the central circulation. If the drug infiltrates into tissues, it may cause local ischemia, leading to tissue injury and ulceration. Epinephrine (and other catecholamines) are inactivated in alkaline solutions and should never be mixed with sodium bicarbonate. In patients with a perfusing rhythm, epinephrine causes tachycardia and often a wide pulse pressure and may produce ventricular ectopy. High infusion doses may produce excessive vasoconstriction, compromising extremity, mesenteric, and renal blood flow and resulting in severe hypertension and tachyarrhythmias.197

**Atropine**

Atropine is discussed in “Treatment of Bradyarrhythmias,” below.

**Vasopressin**

Vasopressin is an endogenous hormone that acts at specific receptors to mediate systemic vasoconstriction (V1 receptor) and reabsorption of water in the renal tubule (V2 receptor). Marked secretion of vasopressin occurs in circulatory shock states and causes relatively selective vasoconstriction of blood vessels in the skin, skeletal muscle, intestine, and fat with relatively less vasoconstriction of the coronary, cerebral, and renal vascular beds. This hemodynamic action produces
<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage (Pediatric)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>0.1 mg/kg</td>
<td>Rapid IV/IO bolus</td>
</tr>
<tr>
<td></td>
<td>Repeat dose: 0.2 mg/kg</td>
<td>Rapid flush to central circulation</td>
</tr>
<tr>
<td></td>
<td>Maximum single dose: 12 mg</td>
<td>Monitor ECG during dose.</td>
</tr>
<tr>
<td>Amiodarone for pulseless VF/VT</td>
<td>5 mg/kg IV/IO</td>
<td>Rapid IV bolus</td>
</tr>
<tr>
<td>Amiodarone for perfusing tachycardias</td>
<td>Loading dose: 5 mg/kg IV/IO</td>
<td>IV over 20 to 60 minutes</td>
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<td></td>
<td>Maximum dose: 15 mg/kg per day</td>
<td>Routine use in combination with drugs prolonging QT interval is not recommended. Hypotension is most frequent side effect.</td>
</tr>
<tr>
<td>Atropine sulfate*</td>
<td>0.02 mg/kg</td>
<td>May give IV, IO or ET.</td>
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<td></td>
<td>Minimum dose: 0.1 mg</td>
<td>Tachycardia and pupil dilation may occur but not fixed dilated pupils.</td>
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<tr>
<td></td>
<td>Maximum single dose: 0.5 mg in child, 1.0 mg in adolescent. May repeat once.</td>
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<tr>
<td>Calcium chloride 10%–100 mg/mL (=27.2 mg/mL elemental Ca)</td>
<td>20 mg/kg (0.2 mL/kg) IV/IO</td>
<td>Give slow IV push for hypocalcemia, hypermagnesemia, calcium channel blocker toxicity, preferably via central vein. Monitor heart rate; bradycardia may occur.</td>
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<tr>
<td>Calcium gluconate 10%–100 mg/mL (=9 mg/mL elemental Ca)</td>
<td>60–100 mg/kg (0.6–1.0 mL/kg) IV/IO</td>
<td>Give slow IV push for hypocalcemia, hypermagnesemia, calcium channel blocker toxicity, preferably via central vein.</td>
</tr>
<tr>
<td>Epinephrine for symptomatic bradycardia*</td>
<td>IV/IO: 0.01 mg/kg (1:10 000, 0.1 mL/kg)</td>
<td>Tachyarrhythmias, hypertension may occur.</td>
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<td></td>
<td>ET: 0.1 mg/kg (1:1000, 0.1 mL/kg)</td>
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<td></td>
<td>First dose:</td>
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<tr>
<td></td>
<td>IV/IO: 0.01 mg/kg (1:10 000, 0.1 mL/kg)</td>
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<td></td>
<td>ET: 0.1 mg/kg (1:1000, 0.1 mL/kg)</td>
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<tr>
<td></td>
<td>Subsequent doses: Repeat initial dose or may increase up to 10 times (0.1 mg/kg, 1:1000, 0.1 mL/kg)</td>
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<td></td>
<td>Administer epinephrine every 3 to 5 minutes.</td>
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<td></td>
<td>IV/IO/ET doses as high as 0.2 mg/kg of 1:1000 may be effective.</td>
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<tr>
<td>Glucose (10% or 25% or 50%)</td>
<td>IV/IO: 0.5–1.0 g/kg</td>
<td>For suspected hypoglycemia; avoid hyperglycemia.</td>
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<td></td>
<td>• 1–2 mL/kg 50%</td>
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<td>• 2–4 mL/kg 25%</td>
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<td>• 5–10 mL/kg 10%</td>
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<tr>
<td>Lidocaine*</td>
<td>IV/IO/ET: 1 mg/kg</td>
<td>Rapid bolus</td>
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<tr>
<td>Lidocaine infusion (start after a bolus)</td>
<td>IV/IO: 20–50 μg/kg per minute</td>
<td>1 to 2.5 mL/kg per hour of 120 mg/100 mL solution or use “Rule of 6” (see Table 3)</td>
</tr>
<tr>
<td>Magnesium sulfate (500 mg/mL)</td>
<td>IV/IO: 25–50 mg/kg, Maximum dose: 2 g per dose</td>
<td>Rapid IV infusion for torsades or suspected hypomagnesemia; 10- to 20-minute infusion for asthma that responds poorly to β-adrenergic agonists.</td>
</tr>
<tr>
<td>Naloxone*</td>
<td>≤ 5 years or ≤ 20 kg: 0.1 mg/kg</td>
<td>For total reversal of narcotic effect. Use small repeated doses (0.01 to 0.03 mg/kg) titrated to desired effect.</td>
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<tr>
<td></td>
<td>&gt; 5 years or &gt; 20 kg: 2.0 mg</td>
<td></td>
</tr>
<tr>
<td>Procainamide for perfusing tachycardias (100 mg/mL and 500 mg/mL)</td>
<td>Loading dose: 15 mg/kg IV/IO</td>
<td>Infusion over 30 to 60 minutes; routine use in combination with drugs prolonging QT interval is not recommended.</td>
</tr>
<tr>
<td>Sodium bicarbonate (1 mEq/mL and 0.5 mEq/mL)</td>
<td>IV/IO: 1 mEq/kg per dose</td>
<td>Infuse slowly and only if ventilation is adequate.</td>
</tr>
</tbody>
</table>

IV indicates intravenous; IO, intraosseous; and ET, endotracheal.

*For endotracheal administration use higher doses (2 to 10 times the IV dose); dilute medication with normal saline to a volume of 3 to 5 mL and follow with several positive-pressure ventilations.
favorable increases in blood flow to the heart and brain in experimental models of cardiac arrest\textsuperscript{198,199} and improved long-term survival compared with epinephrine.\textsuperscript{200} Although adverse effects on splanchnic blood flow are a theoretical concern following large doses of vasopressin, modest declines in adrenal and renal blood flow are seen in experimental animals with no effect on intestinal or hepatic perfusion,\textsuperscript{201} even after repeated doses.\textsuperscript{202}

A small study in adults with VF resistant to defibrillation randomized subjects to receive epinephrine or vasopressin plus epinephrine.\textsuperscript{203} The patients receiving vasopressin plus epinephrine were significantly more likely to survive to hospital admission and for 24 hours. Even low-dose vasopressin infusions demonstrated significant pressor effect in critically ill adults\textsuperscript{204,205} and critically ill infants and children during evaluation for brain death and organ recovery.\textsuperscript{206} Despite promising animal and limited clinical data,\textsuperscript{207} there is no data on the use of vasopressin in pediatric cardiac arrest. Moreover, in a piglet model of prolonged asphyxial cardiac arrest, vasopressin was less effective than epinephrine.\textsuperscript{208} Even though vasopressin is an alternative vasopressor in the treatment of adult shock-refractory VF, there is inadequate data to evaluate its efficacy and safety in infants and children at this time (Class Indeterminate; LOE 2, 6).

**Calcium**

Calcium is essential in myocardial excitation-contraction coupling. However, routine calcium administration does not improve outcome of cardiac arrest.\textsuperscript{209} In addition, several studies implicated cytoplasmic calcium accumulation in the final common pathway of cell death.\textsuperscript{210} Calcium accumulation results from calcium entering cells after ischemia and during reperfusion of ischemic organs; increased cytoplasmic calcium concentration activates intracellular enzyme systems, resulting in cellular necrosis.

Although calcium has been recommended in the treatment of electromechanical dissociation and asystole, experimental evidence for efficacy in either setting is lacking.\textsuperscript{209,211} Therefore, routine administration of calcium in resuscitation of asystolic patients cannot be recommended. Calcium is indicated for treatment of documented hypocalcemia and hyperkalemia,\textsuperscript{212} particularly in hemodynamically compromised patients. Ionized hypocalcemia is relatively common in critically ill children, particularly those with sepsis.\textsuperscript{213,214} Calcium also should be considered for treatment of hypermagnesemia\textsuperscript{215} and calcium channel blocker overdose\textsuperscript{216} (Class IIa; LOE 5, 6).

There is little information about the optimal emergency dose of calcium. The currently recommended dose of 5 to 7 mg/kg of elemental calcium is based on extrapolation from adult data and limited pediatric data.\textsuperscript{217} Calcium chloride 10% (100 mg/mL) is the calcium preparation of choice in children because it provides greater bioavailability of calcium than calcium gluconate.\textsuperscript{217} A dose of 0.2 mL/kg of 10% calcium chloride will provide 20 mg/kg of the salt and 5.4 mg/kg of elemental calcium. The dose should be infused by slow intravenous push over 10 to 20 seconds during cardiac arrest or over 5 to 10 minutes in perfusing patients. In cardiac arrest, the dose may be repeated in 10 minutes if required. Further doses should be based on measured deficits of ionized calcium.

**Magnesium**

Magnesium is a major intracellular cation and serves as a cofactor in >300 enzymatic reactions. The plasma magnesium concentration is composed of bound and unbound fractions in a manner that is similar to that of calcium; approximately 50% of the circulating magnesium is free (i.e., ionized). In critically ill patients the total magnesium concentration may poorly reflect the physiological (ionized) concentration\textsuperscript{218,219}; the latter can be measured with ion-selective electrodes. Particularly in pharmacological concentrations,\textsuperscript{220} magnesium can inhibit calcium channels, which represents some of the potentially therapeutic effects of magnesium. Through inhibition of calcium channels and the subsequent reduction of intracellular calcium concentration, magnesium causes smooth muscle relaxation, which has been used in the treatment of acute severe asthma.\textsuperscript{221} In addition, the effects of magnesium on calcium channels, and perhaps other membrane effects, have been useful in the treatment of torsades de pointes VT.\textsuperscript{222}

The beneficial effect of magnesium in acute asthma is debated; studies report conflicting results.\textsuperscript{221,223,224} In a randomized, prospective, double-blind pediatric trial, children who continued to have poor respiratory function (peak expiratory flow rate <60% of predicted) after 3 nebulized albuterol treatments were randomized to receive magnesium sulfate (25 mg/kg up to 2 g) or placebo.\textsuperscript{221} The children in the magnesium group had significantly greater improvement in pulmonary function and were less likely to be admitted for treatment than the placebo group. The entry criteria for this study may explain why earlier studies failed to show a beneficial effect: the study population was composed of those children who failed routine management with 3 nebulized albuterol treatments before study entry. This observation is consistent with a similarly designed randomized, blinded trial in children\textsuperscript{225} and a randomized, blinded clinical trial in adults showing that magnesium infusion (2 g over 20 minutes) produced a beneficial effect only in the most severely ill patients.\textsuperscript{226} Thus, data does not support the routine use of magnesium in asthma therapy but shows that it may be beneficial in children with severe asthma despite routine medical therapy. A dose of 25 to 50 mg/kg (up to 2 g) may be given safely over 10 to 20 minutes by intravenous infusion.\textsuperscript{225,227} Blood pressure and heart rate should be monitored during infusion. Although some evidence suggests that a threshold serum concentration is needed to produce a beneficial effect,\textsuperscript{228,229} there is insufficient data to recommend trying to achieve a specific serum concentration.

Magnesium has been used in the treatment of a wide range of arrhythmias and was used in post–myocardial infarction patients to reduce ventricular arrhythmias. Data, however, supports only the routine use of magnesium sulfate in patients with documented hypomagnesemia or with torsades de pointes VT.\textsuperscript{222,230} This is a unique polymorphic VT characterized by an ECG appearance of QRS complexes changing in amplitude and polarity so that they appear to rotate around an isoelectric line. It is seen in conditions distinguished by a
long QT interval. Prolongation of the QT interval may occur in congenital conditions (eg, Romano-Ward and Jervell and Lange-Nielsen) or following drug toxicity. Type Iα antiarrhythmics (eg, quinidine and disopyramide), type III (eg, sotalol and amiodarone), tricyclic antidepressants (see discussion below), and digitalis are all reported causes. In addition, unanticipated pharmacokinetic interactions may cause torsades de pointes; the interaction between cisapride and inhibitors of the cytochrome P450 system (eg, clarithromycin or erythromycin) is a recently recognized problem.231 Regardless of the cause, magnesium sulfate in a rapid intravenous infusion (several minutes) of 25 to 50 mg/kg (up to 2 g) is recommended in the setting of torsades de pointes VT.

**Glucose**

Infants have high glucose requirements and low glycogen stores. As a result, during periods of increased energy requirements, such as shock, the infant may become hypoglycemic. For this reason, monitor blood glucose concentrations closely using rapid bedside tests during coma, shock, or respiratory failure. Documented hypoglycemia should be treated with an infusion of a glucose-containing solution. A dose of 2 to 4 mL/kg of 25% glucose (250 mg/mL) will provide 0.5 to 1.0 g/kg; 10% glucose (100 mg/mL) may be used at a dose of 5 to 10 mL/kg to deliver a similar quantity of glucose.

If possible, treat hypoglycemia with a continuous glucose infusion; bolus therapy with hypertonic glucose should be limited if possible because it may contribute to a sharp rise in serum osmolality and may result in an osmotic diuresis. Furthermore, hyperglycemia before cerebral ischemia worsens neurological outcome.169,232 Although the effect of hyperglycemia occurring after cerebral ischemia on neurological function is unknown. Combined administration of glucose, insulin, and potassium after an ischemic insult may be beneficial, based on data in adults showing that this infusion improves outcome and reduces complications after myocardial infarction.233 In the absence of convincing data showing benefit or harm of hyperglycemia after arrest, the current recommendation is to ensure that the blood glucose concentration is at least normal during resuscitation and that hypoglycemia is avoided after resuscitation.

**Sodium Bicarbonate**

Although sodium bicarbonate previously was recommended for the treatment of severe metabolic acidosis in cardiac arrest, in most but not all studies routine sodium bicarbonate administration failed to improve the outcome of cardiac arrest.235 In children, respiratory failure is the major cause of cardiac arrest. Because sodium bicarbonate therapy transiently elevates CO₂ tension, administration of this drug to the pediatric patient during resuscitation may worsen existing respiratory acidosis. For these reasons treatment priorities for the infant or child in cardiac arrest should include assisting ventilation, supplementing oxygen, and restoring effective systemic perfusion (to correct tissue ischemia). Once effective ventilation is ensured and epinephrine plus chest compressions are provided to maximize circulation, use of sodium bicarbonate may be considered for the patient with prolonged cardiac arrest (Class IIb; LOE 6, 7).

Administration of this drug also may be considered when shock is associated with documented severe metabolic acidosis (Class IIb), although clinical trials in acidic critically ill adults failed to show a beneficial effect of sodium bicarbonate on hemodynamics despite improvements in metabolic acidosis.236,237 There is no specific level of acidosis that requires treatment; the decision to administer sodium bicarbonate is determined by the acuity and severity of the acidosis and the child’s circulatory state, among other factors. For example, a child with shock and marked metabolic acidosis from dehydration due to diabetic ketoacidosis does not require sodium bicarbonate in most circumstances and will respond well to fluid resuscitation and insulin administration alone.

Sodium bicarbonate is recommended in the treatment of symptomatic patients with hyperkalemia238 (Class IIa; LOE 6, 7), hypermagnesemia, tricyclic antidepressant overdose, or overdose from other sodium channel blocking agents239 (see “Special Resuscitation Situations” below; Class IIb; LOE 6, 7). Often patients with these metabolic or toxicological disorders will exhibit ECG abnormalities secondary to adverse effects on the heart.

When indicated, the initial dose of sodium bicarbonate is 1 mEq/kg (1 mL/kg of 8.4% solution) intravenously or via the intravenous route (Table 2). A dilute solution (0.5 mEq/mL; 4.2% solution) may be used in neonates to limit the osmotic load, but there is no evidence that the dilute solution is beneficial in older infants or children. Further doses of sodium bicarbonate may be based on blood gas analyses. If such measurements are unavailable, subsequent doses of sodium bicarbonate may be considered after every 10 minutes of continued arrest. Even if available, arterial blood gas analysis may not accurately reflect tissue and venous pH during cardiac arrest or severe shock.240,241 The role of sodium bicarbonate remains unclear in children who have documented postarrest metabolic acidosis.

Excessive sodium bicarbonate administration may have several adverse effects. Resulting metabolic alkalosis produces leftward displacement of the oxyhemoglobin dissociation curve with impaired delivery of oxygen to tissues,242 acute intracellular shift of potassium, decreased plasma ionized calcium concentration, decreased VF threshold,243 and impaired cardiac function. Hypernatremia and hyperosmolality may also result from excessive sodium bicarbonate administration.244 Catecholamines are inactivated by bicarbonate and calcium precipitates when mixed with bicarbonate, so the intravenous tubing must be carefully irrigated with a 5- to 10-mL normal saline bolus after administration of sodium bicarbonate. A normal saline bolus (5 to 10 mL) should be given routinely between infusions of any resuscitation drugs.

**Rhythm Disturbances**

Although primary cardiac events are uncommon in the pediatric age group, the ECGs of all critically ill or injured children should be continuously monitored. In addition to
Figure 6. PALS pulseless arrest algorithm.
detecting cardiac arrhythmias, it is worthwhile to monitor changes in the heart rate in response to therapy. Most pediatric arrhythmias are the consequence of hypoxemia, acidosis, and hypotension rather than the cause of these clinical states, but children with myocarditis or cardiomyopathy are at increased risk of primary arrhythmias, as are children after heart surgery. In addition, a number of drugs taken in therapeutic or toxic amounts may cause arrhythmias. When rhythm is recorded in pediatric cardiac arrest victims in the out-of-hospital, Emergency Department, and hospital settings, the majority have asystole or some form of bradyarythmia, often with a wide QRS complex.\(^2,17\)

Approximately 10% of reported pediatric cardiac arrest patients had VF or pulseless VT.\(^2\) In a relatively large retrospective out-of-hospital pediatric study, VF was observed in approximately 20% of out-of-hospital cardiac arrest victims after exclusion of SIDS patients.\(^175\)

The likelihood of VF increases with age, based on an analysis of out-of-hospital data. In children with nontraumatic arrest, VF was reported in only 3% of children from 0 to 8 years of age but was observed in 17% of victims from 8 to 30 years of age.\(^246\) In the previously noted out-of-hospital study,\(^175\) VF/VT was much more likely in children >9 years of age through adolescence (20%) than in those <4 years old (6.1% incidence if SIDS cases were included). In other out-of-hospital arrest studies, VF or VT occurred in 9% to 15% of children.\(^190,247\)

The likelihood of detecting a ventricular arrhythmia may depend on the response time or other characteristics of the EMS system, since only 4% of 300 children experiencing an out-of-hospital arrest in the Houston metropolitan area had a ventricular arrhythmia identified on EMS arrival.\(^17\) It is important to recognize and treat ventricular arrhythmias early, since the outcome is significantly better when these arrhythmias are promptly defibrillated than the reported outcome of children with asystole or other nonperfusing rhythms.\(^175,190,247\)

The following sections will review rhythm disturbances moving from slow rhythms to fast rhythms then to VF. Although not technically a specific rhythm disturbance, pulseless electrical activity (PEA) will also be discussed (Figure 6). For each rhythm we review the epidemiology, etiology, and treatment.

**Bradyarythmias**

Hypoxemia, hypothermia, acidosis, hypotension, and hypoglycemia may depress normal sinus node function and slow conduction through the myocardium. In addition, excessive vagal stimulation (eg, induced by suctioning or during endotracheal intubation) may produce bradycardia. Finally, central nervous system insults such as increased intracranial pressure or brain stem compression can result in prominent bradycardia. Sinus bradycardia, sinus node arrest with a slow junctional or idioventricular rhythm, and atrioventricular (AV) block are the most common preterminal rhythms observed in infants and children. When bradycardia is due to heart block, consider drug-induced causes, such as digoxin toxicity, and acute inflammatory injury from myocarditis. In addition, infants and children with a history of heart surgery are at increased risk of sick sinus syndrome or heart block secondary to injury to the AV node or conduction system. All slow rhythms that result in hemodynamic instability require immediate treatment (Figure 7).

**Treatment of Bradyarythmias**

In the small infant (<6 months), cardiac output is more dependent on heart rate than in the older infant and child; bradycardia is therefore more likely to cause symptoms in young infants. Clinically significant bradycardia is defined as a heart rate <60 bpm or a rapidly dropping heart rate despite adequate oxygenation and ventilation associated with poor systemic perfusion. Clinically significant bradycardia should be treated in a child of any age. Initial treatment should be directed to ensuring that the infant or child is breathing adequately and to providing supplemental oxygen. If a pharmacological agent is needed, epinephrine is the most useful drug in the treatment of symptomatic bradycardia in an infant or child, except for bradycardia caused by heart block or increased vagal tone (Figure 7; Class Ia; LOE 7, 7, 8). For suspected vagally mediated bradycardia, atropine is the initial drug of choice. If the bradycardia persists after adequate oxygenation and ventilation and responds only transiently or not at all to bolus epinephrine or atropine administration, consider a continuous infusion of epinephrine or dopamine (Figure 7).

Atropine sulfate, a parasympatholytic drug, accelerates sinus or atrial pacemakers and increases AV conduction. Atropine is recommended in the treatment of symptomatic bradycardia caused by AV block or increased vagal activity (Class I), such as vagally mediated bradycardia during attempts at intubation. Although atropine may be used to treat bradycardia accompanied by poor perfusion or hypotension (Class IIb), epinephrine may be more effective in treating bradycardia accompanied by hypotension. When indicated, give atropine to treat bradycardia only after ensuring adequate oxygenation and ventilation and responds only transiently or not at all to bolus epinephrine or atropine administration, consider a continuous infusion of epinephrine or dopamine (Figure 7).

Small doses of atropine may produce paradoxical bradycardia\(^248\); the recommended dose is 0.02 mg/kg, with a minimum dose of 0.1 mg and a maximum single dose of 0.5 mg in a child and 1.0 mg in an adolescent.\(^248\) The dose may be repeated in 5 minutes, to a maximum total dose of 1.0 mg in a child and 2.0 mg in an adolescent. Larger intravascular doses may be required in special resuscitation circumstances (eg, organophosphate poisoning).\(^249\) If intravenous access is not readily available, atropine (0.02 mg/kg) may be administered tracheally,\(^250\) although absorption into the circulation may be unreliable.\(^251\)

Tachycardia may follow administration of atropine, but the agent is generally well tolerated in the pediatric patient. Atropine used to block vagally mediated bradycardia during intubation may have the undesirable effect of masking hypoxemia-induced bradycardia. Therefore, during attempts at intubation, monitor oxygen saturation with pulse oximetry and avoid prolonged attempts at intubation.

In selected cases of bradycardia caused by complete heart block or abnormal function of the sinus node, emergency
transthoracic pacing may be lifesaving. Pacing is not helpful in children with bradycardia secondary to a postarrest hypoxic/ischemic myocardial insult or respiratory failure. Pacing also was not shown to be effective in the treatment of asystole in children.

**Pulseless Electrical Activity**

PEA is a clinical state characterized by organized electrical activity observed on a monitor or ECG in the absence of detectable cardiac output (ie, pulses). This clinical state often represents a preterminal condition that immediately precedes...
asystole. It frequently represents the final organized electrical state of a severely hypoxic, acidic myocardium and is usually characterized on the monitor by a slow, wide-complex rhythm in a child who has experienced a prolonged period of hypoxia, ischemia, or hypercarbia. In this setting treat PEA in the same manner as asystole.

Occasionally PEA is due to a reversible cause that often occurs rapidly and represents a sudden impairment of cardiac output. When seen shortly after onset, the ECG rhythm may appear normal and the heart rate may be increased or be rapidly decreasing, but pulses or other evidence of detectable cardiac output are absent and the child appears lifeless. This subcategory of PEA is often called electromechanical dissociation (EMD). Causes of EMD are seen in Figure 6 (earlier in this segment) and can be recalled as the 4 H’s and 4 T’s. The 4 H’s are severe hypovolemia (eg, in trauma), hypoxemia, hypothermia, and hyperkalemia (and other metabolic imbalances). The 4 T’s are tension pneumothorax, pericardial tamponade, toxins, and pulmonary thromboembolus. If EMD is observed, search for evidence of these reversible causes and correct them if identified.

**Treatment of PEA**

Treat PEA in the same manner as asystole (Figure 6, pulseless arrest algorithm), with the caveat that reversible causes should be identified and corrected. If the patient remains pulseless after you have established an airway, ventilated the lungs, provided supplemental oxygen, and delivered chest compressions, give epinephrine (0.01 mg/kg initial dose). Several of the reversible causes of PEA (ie, hypovolemia, tension pneumothorax, and pericardial tamponade) may be at least partially corrected by the administration of a fluid bolus of normal saline or lactated Ringer’s solution. Tension pneumothorax and pericardial tamponade, however, will also require more definitive therapy with needle aspiration or rapid drainage catheter placement. Check the child’s temperature and perform immediate (ideally bedside) testing of glucose, electrolytes, and acid-base status. In the out-of-hospital setting, early recognition and effective treatment of PEA (and other rhythm disturbances associated with cardiac arrest) are emphasized on the basis of data reporting that a return of spontaneous circulation before arrival in the Emergency Department is associated with improved survival.176,247,254

**Supraventricular Tachycardia**

Supraventricular tachycardia (SVT) is the most common nonarrest arrhythmia during childhood and is the most common arrhythmia that produces cardiovascular instability during infancy. Usually caused by a reentrant mechanism, SVT in infants generally produces a heart rate >220 bpm and sometimes as high as 300 bpm. Lower heart rates may be observed in children during SVT. The QRS complex is narrow (ie, ≤0.08 seconds) in >90% of involved children,255,256 making differentiation between marked sinus tachycardia (ST) due to shock and SVT somewhat difficult, particularly because either rhythm may be associated with poor systemic perfusion.

The following characteristics may aid differentiation between ST and SVT (Figure 8):

- A history consistent with shock (eg, dehydration or hemorrhage) is usually present with ST, whereas the history is often vague and nondescript with SVT.
- The heart rate is usually <220 bpm in infants and <180 bpm in children with ST, whereas infants with SVT typically have a heart rate >220 bpm, and children with SVT will typically have a heart rate >180 bpm.
- P waves may be difficult to identify in both ST and SVT once the ventricular rate exceeds 200 bpm, but they are usually present in infants and children with ST. If P waves are identifiable in ST, they are usually upright in leads I and aVF, whereas in SVT they are negative in leads II, III, and aVF.
- In ST the heart rate varies from beat to beat (variable R-R interval) and is often responsive to stimulation, but there is no beat-to-beat variability in SVT. Termination of SVT is abrupt, whereas the heart rate slows gradually in ST.

Cardiopulmonary stability during episodes of SVT is affected by the child’s age, duration of SVT, prior ventricular function, and ventricular rate. Older children will typically complain of lightheadedness, dizziness, or chest discomfort, or simply note the fast heart rate. In infants, however, very rapid rates may be undetected for long periods until low cardiac output and shock develop. This deterioration in cardiac function occurs secondary to the combination of increased myocardial oxygen demand and limitation in myocardial oxygen delivery during the short diastolic phase associated with very rapid heart rates. If baseline myocardial function is impaired (eg, in a child with a cardiomyopathy), SVT can produce signs of shock in a relatively short time.

**Wide-QRS SVT**

Wide-QRS SVT (ie, SVT with aberrant conduction) is uncommon in infants and children. Correct diagnosis and differentiation from VT depends on careful analysis of at least a 12-lead ECG that may be supplemented by information from an esophageal lead. Obtain a patient and family history to help identify the presence of an underlying condition predisposing to stable VT. Because either SVT or VT can cause hemodynamic instability, do not base assumptions about the mechanism (ie, ventricular versus supraventricular) solely on the hemodynamic status of the patient. In most circumstances, wide-complex tachycardias should be treated as if they are VT (Figure 9).

**Vagal Maneuvers**

In children with milder symptoms who are hemodynamically stable or during preparation for cardioversion or drug therapy, vagal maneuvers may be tried (Class IIa; LOE 4, 5, 7, 8). Success rates with these maneuvers are variable and depend on the presence of underlying conditions in the patient, the patient’s level of cooperation, and the patient’s age. Ice water applied to the face is most effective in infants and young children.257,258 One method uses crushed ice mixed with water in a plastic bag or glove. Use care to apply the ice water mixture to the infant’s face without obstructing ventilation. Other vagal maneuvers (ie,
carotid sinus massage (or Valsalva) may be effective (Class IIb; LOE 5, 7) and appear to be safe on the basis of data obtained largely in older children, adolescents, and adults.259–261 In children one technique for performing a Valsalva maneuver is to have the child blow through a straw.260 Regardless of which vagal maneuver is attempted, obtain a 12-lead ECG before and after the vagal maneuver and monitor the ECG continuously during application of the ice water or vagal maneuver. Note that application of external ocular pressure may be dangerous and should not be used to induce a vagal response.

**Cardioversion**

SVT that causes circulatory instability (eg, congestive heart failure with diminished peripheral perfusion, increased work of breathing and altered level of consciousness, or hypotension) is most expeditiously treated with electrical or chemical cardioversion. Synchronized electrical cardioversion is rec-
Figure 9. PALS tachycardia algorithm for infants and children with rapid rhythm and evidence of poor perfusion.
ommended at a starting dose of 0.5 to 1 J/kg. If vascular access is already available, adenosine may be administered before electrical cardioversion, but do not delay cardioversion if establishment of vascular access (intravenous or intraoosseous) will require >20 to 30 seconds.

Adenosine
When medications are indicated, adenosine is the drug of choice for SVT in children (Class IIa; LOE 2, 3, 7).256,262 If the patient is unstable, do not delay cardioversion to secure vascular access and deliver adenosine. Adenosine is an endogenous nucleoside that acts at specific receptors to cause a temporary block of conduction through the AV node; it interrupts the reentry circuits that involve the AV node. These reentry circuits are the underlying mechanism for the vast majority of SVT episodes in infants and children. Adenosine is very effective; side effects are minimal because its half-life is only 10 seconds. With continuous ECG monitoring, administer 0.1 mg/kg as a rapid intravenous bolus (Table 2). To enhance delivery of the drug to its site of action in the heart, the injection site should be as close to the heart as possible. A 2-syringe technique is recommended, 1 syringe containing the drug and 1 containing a saline flush of at least 5 mL. Because adenosine is metabolized by an enzyme on the surface of red blood cells (adenosine deaminase), a higher dose may be required for peripheral venous administration than if the drug is administered into a central vein.256,262 If there is no effect, the dose may be doubled (0.2 mg/kg). The maximum recommended initial adult dose is 6 mg, and 12 mg is the second maximum dose. A single dose of adenosine should not exceed 12 mg.256,262 Based on experimental data and a case report, adenosine may also be given by the intraoosseous route.263,264

Verapamil Caution and Alternative Agents
Verapamil should not be used to treat SVT in infants because refractory hypotension and cardiac arrest have been reported following its administration (Class III; LOE 5).265,266 and we discourage its use in children because it may cause hypotension and myocardial depression.267 When used in children older than 1 year, verapamil is infused in a dose of 0.1 mg/kg. Procainamide and amiodarone are alternative agents for use in children with SVT and stable hemodynamics (Class IIb).268,269 but they should not be used concurrently with agents that may prolong the QT interval. Therefore, amiodarone and procainamide generally should not be administered together because they both prolong the QT interval (Figure 9).

Treatment of Wide-QRS Tachycardia
The decision to initiate treatment is based on whether the patient is hemodynamically stable. In the absence of a mitigating history, wide-complex tachycardia associated with hemodynamic instability requires urgent treatment, based on the assumption that the rhythm is ventricular in origin (see “Treatment of VT and VF” below). Urgent treatment of a wide-complex tachycardia includes synchronized cardioversion if pulses are present and defibrillation shocks if pulses are lost. Signs of hemodynamic instability include evidence of compromised tissue perfusion and impaired level of consciousness. If the child is hemodynamically stable (ie, has normal perfusion and level of consciousness), treatment can await further diagnostic studies. Early consultation with a pediatric cardiologist or other physician with appropriate expertise is recommended.

Ventricular Tachycardia and Ventricular Fibrillation
VT and VF are uncommon in children. When seen, consider congenital heart disease, cardiomyopathies, or acute inflammatory injury to the heart (eg, myocarditis). In addition, identify and treat reversible causes, including drug toxicity (eg, recreational drugs, tricyclic antidepressants, digoxin overdose, or toxicity from the combination of cisapride and macrolide antibiotics231), metabolic causes (eg, hyperkalemia, hypermagnesemia, hypocalcemia, or hypoglycemia), or hypothermia (see pulseless arrest algorithm, Figure 6).

Treatment of VT and VF
Hemodynamically Stable VT
If the child with VT is hemodynamically stable (ie, is alert with palpable distal pulses), careful evaluation and early consultation with a cardiologist are indicated before any therapy is given. Focus initial efforts on determining the origin of the tachycardia based on analysis of the 12-lead ECG and a carefully obtained history, including family history for ventricular arrhythmias or sudden death. If pharmacological therapy is undertaken, amiodarone (5 mg/kg over 20 to 60 minutes) should be considered (Class IIb; LOE 7). Procainamide (15 mg/kg over 30 to 60 minutes) or lidocaine (1 mg/kg over approximately 2 to 4 minutes) may be considered as alternative agents. A cautious approach is appropriate in children who are hemodynamically stable, because all of these drugs have intrinsic risks. Amiodarone and procainamide can cause hypotension, and procainamide is a potent negative inotrope. Close hemodynamic and ECG monitoring are required during and after the infusion of either agent. As noted previously, amiodarone and procainamide generally should not be administered together because both prolong the QT interval.

Cardioversion for VT With Pulses
In the infant or child with VT and palpable pulses associated with signs of shock (ie, low cardiac output, poor perfusion), immediate synchronized cardioversion is indicated (Figure 9). Depending on the severity of hemodynamic compromise and the patient’s level of consciousness, cardioversion may be provided before vascular access is obtained. If the child is appropriately responsive and not in distress, there is often time to consult a cardiologist, obtain vascular access, and consider administration of sedation before cardioversion. In addition, it is important to consider drug or metabolic causes of the VT, especially in a child without a known predisposing cause for the arrhythmia. The rhythm should be examined for a torsades de pointes appearance. If torsades de pointes is suspected, administer 25 mg/kg of magnesium by a slow intravenous bolus over 10 to 20 minutes.

Pulseless VT/VF
Delivering shocks to produce defibrillation is the definitive therapy (Figure 2) for pulseless VT and VF. In this setting,
deliver shocks immediately. Ventilation, oxygenation, and chest compressions should be delivered and vascular access may be attempted until the defibrillator arrives and is charged, but these interventions should not delay shocks. If the patient fails to defibrillate after 3 shocks (refer to Figure 6), administer intravenous epinephrine in a dose of 0.01 mg/kg (or 0.1 mg/kg for the tracheal route) and attempt defibrillation again within 30 to 60 seconds. If VF or pulseless VT continues after this epinephrine dose plus shock(s) or if VF/pulseless VT recurs, amiodarone (5 mg/kg by rapid intravenous bolus) may be used (Class Indeterminate; LOE 7) followed by another defibrillation attempt within 30 to 60 seconds after closed-chest compression to deliver the drug to its site of action. (Note that the pattern of treatment after the initial 3 shocks is “CPR-drug-shock, CPR-drug-shock.” We recommend no more than 30 to 60 seconds of artificial circulation before the next shock.) The use of amiodarone is based on adult data of “shock-resistant VT/VF” leading to experience with the use of amiodarone in children in the intensive care unit and (see Figures 6, 8, and 9 and Table 2). “Shock resistance” of a ventricular arrhythmia is defined as continued VF or pulseless VT (ie, requiring epinephrine and a fourth precordial shock) or the recurrence of VF/pulseless VT after initial shock(s) caused defibrillation. Amiodarone will not terminate VF, but it can prevent the recurrence of VF after a successful shock. In summary, amiodarone administration in children with VT with a pulse is a Class IIb recommendation, whereas it is Class Indeterminate in VF and pulseless VT.

In the ACLS algorithm for treatment of pulseless VT and VF, shocks may be delivered in clusters of 3, separated by 1 minute of CPR and drug administration. This “CPR-drug-shock-shock-shock, CPR-drug-shock-shock-shock” pattern is an acceptable alternative to the “CPR-drug-shock, CPR-drug-shock” pattern of resuscitation.

Bretylium is no longer considered an appropriate agent because of the risk of hypotension, the lack of demonstrable effectiveness in VT, and the absence of published studies of its use in children (Class III; LOE 7). Because it cannot be administered rapidly, procainamide also is not considered an appropriate agent in VF or pulseless VT therapy. Although sotalol is not available in the United States as an intravenous preparation, intravenous sotalol may be considered in other countries and subsequently may be approved in the United States (Class IIb; LOE 7).

Amiodarone. Amiodarone is a highly lipid-soluble antiarrhythmic with complex pharmacology, making it difficult to classify. The oral form of the drug is poorly absorbed, which makes acute therapy by the oral route largely impractical. However, an intravenous preparation was approved in 1995, and amiodarone increasingly is used for a wide range of both atrial and ventricular arrhythmias in adults and children. Amiodarone is a noncompetitive inhibitor of both α- and β-adrenergic receptors. Secondary to this sympathetic block, intravenous administration of amiodarone produces vasodilatation and AV nodal suppression; the latter results from prolonging the AV nodal refractory period and slowing AV nodal conduction. Amiodarone inhibits the outward potassium current, which prolongs the QT interval. This effect is thought to be its major action in acutely controlling arrhythmias, but it may also increase the propensity for polymorphic ventricular arrhythmias (ie, torsades de pointes tachycardia). Fortunately this appears to be an uncommon complication. Amiodarone also inhibits sodium channels, which slows conduction in the ventricular myocardium and prolongs QRS duration. Amiodarone-induced sodium channel blockade is use dependent, meaning that the drug is more effective at faster heart rates, which probably represents an important mechanism of its effectiveness in SVT and VT. Intravenous dosing recommendations in children are derived from a number of case series. Amiodarone has been used most commonly in children to treat ectopic atrial tachycardia or junctional ectopic tachycardia after cardiac surgery and VT in postoperative patients or children with underlying cardiac disease. For both supraventricular and ventricular arrhythmias, a loading infusion of 5 mg/kg is recommended over several minutes to 1 hour, depending on the need to achieve a rapid drug effect. Repeated doses of 5 mg/kg up to a maximum of 15 mg/kg per day may be used as needed. Because of the high lipid solubility of amiodarone, measurement of drug levels correlates poorly with drug effect. The main acute side effect from intravenous administration is hypotension. Terminal elimination of amiodarone is very prolonged, with a half-life lasting up to 40 days, but this is relatively unimportant with acute loading. Elimination is not dependent on normal renal or hepatic function. Because of its complex pharmacology, poor oral absorption, and potential for long-term adverse effects, a pediatric cardiologist or similarly experienced provider should direct chronic amiodarone therapy. Potential long-term complications include interference with thyroid hormone metabolism leading to hypothyroidism or hyperthyroidism, interstitial pneumonitis, corneal microdeposits, blue-gray skin discoloration, and elevated liver enzyme levels. ARDS is an unusual but potentially life-threatening complication seen in patients receiving chronic amiodarone therapy who undergo a surgical procedure, especially a cardiac or pulmonary procedure. Fortunately this has not been reported in children, but pulmonary fibrosis was reported in an infant receiving chronic therapy. As use of amiodarone becomes more frequent, we encourage reporting the occurrence of this and other complications.

Lidocaine. Lidocaine is a sodium channel blocker that reduces the slope of phase 4 diastolic repolarization, which decreases automaticity and therefore suppresses ventricular arrhythmias. Therapeutic concentrations raise the VF threshold and therefore may protect against re fibrillation after successful defibrillation. Although lidocaine has long been recommended for the treatment of ventricular arrhythmias in infants and children, data suggests that it is not very effective unless the arrhythmia is associated with focal myocardial ischemia. Lidocaine may be considered in children with shock-resistant VF or pulseless VT (Class Indeterminate; LOE 5, 6, 7). The recommended dose is 1 mg/kg by rapid intravenous injection followed by an
Defibrillation, Cardioversion, and External Pacing

Defibrillation

Defibrillation is the untimed (asynchronous) depolarization of the myocardium that successfully terminates VF or pulseless VT. Electric shocks are used to achieve defibrillation; shocks produce a simultaneous depolarization of a critical mass of myocardial cells, which may then allow resumption of spontaneous depolarization, especially if the myocardium is oxygenated and normothermic and acidosis is not excessive. When VF occurs suddenly, an immediate shock is usually effective. If the arrest is prolonged or the child does not respond to the initial attempts at defibrillation, then ventilation, oxygenation, chest compressions, and pharmacological therapy may be needed to improve the metabolic environment of the myocardium (Figure 2). Defibrillation is not effective in the treatment of asystolic arrest.

Infusions are given at a rate of 20 to 50 μg/kg per minute. If there is more than a 15-minute delay between the bolus dose and start of an infusion, a second bolus dose of 0.5 to 1 mg/kg lidocaine may be given to rapidly restore therapeuthic concentrations. Lidocaine toxicity from excessive plasma concentrations may be seen in patients with persistently poor cardiac output and hepatic or renal failure.

Excessive plasma concentrations may cause myocardial and circulatory depression and possible central nervous system symptoms, including drowsiness, disorientation, muscle twitching, or seizures. If reduced lidocaine clearance is expected or suspected, the infusion rate generally should not exceed 20 μg/kg per minute.

Procainamide. Procainamide is a sodium channel blocking antiarrhythmic agent that prolongs the effective refractory period of atria and ventricles and depresses the conduction velocity within the conduction system. This typically produces prolongation of conduction and refractoriness of accessory pathways, but somewhat paradoxically it shortens the effective refractory period of the AV node and increases AV nodal conduction. This may lead to increased heart rates when used to treat ectopic atrial tachycardia. By slowing intraventricular conduction, procainamide prolongs the QT and PR intervals. Procainamide is effective in the treatment of atrial fibrillation, flutter, and SVT. It may be useful when used to treat ectopic atrial tachycardia.

It also has been used to treat or suppress in the treatment of postoperative junctional ectopic atrial fibrillation, flutter, and SVT, and it may be useful in the treatment of postoperative junctional ectopic tachycardia. It has also been used to treat or suppress VT. Despite a long history of use, there is little data on the effectiveness of procainamide compared with other antiarrhythmic agents in children. Since procainamide must be given by a slow infusion to avoid toxicity from heart block, myocardial depression, and prolongation of the QT interval (which predisposes to torsades de pointes tachycardia), procainamide is not indicated in the treatment of VF or pulseless VT. In children with a perfusing rhythm associated with VT, procainamide may be considered (Class IIb; LOE 5, 6, 7; see Figures 6 and 9). Infuse the loading dose of 15 mg/kg over 30 to 60 minutes with continuous monitoring of the ECG and frequent blood pressure monitoring. If the QRS widens to >50% of baseline or hypotension occurs, stop the infusion. Since procainamide increases the likelihood of polymorphous VT developing, it generally should not be used in combination with another agent that prolongs the QT interval, such as amiodarone.

Epinephrine and Vasopressin. A vasoconstrictor regimen may be considered in shock-resistant VT/VF, since if systemic vasoconstriction is inadequate with routine therapy, coronary perfusion is limited and the myocardium is unlikely to respond to shocks. For these reasons high-dose epinephrine (0.1 to 0.2 mg/kg) may be considered in shock-resistant VF/pulseless VT (Class IIb; LOE 5, 6, 7). Data in animals and limited data in adults suggest that vasopressin may be helpful in VF and pulseless VT, but data is insufficient to allow recommendation for use in children (see previous discussion; Class Indeterminate).

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Newer defibrillators use biphasic waveforms; this waveform appears to be effective at lower energy doses.\textsuperscript{7} Although there is no published data in young children, biphasic AEDs may be used in children \(\geq 8\) years (approximately \(>25\) kg body weight) in the out-of-hospital setting (see “AEDs in Children” below). Manual biphasic defibrillators have also been developed. As information on energy dosing becomes available, these defibrillators may be used appropriately in young children.

If the initial 3 defibrillation attempts are unsuccessful, correct acidosis, hypoxemia, or hypothermia if present and administer epinephrine, perform CPR, and attempt defibrillation. If the repeat (fourth) shock is ineffective, administration of amiodarone (Class Indeterminate) is recommended, and lidocaine or high-dose epinephrine (Class IIb) may be considered. Defibrillation should be repeated with 4 J/kg (Figure 2) within 30 to 60 seconds after each drug (CPR-drug-shock, CPR-drug-shock) if VT/VF persists. An alternative therapeutic approach in shock-resistant VF or pulseless VT is CPR, drug administration, and then 3 shocks in succession.

The recommended energy dose of 2 J/kg is appropriate for children up to at least 8 years of age. As discussed below in the section on AEDs in children, the age or size at which a fixed-energy-dose “adult” defibrillator can be used is unknown. In the out-of-hospital setting, it may be reasonable to use adult defibrillation algorithms in children \(\geq 8\) years, and it certainly is reasonable to use adult energy doses in children who weigh at least 50 kg.

Increasing the shock energy dose is not indicated when defibrillation is initially successful but the rhythm deteriorates back to VF. In this situation, adjunctive medications (eg, amiodarone, lidocaine, or sotalol) may improve the success of subsequent defibrillation at the previously effective dose and prevent further recurrences. In addition, reversible causes of VF/VT should be sought and treated in patients with refractory VF/VT (ie, the 4 H’s and 4 T’s; see Figure 6).

### AEDs in Children

In the prehospital setting, AEDs are commonly used in adults to assess cardiac rhythm and to deliver shocks to produce defibrillation. Data suggests that AEDs can accurately detect VF in children of all ages,\textsuperscript{313–315} but there is inadequate data regarding the ability of AEDs to correctly identify tachyarrhythmic rhythms in infants.\textsuperscript{315} Based on available data, AEDs may be considered for rhythm identification (Class IIb; LOE 3, 5) in children \(\geq 8\) years old but are not recommended for younger children or infants. The energy dose delivered by currently available monophasic and biphasic AEDs exceeds the recommended dose of 2 to 4 J/kg for most children \(< 8\) years of age. The median weight of children \(\geq 8\) years typically exceeds 25 kg (a weight of 25 kg corresponds to a body length of approximately 50 inches or 128 cm\textsuperscript{161}). Thus, the delivered initial dose from an AED (150 to 200 J) will be \(< 10\) J/kg for most children \(\geq 8\) years. Animal data suggests that this may be a safe dose, so attempted defibrillation of VF/pulseless VT detected by an AED may be considered in these older children (Class Indeterminate; LOE 6), particularly in the out-of-hospital setting.\textsuperscript{316} Locations that routinely care for children at risk for arrhythmias and cardiac arrest (eg, in-hospital settings) should continue to use defibrillators capable of appropriate energy adjustment. Attempted defibrillation of children younger than approximately 8 years with energy doses typical of AEDs cannot be recommended at this time. Biphasic waveform transthoracic defibrillation requires lower energy and appears to be effective in adults,\textsuperscript{7} but there is inadequate data to recommend a biphasic energy dose for treatment of VF/pulseless VT in children (Class Indeterminate).

### Synchronized Cardioversion

Synchronized cardioversion is the timed depolarization of myocardial cells that successfully restores a stable rhythm. It is used to treat the symptomatic patient with VT or VF (with pulses) accompanied by poor perfusion, hypotension, or heart failure. It also may be used electively in children with stable VT or SVT at the direction of an appropriate cardiology specialist.

The synchronizer circuit on the defibrillator must be activated before each cardioversion attempt. The initial energy dose is approximately 0.5 to 1 J/kg. The dose is increased up to 2 J/kg with subsequent attempts if necessary. If a second shock is unsuccessful or the tachycardia recurs quickly, consider antiarrhythmic therapy before a third shock. Hypoxemia, acidosis, hypoglycemia, or hypothermia should be corrected if the patient fails to respond to attempts at cardioversion.

### Noninvasive (Transcutaneous) Pacing

Noninvasive transcutaneous pacing has been used to treat adults with bradycardia or asystole.\textsuperscript{317,318} Experience with children, however, is limited and does not support a beneficial effect of pacing on outcome of children with cardiac arrest.\textsuperscript{252,253} Since this form of pacing is very uncomfortable, its use is reserved for children with profound symptomatic bradycardia refractory to BLS and ALS (Class IIb; LOE 5, 7), particularly when caused by underlying congenital or acquired heart disease producing complete heart block or sinus node dysfunction.\textsuperscript{252}

Noninvasive pacing requires the use of an external pacing unit and 2 large adhesive-backed electrodes. If the child weighs \(< 15\) kg, pediatric (small or medium) electrodes are recommended.\textsuperscript{252} The negative electrode is placed over the heart on the anterior chest and the positive electrode behind the heart on the back. If the back cannot be used, the positive electrode is placed on the right side of the anterior chest under the clavicle and the negative electrode on the left side of the chest over the fourth intercostal space, in the midaxillary area. Precise placement of electrodes does not appear to be necessary provided that the negative electrode is placed near the apex of the heart.\textsuperscript{319,320}

Either asynchronous ventricular fixed-rate or ventricular-inhibited pacing may be provided; the latter is preferred. It will usually be necessary to adjust pacemaker output to ensure that every pacer impulse results in ventricular depolarization (capture). In general, if smaller electrodes are used, the pacer output required to produce capture will be higher.\textsuperscript{252} If ventricular-inhibited pacing is performed, the sensitivity of
the pacer’s ECG detection must be adjusted so that intrinsic ventricular electric activity is appropriately sensed. To limit discomfort and to ensure a more reliable method of ongoing cardiac pacing, cardiology consultation is indicated if transcutaneous pacing is successful.

**PALS for the Pediatric Trauma Victim**

The principles of resuscitation of the seriously injured child are the same as those for any pediatric patient requiring PALS. Some aspects of pediatric trauma care, however, require emphasis, because improper resuscitation may be a major cause of preventable pediatric trauma death. Common errors in pediatric trauma resuscitation include failure to open and maintain the airway, failure to provide appropriate fluid resuscitation for children (including those with head injury), and failure to recognize and treat internal bleeding. A qualified surgeon should be involved early in the course of the resuscitation. If possible, children with multisystem trauma should be transported rapidly to trauma centers with pediatric expertise. The relative value of aeromedical transport compared with ground transport of children with multiple trauma is unclear and should be evaluated by individual EMS systems. Depending on the characteristics of each EMS system, it is likely that one mode of transport will be favored over the other.

Initial stabilization of the trauma victim involves 2 surveys: the Primary Survey and the Secondary Survey. Each focuses on assessment and treatment of life-threatening conditions. The Primary Survey includes the ABCs of BLS—including meticulous attention to Airway, Breathing, and Circulation—plus a “D” for Disability to evaluate neurological condition and an “E” for Exposure to keep the child warm and expose the skin to look for hidden injuries.

Airway control includes cervical spine immobilization, which must be continued during transport and stabilization in an ALS facility. Immobilization of an infant’s or young child’s cervical spine in a neutral position is challenging because the occiput is large in young children. Immobilization can best be achieved by using a backboard with a recess for the head or using a roll under the back from the shoulders to the buttocks. Semirigid cervical collars are available in a wide variety of sizes. They can help maintain immobilization in children of various sizes. The head and neck should be further immobilized with towel rolls and tape, with secondary immobilization of the child on a spine board (Figure 10).

Breathing support is provided as needed. In the out-of-hospital setting, bag-mask ventilation may enable adequate support of oxygenation and ventilation, particularly when the transport time is short. Endotracheal intubation is indicated if the trauma victim’s respiratory effort is inadequate, airway patency is compromised, or coma is present. Orotracheal intubation in the out-of-hospital setting should be performed only by properly trained and experienced providers. Regardless of the performance site, cervical spine immobilization...
should be addressed during the entire intubation procedure (Figure 11). Cricoid pressure may facilitate intubation when movement of the neck must be avoided. We particularly encourage confirmation of proper tracheal tube placement by use of capnography or exhaled CO$_2$ detection both after intubation and throughout transport (Class IIa), because hypoxemia and hypercarbia will complicate intracranial injury and are associated with poor outcome.

Although initial hyperventilation for patients with head trauma was previously recommended, routine hyperventilation is not associated with an improved outcome in these patients and may increase intrathoracic pressures, adversely affecting venous return and cardiac output. In addition, hyperventilation may adversely affect cerebral perfusion in areas of the brain still responsive to changes in PCO$_2$, leading to local or global brain ischemia. Hyperventilation is no longer routinely recommended (Class III; LOE 3, 5, 6) and should be reserved for situations in which the victim has signs of increased intracranial pressure, such as transtentorial herniation. After intubation of the trauma patient, the goal of ventilatory support is to restore or maintain normal ventilation and good oxygenation.

In the traumatized victim, ventilation may be impaired by tension pneumothorax, open pneumothorax, hemorthorax, or flail chest. Major thoracic injuries may be present in the absence of external evidence of chest trauma because the child’s chest is extremely compliant. Even severe blunt chest trauma may fail to produce rib fractures. Thoracic injuries must be suspected, identified, and treated if there is a history of thoracoabdominal trauma or difficulty in providing effective ventilation.

After the airway is secured, a nasogastric or an orogastric tube should be inserted to prevent or relieve gastric inflation. Maxillofacial trauma and suspicion or confirmation of a basilar skull fracture are contraindications to blind nasogastric tube insertion because intracranial tube migration may result.

Support of circulation in the trauma victim often requires treatment of hemorrhagic shock. Circulatory support of the pediatric trauma victim requires simultaneous control of external hemorrhage, assessment and support of systemic perfusion, and restoration and maintenance of blood volume. Control of external hemorrhage is best accomplished with direct pressure. Blind application of hemostatic clamps and use of tourniquets are contraindicated, except in traumatic external hemorrhage, assessment and support of systemic perfusion, and restoration and maintenance of blood volume. Control of external hemorrhage is best accomplished with direct pressure. Blind application of hemostatic clamps and use of tourniquets are contraindicated, except in traumatic amputation associated with bleeding from a major vessel.

If systemic perfusion is inadequate, provide rapid volume replacement with a bolus of 20 mL/kg of an isotonic crystalloid (eg, normal saline or lactated Ringer’s solution) even if blood pressure is normal. Administer a second bolus (20 mL/kg) rapidly if heart rate, level of consciousness, capillary refill, and other signs of systemic perfusion fail to improve. The presence of hypotension traditionally was assumed to indicate a blood volume loss of ≥20% and the need for urgent volume replacement and blood transfusion; however, minimal data supports this assumption. It is important to note that hypotension also may occur secondary to reversible causes such as a tension pneumothorax or pericardial tamponade, and hypotension may result from a neurological insult (eg, spinal cord injury or massive brain or brain stem injury resulting in loss of sympathetic nervous system control of peripheral vascular tone).

If the poorly perfused victim fails to respond to administration of 40 to 60 mL/kg of crystalloid, transfusion of 10 to 15 mL/kg of blood is indicated. Although type-specific crossmatched blood is preferred, O-negative blood may be used under urgent conditions. The blood should be warmed before transfusion; otherwise, rapid administration may result in significant hypothermia and can result in transient ionized hypocalcemia. Consider intra-abdominal hemorrhage as a cause of continued hemodynamic instability despite adequate oxygenation, ventilation, and fluid resuscitation; surgical exploration may be needed. Undetected hemorrhage, particularly intra-abdominal hemorrhage, is a cause of preventable pediatric trauma mortality.

Evaluation of neurological function (the “D” of Disability) requires application of a rapid neurological assessment, including a Glasgow Coma Scale (GCS) score. This scoring system evaluates eye opening, verbalization, and movement in response to stimulation. Serial assessments with the GCS allow rapid identification of any deterioration in the child’s neurological status.

The “E” portion of the Primary Survey, Exposure, involves maintenance of a neutral thermal environment—keeping the child warm. A second meaning of exposure is to completely examine the child for hidden injuries.

The Secondary ABCD Trauma Survey involves more detailed evaluation and definitive therapy. This includes a head-to-toe assessment that is beyond the scope of these guidelines.

### Special Resuscitation Situations

The principles of PALS introduced earlier in these guidelines are applicable in a wide variety of life-threatening circumstances. There are special situations, however, that require specific interventions that may differ from the routine PALS approach. Often these conditions are suggested by the history surrounding the event, knowledge of the common causes of arrest in various age groups, or rapidly obtained diagnostic tests. The special resuscitation situations covered in this section include toxicological emergencies and submersion/drowning. “Part 8: Advanced Challenges in Resuscitation” presents information on hypothermia, submersion/drowning/near-drowning, electrical injuries, and anaphylactic emergencies. The management principles of these emergencies are similar in adults and children.

### Toxicological Emergencies

Based on data from the National Center for Health Statistics, drug-induced causes of death (eg, poisoning and overdose) are uncommon in younger children but become an important cause of death in the 15- to 24-year-old age group. Similarly, a review of cardiac arrest in children and young adults suggests that toxicological causes are important in the adolescent age group. The most important agents associated with cardiac arrest or requiring PALS are cocaine, narcotics, tricyclic antidepressants, calcium channel blockers, and β-adrenergic blockers.
The initial approach in toxicological emergencies uses basic PALS principles: assess and rapidly ensure adequate oxygenation, ventilation, and circulation. Subsequent priorities include reversing the adverse effects of the toxin, if possible, and preventing further absorption of the agent. Knowledge of the potential agent or recognition of characteristic clinical signs (toxidromes) for a particular toxin can be key to successful resuscitation. Unfortunately, since there are few well-controlled randomized trials of treatments for acute ingestions, most of the following recommendations are based on animal data and case series.

**Cocaine**

Cocaine has complex pharmacological effects, which are made more complex clinically by the varying onset, duration, and magnitude of these effects related to the route of administration and form of cocaine used.\(^{333,334}\) Cocaine binds to the reuptake pump in presynaptic nerves, blocking the uptake of norepinephrine, dopamine, epinephrine, and serotonin from the synaptic cleft. This action leads to the local accumulation of norepinephrine, dopamine, epinephrine, and serotonin at peripheral and central nervous system effects, depending on the receptors being activated. Accumulation of norepinephrine and epinephrine at \(\beta\)-adrenergic receptors results in exhilaration, hallucinations, and hyperthermia. Peripheral \(5HT\)–receptor stimulation results in coronary artery vasospasm.

The most frequent complication of cocaine use leading to hospitalization is acute coronary syndrome producing chest pain and various types of cardiac rhythm disturbances.\(^{334,335}\) Acute coronary syndrome results from the combined effects of cocaine: stimulation of \(\beta\)-adrenergic myocardial receptors increases myocardial oxygen demand, and its \(\alpha\)-adrenergic and \(5HT\) agonist actions cause coronary artery constriction, leading to ischemia. In addition, cocaine stimulates platelet aggregation,\(^{336}\) perhaps through a secondary effect from cocaine-induced increases in circulating epinephrine.\(^{337}\) Besides blocking reuptake of various amines, cocaine is a fast (ie, voltage-dependent) sodium channel inhibitor.\(^{333}\) Sodium channel blockade prolongs the action potential propagation and therefore prolongs the QRS duration and impairs myocardial contractility.\(^{333,338}\) Through the combination of adrenergic and sodium channel effects, cocaine use may cause various tachyarrhythmias, including VT and VF.

Initial treatment of the acute coronary syndrome consists of oxygen administration, continuous ECG monitoring, administration of a benzodiazepine (eg, diazepam or lorazepam; Class IIb; LOE 5, 6), and administration of aspirin and heparin.\(^{339}\) Administration of aspirin and heparin has not been evaluated in clinical trials and is based on the concept of attempting to reverse the platelet-activating effects of cocaine and biochemical manifestations of a procoagulant state. Substantial animal data shows that benzodiazepine administration is important, probably because these drugs have anticonvulsant and central nervous system–depressant effects. There is no benefit and possible harm from the use of phenothiazines and butyrophenones (eg, haloperidol). Because animal experiments also show that hyperthermia is associated with a significant increase in toxicity,\(^{341}\) aggressive cooling is indicated.

Although \(\beta\)-adrenergic blockers are a recommended treatment after myocardial ischemia in adults,\(^{342}\) they are contraindicated in the setting of cocaine intoxication (Class III; LOE 5, 6, 7). In both animal\(^{343}\) and human studies,\(^{344,345}\) the addition of a \(\beta\)-adrenergic blocker results in increased blood pressure and coronary artery constriction. These adverse pharmacological effects are produced by antagonizing cocaine-induced \(\beta\)-adrenergic receptor stimulation, which normally causes vasodilation and counteracts the cocaine-induced increased stimulation of vasoconstricting \(\alpha\)-adrenergic receptors. Although labetalol has mixed \(\alpha\)–\(\beta\)-adrenergic blocking actions, the latter dominates. This agent is not useful in the treatment of cocaine-induced acute coronary syndrome.\(^{346}\)

To reverse coronary vasoconstriction, administration of the \(\alpha\)-adrenergic blocker phentolamine may be considered but should follow oxygen, benzodiazepines, and nitroglycerin\(^{339,347}\) (Class IIb; LOE 5, 6). The optimal dose of phentolamine is not known, and there is a risk of significant hypotension and tachycardia if excessive doses are used, so doses should be titrated to effect beginning with small intravenous infusions. Additional doses are infused after documenting ongoing hypertension or evidence of myocardial ischemia. Suggested doses for hypertension are 0.05 to 0.1 mg/kg intramuscularly or intravenously in a child up to a maximum of 2.5 to 5 mg, as recommended in adults.\(^{348}\) The dose may be repeated every 5 to 10 minutes until blood pressure is controlled. Coronary vasospasm may also respond to nitroglycerin (Class IIa; LOE 5, 6).\(^{349,350}\)

Because cocaine is a sodium channel blocker, consider administration of sodium bicarbonate in a dose of 1 to 2 mEq/kg in the treatment of ventricular arrhythmias. Although controlled human data is lacking, theoretical considerations and animal data\(^{351,352}\) support this recommendation (Class IIb; LOE 5, 6, 7). Conversely, lidocaine, a local anesthetic that inhibits fast sodium channels, potentiates cocaine toxicity in animals.\(^{353}\) Nevertheless, limited clinical experience has not documented adverse effects from lidocaine administration.\(^{354}\) Therefore, lidocaine may be considered in the setting of cocaine-induced myocardial infarction (Class IIb; LOE 5, 6).

Although epinephrine may exacerbate cocaine-induced arrhythmias\(^{355,356}\) and is contraindicated in ventricular arrhythmias if VF or pulseless VT occurs (Class III; LOE 6), epinephrine may be considered to increase coronary perfusion pressure during CPR (Class Indeterminate).

**Tricyclic Antidepressants and Other Sodium Channel Blocking Agents**

Tricyclic antidepressants continue to be a leading cause of morbidity and mortality despite the increasing availability of
safer selective serotonin reuptake inhibitors for the treatment of depression. The toxic effects of tricyclic antidepressant agents result from their inhibition of fast (voltage-dependent) sodium channels in the brain and myocardium. This action is similar to that of other “membrane-stabilizing” agents (also called “quinidine-like” or “local anesthetics”). Besides tricyclic antidepressants, other sodium channel blockers include β-adrenergic blockers (particularly propranolol and sotalol), procainamide, quinidine, local anesthetics (eg, lidocaine), carbamazepine, type Ic antiarrhythmics (eg, flecainide and encainide), and cocaine (see above).338

With serious intoxication, rhythm disturbances are due to prolongation of the action potential produced by inhibition of phase 0 of the action potential, resulting in delayed conduction. This intraventricular conduction delay results in QRS prolongation (particularly the terminal 40 milliseconds357) and a QRS duration ≥100 milliseconds.358 The presence of these ECG abnormalities may be predictive of seizures and ventricular arrhythmia,359 but this predictive effect is not confirmed by all investigators.358,360 More recently an R wave in lead aVR ≥3 mm or an R wave-to-S wave ratio in lead aVR ≥0.7 was reported to be a superior predictor of serious toxicity.361,362 Tricyclic antidepressants also inhibit potassium channels, leading to prolongation of the QT interval. Through blockade of both sodium and potassium channels, high concentrations of tricyclic antidepressants (and other sodium channel blockers) may result in preterminal sinus bradycardia and heart block with junctional or ventricular wide-complex escape beats.338

Treatment of sodium channel blocker toxicity includes protecting the airway, ensuring adequate oxygenation and ventilation, continuous monitoring of the ECG, and administering sodium bicarbonate (Class IIa; LOE 5, 6, 7). Infuse sodium bicarbonate only after the airway is opened and ventilation is ensured. Sodium bicarbonate narrows the QRS complex, shortens the QT interval, and increases myocardial contractility. These actions often suppress ventricular arrhythmias and reverse hypotension.339,363 Experimental data suggests that the antiarrhythmic effect of sodium bicarbonate results from overcoming sodium channel blockade with hypertonic sodium, although the production of alkalosis per se may be important for some of these agents.363,364 Regardless of the exact mechanism, the goal is to raise the sodium concentration and arterial pH. This can be achieved by administering 1- to 2-mEq/kg bolus injections of sodium bicarbonate until the arterial pH is at least 7.45. After bolus administration, sodium bicarbonate may be infused as a solution of 150 mEq NaHCO₃ per liter in D,W titrated to maintain alkalosis. In severe intoxications, consensus recommendations are to increase the pH to a level between 7.50 and 7.55; higher pH values are not recommended because of the risk of adverse effects.338,365 The role of hyperventilation-induced alkalosis is not clear.363,366 and its benefit may be related to the specific agent ingested364, therefore, maintenance of at least normal ventilation is recommended.

If hypotension is present, administer normal saline boluses (10 mL/Kg each) in addition to sodium bicarbonate. Because tricyclic antidepressants block reuptake of norepinephrine at the neuromuscular junction, leading to catecholamine deple-

...tion, a vasopressor may be necessary to maintain adequate vascular tone and blood pressure. Norepinephrine or epinephrine can be effective; anecdotal data supports treatment with norepinephrine rather than dopamine.367,368 The superiority of norepinephrine over dopamine presumably is due to depletion of catecholamines, which will reduce the hemodynamic actions of dopamine because it is partly dependent on releasable stores of norepinephrine.196 Pure β-adrenergic agonists are contraindicated (eg, dobutamine and isoproterenol) because they may worsen hypotension by causing vasodilation. If vasopressors are insufficient to maintain blood pressure, ECMO and cardiopulmonary bypass may be effective,369,370 but they require the rapid availability of equipment and trained personnel. Early identification of at-risk patients and referral to a center capable of providing this therapy should be considered.

If ventricular arrhythmias do not respond to sodium bicarbonate, lidocaine may be considered, although some investigators argue against its use, because it is also a sodium channel blocker353 (Class IIb; LOE 6, 7). Other Class Ia (quinidine, procainamide) and Class Ic (flecainide, propafenone) antiarrhythmic agents are contraindicated because they may exacerbate the cardiac toxicity (Class III; LOE 6, 8). Class III antiarrhythmics (eg, amiodarone and sotalol) prolong the QT interval and thus also are not indicated.365

**Calcium Channel Blocker Toxicity**

The increasing use of calcium channel blockers for the treatment of hypertension and congestive heart failure makes them available for accidental or intentional overdose. Although there are 3 different classes of these agents, based on their relative effects on the myocardium and vascular smooth muscle, in the overdosed patient these selective properties are inconsequential.216 All of these agents bind to calcium channels, thereby inhibiting the influx of calcium into cells. The clinical manifestations of toxicity include bradyarrhythmias (due to inhibition of pacemaker cells and AV block) and hypotension (due to vasodilation and impaired cardiac contractility).216 Altered mental status, including syncope, seizures, and coma, may occur because of cerebral hypoperfusion.

The initial approach to therapy is to provide oxygenation and ventilation, continuously monitor the ECG, and perform frequent clinical assessments, including close monitoring of blood pressure and hemodynamic status. Consider continuous intra-arterial blood pressure monitoring in symptomatic patients. If hypotension occurs, it may respond to normal saline bolus administration in milder cases, but with more severe intoxication it is often unresponsive to fluid administration. To avoid pulmonary edema, limit fluid boluses to 5 to 10 mL/kg, with careful reassessment after each bolus because of the high frequency of myocardial dysfunction in such patients. Calcium is often infused in calcium channel blocker overdose in an attempt to overcome the channel blockade, but case reports suggest only variable effectiveness (Class IIIb; LOE 5, 6, 8).216,371 The optimal dose of calcium is unclear. If used, calcium chloride is the generally recommended salt, because it results in greater elevation of the ionized calcium...
concentration. Doses of 20 mg/kg (0.2 mL/kg) of 10% calcium chloride infused over 5 to 10 minutes may be provided, followed by infusions of 20 to 50 mg/kg per hour if a beneficial effect is observed. Ionized calcium concentrations should be monitored to limit toxicity from hypercalcemia.

High-dose vasopressor therapy (norepinephrine or epinephrine) may be considered on the basis of successful treatment of bradycardia and hypotension associated with severe calcium channel blocker toxicity (Class IIb; LOE 5). High-dose vasopressor infusions require careful monitoring of the patient and titration of the infusion rate to the desired hemodynamic effect. Animal data and a recent small case series suggest that insulin plus glucagon may be beneficial in calcium channel blocker toxicity (Class Indeterminate; LOE 5, 6). Precise dosage recommendations are unavailable. A loading dose of glucose (0.5 g/kg) may be followed by an infusion at 0.5 g/kg per hour. Following the glucose bolus, an insulin bolus of 0.5 to 1.0 U/kg is suggested, followed by 0.5 U/kg per hour. The goal is to maintain the glucose concentration between 100 and 200 mg/dL by titrating the rate of glucose administration. Presumably the beneficial effect of combined insulin-glucose therapy results from better myocardial use of glucose by activation of pyruvate dehydrogenase, which stimulates ATP production through aerobic metabolism. Careful monitoring of glucose concentration is needed to avoid hypoglycemia, the main adverse effect of this therapy. Because insulin and glucose stimulate movement of potassium from the extracellular to the intracellular space, potassium concentrations should be monitored closely, and exogenous potassium infusions are often needed.

**β-Adrenergic Blocker Toxicity**

β-Adrenergic blockers compete with norepinephrine and epinephrine at the β-adrenergic receptor, resulting in bradycardia and decreased cardiac contractility. In severe intoxication, some β-adrenergic blockers have sodium channel blocking effects as well (eg, propranolol and sotalol), leading to prolongation of the QRS and QT interval. Hypotension, usually with bradycardia, and varying degrees of heart block are common clinical manifestations of β-blocker toxicity. Altered mental status, including seizures and coma, may occur, particularly with propranolol. The initial approach to treatment includes providing adequate oxygenation and ventilation, assessing perfusion, and establishing vascular access and treating shock if present. Continuous ECG monitoring and frequent clinical reassessment are also important. To overcome the β-adrenergic blockade, epinephrine infusions may be effective, although very high infusion doses may be needed (Class Indeterminate; LOE 5, 6). On the basis of animal data and case reports, glucagon also may be considered in the treatment of β-adrenergic blocker overdose (Class IIb; LOE 5, 6). In adults and adolescents, 5 to 10 mg of glucagon may be slowly infused over several minutes, followed by an intravenous infusion of 1 to 5 mg per hour. Bolus doses of 1 mg have been used in younger children. The diluent supplied by the manufacturer contains phenol and should not be used when these large bolus doses and subsequent continuous infusions are given, because phenol may cause hypotension, seizures, or arrhythmias. If a dose ≥2 mg is needed, reconstitute the glucagon in sterile water at a final concentration <1 mg/mL.

As with calcium channel blocker overdose, glucose plus insulin also may be useful, with 1 animal study showing that it was superior to glucagon (Class Indeterminate; LOE 6). When an intravenous conduction delay is observed (ie, prolonged QRS interval), sodium bicarbonate may be used, as previously discussed.

β-Adrenergic blockade reduces cytoplasmic calcium concentration and thus reduces inotropy and chronotropy (ie, heart rate). Limited animal data and a few small clinical uncontrolled case series suggest that calcium administration may be beneficial, although other clinical reports suggest that it has no beneficial effect. Calcium may be considered if administration of glucagon and catecholamine is not effective (Class IIb; LOE 5, 6).

**Opioid Toxicity**

Narcotics produce central nervous system depression and may cause hypoventilation, apnea, and respiratory failure requiring PALS. Naloxone is an effective opioid receptor antagonist that has been used in >20 years of clinical experience, and it remains the treatment of choice to reverse narcotic toxicity (Class IIa; LOE 4, 5, 6, 7). Although naloxone administration is generally well tolerated, both animal and clinical data suggest that adverse events may occur, such as ventricular arrhythmias, acute pulmonary edema, asystole, or seizures. The opioid system and adrenergic system are interrelated; opioid antagonists stimulate sympathetic nervous system activity. Moreover, hypopercapnia stimulates the sympathetic nervous system. Animal data suggests that if ventilation is provided to normalize the partial pressure of arterial CO2, before naloxone administration, the sudden rise in epinephrine concentration and its attendant toxic effects are blunted. Thus, ventilation is recommended before the administration of naloxone (Class IIb; LOE 5, 6). The recommended dose of naloxone is 0.1 mg/kg administered intravenously, up to 2 mg in a single dose. Alternatively, to avoid sudden hemodynamic effects from opioid reversal, repeated doses of 0.01 to 0.03 mg/kg may be used. Naloxone may be administered intramuscularly, subcutaneously, or through the tracheal tube, but its onset of action via these alternative routes may be delayed, particularly if the patient is poorly perfused.

**Drowning/Submersion**

Treatment of the submersion victim requires no particular alteration from the PBLs/PALS approach. Resuscitation, particularly rescue breathing, should begin when the child is in the water. The Heimlich maneuver is not indicated before rescue breathing is begun, and it should not be performed unless foreign-body airway obstruction is suspected. The provision of prompt BLS has been linked with improved outcome following resuscitation in children. Poor prognostic indicators after submersion include a prolonged submersion interval in non-icy water, VF on initial rhythm, and absence of perfusing rhythm on arrival in the local
Emergency Department. Signs of increased intracranial pressure that develop subsequent to a submersion injury are consistent with devastating neurological insult, but there is no evidence that invasive monitoring or aggressive treatment of the increased intracranial pressure alters outcome.

**Postresuscitation Stabilization**

The postresuscitation phase begins after initial stabilization of the patient with shock or respiratory failure or after return of spontaneous circulation in a patient who was in cardiac arrest. This phase may include transport to a pediatric tertiary-care facility or intrahospital transport from the Emergency Department or ward and ongoing care in a pediatric intensive care unit. The goals of postresuscitation care are to preserve brain function, avoid secondary organ injury, seek and correct the cause of illness, and enable the patient to arrive at a tertiary-care setting in the best possible physiological state.

Care of the critically ill or injured child is complex, requiring knowledge and experience in the evaluation of all organ systems, assessment and monitoring of physiological functions, and management of multiple organ failure. Postresuscitation stabilization continues assessment and support of the ABCs (airway, breathing, and circulation) and adds attention to preservation of neurological function and avoidance of multisystem organ failure. Frequent reassessment of the patient is necessary because the patient’s hemodynamic status often deteriorates after a brief period of stability.

After stabilization of the airway and support of oxygenation, ventilation, and perfusion, a secondary survey is performed that includes the patient’s bones, joints, and skin. This survey carefully examines the patient for evidence of trauma and assesses the patient’s neurological status. The medical history (allergies, illnesses, medications, and immunizations) and serious but not life-threatening conditions (such as renal and hepatic dysfunction) are then evaluated. Details on the postresuscitation evaluation and preservation of several organ systems are reviewed below.

**Respiratory System**

After resuscitation all children should receive supplemental oxygen until adequate oxygenation is confirmed by direct PaO2 measurement or use of pulse oximetry and until adequate oxygen-carrying capacity (ie, hemoglobin concentration) is confirmed. In the postarrest setting, ongoing evidence of significant respiratory distress with agitation, poor air exchange, cyanosis, or hypoxemia requires support of oxygenation. which is usually achieved by elective intubation and mechanical ventilation. To achieve airway control so that diagnostic studies such as a CT scan can be safely performed, elective endotracheal intubation using appropriate sedation and paralysis (see “Rapid Sequence Intubation”) is sometimes used. After endotracheal intubation, tube position is assessed by clinical examination combined with a confirmatory test such as detection of exhaled CO2 (Class Ib). Ongoing confirmation of tube placement using intermittent or continuous monitoring of exhaled CO2 is also recommended (Class Ib), especially if the patient undergoes interhospital or intrahospital transport. Before patient transport, secure the tracheal tube and confirm the tube position within the trachea by clinical examination and chest x-ray if available. In both hospital and out-of-hospital settings, oxygen saturation and the cardiac rhythm and rate should be continuously monitored, and blood pressure, breath sounds, perfusion, and color should be assessed frequently in intubated patients with a perfusing rhythm.

Reevaluate tracheal tube position and patency in patients who remain agitated despite effective mechanical ventilatory support and each time the patient is moved, such as into or out of a transport vehicle. If the condition of an intubated patient deteriorates, consider several possibilities that can be recalled by the mnemonic DOPE: Displacement of the tube from the trachea, Obstruction of the tube, Pneumothorax, and Equipment failure. If tracheal tube position and patency are confirmed and mechanical ventilation failure and pneumothorax are ruled out, the presence of agitation may require analgesia for pain control (eg, fentanyl or morphine) and/or sedation for confusion, anxiety, or agitation (eg, lorazepam, midazolam, or ketamine). Occasionally, neuromuscular blocking agents (eg, vecuronium or pancuronium) combined with analgesia or sedation are needed to optimize ventilation and minimize the risk of barotrauma or accidental tube dislodgment. In the hospital, continuous capnography is helpful in mechanically ventilated patients to avoid hypventilation or hyperventilation, which may occur inadvertently during transport and diagnostic procedures. Gastric distention may also cause discomfort and interfere with ventilation; if distention develops, an orogastric or nasogastric tube should be inserted.

Initial mechanical or manual ventilation of an intubated patient should provide 100% oxygen at a typical rate of 20 to 30 breaths per minute for infants and 12 to 20 breaths per minute for older children. Provision of effective ventilation depends on the respiratory rate and tidal volume. In general, the delivered tidal volume should be just sufficient to cause the chest to rise. Occasionally, higher rates or tidal volumes may be needed if intrinsic pulmonary disease or intracranial hypertension is present. Conversely, patients with conditions involving air trapping (eg, asthma and bronchiolitis) often require lower respiratory rates to allow prolonged expiratory time. If a mechanical ventilator is being used, initial delivered tidal volumes should be 7 to 10 mL/kg, sufficient to cause visible chest expansion and audible breath sounds over the distal lung fields.

Ventilator peak inspiratory pressure should begin at 20 to 25 cm H2O and should be gradually increased until chest expansion is observed and breath sounds are adequate bilaterally. Higher inspiratory pressures may be needed in the presence of some lung diseases, but avoid peak pressures in excess of 35 cm H2O if possible. To avoid high peak pressure during volume ventilation (ie, delivering a preset volume of gas rather than a preset inspiratory pressure), inspiratory time should be at least 0.6 to 1.0 second; longer times are often useful in conditions characterized by lower-airway obstruction (such as asthma or bronchiolitis) or poor lung compliance (eg, ARDS). A positive end-expiratory pressure of 2 to 5 cm H2O is routinely provided; higher positive end-expiratory pressure may be necessary if diffuse alveolar disease or marked ventilation-perfusion mismatch associated
with hypoxemia is present. Obtain an arterial blood gas analysis after 10 to 15 minutes on the initial ventilatory settings, and make adjustments in ventilatory support accordingly. Correlating the arterial PCO₂ with end-tidal CO₂ and correlating arterial oxygen saturation with pulse oximetry are useful procedures to permit continuous monitoring of ventilation and oxygenation. Perform frequent clinical assessment of the effectiveness of ventilation by observing for agitation, cyanosis, decreased breath sounds, chest wall movement, tachycardia, and spontaneous respiratory efforts that are asynchronous with mechanical ventilation. All intubated patients should be monitored with continuous pulse oximetry.

Transcutaneous oxygen and CO₂ sensors are used in children, particularly neonates and infants, but changes in oxygenation or ventilation are not rapidly detected with these techniques. Conversely, transcutaneous monitors correlate more accurately with arterial blood PCO₂ than end-tidal detectors. Repeated clinical evaluation is crucial also because transcutaneous monitors may be inaccurate or may not function reliably, especially in the presence of hypothermia or poor perfusion.

Cardiovascular System

Persistent circulatory dysfunction is observed frequently after resuscitation from cardiac arrest. Frequent or continuous clinical evaluation is needed to detect evidence of inadequate cardiac output and shock. Maintaining adequate cardiac output and oxygen delivery to tissues is the key to preserving multorgan function. Clinical signs of inadequate systemic perfusion include decreased capillary refill, absent or decreased intensity of distal pulses, altered mental status, cool extremities, tachycardia, decreased urine output, and hypotension. Decreased cardiac output or shock may be secondary to insufficient volume resuscitation, loss of peripheral vascular tone, and/or myocardial dysfunction. Treatment of altered perfusion includes fluid resuscitation, vasoactive agents to increase or decrease vascular resistance, inotropic agents, and/or correction of hypoxia and metabolic disorders. Heart rate, blood pressure, and oximetry monitoring should be continuous, and clinical evaluation should be repeated at least every 5 minutes. Cuff blood pressure measurements may be inaccurate in the child who remains hemodynamically unstable; consider direct arterial monitoring as soon as feasible in patients with continued cardiovascular compromise. Urine output is an important indicator of splanchic organ perfusion; peripheral perfusion, heart rate, and mental status are nonspecific indicators that may be affected by ambient temperature, pain, fear, or neurological function. Blood pressure may be normal despite the presence of shock. For hemodynamically compromised patients, urine output generally should be monitored with an indwelling catheter.

Laboratory evaluation of the patient’s circulatory state includes arterial blood gas analysis and evaluation of serum electrolytes, glucose, and calcium levels. The presence of metabolic (lactic) acidosis suggests the presence of tissue hypoxia caused by hypoxemia or ischemia. If cardiac output is adequate, a repeated arterial blood gas or lactic acid measurement typically shows improved acidosis and a reduced lactate concentration. A chest x-ray may help evaluate intravascular volume; a small heart is consistent with hypovolemia and a large heart is consistent with volume overload or myocardial dysfunction. Similarly, clear lung fields are inconsistent with cardiogenic shock, whereas pulmonary edema suggests heart failure, volume overload, ARDS, or diffuse pneumonia.

Drugs Used to Maintain Cardiac Output

The following section provides general information on the use of vasoactive and inotropic agents to maintain cardiac output and blood pressure in the postarrest period or in children with compromised hemodynamics at risk of cardiac arrest (Table 3). Note that although these agents are widely used, there is no clinical data comparing agents in the postarrest period that documents an advantage for outcome of one or more agents. In addition, the pharmacokinetics and pharmacodynamics (ie, clinical response to a given infusion rate) of these agents vary from patient to patient and even from hour to hour in the same patient. Factors that influence the effects of these agents include the child’s age and maturity, underlying disease process (which influences receptor density and response), metabolic state, acid-base balance, autonomic and endocrine responses, and hepatic and renal function. Therefore, the recommended infusion doses listed below are starting points; the infusions must be adjusted according to measured patient response to achieve the desired effect.

After cardiac arrest or resuscitation from shock, the victim may have ongoing hemodynamic compromise secondary to a combination of inadequate cardiac pumping function, excessively increased systemic or pulmonary vascular resistance, or very low systemic vascular resistance. The last is most common in the patient with septic shock, although recent data shows that most children with fluid-refractory septic shock have high rather than low systemic vascular resistance and poor myocardial pumping function. Children with cardiogenic shock typically have poor myocardial function and a compensatory increase in systemic and pulmonary vascular resistance as the body attempts to maintain an adequate blood pressure.

The classes of agents used to maintain circulatory function can be divided into inotropes, vasopressors, and vasodilators. Inotropes increase cardiac pumping function and often increase heart rate as well. Vasopressors increase systemic and pulmonary vascular resistance; they are most commonly used in children with inappropriately low systemic vascular resistance. Vasodilators are designed to reduce systemic and pulmonary vascular resistance. Although they do not directly increase pumping function, vasodilators reduce ventricular afterload, which often improves stroke volume and therefore cardiac output. They are the only class of agents that can increase cardiac output and simultaneously reduce myocardial oxygen demand.

Optimal use of these agents requires knowledge of the patient’s cardiovascular physiology, which is not always clearly discerned from the clinical examination. Invasive hemodynamic monitoring, including measurement of central venous pressure, pulmonary capillary wedge pressure, and cardiac output, may be needed. Furthermore, a number of the vasoactive agents have different hemodynamic effects at different infusion rates. For example, at low infusion rates,
epinephrine is a potent inotrope and lowers systemic vascular resistance through a prominent action on vascular \( \beta \)-adrenergic receptors. At higher infusion rates, epinephrine remains a potent inotrope and increases systemic vascular resistance by activating vascular \( \alpha \)-adrenergic receptors. Since the pharmacokinetic and pharmacodynamic responses are not uniform across ages and across different diseases, careful monitoring of the patient’s response to vasoactive agents is needed for optimal use.

**Epinephrine**

An epinephrine infusion is indicated in the treatment of shock with diminished systemic perfusion from any cause that is unresponsive to fluid resuscitation. Epinephrine is a potent inotrope and typically is infused at a rate sufficient to increase systemic vascular resistance and therefore blood pressure. Epinephrine is also a potent chronotrope (ie, it increases heart rate). It may be useful in patients with hemodynamically significant bradycardia that is unresponsive to oxygenation and ventilation. Epinephrine may be preferable to dopamine in patients with marked circulatory instability, particularly in infants (see “Dopamine,” below). Infusions are prepared as listed in Table 3. The infusion is generally initiated at 0.1 to 0.3 \( \mu \)g/kg per minute and is titrated up to 1 \( \mu \)g/kg per minute based on the observed hemodynamic effects (see also Table 2). Epinephrine should be infused only into a secure intravenous line because tissue infiltration may cause local ischemia and ulceration. Epinephrine also may cause atrial or ventricular tachyarrhythmias, severe hypertension, and metabolic changes. Metabolic changes consist of hyperglycemia, increased lactate concentration, and hypokalemia.

**Dopamine**

Dopamine is an endogenous catecholamine with complex cardiovascular effects. At low infusion rates (0.5 to 2 \( \mu \)g/kg per minute), dopamine typically increases renal and splanchnic blood flow with little effect on systemic hemodynamics, although increases in blood pressure and cardiac output were observed in neonates after infusions as low as 0.5 to 1.0 \( \mu \)g/kg per minute. At infusion rates >5 \( \mu \)g/kg per minute, dopamine can result in both direct stimulation of cardiac \( \beta \)-adrenergic receptors and indirect stimulation through the release of norepinephrine stored in cardiac sympathetic nerves. Myocardial norepinephrine stores are depleted in chronic congestive heart failure and also may be diminished in infants because sympathetic nervous system myocardial

### Table 3. PALS Medications to Maintain Cardiac Output and for Postresuscitation Stabilization

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose Range</th>
<th>Comment</th>
<th>Preparation*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amrinone</td>
<td>IV/IO loading dose: 0.75–1.0 mg/kg IV over 5 minutes; may repeat 2 times</td>
<td>Inodilator</td>
<td>6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td></td>
<td>IV/IO infusion: 5–10 ( \mu )g/kg per minute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>IV/IO infusion: 2–20 ( \mu )g/kg per minute</td>
<td>Inotrope; vasodilator</td>
<td>6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Dopamine</td>
<td>IV/IO infusion: 2–20 ( \mu )g/kg per minute</td>
<td>Inotrope; chronotrope; renal and splanchnic vasodilator in lower doses; pressor in higher doses</td>
<td>6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>IV/IO infusion: 0.1–1.0 ( \mu )g/kg per minute</td>
<td>Inotrope; chronotrope; vasodilator in lower doses and pressor in higher doses</td>
<td>0.6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 0.1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>IV/IO loading dose: 1 mg/kg IV/IO infusion: 20–50 ( \mu )g/kg per minute</td>
<td>Antiarrhythmic, mild negative inotrope. Use lower infusion rate if poor cardiac output or poor hepatic function.</td>
<td>60 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 10 ( \mu )g/kg per minute or alternative premix 120 mg/100 mL at 1 to 2.5 mL/kg per hour</td>
</tr>
<tr>
<td>Milrinone</td>
<td>IV/IO loading dose: 50–75 ( \mu )g/kg IV/IO infusion: 0.5–0.75 ( \mu )g/kg per minute</td>
<td>Inodilator</td>
<td>0.6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 0.1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>IV/IO infusion: 0.1–2.0 ( \mu )g/kg per minute</td>
<td>Vasopressor</td>
<td>0.6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 0.1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Prostaglandin E1</td>
<td>IV/IO infusion: 0.05–0.1 ( \mu )g/kg per minute</td>
<td>Maintains patency of ductus arteriosus in cyanotic congenital heart disease. Monitor for apnea, hypotension, and hypoglycemia.</td>
<td>0.3 × body weight (in kg) = No. of mg diluted to total 50 mL; then 1 mL/h delivers 0.1 ( \mu )g/kg per minute</td>
</tr>
<tr>
<td>Sodium nitroprusside</td>
<td>IV/IO infusion: 1–8 ( \mu )g/kg per minute</td>
<td>Vasodilator</td>
<td>6 × body weight (in kg) = No. of mg diluted to total 100 mL; then 1 mL/h delivers 1 ( \mu )g/kg per minute</td>
</tr>
</tbody>
</table>

IV indicates intravenous; IO, intraosseous.

*Most infusions may be calculated on the basis of the “Rule of 6” as illustrated in the table. Alternatively, a standard concentration may be used to provide more dilute or more concentrated drug solution, but then an individual dose must be calculated for each patient and each infusion rate as follows: Infusion rate (mL/h) = \( \text{[weight (kg) × dose (\( \mu \)g/kg per minute) × 60 min/h]/concentration (\( \mu \)g/mL)} \). Diluent may be 5% dextrose in water, 5% dextrose in half-normal saline, normal saline, or Ringer’s lactate unless noted otherwise.
innervation is incomplete during the first months of life. In either condition the inotropic action of dopamine may be reduced. Consistent with observations in animals, dopamine tends to increase pulmonary vascular resistance in children after cardiac surgery, particularly if their pulmonary vascular resistance was elevated at baseline.

Since it possesses inotropic and vasopressor effects, dopamine is used in the treatment of circulatory shock following resuscitation or when shock is unresponsive to fluid administration and is characterized by a low systemic vascular resistance (Class Ib; LOE 5, 6, 7). Dopamine must be infused through a secure intravenous line. Infusions (Table 3) are usually begun at 2 to 5 μg/kg per minute and may be increased to 10 to 20 μg/kg per minute in an effort to improve blood pressure, perfusion, and urine output. Infusion rates exceeding 20 μg/kg per minute may result in excessive vasoconstriction and a loss of renal vasodilating effects, although as previously noted there is substantial interpatient variability in kinetics and response. If further inotropic support is needed, either epinephrine or dobutamine may be preferable to a dopamine infusion of >20 μg/kg per minute. If further vasopressor support is needed to maintain blood pressure despite high-dose dopamine infusion, norepinephrine or epinephrine is generally preferred. Although not a concern after short-term use, if dopamine infusions are used for several days it may adversely affect thyroid function by inhibiting thyrotropin-stimulating hormone release from the pituitary gland.

Dopamine infusions may produce tachycardia, vasoconstriction, and ventricular ectopy. Infiltration of dopamine into tissues can produce local tissue necrosis. Dopamine and other catecholamines are partially inactivated in alkaline solutions and therefore should not be mixed with sodium bicarbonate.

**Dobutamine Hydrochloride**

Dobutamine hydrochloride is a synthetic catecholamine with a relatively selective effect on β1-adrenergic receptors and a lesser effect on β2-adrenergic receptors. Thus, dobutamine is a relatively selective inotrope, increasing myocardial contractility and usually decreasing peripheral vascular tone. It is effective in improving cardiac output and blood pressure in neonates and children. Dobutamine may be particularly useful in the treatment of low cardiac output secondary to poor myocardial function, such as following cardiac arrest. Dobutamine is usually infused in a dose range of 2 to 20 μg/kg per minute (Tables 2 and 3). Higher infusion rates may produce tachycardia or ventricular ectopy. Pharmacokinetics and clinical responses to specific dobutamine doses vary widely among pediatric patients, so the drug must be titrated according to individual patient response.

**Norepinephrine**

Norepinephrine is the neurotransmitter released from sympathetic nerves; it is therefore a potent inotropic agent that also activates peripheral α- and β-adrenergic receptors. At the infusion rates used clinically, α-adrenergic effects predominate and result in both the beneficial and adverse effects of norepinephrine. Since it is a potent vasoconstricting agent, norepinephrine is reserved for children with low systemic vascular resistance that is unresponsive to fluid resuscitation. This is most commonly seen in children with septic shock but also may be seen in spinal shock and anaphylaxis. Although intuitive reasoning would suggest that norepinephrine will worsen renal and splanchnic perfusion secondary to its vasoconstrictive actions, clinical data in adults shows that it improves splanchnic perfusion and renal function in hypotensive patients with septic shock, particularly if combined with dobutamine. Furthermore, infusing low doses of dopamine with norepinephrine appears to increase splanchnic blood flow and urine output, providing some degree of protection from excessive vasoconstriction.

Certainly urine output and the magnitude of metabolic acidosis should be monitored carefully during a norepinephrine infusion.

Prepare norepinephrine infusions as noted in Table 3 and infuse at rates of 0.1 to 2 μg/kg per minute. Adjust the infusion rate to achieve the desired change in blood pressure and perfusion. Since norepinephrine increases systemic vascular resistance and blood pressure, its expected chronotropic effect on heart rate is reduced and the heart rate may actually decrease despite β-adrenergic stimulation. The main toxicities are hypertension, organ ischemia (including distal extremity vascular beds), and arrhythmias. Norepinephrine should be infused through a secure vascular line, preferably one that is placed centrally.

**Sodium Nitroprusside**

Sodium nitroprusside is a vasodilator that reduces tone in all vascular beds by stimulating local nitric oxide production. It has no direct effect on the myocardium when infused at therapeutic doses, but cardiac output often increases following nitroprusside administration because systemic and pulmonary vascular resistance (ie, ventricular afterload) fall. Sodium nitroprusside is indicated in the treatment of shock or low cardiac output states characterized by high vascular resistance. It is also used in the treatment of severe hypotension. Although its vasodilating action may seem to contraindicate its use in patients with low blood pressure, in cardiogenic shock the ability of sodium nitroprusside to increase stroke volume usually more than offsets the decrease in systemic vascular resistance so that blood pressure is stabilized or increased. This is seen in the following equation describing the relationship between these hemodynamic parameters: BP=CO×SVR, where BP is blood pressure, CO is cardiac output, and SVR is systemic vascular resistance. If the increase in cardiac output is proportionately larger than the fall in systemic vascular resistance induced by sodium nitroprusside (or other vasodilators), blood pressure will increase rather than decrease. If the patient is volume depleted, sodium nitroprusside is contraindicated, because hypotension is likely.

Since sodium nitroprusside is rapidly metabolized, it must be infused continuously. The drug must be prepared in dextrose in water and cannot be infused with a saline-containing solution. This may create the need for a separate infusion site. Infusions are typically started at 1 μg/kg per minute and adjusted as needed up to 8 μg/kg per minute. Nitroprusside undergoes metabolism by endothelial cells and red blood cells, releasing nitric oxide and cyanide. The latter is rapidly metabolized in the liver to thiocyanate, provided that hepatic function is adequate. High infusion rates or diminished hepatic function may exceed the ability of the liver to metabolize cyanide, resulting in clinical
Inodilators

This class of agents combines inotropic stimulation of the heart with vasodilation of the systemic and pulmonary vascular beds. The agents currently available are amrinone and milrinone. Unlike catecholamines, inodilators do not depend on activation of receptors. Instead, these agents inhibit phosphodiesterase type III, which results in an increase in the intracellular concentration of cAMP. In the myocardium, cAMP acts as a second messenger increasing cardiac contractility; heart rate is increased to a lesser extent because phosphodiesterase type III is more prevalent in myocytes and vascular smooth muscle than it is in the pacemaker cells of the heart. Indeed, the action of inodilators is most notable in vascular smooth muscle, so this class of agents acts much like a combination of sodium nitroprusside and a selective inotrope such as dobutamine.

Inodilators are used to treat children with myocardial dysfunction and increased systemic or pulmonary vascular resistance. They are used for conditions such as congestive heart failure in postoperative cardiac surgical patients or patients with dilated cardiomyopathy and even in selected children with septic shock and myocardial dysfunction with a combination of sodium nitroprusside and a selective inotrope such as dobutamine.

Amrinone is given as a loading dose of approximately 0.75 to 1 mg/kg over 5 minutes. If the patient tolerates the load, it may be repeated up to 2 times to a total load of 3 mg/kg followed by an infusion of 5 to 10 μg/kg per minute. There is a 6-fold variation in amrinone pharmacokinetics in children, making it difficult to predict the optimal infusion rate. In infants <4 weeks of age and in patients with renal dysfunction, amrinone clearance will be low, leading to a greater risk of toxicity. If hypotension occurs during the loading dose, give 5 to 10 mL/kg of normal saline or other appropriate fluid and position the patient flat or head down if the patient can tolerate this position. If the patient remains hypotensive despite fluid loading, then a vasopressor agent needs to be used, and no further loading of amrinone should be given. For short-term stabilization, the patient may be treated with just a loading dose without an infusion. If the patient’s renal function is more severely affected than recognized initially, the amrinone concentration will accumulate during an infusion, resulting in excessive vasodilation and hypotension that may not present until ≥12 to 24 hours after the initiation of an amrinone infusion. The other major side effect of amrinone is increased platelet destruction, so the platelet count should be checked every 12 to 24 hours when starting an amrinone infusion.

Milrinone is a newer inodilator agent that is also cleared by the kidney, but because it has a shorter half-life than amrinone, it is often preferred. Milrinone also has less effect on platelets. Milrinone has been used in children to increase cardiac output and decrease systemic vascular resistance in septic shock; these effects require that the patient is adequately fluid resuscitated and has an elevated systemic vascular resistance. Based on pharmacokinetic data, milrinone initially is given as a bolus of 50 to 75 μg/kg followed by an infusion of 0.5 to 0.75 μg/kg per minute.

Neurological Preservation

Central nervous system dysfunction may either contribute to or result from a cardiac arrest. The key to preserving neurological function is the rapid restoration and maintenance of adequate oxygen delivery to the brain and avoidance of secondary injury to the neurons. Therefore, if there is evidence of significant central nervous system depression that may prevent adequate airway protection or respiratory drive, intubation and controlled ventilation are recommended. Data does not support the routine use of hyperventilation in brain-injured patients. Indeed, data suggests that hyperventilation may impair neurological outcome, most likely because of a combination of adverse effects on cardiac output, cerebral venous return, and cerebral vascular tone.

Recent data suggests that postarrest or postischemia hypothermia (core temperatures of 33°C to 36°C) may have beneficial effects on neurological function. There is insufficient data, however, to recommend the routine application of hypothermia (Class Indeterminate), but postarrest patients with core temperatures <37.5°C should not be actively rewarmed (Class IIb) unless the core temperature is <33°C, in which case they should be rewarmed to 34°C (Class IIb). Conversely, increased core temperature increases metabolic demand by 10% to 13% for each degree Celsius increase in temperature above normal. Since increasing metabolic demand may worsen neurological injury, it is not surprising that the presence of fever following brain injury is associated with worsened neurological outcome in adults with cerebral ischemia. In the brain-injured patient or in the postarrest patient with compromised cardiac output, correct toxicity. Furthermore, the hepatic metabolite thiocyanate must be renally excreted. In patients with poor renal function, thiocyanate may accumulate, leading to central nervous system dysfunction that ranges from irritability to seizures, abdominal pain, nausea, and vomiting. Thiocyanate levels should be measured in patients receiving prolonged sodium nitroprusside infusions, particularly if the infusion rate exceeds 2 μg/kg per minute.
hyperthermia with active cooling to achieve a normal core temperature (Class IIa; LOE 5, 6, 7). Prevent shivering because it will increase metabolic demand. Sedation may be adequate to control shivering, but neuromuscular blockade may be needed. Seizures may occur at any time after a significant hypoxic-ischemic insult to the brain, such as that following a cardiac arrest. If seizures occur, search for a correctable metabolic cause such as hypoglycemia or an electrolyte disturbance. Because seizures greatly increase cerebral metabolic demand at a time when cerebral blood flow may be compromised, aggressive treatment of these postischemia seizures is indicated. Initial control of the seizures is typically best achieved with the use of a benzodiazepine such as lorazepam, diazepam, or midazolam. Although the concept seems rational, there is no clinical data supporting the routine administration of an antiepileptic to prevent postarrest seizures. Conversely, if the postarrest or head-injured patient requires neuromuscular blockade, a cerebral function monitor is needed to detect seizure activity. If a cerebral function monitor is unavailable, the patient may be loaded with an anticonvulsant such as phenytoin, fosphenytoin, or phenobarbital in an attempt to prevent unrecognized seizures and further brain injury.

Renal System
Decreased urine output (<1.0 mL/kg per hour in infants and children or <30 mL per hour in adolescents) in the postresuscitation period may result from prerenal causes (such as dehydration and inadequate systemic perfusion), renal ischemic damage, or a combination of these conditions. Determine baseline serum urea nitrogen and creatinine values as soon as possible. Volume depletion may be treated with additional fluid administration (see “Intravascular Fluids”). Treat myocardial dysfunction with vasoactive drug therapy as described in the drug section. Nephrotoxic and renally excreted medications should be avoided or administered cautiously until renal status is determined. For example, pancuronium administration may result in very prolonged neuromuscular blockade, because it is renally excreted.

Gastrointestinal System
If bowel sounds are absent, abdominal distention is present, or the patient requires mechanical ventilation, an orogastric or nasogastric tube should be inserted to prevent or treat gastric distention. Blind nasogastric tube placement is contraindicated in the patient with serious facial trauma or basilar skull fracture because intracranial tube migration may result.328

General Postresuscitation Care
Once the patient’s cardiopulmonary status is stable, change intraosseous lines to intravenous ones and secure all intravenous lines. Splint any apparent fractures. The underlying cause of the arrest (infection, ingestion, etc) should be treated if known. Because hypoglycemia and hypothermia are frequently observed, monitor serum glucose level and core body temperature frequently and take corrective measures as needed. Recommended guidelines for treatment (Table 4) and equipment (Table 5) for stabilization of seriously ill or injured children may be consulted.429

<table>
<thead>
<tr>
<th>TABLE 4. Summary of Postresuscitation Care</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intervention</td>
</tr>
<tr>
<td>Airway</td>
</tr>
<tr>
<td>Tracheal intubation with confirmation of tube position and repeat confirmation on movement/transport</td>
</tr>
<tr>
<td>Secure tube before transport</td>
</tr>
<tr>
<td>Gastric decompression</td>
</tr>
<tr>
<td>Breathing</td>
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<tr>
<td>100% inspired oxygen</td>
</tr>
<tr>
<td>Provide mechanical ventilation targeting normal ventilation goals (P&lt;sub&gt;CO&lt;/sub&gt;&lt;sub&gt;2&lt;/sub&gt;: 35 to 40 mm Hg)</td>
</tr>
<tr>
<td>Monitor continuous pulse oximetry and exhaled CO&lt;sub&gt;2&lt;/sub&gt; (or capnography) if available</td>
</tr>
<tr>
<td>Circulation</td>
</tr>
<tr>
<td>Ensure adequate intravascular volume (volume titration)</td>
</tr>
<tr>
<td>Optimize myocardial function and systemic perfusion (inotropes, vasopressors, vasodilators)</td>
</tr>
<tr>
<td>Monitor capillary refill, blood pressure, continuous ECG, urine output; measure arterial blood gas and lactate to assess degree of acidosis, if available</td>
</tr>
<tr>
<td>Ideally maintain 2 routes of functional vascular access</td>
</tr>
<tr>
<td>Disability</td>
</tr>
<tr>
<td>Perform rapid secondary survey including brief neurological assessment</td>
</tr>
<tr>
<td>Avoid hyperglycemia, treat hypoglycemia (monitor glucose)</td>
</tr>
<tr>
<td>If seizures are observed, medicate with anticonvulsant agents</td>
</tr>
<tr>
<td>Obtain laboratory studies (if available): arterial blood gases, glucose, electrolytes, hematocrit, chest radiograph</td>
</tr>
<tr>
<td>Exposure</td>
</tr>
<tr>
<td>Avoid and correct hypothermia (monitor temperature)</td>
</tr>
<tr>
<td>Avoid profound hypothermia &lt;33°C</td>
</tr>
</tbody>
</table>

Be sure to communicate interventions and status of the patient to family and to transport and receiving providers.

Interhospital Transport
Ideally, postresuscitation care is provided by trained medical personnel in specialized pediatric intensive care units. Transportation to these units should be coordinated with the receiving unit to ensure that the child is safely delivered to a pediatric tertiary-care facility in stable or improved condition.430 To reduce the likelihood of complications during transport, the transport team members preferably should receive training and experience in the care of critically ill and injured children29,431 and should be supervised by a physician with experience and training in pediatric emergency medicine or pediatric critical care. The mode of transport as well as the composition of the team should be established for each EMS system, based on the care required by an individual patient.432 In general, if a pediatric and adult team are available within the same time frame, the pediatric team is preferred. The weather, distance, and the patient’s condition will determine the selection of surface ambulance, fixed-wing aircraft, or helicopter. Equipment that should be available for transport of children is listed in Table 6.

Family Presence During Resuscitation
According to surveys in the United States and the United Kingdom,433–438 most family members would like to be present during the attempted resuscitation of a loved one. Parents and providers of care for chronically ill children are often knowledgeable about and comfortable with medical equipment and emergency procedures. Family members with
no medical background report that being at the side of a loved one and saying goodbye during the final moments of life are extremely comforting. Parents or family members often fail to ask if they can be present, but healthcare providers should offer the opportunity whenever possible. Family members present during resuscitation report that it helped their adjustment to the death of the loved one, and most indicate they would participate again. Standardized psychological examinations suggest that family members present during resuscitation show less anxiety and depression and more constructive grieving behavior than family members not present during the resuscitation.

When family members are present during resuscitative efforts, resuscitation team members should be sensitive to the presence of the family member. When family members are present during an in-hospital resuscitation, if possible one person should remain with the family member to answer questions, clarify information, and provide comfort.

**TABLE 5. Suggested Equipment, Supplies, and Drugs for EMS ALS Responders**

<table>
<thead>
<tr>
<th>Equipment</th>
<th>Supplies</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Backboard and spine board (preferably with head well)</td>
<td>Arm boards (6, 8, and 15 in)</td>
<td>Albuterol in 2.5-mg unit doses, or equivalent bronchodilator for inhalation</td>
</tr>
<tr>
<td>Blood pressure cuffs in newborn, infant, child, and adult sizes</td>
<td>*Tracheal tubes, cuffed (6.0, 6.5, 7.0, 7.5, and 8.0 mm)</td>
<td>Adenosine</td>
</tr>
<tr>
<td>Exhaled CO2 monitor</td>
<td>*Tracheal tubes, uncuffed (2.5, 3.0, 3.5, 4.0, 4.5, 5.0, 5.5 mm)</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>Semirigid cervical collars in several pediatric sizes</td>
<td>Intraosseous infusion needles</td>
<td>Atropine sulfate</td>
</tr>
<tr>
<td>*Laryngoscope with straight blades, Nos. 0, 1, 2, and 3 (curved blades may be used in size 2 and 3)</td>
<td>Minidrip intravenous burettes</td>
<td>Benzodiazepine (for seizure control; eg, diazepam, lorazepam, midazolam)</td>
</tr>
<tr>
<td>Monitor/defibrillator (including small paddles/electrodes optional)</td>
<td>Nasal cannulas in infant and child sizes</td>
<td>Calcium chloride or calcium gluconate</td>
</tr>
<tr>
<td>External pacemaker</td>
<td>Extraoral saline or lactated Ringer’s solution</td>
<td>Dopamine</td>
</tr>
<tr>
<td>Oxygen source</td>
<td>Oral airways, 0–5</td>
<td>Diphenhydramine</td>
</tr>
<tr>
<td>Pediatric self-inflating bag-mask resuscitator with newborn, infant, and child masks</td>
<td>Over-the-needle catheters (24-, 22-, 20-, 18-, 16-, 14-gauge)</td>
<td>Epinephrine 1 mg/mL (1:1000)</td>
</tr>
<tr>
<td>Pediatric femur splint</td>
<td>Pediatric nonrebreathing mask</td>
<td>Epinephrine 1 mg/10 mL (1:10 000)</td>
</tr>
<tr>
<td>Pulse oximeter</td>
<td>Prostaglandin E1 (optional)</td>
<td>Flumazenil (benzodiazepine antagonist) (optional)</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>Sodium bicarbonate</td>
<td>Glucose 50% and/or 25% and/or 10%</td>
</tr>
<tr>
<td>Stiff cervical collars in infant and child sizes</td>
<td>Steroid (eg, dexamethasone, methylprednisolone)</td>
<td>Glucagon</td>
</tr>
<tr>
<td>Tracheal tube placement confirmation devices</td>
<td></td>
<td>Insulin</td>
</tr>
<tr>
<td>Standard precautions equipment</td>
<td></td>
<td>Lidocaine</td>
</tr>
<tr>
<td>Portable suction capability</td>
<td></td>
<td>Magnesium sulfate</td>
</tr>
<tr>
<td><strong>Equipment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Supplies</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Drugs</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TABLE 5. Suggested Equipment, Supplies, and Drugs for EMS ALS Responders</strong></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Termination of Resuscitative Efforts**

Despite the best efforts of healthcare providers, most children experiencing a cardiac arrest will not survive. There may be transient return of spontaneous circulation, with death occurring subsequently in the intensive care unit. Alternatively, some children will not respond to prolonged efforts. If a child fails to respond to at least 2 doses of epinephrine with a return of spontaneous circulation, the child is unlikely to survive. In the absence of recurring or refractory VF or VT, history of a toxic drug exposure, or a primary hypothermic insult, resuscitative efforts may be discontinued if there is no return of spontaneous circulation despite ALS interventions. In general, this requires no more than 30 minutes. Further discussion on the ethics of resuscitation is contained in Part 2.
TABLE 6. Suggested Equipment for Pediatric Transport

<table>
<thead>
<tr>
<th>ALS</th>
<th>BLS</th>
<th>Pediatric Kit</th>
</tr>
</thead>
<tbody>
<tr>
<td>x</td>
<td>x</td>
<td>Standard precaution equipment</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Oxygen source</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Neontal-infant masks</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric self-inflating bag-mask resuscitator</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric masks in 3 sizes</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Nasal cannulas, infant and child</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Oral airways, 00-5</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>*Tracheal tubes, uncuffed 2.5 to 5.5 mm and cuffed 6.0 to 8.0 mm</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Intubating stylet, 6F</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Bulb syringe</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Suction catheters (6F, 8F, 10F, 12F, and 14F)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>*Laryngoscope blades, straight Nos. 0, 1, 2, and 3 and curved Nos. 2, 3, and 4 (optional)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Blood pressure cuffs, infant and child</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Buretrol (Metriset)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Over-the-needle catheters, 24- to 16-gauge</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Butterfly cannulas, 23- to 19-gauge</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Tourniquets, infant and child</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Intravenous needles, 18- to 15-gauge</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric Magill forceps</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric defibrillator paddles/electrodes</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric ECG electrodes</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric traction splint</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Cervical immobilization devices (eg, semirigid collars, wedge)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Extrication device short board</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Swaddler or immobilization device</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Cord clamps</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Point-of-care glucose analysis capability</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Gastric decompression tube, 8F to 16F</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Meconium aspirator</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pulse oximeter and transport ECG monitor</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Tracheal tube placement confirmation equipment (CO₂ detection/isophagel detector)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Stethoscope</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Portable suction device</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Obstetric pack</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Thermal blanket</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Water-soluble lubricant</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Infant car seat</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Nasopharyngeal airways (18F to 34F)</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Glasgow Coma Scale score reference</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Pediatric trauma score reference</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Hand-held nebulizer</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Length-weight-based drug dosing reference</td>
</tr>
<tr>
<td>x</td>
<td>x</td>
<td>Resuscitation drugs and IV fluids that meet local standards of care</td>
</tr>
</tbody>
</table>

*If within the scope of trained providers practice.

Future Directions

These guidelines more clearly indicate the quality of evidence for our recommendations than heretofore. There are few recommendations that have sufficient evidence to merit a Class IIa status, much less a Class I status. This observation represents an opportunity and a call to action to obtain better information to guide future guideline developers. Since the rate of cardiac arrest in infants and children is relatively low, a single center is unlikely to gather sufficient data to answer some of the important questions. Instead, a multi-institutional effort is needed to collect data using a consistent set of definitions. The AHA is sponsoring the development of a National Registry for Cardiopulmonary Resuscitation (NRCPR) that should help address this need for data. We encourage widespread participation, which will lead to improved evidence-based guidelines.

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Part 10: Pediatric Advanced Life Support


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Part 10: Pediatric Advanced Life Support

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