Part 12: Pediatric Advanced Life Support

Web-based Integrated 2010 & 2015 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Key Words: arrhythmia cardiopulmonary resuscitation pediatrics

1 Highlights & Introduction

1.1 Highlights

Summary of Key Issues and Major Changes

Many key issues in the review of the pediatric advanced life support literature resulted in refinement of existing recommendations rather than in new recommendations. New information or updates are provided about fluid resuscitation in febrile illness, atropine use before tracheal intubation, use of amiodarone and lidocaine in shock-refractory VF/pVT, TTM after resuscitation from cardiac arrest in infants and children, and post–cardiac arrest management of blood pressure.

- In specific settings, when treating pediatric patients with febrile illnesses, the use of restrictive volumes of isotonic crystalloid leads to improved survival. This contrasts with traditional thinking that routine aggressive volume resuscitation is beneficial.
- Routine use of atropine as a premedication for emergency tracheal intubation in non-neonates, specifically to prevent arrhythmias, is controversial. Also, there are data to suggest that there is no minimum dose required for atropine for this indication.
- If invasive arterial blood pressure monitoring is already in place, it may be used to adjust CPR to achieve specific blood pressure targets for children in cardiac arrest.
- Amiodarone *or* lidocaine is an acceptable antiarrhythmic agent for shock-refractory pediatric VF and pVT in children.
- Epinephrine continues to be recommended as a vasopressor in pediatric cardiac arrest.
- For pediatric patients with cardiac diagnoses and IHCA in settings with existing extracorporeal membrane oxygenation protocols, ECPR may be considered.
- Fever should be avoided when caring for comatose children with ROSC after OHCA. A large randomized trial of therapeutic hypothermia for children with OHCA showed no difference in outcomes whether a period of moderate therapeutic hypothermia (with temperature maintained at 32°C to 34°C) or the strict maintenance of normothermia (with temperature maintained 36°C to 37.5°C) was provided.
- Several intra-arrest and post–cardiac arrest clinical variables were examined for prognostic significance. No single variable was identified to be sufficiently reliable to predict outcomes. Therefore, caretakers should consider multiple factors in trying to predict outcomes during cardiac arrest and in the post-ROSC setting.
- After ROSC, fluids and vasoactive infusions should be used to maintain a systolic blood pressure above the fifth percentile for age.
- After ROSC, normoxemia should be targeted. When the necessary equipment is available, oxygen administration should be weaned to target an oxyhemoglobin saturation of 94% to 99%. Hypoxemia should be strictly avoided. Ideally, oxygen should be titrated to a value appropriate to the specific patient condition. Likewise, after ROSC, the child's $Paco_2$ should be targeted to a level appropriate to each patient's condition. Exposure to severe hypercapnia or hypocapnia should be avoided.

Recommendations for Fluid Resuscitation

2015 (New): Early, rapid IV administration of isotonic fluids is widely accepted as a cornerstone of therapy for septic shock. Recently, a large randomized controlled trial of fluid resuscitation conducted in children with severe febrile illnesses in a resource-limited setting found worse outcomes to be associated with IV fluid boluses. For children in shock, an initial fluid bolus of 20 mL/kg is reasonable. However, for children with febrile illness in settings with limited access to critical care resources (ie, mechanical ventilation and inotropic support), administration of bolus IV fluids should be undertaken with extreme caution, as it may be harmful. Individualized

treatment and frequent clinical reassessment are emphasized.

Why: This recommendation continues to emphasize the administration of IV fluid for children with septic shock. Additionally, it emphasizes individualized treatment plans for each patient, based on frequent clinical assessment before, during, and after fluid therapy is given, and it presumes the availability of other critical care therapies. In certain resource-limited settings, excessive fluid boluses given to febrile children may lead to complications where the appropriate equipment and expertise might not be present to effectively address them.

Atropine for Endotracheal Intubation

2015 (Updated): There is no evidence to support the *routine* use of atropine as a premedication to prevent bradycardia in emergency pediatric intubations. It may be considered in situations where there is an increased risk of bradycardia. There is no evidence to support a minimum dose of atropine when used as a premedication for emergency intubation.

2010 (Old): A minimum atropine dose of 0.1 mg IV was recommended because of reports of paradoxical bradycardia occurring in very small infants who received low doses of atropine.

Why: Recent evidence is conflicting as to whether atropine prevents bradycardia and other arrhythmias during emergency intubation in children. However, these recent studies did use atropine doses less than 0.1 mg without an increase in the likelihood of arrhythmias.

Invasive Hemodynamic Monitoring During CPR

2015 (Updated): If invasive hemodynamic monitoring is in place at the time of a cardiac arrest in a child, it may be reasonable to use it to guide CPR quality.

2010 (Old): If the patient has an indwelling arterial catheter, the waveform can be used as feedback to evaluate hand position and chest compression depth. Compressing to a specific systolic blood pressure target has not been studied in humans but may improve outcomes in animals.

Why: Two randomized controlled trials in animals found improvements in ROSC and survival to completion of the experiment when CPR technique was adjusted on the basis of invasive hemodynamic monitoring. This has yet to be studied in humans.

Antiarrhythmic Medications for Shock-Refractory VF or Pulseless VT

2015 (Updated): Amiodarone or lidocaine is equally acceptable for the treatment of shock refractory VF or pVT in children.

2010 (Old): Amiodarone was recommended for shock-refractory VF or pVT. Lidocaine can be given if amiodarone is not available.

Why: A recent, retrospective, multi-institution registry of in-patient pediatric cardiac arrest showed that, compared with amiodarone, lidocaine was associated with higher rates of ROSC and 24-hour survival. However, neither lidocaine nor amiodarone administration was associated with improved survival to hospital discharge.

Vasopressors for Resuscitation

2015 (Updated): It is reasonable to give epinephrine during cardiac arrest.

2010 (Old): Epinephrine should be given for pulseless cardiac arrest.

Why: The recommendation about epinephrine administration during cardiac arrest was downgraded slightly in Class of Recommendation. There are no high-quality pediatric studies showing the effectiveness of any vasopressors in cardiac arrest. Two pediatric observational studies were inconclusive, and 1 randomized, out-of-hospital adult study found that epinephrine was associated with improved ROSC and survival to hospital admission but not to hospital discharge.

ECPR Compared With Standard Resuscitation

2015 (Updated): ECPR may be considered for children with underlying cardiac conditions who have an IHCA,

provided appropriate protocols, expertise, and equipment are available.

2010 (Old): Consider early activation of extracorporeal life support for a cardiac arrest that occurs in a highly supervised environment, such as an intensive care unit, with the clinical protocols in place and the expertise and equipment available to initiate it rapidly. Extracorporeal life support should be considered only for children in cardiac arrest refractory to standard resuscitation attempts, with a potentially reversible cause of arrest.

Why: OHCA in children was not considered. For pediatric IHCA, there was no difference in overall survival comparing ECPR to CPR without extracorporeal membrane oxygenation. One retrospective registry review showed better outcome with ECPR for patients with cardiac disease than for those with noncardiac disease.

Targeted Temperature Management

2015 (Updated): For children who are comatose in the first several days after cardiac arrest (in-hospital or out-ofhospital), temperature should be monitored continuously and fever should be treated aggressively.

For comatose children resuscitated from OHCA, it is reasonable for caretakers to maintain either 5 days of normothermia (36°C to 37.5°C) or 2 days of initial continuous hypothermia (32°C to 34°C) followed by 3 days of normothermia.

For children remaining comatose after IHCA, there are insufficient data to recommend hypothermia over normothermia.

2010 (Old): Therapeutic hypothermia (32°C to 34°C) may be considered for children who remain comatose after resuscitation from cardiac arrest. It is reasonable for adolescents resuscitated from witnessed out-of-hospital VF arrest.

Why: A prospective, multicenter study of pediatric OHCA victims randomized to receive either therapeutic hypothermia (32°C to 34°C) or normothermia (36°C to 37.5°C) showed no difference in functional outcome at 1 year between the 2 groups. This and other observational studies demonstrated no additional complications in the group treated with therapeutic hypothermia. Results are currently pending from a large, multicenter, randomized controlled trial of therapeutic hypothermia for patients who are comatose after ROSC following pediatric IHCA (see Therapeutic Hypothermia After Pediatric Cardiac Arrest website: www.THAPCA.org.

Intra-arrest and Postarrest Prognostic Factors

2015 (Updated): Multiple factors should be considered when trying to predict outcomes of cardiac arrest. Multiple factors play a role in the decision to continue or terminate resuscitative efforts during cardiac arrest and in the estimation of potential for recovery after cardiac arrest.

2010 (Old): Practitioners should consider multiple variables to prognosticate outcomes and use judgment to titrate efforts appropriately.

Why: No single intra-arrest or post–cardiac arrest variable has been found that reliably predicts favorable or poor outcomes

Post–Cardiac Arrest Fluids and Inotropes

2015 (New): After ROSC, fluids and inotropes/vasopressors should be used to maintain a systolic blood pressure above the fifth percentile for age. Intra-arterial pressure monitoring should be used to continuously monitor blood pressure and identify and treat hypotension.

Why: No studies were identified that evaluated specific vasoactive agents in post-ROSC pediatric patients. Recent observational studies found that children who had post-ROSC hypotension had worse survival to hospital discharge and worse neurologic outcome.

Post–Cardiac Arrest Pao_2 and $Paco_2$

2015 (Updated): After ROSC in children, it may be reasonable for rescuers to titrate oxygen administration to achieve normoxemia (oxyhemoglobin saturation of 94% or above). When the requisite equipment is available, oxygen should be weaned to target an oxyhemoglobin saturation within the range of 94% to 99%. The goal should be to strictly avoid hypoxemia while maintaining normoxemia. Likewise, post-ROSC ventilation strategies

in children should target a $PaCO_2$ that is appropriate for each patient while avoiding extremes of hypercapnia or hypocapnia.

2010 (Old): Once circulation is restored, if appropriate equipment is in place, it may be reasonable to wean the fraction of inspired oxygen to maintain an oxyhemoglobin saturation of 94% or greater. No recommendations were made about $PaCO_2$.

Why: A large observational pediatric study of IHCA and OHCA found that normoxemia (defined as PaO_2 60 to 300 mm Hg) was associated with improved survival to pediatric intensive care unit discharge, compared with hyperoxemia (PaO_2 greater than 300 mm Hg). Adult and animal studies show increased mortality associated with hyperoxemia. Likewise, adult studies after ROSC demonstrate worse patient outcomes associated with hypocapnia.

1.2 Introduction - Updated

These *Web-based Integrated Guidelines* incorporate the relevant recommendations from 2010 and the new or updated recommendations from 2015.

Over the past 13 years, survival to discharge from pediatric inhospital cardiac arrest (IHCA) has markedly improved. From 2001 to 2013, rates of return of spontaneous circulation (ROSC) from IHCA increased significantly from 39% to 77%, and survival to hospital discharge improved from 24% to 36% to 43% (Girotra et al and personal communication with Paul Chan, MD, MSc, April 3, 2015). In a single center, implementation of an intensive care unit (ICU)–based interdisciplinary debriefing program improved survival with favorable neurologic outcome from 29% to 50%.[1] Furthermore, new data show that prolonged cardiopulmonary resuscitation (CPR) is not futile: 12% of patients receiving CPR in IHCA for more than 35 minutes survived to discharge, and 60% of the survivors had a favorable neurologic outcome.[2] This improvement in survival rate from IHCA can be attributed to multiple factors, including emphasis on high-quality CPR and advances in post-resuscitation care. Over the past decade, the percent of cardiac arrests occurring in an ICU setting has increased (87% to 91% in 2000 to 2003 to 94% to 96% in 2004 to 2010).[3] While rates of survival from pulseless electrical activity and asystole have increased, there has been no change in survival rates from in-hospital ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT).

Conversely, survival from out-of-hospital cardiac arrest (OHCA) has not improved as dramatically over the past 5 years. Data from 11 US and Canadian hospital emergency medical service systems (the Resuscitation Outcomes Consortium) during 2005 to 2007 showed age-dependent discharge survival rates of 3.3% for infants (less than 1 year), 9.1% for children (1 to 11 years), and 8.9% for adolescents (12 to 19 years).[4] More recently published data (through 2012) from this network demonstrate 8.3% survival to hospital discharge across all age groups, with 10.5% survival for children aged 1 to 11 years and 15.8% survival for adolescents aged 12 to 18 years.[5]

2 Evidence Evaluation Process Informing The Guidelines Update - Updated

The American Heart Association (AHA) Emergency Cardiovascular Care (ECC) Committee uses a rigorous process to review and analyze the peer-reviewed published scientific evidence supporting the AHA Guidelines for CPR and ECC, including this update. In 2000, the AHA began collaborating with other resuscitation councils throughout the world, via the International Liaison Committee on Resuscitation (ILCOR), in a formal international process to evaluate resuscitation science. This process resulted in the publication of the International Consensus on CPR and ECC Science With Treatment Recommendations (CoSTR) in 2005 and 2010 [6,7] These publications provided the scientific support for AHA Guidelines revisions in those years.

In 2011, the AHA created an online evidence review process, the Scientific Evidence Evaluation and Review System (SEERS), to support ILCOR systematic reviews for 2015 and beyond. This new process includes the use of Grading of Recommendations Assessment, Development, and Evaluation (GRADE) software to create systematic reviews that will be available online and used by resuscitation councils to develop their guidelines for CPR and ECC. The drafts of the online reviews were posted for public comment, and ongoing reviews will be accessible to the public (https://volunteer.heart.org/ apps/pico/Pages/default.aspx).

The AHA process for identification and management of potential conflicts of interest was used, and potential conflicts for writing group members are listed at the end of each Part of the *2015 AHA Guidelines Update for CPR and ECC*. For additional information about this systematic review or management of the potential conflicts of interest, see “Part 2: Evidence Evaluation and Management of Conflicts of Interest” in this supplement and the

related article "Part 2: Evidence Evaluation and Management of Conflict of Interest" in the 2015 CoSTR publication.[8,9]

This update to the *2010 AHA Guidelines for CPR and ECC* for pediatric advanced life support (PALS) targets key questions related to pediatric resuscitation. Areas of update were selected by a group of international pediatric resuscitation experts from ILCOR, and the questions encompass resuscitation topics in prearrest care, intra-arrest care, and postresuscitation care. The ILCOR Pediatric Life Support Task Force experts reviewed the topics addressed in the 2010 Guidelines for PALS and, based on in-depth knowledge of new research developments, formulated 18 questions for further systematic evaluation.[10] Three questions that address pediatric basic life support appear in "Part 11: Pediatric Basic Life Support and Cardiopulmonary Resuscitation Quality."

Beginning with the publication of the 2015 CoSTR, the ILCOR evidence evaluation process will be continuous, rather than "batched" into 5-year cycles. The goal of this continuous evidence review is to improve survival from cardiac arrest by shortening the time between resuscitation science discoveries and their application in resuscitation practice. As additional resuscitation topics are prioritized and reviewed, these Guidelines may be updated again. When the evidence supports sufficient changes to the Guidelines or a change in sequence or treatments that must be woven throughout the Guidelines, then the Guidelines will be revised completely.

Because the *2015 AHA Guidelines Update for CPR and ECC* represents the first update to the previous Guidelines, recommendations from both this 2015 Guidelines Update and the 2010 Guidelines are contained in the Appendix. If the 2015 ILCOR review resulted in a new or significantly revised Guidelines recommendation, that recommendation will be labeled as *New or Updated*.

As with all AHA Guidelines, each 2015 recommendation is labeled with a Class of Recommendation (COR) and a Level of Evidence (LOE). This update uses the newest AHA COR and LOE classification system, which contains modifications of the Class III recommendation and introduces LOE B-R (randomized studies) and B-NR (nonrandomized studies) as well as LOE C-LD (limited data) and LOE C-EO (consensus of expert opinion).

These PALS recommendations are informed by the rigorous systematic review and consensus recommendations of the ILCOR Pediatric Task Force, and readers are referred to the complete consensus document in the 2015 CoSTR.[11,12] In the online version of this document, live links are provided so the reader can connect directly to the systematic reviews on the SEERS website. These links are indicated by a superscript combination of letters and numbers (eg, Peds 397). We encourage readers to use the links and review the evidence and appendixes, including the GRADE tables.

The 2015 Guidelines Update for PALS includes science review in the following subjects:

Prearrest Care

• Effectiveness of **medical emergency teams or rapid response teams** to improve outcomes

• Effectiveness of a **pediatric early warning score (PEWS)** to improve outcomes

• **Restrictive volume of isotonic crystalloid** for resuscitation from septic shock

• **Use of atropine as a premedication** in infants and children requiring emergency tracheal intubation

• Treatment for infants and children with **myocarditis or dilated cardiomyopathy and impending cardiac arrest**

Intra-arrest Care

• Effectiveness of **extracorporeal membrane oxygenation (ECMO) resuscitation** compared to standard resuscitation without ECMO

• Targeting a **specific end-tidal CO_2 ($ETCO_2$) threshold** to improve chest compression technique

• Reliability of **intra-arrest prognostic factors** to predict outcome

• Use of **invasive hemodynamic monitoring during CPR** to titrate to a specific systolic/diastolic blood pressure to improve outcomes

• Effectiveness of NO **vasopressor** compared with ANY vasopressors for resuscitation from cardiac arrest

• Use of amiodarone compared with **lidocaine** for **shockrefractory VF or pVT**

• Optimal **energy dose** for **defibrillation**

Postarrest Care

• Use of **targeted temperature management** to improve outcomes

• Use of a **targeted Pao_2 strategy** to improve outcomes

• Use of a **specific $Paco_2$ target** to improve outcomes

• Use of **parenteral fluids and inotropes and/or vasopressors** to maintain targeted measures of perfusion such as blood pressure to improve outcomes

• Use of **electroencephalograms (EEGs)** to accurately predict outcomes

• Use of any **specific post–cardiac arrest factors** to accurately predict outcomes

As noted above, these Web-based Integrated Guidelines incorporate all the recommendations from both 2010 Guidelines and the 2015 Guidelines Update.

3 Prearrest Care - Updated

3.1 Medical Emergency Team/Rapid Response Team - Updated PEDS 397

Medical emergency team or rapid response team activation by caregivers or parents ideally occurs as a response to changes noted in a patient's condition and may prevent cardiac or respiratory arrest. Several variables, including the composition of the team, the type of patient, the hospital setting, and the confounder of a wider "system benefit," further complicate objective analyses.

3.1.1 2015 Evidence Summary

Observational data have been contradictory and have not consistently shown a decreased incidence of cardiac and/or respiratory arrest outside of the ICU setting.[13-15] The data addressing effects on hospital mortality were inconclusive.[15-20]

3.1.2 2015 Recommendation—Updated

Pediatric medical emergency team/rapid response team systems may be considered in facilities where children with high-risk illnesses are cared for on general in-patient units. (Class IIb, LOE C-LD)

3.2 Pediatric Early Warning Scores - Updated PEDS 818

In-hospital pediatric cardiac or respiratory arrest can potentially be averted by early recognition of and intervention for the deteriorating patient. The use of scoring systems might help to identify such patients sufficiently early so as to enable effective intervention.

3.2.1 2015 Evidence Summary

There is no evidence that the use of PEWS outside of the pediatric ICU setting reduces hospital mortality. In 1 observational study, PEWS use was associated with a reduction in cardiac arrest rate when used in a single hospital with an established medical emergency team system.[21]

3.2.2 2015 Recommendation—New

The use of PEWS may be considered, but its effectiveness in the in-hospital setting is not well established. (Class IIb, LOE C-LD)

3.3 Fluid Resuscitation in Septic Shock - Updated PEDS 545

This update regarding intravenous fluid resuscitation in infants and children in septic shock in all settings addressed 2 specific therapeutic elements: (1) Withholding the use of bolus fluids was compared with the use of bolus fluids, and (2) noncrystalloid was compared with crystalloid fluids.

Early and rapid administration of intravenous fluid to reverse decompensated shock, and to prevent progression from compensated to decompensated shock, has been widely accepted based on limited observational studies.[22] Mortality from pediatric sepsis has declined in recent years, during which guidelines and publications have emphasized the role of early rapid fluid administration (along with early antibiotic and vasopressor therapy, and careful cardiovascular monitoring) in treating septic shock.[23,24] Since the 2010 Guidelines, a large randomized controlled trial of fluid resuscitation in pediatric severe febrile illness in a resource-limited setting found intravenous fluid boluses to be harmful.[25]This new information, contradicting long-held beliefs and practices, prompted careful analysis of the effect of fluid resuscitation on many outcomes in specific infectious illnesses.

3.3.1 2015 Evidence Summary

Specific infection-related shock states appear to behave differently with respect to fluid bolus therapy. Evidence was not considered to be specific to a particular setting, after determining that “resource-limited setting” is difficult to define and can vary greatly even within individual health systems and small geographic regions.

The evidence regarding the impact of restricting fluid boluses during resuscitation on outcomes in pediatric septic shock is summarized in Table 1. There were no studies for many specific combinations of presenting illness and outcome. In the majority of scenarios, there was no benefit to restricting fluid boluses during resuscitation.

Table 1: Evidence for the Use of Restrictive Volume of Intravenous Fluid Resuscitation, Compared With Unrestrictive Volume

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Evidence for the Use of Restrictive Volume of Intravenous Fluid Resuscitation, Compared With Unrestrictive Volume

	Studies	Survival to Hospital Discharge	Need for Transfusion or Diuretics	Need for Rescue Fluid	Mechanical Ventilation or Vasopressor	Time to Resolution of Shock	Total IV Fluids
Severe sepsis/septic shock	Santhanam 2008; Carcillo 1991	No Benefit	No Benefit	No Studies Available	No Benefit	No Benefit	No Studies Available
Severe malaria	Maitland 2005; Maitland 2005	No Benefit	No Benefit	Harm	No Studies Available	No Benefit	No Benefit

	Studies	Survival to Hospital Discharge	Need for Transfusion or Diuretics	Need for Rescue Fluid	Mechanical Ventilation or Vasopressor	Time to Resolution of Shock	Total IV Fluids
Severe febrile illness with some but not all signs of shock	Maitland 2011; Maitland 2013	Benefit	No Benefit	No Studies Available	No Studies Available	Harm	No Benefit

Evidence for the use of restrictive volume of intravenous fluid resuscitation, compared with unrestrictive volume, by presenting illness and outcome. Benefit indicates that studies show a benefit to restricting fluid volume, No Benefit indicates that there is no benefit to restricting fluid volume, and Harm indicates that there is harm associated with restricting fluid volume. No Studies Available indicates no studies are available for a particular illness/outcome combination.

The most important exception is that in 1 large study, restriction of fluid boluses conveyed a benefit for survival to both 48 hours and 4 weeks after presentation. This study was conducted in sub-Saharan Africa, and inclusion criteria were severe febrile illness complicated by impaired consciousness (prostration or coma), respiratory distress (increased work of breathing), or both, and with impaired perfusion, as evidenced by 1 or more of the following: a capillary refill time of 3 or more seconds, lower limb temperature gradient, weak radialpulse volume, or severe tachycardia. In this study, administration of 20 mL/kg or 40 mL/kg in the first hour was associated with decreased survival compared with the use of maintenance fluids alone.[25] Therefore, it appears that in this specific patient population, where critical care resources including inotropic and mechanical ventilator support were limited, bolus fluid therapy resulted in higher mortality.

The use of noncrystalloid fluid was compared with crystalloid fluid for the same diseases and outcomes listed in the preceding paragraph.[25-31] Evidence is summarized in Table 2. In most scenarios, there was no benefit to noncrystalloids over crystalloids. In patients with Dengue shock, a benefit was conferred in using noncrystalloid compared with crystalloid fluid for the outcome of time to resolution of shock.[30]

Table 2: 2015 - Noncrystalloid vs Crystalloid IV Fluid

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Noncrystalloid vs Crystalloid IV Fluid								
	Studies	**Survival to Hospital Discharge**	**Need for Other Treatment**	**Need for Rescue Fluid**	**Mechanical Ventilation or Vasopressor**	**Time to Resolution of Shock**	**Total IV Fluids**	**Hospital Duration of Stay**
Severe sepsis/ septic shock	Upadhyay 2005	No Benefit	No Benefit	No Studies Available	No Benefit	No Benefit	No Studies Available	No Studies Available
Severe malaria	Maitland 2003; Maitland 2005	No Studies Available	No Benefit	No Studies Available	No Studies Available	No Benefit	No Studies Available	No Studies Available

	Studies	Survival to Hospital Discharge	Need for Other Treatment	Need for Rescue Fluid	Mechanical Ventilation or Vasopressor	Time to Resolution of Shock	Total IV Fluids	Hospital Duration of Stay
Dengue shock	Cifra 2003; Dung 1999; Ngo 2001; Wills 2005	No Benefit	No Benefit	No Benefit	No Studies Available	Benefit	No Benefit	No Benefit
Severe febrile illness with some but not all signs of shock	Maitland 2011	No Benefit	No Benefit	No Benefit	No Studies Available	No Benefit	No Benefit	No Studies Available

Evidence for the use of noncrystalloid intravenous fluid resuscitation, compared with crystalloid, by presenting illness and outcome. *Benefit* indicates that studies show a benefit to the use of noncrystalloid intravenous fluid resuscitation compared with crystalloid, and *No Benefit* indicates that there is no benefit to the use of noncrystalloid intravenous fluid resuscitation compared with crystalloid. *No Studies Available* indicates no studies are available for a particular illness/outcome combination.

3.3.2 2015 Recommendations—New

Administration of an initial fluid bolus of 20 mL/kg to infants and children with shock is reasonable, including those with conditions such as severe sepsis (Class IIa, LOE C-LD), severe malaria and Dengue. (Class IIb, LOE B-R)

When caring for children with severe febrile illness (such as those included in the FEAST trial26) in settings with limited access to critical care resources (ie, mechanical ventilation and inotropic support), administration of bolus intravenous fluids should be undertaken with extreme caution because it may be harmful. (Class IIb, LOE B-R)

Providers should reassess the patient after every fluid bolus. (Class I, LOE C-EO)

Either isotonic crystalloids or colloids can be effective as the initial fluid choice for resuscitation. (Class IIa, LOE B-R)

This recommendation takes into consideration the important work of Maitland et al,[25] which found that fluid boluses as part of resuscitation are not safe for all patients in all settings. This study showed that the use of fluid boluses as part of resuscitation increased mortality in a specific population in a resource-limited setting, without access to some critical care interventions such as mechanical ventilation and inotrope support.

The spirit of this recommendation is a continued emphasis on fluid resuscitation for both compensated (detected by physical examination) and decompensated (hypotensive) septic shock. Moreover, emphasis is also placed on the use of individualized patient evaluation before the administration of intravenous fluid boluses, including physical examination by a clinician and frequent reassessment to determine the appropriate volume of fluid resuscitation. The clinician should also integrate clinical signs with patient and locality-specific information about prevalent diseases, vulnerabilities (such as severe anemia and malnutrition), and available critical care

resources.

Early assisted ventilation may be considered as part of a protocol-driven strategy for septic shock. (Class IIb, LOE C)

Etomidate has been shown to facilitate endotracheal intubation in infants and children with minimal hemodynamic effect, but do not use it routinely in pediatric patients with evidence of septic shock. (Class III, LOE B)

Adrenal suppression is seen after administration of etomidate in children[32] and adults.[33] In children and adults with septic shock, etomidate administration is associated with a higher mortality rate.[32,34]

3.4 Hypovolemic Shock

Use an isotonic crystalloid solution (eg, lactated Ringer's solution or normal saline) as the initial fluid for the treatment of shock. (Class I, LOE A)

There is no added benefit in using colloid (eg, albumin) during the early phase of resuscitation.[35,36]

Treat signs of shock with a bolus of 20 mL/kg of isotonic crystalloid even if blood pressure is normal. (Class IIb, LOE C)

Crystalloids may have an associated survival benefit over colloid for children with shock secondary to general trauma, traumatic brain injury, and burns.[36-39] There is no evidence to support the use of a specific isotonic crystalloid. Give additional boluses (20 mL/kg) if systemic perfusion fails to improve. There are insufficient data to make a recommendation for or against use of hypertonic saline for shock associated with head injuries or hypovolemia.[40,41]

There is insufficient evidence in infants and children to make a recommendation about the best timing or extent of volume resuscitation for children with hemorrhagic shock following trauma.

3.5 Dilated Cardiomyopathy or Myocarditis - Updated PEDS 819

Optimal care of a critically ill infant or child with dilated cardiomyopathy or myocarditis should avert cardiac arrest. While significant global experience exists with the care of these patients, the evidence base is limited. The ILCOR systematic review ultimately restricted its analysis to patients with myocarditis and did not include the use of ventricular assist devices.

3.5.1 2015 Evidence Summary

No literature was identified evaluating best prearrest management strategies (including anesthetic technique) for infants and children with dilated cardiomyopathy or myocarditis. Limited observational data support the pre-cardiac arrest use of ECMO in children with acute fulminant myocarditis.[42]

3.5.3 2015 Recommendation—New

Venoarterial ECMO use may be considered in patients with acute fulminant myocarditis who are at high risk of imminent cardiac arrest. (Class IIb, LOE C-EO)

Optimal outcomes from ECMO are achieved in settings with existing ECMO protocols, expertise, and equipment.

3.6 Toxicological Emergencies

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

3.6.1 Local Anesthetic

Local anesthetics are used topically, intravenously, subcutaneously, and in epidural or other catheters for delivery of regional analgesia. The toxicity of local anesthetics is well recognized in children; they may cause changes in mental status, seizures, arrhythmias, or even cardiac arrest in settings of overdose or inadvertent vascular administration. Multiple case reports, including some pediatric reports, have described successful treatment of local anesthetic toxicity with intravenous lipid emulsion.[43]

3.6.2 Cocaine

Acute coronary syndrome, manifested by chest pain and cardiac rhythm disturbances (including VT and VF), is the most frequent cocaine-related reason for hospitalization in adults.[44,45] Cocaine also may prolong the action potential and QRS duration and impairs myocardial contractility.[46,47]

3.6.2.1 Treatment

Hyperthermia, which may result from cocaine-induced hypermetabolism, is associated with an increase in toxicity;[48] therefore treat elevated temperature aggressively.

For coronary vasospasm consider nitroglycerin (Class IIa, LOE C), a benzodiazepine, and phentolamine (an ?-adrenergic antagonist). (Class IIb, LOE C)

Do not give ?-adrenergic blockers. (Class III, LOE C)

For ventricular arrhythmia, consider sodium bicarbonate (1 to 2 mEq/kg) administration in addition to standard treatment. (Class IIb, LOE C)

3.6.3 Tricyclic Antidepressants and Other Sodium Channel Blockers

Toxic doses cause cardiovascular abnormalities, including intraventricular conduction delays, heart block, bradycardia, prolongation of the QT interval, ventricular arrhythmias (including torsades de pointes, VT, and VF), hypotension, seizures,[47,49] and a depressed level of consciousness.

3.6.3.1 Treatment

Give 1 to 2 mEq/kg intravenous boluses of sodium bicarbonate until arterial pH is >7.45; then provide an infusion of 150 mEq NaHCO3 per liter of D5W to maintain alkalosis. In cases of severe intoxication increase the pH to 7.50 to 7.55.[47,50]

Do not administer Class IA (quinidine, procainamide), Class IC (flecainide, propafenone), or Class III (amiodarone and sotalol) antiarrhythmics, which may exacerbate cardiac toxicity. (Class III, LOE C)

For hypotension, give boluses (10 mL/kg each) of normal saline. If hypotension persists, epinephrine and norepinephrine are more effective than dopamine in raising blood pressure.[51,52]

Consider ECMO if high-dose vasopressors do not maintain blood pressure.[53,54]

3.6.4 Calcium Channel Blockers

Manifestations of toxicity include hypotension, ECG changes (prolongation of the QT interval, widening of the QRS, and right bundle branch block), arrhythmias (bradycardia, SVT, VT, torsades de pointes, and VF),[55] seizures, and altered mental status.

3.6.4.1 Treatment

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

The effectiveness of calcium administration is variable. (Class IIb, LOE C)

Infuse 20 mg/kg (0.2 mL/kg) of 10% calcium chloride intravenously over 5 to 10 minutes; if there is a beneficial effect, give an infusion of 20 to 50 mg/kg per hour. Monitor serum ionized calcium concentration to prevent hypercalcemia. It is preferable to administer calcium chloride via a central venous catheter; use caution when infusing into a peripheral IV because infiltration can cause severe tissue injury. If no central venous catheter is available, infuse calcium gluconate through a secure peripheral IV.

For bradycardia and hypotension, consider vasopressors and inotropes such as norepinephrine or epinephrine. (Class IIb, LOE C)

There are insufficient data to recommend for or against an infusion of insulin and glucose[56-59] or sodium bicarbonate.

3.6.5 Beta-Adrenergic Blockers

Toxic doses of ?-adrenergic blockers cause bradycardia, heart block, and decreased cardiac contractility, and some (eg, propranolol and sotalol) may also prolong the QRS and the QT intervals.[59-62]

3.6.5.1 Treatment

High-dose epinephrine infusion may be effective. (Class IIb, LOE C)

Consider glucagon. In adolescents infuse 5 to 10 mg of glucagon over several minutes followed by an IV infusion of 1 to 5 mg/hour. (Class IIb, LOE C)

Consider an infusion of glucose and insulin. (Class IIb, LOE C)

There are insufficient data to make a recommendation for or against using calcium. (Class IIb, LOE C)

Calcium may be considered if glucagon and catecholamines are ineffective. (Class IIb, LOE C)

3.6.6 Opioids

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

3.6.6.1 Treatment

Support of oxygenation and ventilation is the initial treatment for severe respiratory depression from any

cause. (Class I)

Naloxone reverses the respiratory depression of narcotic overdose, but in persons with long-term addictions or cardiovascular disease, naloxone may markedly increase heart rate and blood pressure and cause acute pulmonary edema, cardiac arrhythmias (including asystole), and seizures. (Class I, LOE B)

Ventilation before administration of naloxone appears to reduce these adverse effects.[63] Intramuscular administration of naloxone may lower the risk by slowing the onset of drug effect.

3.7 Atropine for Premedication During Emergency Intubation - Updated PEDS 821

Bradycardia commonly occurs during emergency pediatric intubation, resulting from hypoxia/ischemia, as a vagal response to laryngoscopy, as a reflex response to positive pressure ventilation, or as a pharmacologic effect of some drugs (eg, succinylcholine or fentanyl). Practitioners have often tried to blunt this bradycardia with prophylactic premedication with atropine.

3.7.1 2015 Evidence Summary

The evidence regarding the use of atropine during emergency intubation has largely been observational, including extrapolation from experience with elective intubation in the operating suite. More recent in-hospital literature involves larger case series of critically ill neonates, infants, and children undergoing emergency intubation.[64-66]

There is no evidence that preintubation use of atropine improves survival or prevents cardiac arrest in infants and children. Observational data suggest that it increases the likelihood of survival to ICU discharge in children older than 28 days.[64] Evidence is conflicting as to whether preintubation atropine administration reduces the incidence of arrhythmias or postintubation shock.[65, 66]

In past Guidelines, a minimum atropine dose of 0.1 mg IV was recommended after a report of paradoxical bradycardia observed in very small infants who received very low atropine doses.[67] However, in 2 of the most recent case series cited above, preintubation doses of 0.02 mg/kg, with no minimum dose, were shown to be effective.[64,65]

3.7.2 2015 Recommendations—New

The available evidence does not support the routine use of atropine preintubation of critically ill infants and children.

It may be reasonable for practitioners to use atropine as a premedication in specific emergency intubations when there is higher risk of bradycardia (eg, when giving succinylcholine as a neuromuscular blocker to facilitate intubation). (Class IIb, LOE C-LD)

A dose of 0.02 mg/kg of atropine with no minimum dose may be considered when atropine is used as a premedication for emergency intubation. (Class IIb, LOE C-LD)

This new recommendation applies only to the use of atropine as a premedication for infants and children during emergency intubation.

4 Intra-arrest Care - Updated

4.1 BLS Considerations During PALS

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

4.1.1 Simultaneous Actions

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

Chest compressions should be immediately started by one rescuer, while a second rescuer prepares to start ventilations with a bag and mask. Ventilation is extremely important in pediatrics because of the large percentage of asphyxial arrests in which best results are obtained by a combination of chest compressions and ventilations.[68] Unfortunately ventilations are sometimes delayed because equipment (bag, mask, oxygen, airway) must be mobilized. Chest compressions require only the hands of a willing rescuer.

Therefore, start CPR with chest compressions immediately, while a second rescuer prepares to provide ventilations. (Class I, LOE C)

The effectiveness of PALS is dependent on high-quality CPR, which requires an adequate compression rate (at least 100 compressions/min), an adequate compression depth (at least one third of the AP diameter of the chest or approximately 1 ½ inches [4 cm] in infants and approximately 2 inches [5 cm] in children), allowing complete recoil of the chest after each compression, minimizing interruptions in compressions, and avoiding excessive ventilation. Reasons for not performing high-quality CPR include rescuer inattention to detail, rescuer fatigue, and long or frequent interruptions to secure the airway, check the heart rhythm, and move the patient.[69] Optimal chest compressions are best delivered with the victim on a firm surface.[70,71]

While one rescuer performs chest compressions and another performs ventilations, other rescuers should obtain a monitor/defibrillator, establish vascular access, and calculate and prepare the anticipated medications.

4.1.2 Monitored Patients

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

4.1.3 Respiratory Failure

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

An increased respiratory rate, particularly with signs of distress (eg, increased respiratory effort including nasal flaring, retractions, seesaw breathing, or grunting)

An inadequate respiratory rate, effort, or chest excursion (eg, diminished breath sounds or gasping), especially if mental status is depressed

Cyanosis with abnormal breathing despite supplementary oxygen

4.1.4 Shock

Shock results from inadequate blood flow and oxygen delivery to meet tissue metabolic demands. The most common type of shock in children is hypovolemic, including shock due to hemorrhage. Distributive, cardiogenic, and obstructive shock occur less frequently. Shock progresses over a continuum of severity, from a

compensated to a decompensated state. Compensatory mechanisms include tachycardia and increased systemic vascular resistance (vasoconstriction) in an effort to maintain cardiac output and perfusion pressure respectively. Decompensation occurs when compensatory mechanisms fail and results in hypotensive shock.

Typical signs of compensated shock include

– Tachycardia

– Cool and pale distal extremities

– Prolonged (>2 seconds) capillary refill (despite warm ambient temperature)

– Weak peripheral pulses compared with central pulses

– Normal systolic blood pressure

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

– Depressed mental status

– Decreased urine output

– Metabolic acidosis

– Tachypnea

– Weak central pulses

– Deterioration in color (eg, mottling, see below)

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

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

Capillary refill time alone is not a good indicator of circulatory volume, but a capillary refill time >2 seconds is a useful indicator of moderate dehydration when combined with decreased urine output, absent tears, dry mucous membranes, and a generally ill appearance. Capillary refill time is influenced by ambient temperature,[72] site, and age and its interpretation can be influenced by lighting.[73]

Tachycardia is a common sign of shock, but it can also result from other causes, such as pain, anxiety, and fever.

Pulses are weak in hypovolemic and cardiogenic shock, but may be bounding in anaphylactic, neurogenic, and septic shock.

Blood pressure may be normal in a child with compensated shock but may decline rapidly when the child decompensates. Like the other signs, hypotension must be interpreted within the context of the entire clinical picture.

There are several sources of data that use large populations to identify the 5th percentile for systolic blood pressure at various ages.[74,75] For purposes of these guidelines, hypotension is defined as a ***systolic*** blood pressure:

<60 mm Hg in term neonates (0 to 28 days)

<70 mm Hg in infants (1 month to 12 months)

<70 mm Hg + (2 × age in years) in children 1 to 10 years

<90 mm Hg in children ?10 years of age

4.1.5 Airway

4.1.5.1 Oropharyngeal and Nasopharyngeal Airways

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

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

4.1.5.2 Laryngeal Mask Airway (LMA)

Although several supraglottic devices have been used in children, clinical studies of devices other than the LMA in pediatric patients are limited.

When bag-mask ventilation (see "Bag-Mask Ventilation," below) is unsuccessful and when endotracheal intubation is not possible, the LMA is acceptable when used by experienced providers to provide a patent airway and support ventilation. (Class IIa, LOE C)

LMA insertion is associated with a higher incidence of complications in young children compared with older children and adults.[76-81]

4.1.6 Oxygen

It is reasonable to ventilate with 100% oxygen during CPR because there is insufficient information on the optimal inspired oxygen concentration. (Class IIa, LOE C)

Once the circulation is restored, monitor systemic oxygen saturation. It may be reasonable, when the appropriate equipment is available, to titrate oxygen administration to maintain the oxyhemoglobin saturation ?94%. Provided appropriate equipment is available, once ROSC is achieved, adjust the FIO_2 to the minimum concentration needed to achieve an arterial oxyhemoglobin saturation at least 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery.

Since an arterial oxyhemoglobin saturation of 100% may correspond to a PaO2 anywhere between ?80 and 500 mmHg, in general it is appropriate to wean the FIO2 when saturation is 100%, provided the oxyhemoglobin saturation can be maintained ?94%. (Class IIb, LOE C)

Remember that adequate oxygen delivery requires not only adequate arterial oxyhemoglobin saturation but also adequate hemoglobin concentration and cardiac output.

4.1.7 Pulse Oximetry

If the patient has a perfusing rhythm, monitor oxyhemoglobin saturation continuously with a pulse oximeter because clinical recognition of hypoxemia is not reliable.[82] Pulse oximetry may, however, also be unreliable in patients with poor peripheral perfusion, carbon monoxide poisoning, or methemoglobinemia.

4.1.7.1 Bag-Mask Ventilation

Bag-mask ventilation can be as effective, and may be safer, than endotracheal tube ventilation for short periods during out-of-hospital resuscitation.[83-90]

In the prehospital setting it is reasonable to ventilate and oxygenate infants and children with a bag-mask device, especially if transport time is short. (Class IIa, LOE B)

Bag-mask ventilation requires training and periodic retraining in selecting a correct mask size, maintaining an open airway, providing a tight seal between mask and face, providing ventilation, and assessing effectiveness of ventilation (see Part 11, Pediatric Basic Life Support).

4.1.7.1.1 Precautions

Use only the force and tidal volume needed to just make the chest rise visibly (Class I, LOE C); avoid delivering excessive ventilation during cardiac arrest. (Class III, LOE C)

Evidence shows that cardiac arrest victims frequently receive excessive ventilation.[69,91-93] Excessive ventilation during cardiac arrest increases intrathoracic pressure, which impedes venous return, thus reducing cardiac output and cerebral and coronary blood flow. These effects will reduce the likelihood of ROSC.[92] In addition, excessive ventilation may cause air trapping and barotrauma in patients with small airway obstruction. It also increases the risk of stomach inflation, regurgitation, and aspiration.

If the infant or child is not intubated, pause after 30 chest compressions (1 rescuer) or after 15 chest compressions (2 rescuers) to give 2 ventilations (mouth-to-mouth, mouth-to-mask, or bag-mask).

Deliver each breath with an inspiratory time of approximately 1 second. If the infant or child is intubated, ventilate at a rate of about 1 breath every 6 seconds (10 times per minute) without interrupting chest compressions.

It may be reasonable to do the same if an LMA is in place. (Class IIb, LOE C)

In the victim with a perfusing rhythm but absent or inadequate respiratory effort, give 1 breath every 3 to 5 seconds (12 to 20 breaths per minute), using the higher rate for the younger child. (Class I, LOE C)

One way to achieve that rate with a ventilating bag is to use the mnemonic “squeeze-release-release” at a normal speaking rate.[83,94]

4.1.8 Two-Person Bag-Mask Ventilation

A 2-person ventilation technique may be preferable when personnel are available and may be more effective than ventilation by a single rescuer if the patient has significant airway obstruction, poor lung compliance, or the rescuer has difficulty in creating a tight mask-to-face seal.[95,96] One rescuer uses both hands to maintain an open airway with a jaw thrust and a tight mask-to-face seal while the other compresses the ventilation bag. Both rescuers should observe the victim’s chest to ensure chest rise.

4.1.9 Gastric Inflation

Gastric inflation may interfere with effective ventilation[97] and cause regurgitation, aspiration of stomach contents, and further ventilatory compromise. The risk of gastric inflation can be decreased by

Avoiding excessive peak inspiratory pressures by ventilating slowly and giving only enough tidal volume to just achieve visible chest rise.[83]

Applying cricoid pressure in an unresponsive victim to reduce air entry into the stomach. (Class IIa, LOE B)

This may require a third rescuer if cricoid pressure cannot be applied by the rescuer who is securing the bag to the face.

Avoid excessive cricoid pressure so as not to obstruct the trachea. (Class III, LOE B)

Passing a nasogastric or orogastric tube to relieve gastric inflation, especially if oxygenation and ventilation are compromised. Pass the tube after intubation because a gastric tube interferes with gastroesophageal sphincter function, allowing regurgitation during intubation. If a gastrostomy tube is present, vent it during bag-mask ventilation to allow gastric decompression.

4.1.10 Ventilation With an Endotracheal Tube

Endotracheal intubation in infants and children requires special training because the pediatric airway anatomy differs from that of the adult. The likelihood of successful endotracheal tube placement with minimal complications is related to the length of training, supervised experience in the operating room and in the field,[98, 99] adequate ongoing experience,[100] and use of rapid sequence intubation (RSI).[101,102]

4.1.11 Ventilation With a Tracheostomy or Stoma

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

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

4.1.12 Rapid Sequence Intubation (RSI)

To facilitate emergency intubation and reduce the incidence of complications, skilled, experienced providers may use sedatives, neuromuscular blocking agents, and other medications to rapidly sedate and neuromuscularly block the pediatric patient.[103]

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

Actual body weight, rather than ideal body weight, should be used for some non-resuscitation medications (eg, succinylcholine).[104-119]

4.1.13 Cricoid Pressure During Intubation

There is insufficient evidence to recommend routine cricoid pressure application to prevent aspiration during endotracheal intubation in children.

Do not continue cricoid pressure if it interferes with ventilation or the speed or ease of intubation. (Class III, LOE C)

4.1.14 Cuffed Versus Uncuffed Endotracheal Tubes

Both cuffed and uncuffed endotracheal tubes are acceptable for intubating infants and children. (Class IIa, LOE C)

In the operating room, cuffed endotracheal tubes are associated with a higher likelihood of correct selection of tube size, thus achieving a lower reintubation rate with no increased risk of perioperative complications.[120-122] In intensive care settings the risk of complications in infants and in children is no greater with cuffed tubes than with noncuffed tubes.[123-125] Cuffed endotracheal tubes may decrease the risk of aspiration.[126] If cuffed endotracheal tubes are used, cuff inflating pressure should be monitored and limited according to manufacturer's instruction (usually less than 20 to 25 cm H_2O).

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

4.1.15 Endotracheal Tube Size

Length-based resuscitation tapes are helpful and more accurate than age-based formula estimates of endotracheal tube size for children up to approximately 35 kg,[111,127,128] even for children with short stature.[129]

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

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

Uncuffed endotracheal tube ID (mm) = 4+(age/4)

If a cuffed tube is used for emergency intubation of an infant less than 1 year of age, it is reasonable to select a 3.0 mm ID tube.

For children between 1 and 2 years of age, it is reasonable to use a cuffed endotracheal tube with an internal diameter of 3.5 mm. (Class IIa, LOE B)

After age 2 it is reasonable to estimate tube size with the following formula. (Class IIa, LOE B)

Cuffed endotracheal tube ID (mm) = 3.5+(age/4)

4.1.16 Verification of Endotracheal Tube Placement

There is a risk of endotracheal tube misplacement (ie, in the esophagus, the pharynx above the vocal cords, or a mainstem bronchus) and an ongoing risk of displacement or obstruction,[83,130] especially during patient transport.[131]

Since no single confirmation technique, including clinical signs or the presence of water vapor in the tube, is completely reliable, use both clinical assessment and confirmatory devices to verify proper tube placement immediately after intubation, again after securing the endotracheal tube, during transport, and each time the patient is moved (eg, from gurney to bed). (Class I, LOE B)

The following are methods for confirming correct position:

Look for bilateral chest movement and listen for equal breath sounds over both lung fields, especially over the axillae.

Listen for gastric insufflation sounds over the stomach. They should *not* be present if the tube is in the trachea.[132]

Check for exhaled CO_2 (see "Exhaled or End-Tidal CO_2 Monitoring," below).

If there is a perfusing rhythm, check oxyhemoglobin saturation with a pulse oximeter. Remember that following hyperoxygenation, the oxyhemoglobin saturation detected by pulse oximetry may not decline for as long as 3 minutes even without effective ventilation.[133,134]

If you are still uncertain, perform direct laryngoscopy and visualize the endotracheal tube to confirm that it lies between the vocal cords.

In hospital settings, perform a chest x-ray to verify that the tube is not in a bronchus and to identify proper position in the midtrachea.

After intubation, secure the tube; there is insufficient evidence to recommend any single method. After securing the tube, maintain the patient's head in a neutral position; neck flexion may push the tube farther into the airway, and extension may pull the tube out of the airway.[135,136]

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

Displacement of the tube

Obstruction of the tube

Pneumothorax

Equipment failure

4.1.17 Exhaled or End-Tidal CO2 ($ETCO_2$) Monitoring

When available, exhaled CO2 detection (capnography or colorimetry) is recommended as confirmation of tracheal tube position for neonates, infants, and children with a perfusing cardiac rhythm in all settings (eg, prehospital, emergency department [ED], ICU, ward, operating room) (Class I, LOE C) and during intrahospital or interhospital transport. (Class IIb, LOE C)

Remember that a color change or the presence of a capnography waveform confirms tube position in the airway but does not rule out right mainstem bronchus intubation.

During cardiac arrest, if exhaled CO2 is not detected, confirm tube position with direct laryngoscopy because the absence of CO2 may reflect very low pulmonary blood flow rather than tube misplacement. (Class IIa, LOE C)

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

If the detector is contaminated with gastric contents or acidic drugs (eg, endotracheally administered epinephrine), a consistent color rather than a breath-to-breath color change may be seen.

An intravenous (IV) bolus of epinephrine[137] may transiently reduce pulmonary blood flow and exhaled CO_2 below the limits of detection.[138]

Severe airway obstruction (eg, status asthmaticus) and pulmonary edema may impair CO_2 elimination below the limits of detection.[138,139-141]

A large glottic air leak may reduce exhaled tidal volume through the tube and dilute CO_2 concentration.

4.1.18 Esophageal Detector Device (EDD)

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

4.1.19 Transtracheal Catheter Oxygenation and Ventilation

Transtracheal catheter oxygenation and ventilation may be considered for patients with severe airway obstruction above the level of the cricoid cartilage if standard methods to manage the airway are unsuccessful. Note that transtracheal ventilation primarily supports oxygenation as tidal volumes are usually too small to effectively remove carbon dioxide.

This technique is intended for temporary use while a more effective airway is obtained. Attempt this procedure only after proper training and with appropriate equipment. (Class IIb, LOE C)

4.1.20 Suction Devices

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

4.1.21 CPR Guidelines for Newborns With Cardiac Arrest of Cardiac Origin

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

Newborns who require CPR in other settings (eg, prehospital, ED, pediatric intensive care unit [PICU], etc.), should receive CPR according to infant guidelines: 2 rescuers provide continuous chest compressions with asynchronous ventilations if an advanced airway is in place and a 15:2 ventilation-to-compression ratio if no advanced airway is in place. (Class IIb, LOE C)

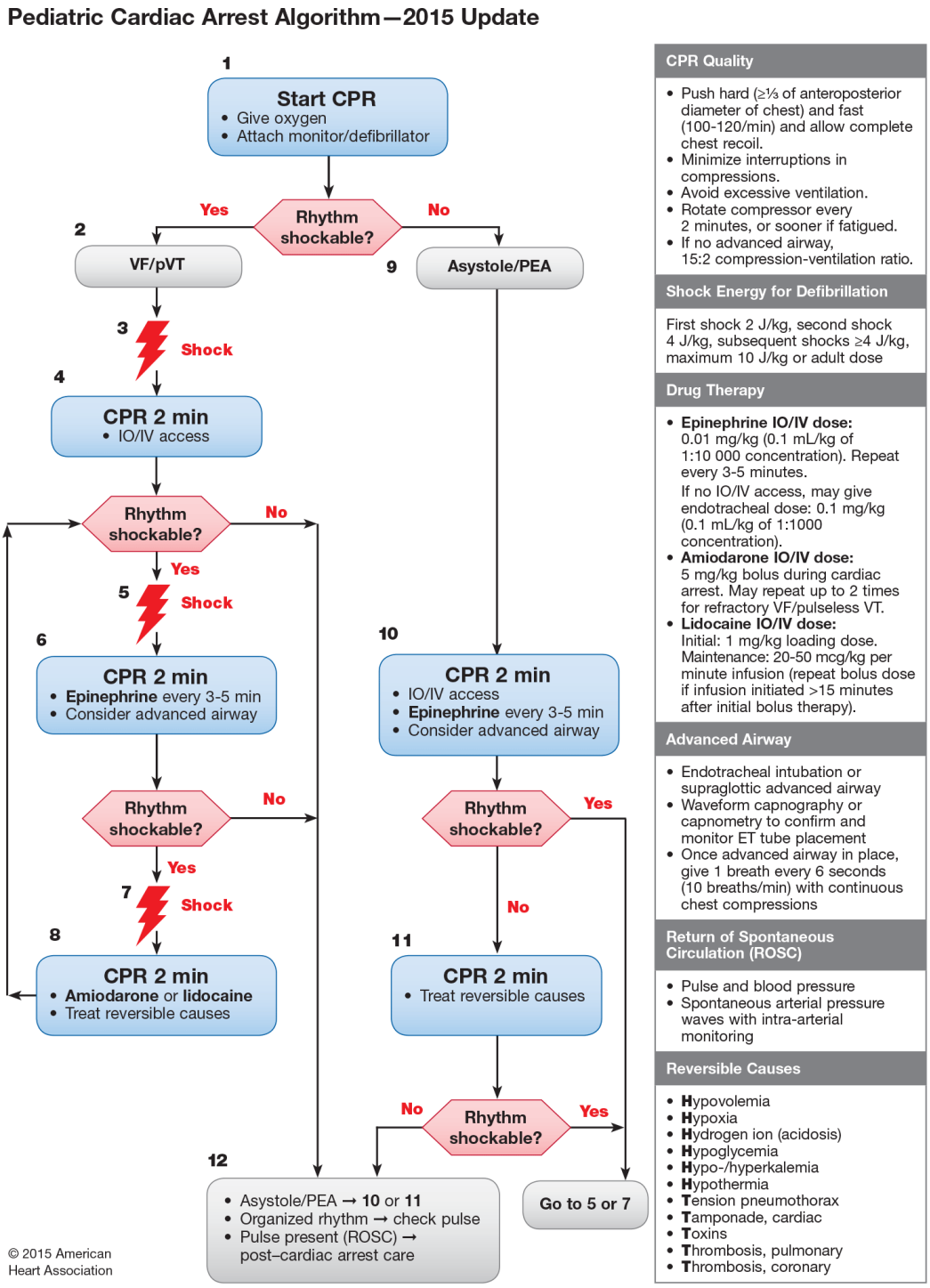
It is reasonable to resuscitate newborns with a primary cardiac etiology of arrest, regardless of location, according to infant guidelines, with emphasis on chest compressions. (Class IIa, LOE C)

For further information, please refer to Part 11: Pediatric Basic Life Support, and Part 13: Neonatal Resuscitation.

4.2 Pulseless Arrest

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

Figure 1: Pediatric Cardiac Arrest Algorithm—2015 Update



(Step 1) As soon as the child is found to be unresponsive with no breathing, call for help, send for a defibrillator (manual or AED), and start CPR (with supplementary oxygen if available). Attach ECG monitor or AED pads as soon as available. Throughout resuscitation, emphasis should be placed on provision of high-quality CPR (providing chest compressions of adequate rate and depth, allowing complete chest recoil after each compression, minimizing interruptions in compressions and avoiding excessive ventilation).

While CPR is being given, determine the child's cardiac rhythm from the ECG or, if you are using an AED, the device will tell you whether the rhythm is "shockable" (eg, VF or rapid pVT) or "not shockable" (eg, asystole or PEA). It may be necessary to temporarily interrupt chest compressions to determine the child's rhythm. Asystole and bradycardia with a wide QRS are most common in asphyxial arrest.[142] VF and PEA are less common[143] but VF is more likely to be present in older children with sudden witnessed arrest.

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

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

(Step 10) Continue CPR with as few interruptions in chest compressions as possible. A second rescuer obtains vascular access and delivers epinephrine, 0.01 mg/kg (0.1 mL/kg of 1:10 000 solution) maximum of 1 mg (10 mL), while CPR is continued.

The same epinephrine dose is repeated every 3 to 5 minutes. (Class I, LOE B)

There is no survival benefit from high-dose epinephrine, and it may be harmful, particularly in asphyxia. (Class III, LOE B)

High-dose epinephrine may be considered in exceptional circumstances, such as ?-blocker overdose. (Class IIb, LOE C)

Once an advanced airway is in place, 1 rescuer should give continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The second rescuer delivers ventilations at a rate of 1 breath every 6 seconds (10 breaths per minute). Rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions. Check rhythm every 2 minutes with minimal interruptions in chest compressions. If the rhythm is "nonshockable" continue with cycles of CPR and epinephrine administration until there is evidence of ROSC or you decide to terminate the effort. If at any time the rhythm becomes "shockable," give a shock (Step 7) and immediately resume chest compressions for 2 minutes before rechecking the rhythm. Minimize time between chest compressions and shock delivery (ie, check rhythm and deliver shocks immediately after compressions rather than after rescue breaths, if possible) and between shock delivery and resumption of chest compressions.

Search for and treat reversible causes.

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

Defibrillation is the definitive treatment for VF with an overall survival rate of 17% to 20%. (Class I, LOE B)

Survival is better in primary than in secondary VF.[144] In adults, the probability of survival declines by 7% to 10% for each minute of arrest without CPR and defibrillation.[145] Survival is better if early, high-quality CPR is provided with minimal interruptions. Outcome of shock delivery is best if rescuers minimize the time between last compression and shock delivery, so rescuers should be prepared to coordinate (brief) interruptions in chest compressions to deliver shocks, and should resume compressions immediately after shock delivery.

4.3 Monitoring

4.3.1 End-Tidal CO2 Monitoring to Guide CPR Quality - Updated PEDS 827

High-quality CPR is associated with improved outcomes after cardiac arrest. Animal data support a direct association between $ETCO_2$ and cardiac output. Capnography is used during pediatric cardiac arrest to monitor

for ROSC as well as CPR quality. The 2010 Guidelines recommended that if the partial pressure of $ETCO_2$ is consistently less than 15 mmHg, efforts should focus on improving CPR quality, particularly improving chest compressions and ensuring that the victim does not receive excessive ventilation.

4.3.1.1 2015 Evidence Summary

There is no pediatric evidence that $ETCO_2$ monitoring improves outcomes from cardiac arrest. One pediatric animal study showed that $ETCO_2$ -guided chest compressions are as effective as standard chest compressions optimized by marker, video, and verbal feedback for achieving ROSC.[146] A recent study in adults found that $ETCO_2$ values generated during CPR were significantly associated with chest compression depth and ventilation rate.[147]

4.3.1.2 2015 Recommendation—New

ETCO2 monitoring may be considered to evaluate the quality of chest compressions, but specific values to guide therapy have not been established in children. (Class IIb, LOE C-LD)

4.3.2 Electrocardiography

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

4.3.3 Echocardiography

There is insufficient evidence for or against the routine use of echocardiography in pediatric cardiac arrest.

When appropriately trained personnel are available, echocardiography may be considered to identify patients with potentially treatable causes of the arrest, particularly pericardial tamponade and inadequate ventricular filling. (Class IIb, LOE C)

Minimize interruption of CPR while performing echocardiography.

4.3.4 Invasive Hemodynamic Monitoring During CPR - Updated PEDS 826

Children often have cardiac arrests in settings where invasive hemodynamic monitoring already exists or is rapidly obtained. If a patient has an indwelling arterial catheter, the waveform can be used as feedback to evaluate chest compressions.

4.3.4.1 2015 Evidence Summary

Adjusting chest compression technique to a specific systolic blood pressure target has not been studied in humans. Two randomized controlled animal studies showed increased likelihood of ROSC and survival to completion of experiment with the use of invasive hemodynamic monitoring.[148,149]

4.3.4.2 2015 Recommendation—New

For patients with invasive hemodynamic monitoring in place at the time of cardiac arrest, it may be reasonable for rescuers to use blood pressure to guide CPR quality. (Class IIb, LOE C-EO)

Specific target values for blood pressure during CPR have not been established in children.

4.3.5 Vascular Access

Vascular access is essential for administering medications and drawing blood samples. Obtaining peripheral venous access can be challenging in infants and children during an emergency; intraosseous (IO) access can be quickly established with minimal complications by providers with varied levels of training.[150-157] Limit the time spent attempting to establish peripheral venous access in a critically ill or injured child.[158]

4.3.6 Intraosseous (IO) Access

IO access is a rapid, safe, effective, and acceptable route for vascular access in children, and it is useful as the initial vascular access in cases of cardiac arrest. (Class I, LOE C)

All intravenous medications can be administered intraosseously, including epinephrine, adenosine, fluids, blood products,[159,160] and catecholamines.[161] Onset of action and drug levels for most drugs are comparable to venous administration.[162] IO access can be used to obtain blood samples for analysis including for type and cross match and blood gases during CPR,[163]but acid-base analysis is inaccurate after sodium bicarbonate administration via the IO cannula.[164] Use manual pressure or an infusion pump to administer viscous drugs or rapid fluid boluses;[165,166] follow each medication with a saline flush to promote entry into the central circulation.

4.3.7 Venous Access

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

4.3.8 Endotracheal Drug Administration

Vascular access (IO or IV) is the preferred method for drug delivery during CPR, but if it is not possible, lipid-soluble drugs, such as lidocaine, epinephrine, atropine, and naloxone (mnemonic “LEAN”)[167,168] can be administered via an endotracheal tube.[169] However, the effects may not be uniform with tracheal as compared with intravenous administration. One study of children in cardiac arrest[170] demonstrated similar ROSC and survival rates regardless of the method of drug delivery, while three studies of adults in cardiac arrest[171-173] demonstrated reduced ROSC and survival to hospital discharge with tracheal administration of epinephrine compared to vascular delivery. If CPR is in progress, stop chest compressions briefly, administer the medications, and follow with a flush of at least 5 mL of normal saline and 5 consecutive positive-pressure ventilations.[174] Optimal endotracheal doses of medications are unknown; in general expert consensus recommends doubling or tripling the dose of lidocaine, atropine or naloxone given via the ETT. For epinephrine, a dose ten times the intravenous dose (0.1 mg/kg or 0.1 mL/kg of 1:1,000 concentration) is recommended (see Table 3).

Table 3: 2010 - Medications for Pediatric Resuscitation

Open table in a new window

Medications for Pediatric Resuscitation		
Medication	**Dose**	**Remarks**
Adenosine	0.1 mg/kg (maximum 6 mg) Second dose: 0.2 mg/kg (maximum 12 mg)	Monitor ECG Rapid IV/IO bolus with flush

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

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

The effectiveness of endotracheal epinephrine during cardiac arrest is controversial. Some studies showed it to be as effective as vascular administration[170,175,176] while other studies have not found it to be as effective.[171-173,177] Animal studies[178-183] suggested that a higher dose of epinephrine is required for endotracheal than for intravascular administration because the lower epinephrine concentrations achieved when the drug is delivered by the endotracheal route may produce predominant transient peripheral ?2-adrenergic vasodilating effects. These effects can be detrimental, and cause hypotension, lower coronary artery perfusion pressure and flow,

and a reduced potential for ROSC.

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

4.4 Extracorporeal CPR for In-Hospital Pediatric Cardiac Arrest - Updated PEDS 407

The 2010 AHA PALS Guidelines suggested the use of ECMO when dealing with pediatric cardiac arrest refractory to conventional interventions and when managing a reversible underlying disease process. Pediatric OHCA was not considered for the 2015 ILCOR systematic review.

4.4.1 2015 Evidence Summary

Evidence from 4 observational studies of pediatric IHCA has shown no overall benefit to the use of CPR with ECMO (ECPR) compared to CPR without ECMO.[184-187] Observational data from a registry of pediatric IHCA showed improved survival to hospital discharge with the use of ECPR in patients with surgical cardiac diagnoses.[188] For children with underlying cardiac disease, when ECPR is initiated in a critical care setting, long-term survival has been reported even after more than 50 minutes of conventional CPR.[189] When ECPR is used during cardiac arrest, the outcome for children with underlying cardiac disease is better than for those with noncardiac disease.[190]

4.4.2 2015 Recommendation—New

ECPR may be considered for pediatric patients with cardiac diagnoses who have IHCA in settings with existing ECMO protocols, expertise, and equipment. (Class IIb, LOE C-LD)

4.5 Intra-arrest Prognostic Factors for Cardiac Arrest - Updated PEDS 814

Accurate and reliable prognostication during pediatric cardiac arrest would allow termination of CPR in patients where CPR is futile, while encouraging continued CPR in patients with a potential for good recovery.

4.5.1 2015 Evidence Summary

For infants and children with OHCA, age less than 1 year,[4,191] longer durations of cardiac arrest[192-194] and presentation with a nonshockable as opposed to a shockable rhythm[4,191,193] are all predictors of poor patient outcome. For infants and children with IHCA, negative predictive factors include age greater than 1 year[2] and longer durations of cardiac arrest.[2,195-197] The evidence is contradictory as to whether a nonshockable (as opposed to shockable) initial cardiac arrest rhythm is a negative predictive factor in the in-hospital setting.[2,198,199]

4.5.2 2015 Recommendation—New

Multiple variables should be used when attempting to prognosticate outcomes during cardiac arrest. (Class I, LOE C-LD)

Although there are factors associated with better or worse outcomes, no single factor studied predicts outcome with sufficient accuracy to recommend termination or continuation of CPR.

4.6 Vasopressors During Cardiac Arrest - Updated PEDS 424

During cardiac arrest, vasopressors are used to restore spontaneous circulation by optimizing coronary perfusion and to help maintain cerebral perfusion. However, they also cause intense vasoconstriction and increase myocardial oxygen consumption, which might be detrimental.

4.6.1 2015 Evidence Summary

There are no pediatric studies that demonstrate the effectiveness of any vasopressors (epinephrine, or combination of vasopressors) in cardiac arrest. Two pediatric observational out-of-hospital studies[200,201] had too many confounders to determine if vasopressors were beneficial. One adult OHCA randomized controlled trial [202] showed epinephrine use was associated with increased ROSC and survival to hospital admission but no

improvement in survival to hospital discharge.

4.6.2 2015 Recommendation—New

It is reasonable to administer epinephrine in pediatric cardiac arrest. (Class IIa, LOE C-LD)

4.7 Amiodarone and Lidocaine for Shock-Refractory VF and pVT - Updated PEDS 825

The 2005 and 2010 Guidelines recommended administering amiodarone in preference to lidocaine for the management of VF or pVT. This recommendation was based predominantly on pediatric case series or extrapolation from adult studies that used short-term outcomes.

4.7.1 2015 Evidence Summary

New pediatric observational data[203] showed improved ROSC with the use of lidocaine as compared with amiodarone. Use of lidocaine compared with no lidocaine was significantly associated with an increased likelihood of ROSC. The same study did not show an association between lidocaine or amiodarone use and survival to hospital discharge.

4.7.2 2015 Recommendation—New

For shock-refractory VF or pVT, either amiodarone or lidocaine may be used. (Class IIb, LOE C-LD)

The Pediatric Cardiac Arrest Algorithm (Figure 1) reflects this change.

4.8 Defibrillators (2015/2010)

Defibrillators are either manual or automated (AED), with monophasic or biphasic waveforms.

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

The following should be considered when using a manual defibrillator:

4.8.1 Paddle Size

In general, manual defibrillators have two sizes of hand-held paddles: adult and infant. The infant paddles may slide over or be located under the adult paddles. Manual defibrillators can also be used with hands-free pads that are self adhesive. Use the largest paddles or self-adhering electrodes[204-206] that will fit on the child's chest without touching (when possible, leave about 3 cm between the paddles or electrodes). Paddles and self-adhering pads appear to be equally effective.[207] Self-adhering pads should be pressed firmly on the chest so that the gel on the pad completely touches the child's chest. An appropriate paddle or self-adhesive pad size is

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

"Infant" size for infants <10 kg

4.8.2 Interface

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

4.8.3 Paddle Position

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

Place manual paddles over the right side of the upper chest and the apex of the heart (to the left of the nipple over the left lower ribs) so the heart is between the two paddles. Apply firm pressure. There is no advantage in an anterior-posterior position of the paddles.[207]

4.8.4 Energy Doses for Defibrillation - Updated PEDS 405

The 2015 ILCOR systematic review addressed the dose of energy for pediatric manual defibrillation during cardiac arrest. Neither the energy dose specifically related to automated external defibrillators, nor the energy dose for cardioversion was evaluated in this evidence review.

4.8.4.1 2015 Evidence Summary

Two small case series demonstrated termination of VF/pVT with either 2 J/kg[208] or 2 to 4 J/kg.[209] In 1 observational study of IHCA,[210] a higher initial energy dose of more than 3 to 5 J/kg was less effective than 1 to 3 J/kg in achieving ROSC. One small observational study of IHCA[211] showed no benefit in achieving ROSC with a specific energy dose for initial defibrillation. Three small observational studies of IHCA and OHCA[209,211,212] showed no survival to discharge advantage of any energy dose compared with 2 to 4 J/kg for initial defibrillation.

4.8.4.2 2015 Recommendations—Updated

It is reasonable to use an initial dose of 2 to 4 J/kg of monophasic or biphasic energy for defibrillation (Class IIa, LOE C-LD), but for ease of teaching, an initial dose of 2 J/kg may be considered. (Class IIb, LOE C-EO)

For refractory VF, it is reasonable to increase the dose to 4 J/kg. (Class IIa, LOE C-LD)

For subsequent energy levels, a dose of 4 J/kg may be reasonable and higher energy levels may be considered, though not to exceed 10 J/kg or the adult maximum dose. (Class IIb, LOE C-LD)

4.8.5 AEDs

Many AEDs can accurately detect VF in children of all ages.[213,214-216] They can differentiate “shockable” from “nonshockable” rhythms with a high degree of sensitivity and specificity.[214,215] It is recommended that systems and institutions that have AED programs and that care for children should use AEDs with a high specificity to recognize pediatric shockable rhythms and a pediatric attenuating system that can be used for infants and children up to approximately 25 kg (approximately 8 years of age).[217,218]

If an AED with an attenuator is not available, use an AED with standard electrodes. (Class IIa, LOE C)

In infants <1 year of age a manual defibrillator is preferred. If a manual defibrillator is not available, an AED with a dose attenuator may be used.

An AED without a dose attenuator may be used if neither a manual defibrillator nor one with a dose attenuator is available. (Class IIb, LOE C)

4.8.6 Integration of Defibrillation With Resuscitation Sequence

Please refer to Figure 1: Pediatric Cardiac Arrest Algorithm—2015 Update.

The following are important considerations:

Provide CPR until the defibrillator is ready to deliver a shock; after shock delivery, resume CPR, beginning with chest compressions. Minimize interruptions of chest compressions. In adults with prolonged arrest[219,220] and in animal models,[221] defibrillation is more likely to be successful after a period of effective chest compressions. Ideally chest compressions should be interrupted only for ventilations (until an advanced airway is in place), rhythm check, and shock delivery. If a “shockable” rhythm is still present, continue chest compressions after a

rhythm check (when possible) while the defibrillator is charging (so chest compressions are delivered until shock delivery).

(Step 3) Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR, beginning with chest compressions. If 1 shock fails to eliminate VF, the incremental benefit of another immediate shock is low, and resumption of CPR is likely to confer a greater value than another shock. CPR may provide coronary perfusion, increasing the likelihood of defibrillation with a subsequent shock. It is important to minimize the time between chest compressions and shock delivery and between shock delivery and resumption of postshock compressions.

(Step 4) Continue CPR for about 2 minutes. In in-hospital settings with continuous invasive monitoring, this sequence may be modified at the expert provider's discretion. If sufficient rescuers are present, obtain vascular (IO or IV) access.

After 2 minutes of CPR, check the rhythm; recharge the defibrillator to a higher dose (4 J/kg).

(Step 5) If a "shockable" rhythm persists, give another shock (4 J/kg). If rhythm is "nonshockable," continue with the asystole/PEA algorithm (Steps 10 and 11).

(Step 6) Immediately resume chest compressions. Continue CPR for approximately 2 minutes. During CPR give epinephrine 0.01 mg/kg (0.1 mL/kg of 1:10 000 concentration), maximum of 1 mg every 3 to 5 minutes. (Class I, LOE B)

It is helpful if a third rescuer prepares the drug doses *before* the rhythm is checked so epinephrine can be administered as soon as possible. Epinephrine should be administered during chest compressions, but the timing of drug administration is less important than the need to minimize interruptions in chest compressions. Just prior to the rhythm check, the rescuer operating the defibrillator should prepare to recharge the defibrillator (4 J/kg or more with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower).

Check the rhythm

(Step 7) If the rhythm is "shockable," deliver another shock (4 J/kg or more with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower) and immediately resume CPR (beginning with chest compressions).

(Step 8) While continuing CPR, give either amiodarone or lidocaine.

If at any time the rhythm check shows a "nonshockable" rhythm, proceed to the "Pulseless Arrest" sequence (Steps 10 or 11).

Once an advanced airway is in place, 2 rescuers no longer deliver cycles of CPR (ie, compressions interrupted by pauses for ventilation). Instead, the compressing rescuer gives continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The rescuer delivering ventilation provides about 1 breath every 6 seconds (10 breaths per minute). Two or more rescuers should rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions.

If defibrillation successfully restores an organized rhythm (or there is other evidence of ROSC, such as an abrupt rise in $ETCO_2$ or visible pulsations on an arterial waveform), check the child's pulse to determine if a perfusing rhythm is present. If a pulse is present, continue with postresuscitation care.

If defibrillation is successful but VF recurs, resume CPR and give another bolus of amiodarone before trying to defibrillate with the previously successful shock dose.

Search for and treat reversible causes

4.8.7 Torsades de Pointes

This polymorphic VT is associated with a long QT interval, which may be congenital or may result from toxicity with type IA antiarrhythmics (eg, procainamide, quinidine, and disopyramide) or type III antiarrhythmics (eg, sotalol and amiodarone), tricyclic antidepressants (see below), digitalis, or drug interactions.[222,223]

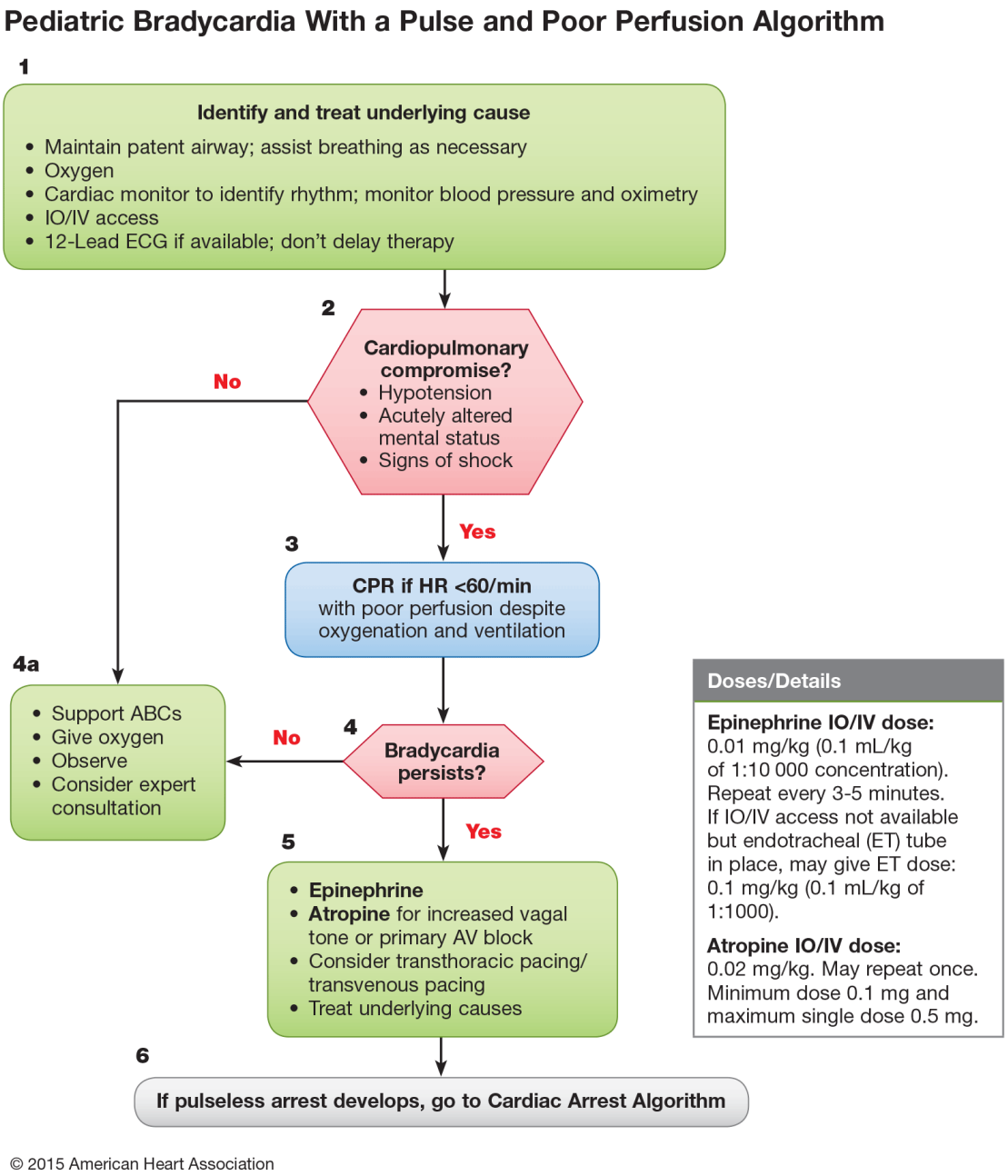
4.8.7.1 Treatment

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

4.8.8 Bradycardia

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

Figure 2: Pediatric Bradycardia With a Pulse and Poor Perfusion Algorithm



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

(**Box 1**) Support a patent airway, breathing, and circulation as needed. Administer oxygen, attach an ECG monitor/defibrillator, and obtain vascular access.

(**Box 2**) Reassess the patient to determine if bradycardia persists and is still causing cardiorespiratory symptoms despite adequate oxygenation and ventilation.

(**Box 4a**) If pulses, perfusion, and respirations are adequate, no emergency treatment is necessary. Monitor and proceed with evaluation.

(**Box 3**) If heart rate is <60 beats per minute with poor perfusion despite effective ventilation with oxygen, start CPR.

(**Box 4**) After 2 minutes reevaluate the patient to determine if bradycardia and signs of hemodynamic compromise persist. Verify that the support is adequate (eg, check airway, oxygen source, and effectiveness of ventilation).

(**Box 5**) Medications and pacing:

Continue to support airway, ventilation, oxygenation, and chest compressions. (Class I, LOE B)

If bradycardia persists or responds only transiently, give epinephrine IV (or IO) 0.01 mg/kg (0.1 mL/kg of 1:10 000 solution) or if IV/IO access not available, give endotracheally 0.1 mg/kg (0.1 mL/kg of 1:1 000 solution). (Class I, LOE B)

If bradycardia is due to increased vagal tone or primary AV conduction block (ie, not secondary to factors such as hypoxia), give IV/IO atropine 0.02 mg/kg or an endotracheal dose of 0.04 to 0.06 mg/kg. (Class I, LOE C)

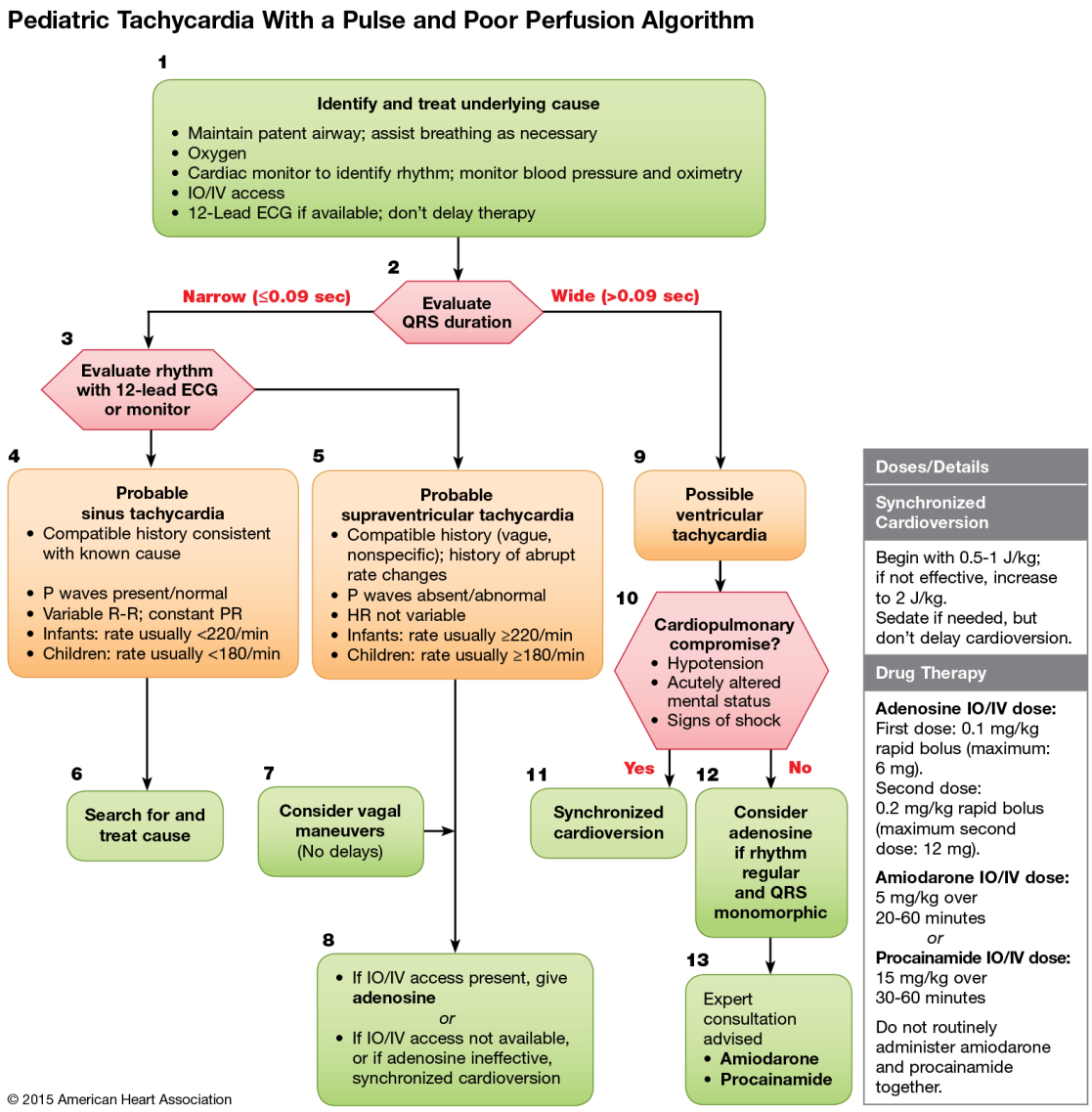
Emergency transcutaneous pacing may be lifesaving if the bradycardia is due to complete heart block or sinus node dysfunction unresponsive to ventilation, oxygenation, chest compressions, and medications, especially if it is associated with congenital or acquired heart disease. (Class IIb, LOE C)

Pacing is not useful for asystole[224,225] or bradycardia due to postarrest hypoxic/ischemic myocardial insult or respiratory failure.

4.8.9 Tachycardia

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

Figure 3: Pediatric Tachycardia With a Pulse and Poor Perfusion Algorithm



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

(Figure 3 **Box 1**) If pulses are palpable and the patient has adequate perfusion

–Assess and support airway, breathing, and circulation

–Provide oxygen.

–Attach monitor/defibrillator.

–Obtain vascular access.

–Evaluate 12-lead ECG and assess QRS duration (Figure 3 **Box 2**).

4.8.9.1 Narrow-Complex (?0.09 Second) Tachycardia

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

4.8.9.2 Supraventricular Tachycardia (Box 5)

Monitor rhythm during therapy to evaluate the effect of interventions. The choice of therapy is determined by the patient's degree of hemodynamic instability.

Attempt vagal stimulation (Box 7) first, unless the patient is hemodynamically unstable or the procedure will unduly delay chemical or electric cardioversion. (Class IIa, LOE C)

In infants and young children, apply ice to the face without occluding the airway.[226,227]

In older children, carotid sinus massage or Valsalva maneuvers are safe.[228-230]

One method for performing a Valsalva maneuver is to have the child blow through a narrow straw.[229]Do not apply pressure to the eye because this can damage the retina.

Pharmacologic cardioversion with adenosine (Box 8) is very effective with minimal and transient side effects.[231-235]

If IV/IO access is readily available, adenosine is the drug of choice. (Class I, LOE C)

Side effects are usually transient.[231-235]Administer IV/IO adenosine 0.1 mg/kg using 2 syringes connected to a T-connector or stopcock; give adenosine rapidly with 1 syringe and immediately flush with ?5 mL of normal saline with the other.

An IV/IO dose of Verapamil, 0.1 to 0.3 mg/kg is also effective in terminating SVT in older children, but it should not be used in infants without expert consultation because it may cause potential myocardial depression, hypotension, and cardiac arrest. (Class III, LOE C)

If the patient is hemodynamically unstable or if adenosine is ineffective, perform electric synchronized cardioversion (Box 8).

Use sedation, if possible. Start with a dose of 0.5 to 1 J/kg. If unsuccessful, increase the dose to 2 J/kg. (Class IIb, LOE C)

If a second shock is unsuccessful or the tachycardia recurs quickly, consider amiodarone or procainamide before a third shock.

Consider amiodarone 5 mg/kg IO/IV or procainamide 15 mg/kg IO/IV236 for a patient with SVT unresponsive to vagal maneuvers and adenosine and/or electric cardioversion; for hemodynamically stable patients, expert consultation is strongly recommended prior to administration. (Class IIb, LOE C)

Both amiodarone and procainamide must be infused slowly (amiodarone over 20 to 60 minutes and procainamide over 30 to 60 minutes), depending on the urgency, while the ECG and blood pressure are monitored. If there is no effect and there are no signs of toxicity, give additional doses (Table 3). Avoid the simultaneous use of amiodarone and procainamide without expert consultation.

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

Wide-complex tachycardia often originates in the ventricles (ventricular tachycardia) but may be supraventricular in origin.[236]

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

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

Adenosine may be useful in differentiating SVT from VT and converting wide-complex tachycardia of supraventricular origin (Box 12). Adenosine should be considered only if the rhythm is regular and the QRS is monomorphic. Do not use adenosine in patients with known Wolff-Parkinson-White syndrome and wide-complex tachycardia.

Consider electric cardioversion after sedation using a starting energy dose of 0.5 to 1 J/kg. If that fails, increase the dose to 2 J/kg (Box 11). (Class IIb, LOE C)

Consider pharmacologic conversion with either intravenous amiodarone (5 mg/kg over 20 to 60 minutes) or procainamide (15 mg/kg given over 30 to 60 minutes) while monitoring ECG and blood pressure. Stop or slow the infusion if there is a decline in blood pressure or the QRS widens (Box 13). Expert consultation is strongly recommended prior to administration.

In hemodynamically unstable patients:

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

4.9 Special Resuscitation Situations

4.9.1 Trauma

Some aspects of trauma resuscitation require emphasis because improperly performed resuscitation is a major cause of preventable pediatric deaths.[237]

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

The following are special aspects of trauma resuscitation:

When the mechanism of injury is compatible with cervical spinal injury, restrict motion of the cervical spine and avoid traction or movement of the head and neck. Open and maintain the airway with a jaw thrust, and do not tilt the head.

If the airway cannot be opened with a jaw thrust, use a head tilt–chin lift because you must establish a patent airway. Because of the disproportionately large head of infants and young children, optimal positioning may require recessing the occiput[238] or elevating the torso to avoid undesirable backboard-induced cervical flexion.[238,239]

Do not routinely hyperventilate even in case of head injury. (Class III, LOE C)

Intentional brief hyperventilation may be used as a temporizing rescue therapy if there are signs of impending brain herniation (eg, sudden rise in measured intracranial pressure, dilation of one or both pupils with decreased response to light, bradycardia, and hypertension).

Suspect thoracic injury in all thoraco-abdominal trauma, even in the absence of external injuries. Tension pneumothorax, hemothorax, or pulmonary contusion may impair oxygenation and ventilation.

If the patient has maxillofacial trauma or if you suspect a basilar skull fracture, insert an orogastric rather than a nasogastric tube. (Class IIa, LOE C)

In the very select circumstances of children with cardiac arrest from penetrating trauma with short transport times, consider performing resuscitative thoracotomy. (Class IIb, LOE C)

Consider intra-abdominal hemorrhage, tension pneumothorax, pericardial tamponade, and spinal cord injury in infants and children, and intracranial hemorrhage in infants, as causes of shock.[240,241]

4.9.2 Single Ventricle

Standard prearrest and arrest resuscitation procedures should be followed for infants and children with single ventricle anatomy following Stage I palliation or in the infant or neonate with a univentricular heart and a shunt to augment pulmonary blood flow. Heparin may be considered for infants with a systemic-pulmonary artery shunt or right ventricular-pulmonary artery shunt. Following resuscitation from cardiac arrest, oxygen administration should be adjusted to balance systemic and pulmonary blood flow, targeting an oxyhemoglobin saturation (SpO_2) of approximately 80%. End-tidal CO_2 ($ETCO_2$) in the single-ventricle patient during cardiac arrest may not be a reliable indicator of CPR quality because pulmonary blood flow changes rapidly and does not necessarily reflect cardiac output during CPR.[242]

Neonates in a prearrest state due to elevated pulmonary-to-systemic flow ratio prior to Stage I repair might benefit from a PaCO2 of 50 to 60 mm Hg, which can be achieved during mechanical ventilation by reducing minute ventilation, increasing the inspired fraction of CO2, or administering opioids with or without chemical paralysis. (Class IIb, LOE B)

Neonates in a low cardiac output state following stage I repair may benefit from systemic vasodilators such as ?-adrenergic antagonists (eg, phenoxybenzamine) to treat or ameliorate increased systemic vascular resistance, improve systemic oxygen delivery, and reduce the likelihood of cardiac arrest. (Class IIa, LOE B)

Other drugs that reduce systemic vascular resistance (eg, milrinone or nipride) may also be considered for patients with excessive Qp:Qs. (Class IIa, LOE B)

Following Stage I repair, evaluation of oxygen delivery and extraction (eg, using central venous oxygen saturation [ScvO2] and near-infrared spectroscopy) may help identify evolving changes in hemodynamics that may herald impending cardiac arrest.[243-245]

During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with single ventricle anatomy who have undergone Stage I procedure. (Class IIa, LOE B)

Hypoventilation may improve oxygen delivery in patients in a prearrest state with Fontan or hemi-Fontan/bidirectional Glenn (BDG) physiology. (Class IIa, LOE B)

Negative-pressure ventilation may improve cardiac output. (Class IIa, LOE C)

During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with Fontan physiology. (Class IIa, LOE C)

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

4.9.3 Pulmonary Hypertension

Standard PALS, including oxygenation and ventilation, should be provided to patients with pulmonary hypertension and a cardiopulmonary arrest. It may be beneficial to attempt to correct hypercarbia. Administration of a bolus of isotonic fluid may be useful to maintain preload to the systemic ventricle.

If intravenous or inhaled therapy to decrease pulmonary hypertension has been interrupted, reinstitute it. (Class IIa, LOE C)

Consider administering inhaled nitric oxide (iNO) or aerosolized prostacyclin or analogue to reduce pulmonary vascular resistance. (Class IIa, LOE C))

If iNO is not available, consider giving an intravenous bolus of prostacyclin. (Class IIa, LOE C)

ECMO may be beneficial if instituted early in the resuscitation. (Class IIa, LOE C)

4.10 Family Presence During Resuscitation

Family presence during CPR is increasingly common, and most parents would like to be given the opportunity to be present during resuscitation of their child.[246-255] Studies show that family members who are present at a resuscitation would recommend it to others.[246,247,249,255,256] Parents of chronically ill children are comfortable with medical equipment and emergency procedures, but even family members with no medical background who were at the side of a loved one to say goodbye during the final moments of life believe that their presence was beneficial to the patient,[246-248,250] comforting for them,[246-249,252-255,257] and helpful in their adjustment[247-249,256,258,257,259] and grieving process.[259] Standardized psychological examinations suggest that, compared with those not present, family members present during attempted resuscitations have less anxiety and depression and more constructive grieving behavior.[259] Parents or family members often fail to ask, but healthcare providers should offer the opportunity in most situations.[260,261,262]

Whenever possible, provide family members with the option of being present during resuscitation of an infant or child) (Class I, LOE B)

Family presence during resuscitation, in general, is not disruptive,[248,256,263,257,264,265] and does not create stress among staff or negatively affect their performance.[246,248,264,266]

If the presence of family members creates undue staff stress or is considered detrimental to the resuscitation, then family members should be respectfully asked to leave. (Class IIa, LOE C)

Members of the resuscitation team must be sensitive to the presence of family members, and one person should be assigned to remain with the family to comfort, answer questions, and support the family.[267]

4.11 Termination of Resuscitative Efforts

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

Clinical variables associated with survival include length of CPR, number of doses of epinephrine, age, witnessed versus unwitnessed cardiac arrest, and the first and subsequent rhythm.

[144,268,269-143,271,272,273,274-278] None of these associations, however, predict outcome. Witnessed collapse, bystander CPR, and a short interval from collapse to arrival of professionals improve the chances of a successful resuscitation. Intact survival has been documented after unusually prolonged in-hospital resuscitation.[143,279,280,273,281,282]

5 Postarrest Care Updates - Updated

5.1 Postresuscitation Stabilization (Post Cardiac Arrest Care) - Updated

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

5.1.1 Respiratory System - Updated

5.1.1.1 Post–Cardiac Arrest Oxygenation - Updated PEDS 544

Animal studies suggest that elevated levels of tissue Po2 after ROSC (hyperoxia) contribute to oxidative stress that may potentiate the postresuscitation syndrome, while some adult studies show associations between hyperoxemia and increased mortality.[283,284]

5.1.1.1.1 2015 Evidence Summary

Three small observational studies of pediatric IHCA and OHCA survivors[285-287] did not show an association between elevated Pao2 and outcome. In a larger observational study of 1427 pediatric IHCA and OHCA victims who survived to pediatric ICU admission,[288] after adjustment of confounders, the presence of normoxemia (defined as a Pao2 60 mmHg or greater and less than 300 mmHg) when compared with hyperoxemia (Pao2 greater than 300 mmHg) after ROSC was associated with improved survival to pediatric ICU discharge.

5.1.1.1.2 2015 Recommendations—New

It may be reasonable for rescuers to target normoxemia after ROSC. (Class IIb, LOE B-NR)

Because an arterial oxyhemoglobin saturation of 100% may correspond to a Pao_2 anywhere between 80 and approximately 500 mmHg, it may be reasonable—when the necessary equipment is available—for rescuers to wean oxygen to target an oxyhemoglobin saturation of less than 100%, but 94% or greater. The goal of such an approach is to achieve normoxemia while ensuring that hypoxemia is strictly avoided. Ideally, oxygen is titrated to a value appropriate to the specific patient condition.

5.1.1.2 Post–Cardiac Arrest $Paco_2$ - Updated PEDS 815

Cerebral vascular autoregulation may be abnormal after ROSC. Adult data show an association between post-ROSC hypocapnia and worse patient outcomes.[289,290] In other types of pediatric brain injury, hypocapnia is associated with worse clinical outcomes.[291-294]

5.1.1.2.1 2015 Evidence Summary

There were no studies in children after cardiac arrest specifically comparing ventilation with a predetermined Paco2 target. One small observational study of both pediatric IHCA and OHCA[285] demonstrated no association between hypercapnia (Paco2 greater than 50 mmHg) or hypocapnia (Paco2 less than 30 mmHg) and outcome. However, in an observational study of pediatric IHCA,[287] hypercapnia (Paco2 50 mmHg or greater) was associated with worse survival to hospital discharge.

5.1.1.2.2 2015 Recommendation-Updated

It is reasonable for practitioners to target a Paco2 after ROSC that is appropriate to the specific patient condition, and limit exposure to severe hypercapnia or hypocapnia. (Class IIb, LOE C-LD)

Monitor exhaled CO2 (ETCO2), especially during transport and diagnostic procedures. (Class IIa, LOE B)

5.1.2 Cardiovascular System - Updated

Monitor heart rate and blood pressure. Repeat clinical evaluations at frequent intervals until the patient is stable. Consider monitoring urine output with an indwelling catheter. A 12-lead ECG may be helpful in establishing the cause of the cardiac arrest.

Remove the IO access after alternative (preferably 2) secure venous catheters are placed. Monitor venous or arterial blood gas analysis and serum electrolytes, glucose, and calcium concentrations. A chest x-ray should be performed to evaluate endotracheal tube position, heart size, and pulmonary status. Consider obtaining arterial lactate and central venous oxygen saturation to assess adequacy of tissue oxygen delivery.

5.1.2.1 Post–Cardiac Arrest Fluids and Inotropes - Updated PEDS 820

Myocardial dysfunction and vascular instability are common after resuscitation from cardiac arrest.[295-301]

5.1.2.1.1 2015 Evidence Summary

Three small observational studies involving pediatric IHCA and OHCA[302-304] demonstrated worse survival to hospital discharge when children were exposed to post-ROSC hypotension. One of these studies[302] associated post-ROSC hypotension (defined as a systolic blood pressure less than fifth percentile for age) after IHCA with lower likelihood of survival to discharge with favorable neurologic outcome. There are no studies evaluating the benefit of specific vasoactive agents after ROSC in infants and children.

5.1.2.1.2 2015 Recommendations—New

After ROSC, we recommend that parenteral fluids and/or inotropes or vasoactive drugs be used to maintain a systolic blood pressure greater than fifth percentile for age. (Class I, LOE C-LD)

When appropriate resources are available, continuous arterial pressure monitoring is recommended to identify and treat hypotension. (Class I, LOE C-EO)

5.1.2.2 Drugs Used to Maintain Cardiac Output

Table 4: 2010 - Medications to Maintain Cardiac Output and for Postresuscitation Stabilization

Open table in a new window

Medications to Maintain Cardiac Output and for Postresuscitation Stabilization		
Medication	**Dose Range**	**Comment**
Inamrinone	0.75–1 mg/kg IV/IO over 5 minutes; may repeat × 2 then: 5–10 mcg/kg per minute	Inodilator
Dobutamine	2–20 mcg/kg per minute IV/IO	Inotrope; vasodilator

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

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

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

Systemic and pulmonary vascular resistances are often increased initially, except in some cases of septic shock.[305] The postarrest effects on the cardiovascular system may evolve over time, with an initial hyperdynamic state replaced by worsening cardiac function. Therefore in infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest, it is reasonable to administer vasoactive drugs titrated to improve myocardial function and organ perfusion.

5.1.2.2.1 Epinephrine

Low-dose infusions (<0.3 mcg/kg per minute) generally produce ?-adrenergic actions (tachycardia, potent inotropy, and decreased systemic vascular resistance). Higher-dose infusions (>0.3 mcg/kg per minute) cause ?-adrenergic vasoconstriction.[306,307] Because there is great interpatient variability in response,[308,309] titrate the drug to the desired effect. Epinephrine or norepinephrine may be preferable to dopamine in patients (especially infants) with marked circulatory instability and decompensated shock.[310]

5.1.2.2.2 Dopamine

Dopamine can produce direct dopaminergic effects and indirect ?- and ?-adrenergic effects through stimulation of norepinephrine release.

Titrate dopamine to treat shock that is unresponsive to fluids and when systemic vascular resistance is low. (Class IIb, LOE C)

Titrate dopamine to treat shock that is unresponsive to fluids and when systemic vascular resistance is low (

***Class IIb, LOE C*)**.[420,435]

Typically a dose of 2 to 20 mcg/kg per minute is used. Although low-dose dopamine infusion has been frequently recommended to maintain renal blood flow or improve renal function, data do not show benefit from such therapy.[311,312] At higher doses (>5 mcg/kg per minute), dopamine stimulates cardiac ?-adrenergic receptors, but this effect may be reduced in infants and in patients with chronic congestive heart failure. Infusion rates >20 mcg/kg per minute may result in excessive vasoconstriction.[306,307] In one study in single ventricle postoperative cardiac patients, dopamine increased oxygen consumption while not improving blood pressure or cardiac output.[313]

5.1.2.2.3 Dobutamine Hydrochloride

Dobutamine has a relatively selective effect on ?1- and ?2-adrenergic receptors due to effects of the two isomers; one is an ?-adrenergic agonist, and the other is an ?-adrenergic antagonist.[314]Dobutamine increases myocardial contractility and can decrease peripheral vascular resistance. Titrate the infusion[308,315,316] to improve cardiac output and blood pressure due to poor myocardial function.[316]

5.1.2.2.4 Norepinephrine

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

5.1.2.2.5 Sodium Nitroprusside

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

5.1.2.2.6 Inodilators

Inodilators (inamrinone and milrinone) augment cardiac output with little effect on myocardial oxygen demand.

It is reasonable to use an inodilator in a highly monitored setting for treatment of myocardial dysfunction with increased systemic or pulmonary vascular resistance. (Class IIa, LOE B)

Administration of fluids may be required secondary to vasodilatory effects.

Inodilators have a long half-life with a delay in reaching a steady-state hemodynamic effect after the infusion rate is changed (18 hours with inamrinone and 4.5 hours with milrinone). In cases of toxicity the cardiovascular effects may persist for several hours even after the infusion is discontinued.

5.1.3 Neurologic System - Updated

5.1.3.1 Post–Cardiac Arrest Temperature Management - Updated PEDS 387

Data suggest that fever after pediatric cardiac arrest is common and is associated with poor outcomes.[317] The 2010 AHA PALS Guidelines suggested a role for targeted temperature management after pediatric cardiac arrest (fever control for all patients, therapeutic hypothermia for some patients), but the recommendations were based predominantly on extrapolation from adult and asphyxiated newborn data.

5.1.3.1.1 2015 Evidence Summary

A large multi-institutional, prospective, randomized study of pediatric patients (aged 2 days to 18 years) with OHCA found no difference in survival with good functional outcome at 1 year and no additional complications in comatose patients who were treated with therapeutic hypothermia (32°C to 34°C), compared to those treated with normothermia (36°C to 37.5°C).[318]Observational data of pediatric patients resuscitated from IHCA or OHCA [319,320] have also shown that ICU duration of stay, neurologic outcomes, and mortality are unchanged with the use of therapeutic hypothermia. Only 1 small study of therapeutic hypothermia in survivors of pediatric asphyxial cardiac arrest[321] showed an improvement in mortality at hospital discharge, but with no difference in neurologic outcomes. Results are pending from a large multicenter randomized controlled trial of targeted temperature management for pediatric patients with IHCA (see Therapeutic Hypothermia After Cardiac Arrest).

5.1.3.1.2 2015 Recommendations—New

For infants and children remaining comatose after OHCA, it is reasonable either to maintain 5 days of continuous normothermia (36°C to 37.5°C) or to maintain 2 days of initial continuous hypothermia (32°C to 34°C) followed by 3 days of continuous normothermia. (Class IIa, LOE B-R)

Continuous measurement of temperature during this time period is recommended. (Class I, LOE B-NR)

For infants and children remaining comatose after IHCA, there is insufficient evidence to recommend cooling over normothermia.

Fever (temperature 38°C or higher) should be aggressively treated after ROSC. (Class I, LOE B-NR)

5.1.4 Renal System

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

5.2 Prognostication - Updated

5.2.1 Postresuscitation Use of EEG for Prognosis - Updated PEDS 822

Early and reliable prognostication of neurologic outcome in pediatric survivors of cardiac arrest is essential to enable effective planning and family support (whether it be to continue or discontinue life-sustaining therapy).

5.2.1.1 2015 Evidence Summary

Observational data from 2 small pediatric studies[322,323] showed that a continuous and reactive tracing on an EEG performed in the first 7 days after cardiac arrest was associated with a significantly higher likelihood of good neurologic outcome at hospital discharge, while an EEG demonstrating a discontinuous or isoelectric tracing was associated with a poorer neurologic outcome at hospital discharge. There are no data correlating EEG findings with neurologic outcome after hospital discharge.

5.2.1.2 2015 Recommendation—New

EEGs performed within the first 7 days after pediatric cardiac arrest may be considered in prognosticating neurologic outcome at the time of hospital discharge but should not be used as the sole criterion. (Class IIb, LOE C-LD)

5.2.2 Predictive Factors After Cardiac Arrest - Updated PEDS 813

Several post-ROSC factors have been studied as possible predictors of survival and neurologic outcome after pediatric cardiac arrest. These include pupillary responses, the presence of hypotension, serum neurologic biomarkers, and serum lactate.

5.2.2.1 2015 Evidence Summary

Four observational studies supported the use of pupillary reactivity at 12 to 24 hours after cardiac arrest in predicting survival to discharge,[193,197,323,324] while 1 observational study found that reactive pupils 24 hours after cardiac arrest were associated with improved survival at 180 days with favorable neurologic outcome.[325]

Several serum biomarkers of neurologic injury have been considered for their prognostic value. Two small

observational studies found that lower neuron-specific enolase and S100B serum levels after arrest were associated with improved survival to hospital discharge and with improved survival with favorable neurologic outcome.[325,326]

One observational study found that children with lower lactate levels in the first 12 hours after arrest had an improved survival to hospital discharge.[327]

5.2.2.2 2015 Recommendation—New

The reliability of any 1 variable for prognostication in children after cardiac arrest has not been established. Practitioners should consider multiple factors when predicting outcomes in infants and children who achieve ROSC after cardiac arrest. (Class I, LOE C-LD)

5.3 Interhospital Transport

Ideally postresuscitation care should be provided by a trained team from a pediatric tertiary care facility. Contact such a team as early as possible during the resuscitation attempt and coordinate transportation with the receiving unit.[328]Transport team members should be trained and experienced in the care of critically ill and injured children[131,329] and supervised by a pediatric emergency medicine or pediatric critical care physician. The mode of transport and composition of the team should be established for each system based on the care required by each patient.[330]

Monitor exhaled CO2 (qualitative colorimetric detector or capnography) during interhospital or intrahospital transport of intubated patients. (Class IIa, LOE B)

5.4 Sudden Unexplained Deaths

Increasing evidence demonstrates that some cases of sudden infant death syndrome (SIDS) and sudden death in older children and young adults may be associated with genetic mutations causing cardiac ion channelopathies. Channelopathies are dysfunctional myocyte ion channels that result in abnormal movement of electrolytes into and/or out of the cell and predispose the heart to arrhythmia.[331-340] Mutations causing cardiac ion channelopathies are found in 2% to 10% of victims[331-337] and in 14% to 20% of young adults with sudden death in whom the cause of death is not evident in a routine autopsy.[338-340] Clinical and laboratory (eg, ECG, molecular-genetic screening) investigations of first- and second-degree relatives of patients with sudden unexplained death reported inherited, arrhythmogenic disease in 22% to 53% of families.[341-344]

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

All infants, children, and young adults with sudden unexpected death should, where resources allow, have an unrestricted, complete autopsy, preferably performed by a pathologist with training and experience in cardiovascular pathology. Consider appropriate preservation and genetic analysis of tissue to determine the presence of a channelopathy.

Refer families of patients that do not have a cause of death found on autopsy to a healthcare provider or center with expertise in arrhythmias. (Class I, LOE C)

6 Emergency Fluids and Medications

6.1 Estimating Weight

In the out-of-hospital setting, a child's weight is often unknown, and even experienced personnel may not be able to estimate it accurately.[108]Tapes with precalculated doses printed at various patient lengths have been clinically validated[108,111,127] and are more accurate than age-based or observer (parent or provider) estimate-based methods in the prediction of body weight.[104-111] Body habitus may also be an important consideration.[104,106,112,113]

6.2 Medication Dose Calculation

To calculate the dose of resuscitation medications, use the child's weight if it is known.

If the child's weight is unknown, it is reasonable to use a body length tape with precalculated doses. (Class IIa, LOE C)

It is unclear if an adjustment in the calculation of resuscitation medications is needed in obese children. Use of the actual body weight in calculation of drug doses in obese patients may result in potentially toxic doses. Length-based tapes estimate the 50th percentile weight for length (ie, ideal body weight), which may, theoretically, result in inadequate doses of some medications in obese patients. Despite these theoretical considerations, there are no data regarding the safety or efficacy of adjusting the doses of resuscitation medications in obese patients.

Therefore, regardless of the patient's habitus, use the actual body weight for calculating initial resuscitation drug doses or use a body length tape with precalculated doses. (Class IIb, LOE C)

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

6.3 Medications

See Table 3

Table 3: 2010 - Medications for Pediatric Resuscitation

Open table in a new window

Medications for Pediatric Resuscitation		
Medication	**Dose**	**Remarks**
Adenosine	0.1 mg/kg (maximum 6 mg) Second dose: 0.2 mg/kg (maximum 12 mg)	Monitor ECG Rapid IV/IO bolus with flush
Amiodarone	5 mg/kg IV/IO; may repeat twice up to 15 mg/kg Maximum single dose 300 mg	Monitor ECG and blood pressure; adjust administration rate to urgency (IV push during cardiac arrest, more slowly–over 20–60 minutes with perfusing rhythm). Expert consultation strongly recommended prior to use when patient has a perfusing rhythmUse caution when administering with other drugs that prolong QT (obtain expert consultation)
Atropine	0.02 mg/kg IV/IO 0.04–0.06 mg/kg ET* Repeat once if neededMaximum single dose: 0.5 mg	Higher doses may be used with organophosphate poisoning
Calcium Chloride (10%)	20 mg/kg IV/IO (0.2 mL/kg) Maximum single dose 2 g	Administer slowly

Medication	Dose	Remarks
Epinephrine	0.01 mg/kg (0.1 mL/kg 1:10 000) IV/IO 0.1 mg/kg (0.1 mL/kg 1:1000) ET* Maximum dose 1 mg IV/IO; 2.5 mg ET	May repeat every 3–5 minutes
Glucose	0.5–1 g/kg IV/IO	Newborn: 5–10 mL/kg $D_{10}W$ Infants and Children: 2–4 mL/kg $D_{25}W$ Adolescents: 1–2 mL/kg $D_{50}W$
Lidocaine	Bolus: 1 mg/kg IV/IO Infusion: 20–50 mcg/kg/minute	
Magnesium Sulfate	25–50 mg/kg IV/IO over 10–20 minutes, faster in torsades de pointes Maximum dose 2 g	
Naloxone	Full Reversal: <5 y or ?20 kg: 0.1 mg/kg IV/IO/ET* ?5y or >20 kg: 2 mg IV/IO/ET*	Use lower doses to reverse respiratory depression associated with therapeutic opioid use (1–5 mcg/kg titrate to effect)
Procainamide	15 mg/kg IV/IO Adult Dose: 20 mg/min IV infusion to total maximum dose of 17 mg/kg	Monitor ECG and blood pressure; Give slowly–over 30–60 minutes. Use caution when administering with other drugs that prolong QT (obtain expert consultation)
Sodium bicarbonate	1 mEq/kg per dose IV/IO slowly	After adequate ventilation

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

6.3.1 Adenosine

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

6.3.2 Amiodarone

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

6.3.2.1 Precautions

Monitor blood pressure and electrocardiograph (ECG) during intravenous administration of amiodarone. If the patient has a perfusing rhythm, administer the drug as slowly (over 20 to 60 minutes) as the patient's clinical condition allows; if the patient is in VF/pulseless VT, give the drug as a rapid bolus. Amiodarone causes hypotension through its vasodilatory property, and the severity is related to the infusion rate; hypotension is less common with the aqueous form of amiodarone.[345] Decrease the infusion rate if there is prolongation of the QT interval or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Other potential complications of amiodarone include bradycardia and torsades de pointes ventricular tachycardia. Amiodarone should not be administered together with another drug that causes QT prolongation, such as procainamide, without expert consultation.

6.3.3 Atropine

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

6.3.3.1 Precautions

Larger than recommended doses may be required in special circumstances such as organophosphate poisoning [346] or exposure to nerve gas agents.

6.3.4 Calcium

Calcium administration is not recommended for pediatric cardiopulmonary arrest in the absence of documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia. (Class III, LOE B)

Routine calcium administration in cardiac arrest provides no benefit[347-358] and may be harmful.[347-349]

If calcium administration is indicated during cardiac arrest, either calcium chloride or calcium gluconate may be considered. Hepatic dysfunction does not appear to alter the ability of calcium gluconate to raise serum calcium levels.[359] In critically ill children, calcium chloride may be preferred because it results in a greater increase in ionized calcium during the treatment of hypocalcemia.[360] In the nonarrest setting, if the only venous access is peripheral, calcium gluconate is recommended because it has a lower osmolality than calcium chloride and is therefore less irritating to the vein.

6.3.5 Epinephrine

The ?-adrenergic-mediated vasoconstriction of epinephrine increases aortic diastolic pressure and thus coronary perfusion pressure, a critical determinant of successful resuscitation from cardiac arrest.[361,362] At low doses, the ?-adrenergic effects may predominate, leading to decreased systemic vascular resistance; in the doses used during cardiac arrest, the vasoconstrictive ?-effects predominate.

6.3.5.1 Precautions

Do not administer catecholamines and sodium bicarbonate simultaneously through an IV catheter or tubing because alkaline solutions such as the bicarbonate inactivate the catecholamines.

In patients with a perfusing rhythm, epinephrine causes tachycardia; it may also cause ventricular ectopy, tachyarrhythmias, vasoconstriction, and hypertension.

6.3.6 Glucose

Because infants have a relatively high glucose requirement and low glycogen stores, they may develop hypoglycemia when energy requirements rise.[363]

Check blood glucose concentration during the resuscitation and treat hypoglycemia promptly. (Class I, LOE C)

6.3.7 Lidocaine

Lidocaine decreases automaticity and suppresses ventricular arrhythmias.[364]

6.3.7.1 Precautions

Lidocaine toxicity includes myocardial and circulatory depression, drowsiness, disorientation, muscle twitching, and seizures, especially in patients with poor cardiac output and hepatic or renal failure.[365,366]

6.3.8 Magnesium

Magnesium is indicated for the treatment of documented hypomagnesemia or for torsades de pointes (polymorphic VT associated with long QT interval). There is insufficient evidence to recommend for or against the routine administration of magnesium during cardiac arrest.[367-369]

6.3.8.1 Precautions

Magnesium produces vasodilation and may cause hypotension if administered rapidly.

6.3.9 Procainamide

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

6.3.9.1 Precautions

There is limited clinical data on using procainamide in infants and children.[370-372] Infuse procainamide very slowly (over 30 to 60 minutes) while monitoring the ECG and blood pressure. Decrease the infusion rate if there is prolongation of the QT interval, or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Do not administer together with another drug causing QT prolongation, such as amiodarone, without expert consultation. Prior to using procainamide for a hemodynamically stable patient, expert consultation is strongly recommended.

6.3.10 Sodium Bicarbonate

Routine administration of sodium bicarbonate is not recommended in cardiac arrest. (Class III, LOE B)

Sodium bicarbonate may be administered for treatment of some toxidromes (see “Toxicological Emergencies,” below) or special resuscitation situations such as hyperkalemic cardiac arrest.

6.3.10.1 Precautions

During cardiac arrest or severe shock, arterial blood gas analysis may not accurately reflect tissue and venous acidosis.[373,374] Excessive sodium bicarbonate may impair tissue oxygen delivery;[375]cause hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality;[376,377] decrease the VF threshold;[378]and impair cardiac function.

6.3.11 Vasopressin

There is insufficient evidence to make a recommendation for or against the routine use of vasopressin during cardiac arrest. Pediatric[379-381] and adult[382,383] case series/reports suggested that vasopressin[379] or its long-acting analog, terlipressin,[380,381] may be effective in refractory cardiac arrest when standard therapy fails. A large pediatric NRCPR case series, however, suggested that vasopressin is associated with lower ROSC, and a trend toward lower 24-hour and discharge survival.[384]A preponderance of controlled trials in adults do not demonstrate a benefit.[385-390]

7 Authorship and Disclosures - Updated

7.1 2015 Writing Team

Allan R. de Caen, Chair; Marc D. Berg; Leon Chameides; Cheryl K. Gooden; Robert W. Hickey; Halden F. Scott; Robert M. Sutton; Janice A. Tijssen; Alexis Topjian; Élise W. van der Jagt; Stephen M. Schexnayder; Ricardo A. Samson

Table 5: Part 12: Pediatric Advanced Life Support: 2015 Guidelines Update Writing Group Disclosures

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Part 12: Pediatric Advanced Life Support: 2015 Guidelines Update Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honora	Expert Witness	Ownership Interest	Consultant/Advis Board	Other
Allan R. de Caen	University of Alberta Stollery Children's Hospital	None	None	None	None	None	None	None
Marc D. Berg	University of Arizona	None	None	None	None	None	None	None
Leon Chameides	Connecticut Children's Medical Center	None	None	None	None	None	None	None
Cheryl K. Gooden	Mount Sinai Medical Center	None	None	None	None	None	None	None
Robert W. Hickey	Children's Hospital of Pittsburgh	None	None	None	None	None	None	None
Halden F. Scott	Children's Hospital Colorado	None	None	None	None	None	None	None
Robert M. Sutton	The Children's Hospital of Philadelphia; University of Pennsylvania School of Medicine	NIH†	None	Zoll Medical Sales Meeting Lecture (Speaking Honoraria)*	Webber and Gallagher*	None	None	None
Janice A. Tijssen	London Health Services Center	AMOSO Opportunities Fund*	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honora	Expert Witness	Ownership Interest	Consultant/Advis Board	Other
Alexis Topjian	The Children's Hospital of Philadelphia; University of Pennsylvania School of Medicine	NIH†	None	None	Expert witness for defense and plantiff*	None	None	None
E?lise W. van der Jagt	University of Rochester School of Medicine	NHLBI*	None	None	None	None	None	None
Consultants								
Ricardo A. Samson	The University of Arizona	None	None	None	None	None	American Heart Association†	None
Stephen M. Schexnayder	University of Arkansas; Arkansas Children's Hospital	None	None	None	Arkansas Dept. of Human Services*; Lewis Thomason*; University of Chicago*	None	American Heart Association†	None

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

7.2 2010 Writing Team

Monica E. Kleinman, Chair; Leon Chameides; Stephen M. Schexnayder; Ricardo A. Samson; Mary Fran Hazinski; Dianne L. Atkins; Marc D. Berg; Allan R. de Caen; Ericka L. Fink; Eugene B. Freid; Robert W. Hickey; Bradley S. Marino; Vinay M. Nadkarni; Lester T. Proctor; Faiqa A. Qureshi; Kennith Sartorelli; Alexis Topjian; Elise W. van der Jagt; Arno L. Zaritsky

Table 6: 2010 - Guidelines Part 14: PALS Writing Group Disclosures

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2010 Guidelines Part 14: PALS Writing Group Disclosures

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Monica E. Kleinman	Children's Hospital Anesthesia Foundation–Senior Associate in Critical Care Medicine	None	None	None	None	None	None
Leon Chameides	Emeritus Director Pediatric Cardiology, Clinical Professor, University of Connecticut	None	None	None	None	None	None
Stephen M. Schexnayder	University of Arkansas for Medical Sciences—Professor/ Division Chief; †AHA Compensated Consultant as Associate Senior Science Editor	*Pharmacokinetics of Proton Pumps inhibitors in Critically Ill patients	None	None	None	None	*Expert witness in several cases involving pediatric critical care & emergency medicine
Ricardo A. Samson	The University of Arizona: clinical care, teaching and research related to the field of Pediatric Cardiology in academic setting- Professor	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Mary Fran Hazinski	Vanderbilt University School of Nursing—Professor; AHA ECC Product Development—Senior Science Editor- † Significant compensation as a paid AHA consultant to help develop and edit the 2010 AHA Guidelines for CPR and ECC.	None	None	None	None	None	None
Dianne L. Atkins	University of Iowa—Profess * Compensated worksheet editor for the 2010 AHA Guidelines. Money is divided 2/3 to my institution and 1/3 to me.	None	None	None	None	None	*Defense expert witness for episode of ventricular fibrillation in a 2 year old child. Attorney are Buckley and Thereoux of Princeton, New Jersey
Marc D. Berg	University of Arizona - Staff Intensivist; Asso. Prof. Clinical Pediatrics, Attending Intensivist, Pediatric Critical Care Medicine	None	None	Travel expenses defrayed with an honorarium of $4000 for speaking at 13th Asian Australasian Congress of Anesthesiologists, Fukuoka, Japan 6/2010	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Allan R. de Caen	Self employed, pediatric intensivist	None	None	None	None	None	*Medical expert for Canadian Medical ProtectiveAssoc
Ericka L. Fink	Children's Hospital of Pittsburgh of UPMC–Assistant Professor	†National Institutes of Health, NINDS K23, Laerdal Foundation, and Children's Hospital of Pittsburgh Clinical and Translational Science Institute grants to study duration of hypothermia after pediatric cardiac arrest.	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Eugene B. Freid	Nemours Childrens Clinics–Anesth and Intensivist	None	None	*$1500.00 from University of North Carolina to Nemours Childrens Clinics for 3 lectures at annual anesthesiology conference-lectures related to anesthesia management of patients with cancer, operating room ventilators & postoperative nausea/vomitir No direct conflicts with Pediatric Life support topics	None	None	None
Robert W. Hickey	University of Pittsburgh–Pediatric Emergency Medicine Physician	†NIH sponsored research on the effect of cyclopentenone prostaglandins upon post-ischemic brain.	None	None	None	None	*Occasional expert witness (1–2 times per year)
Bradley S. Marino	Cincinnati Children's Hospital Medical Center–Associ Professor of Pediatrics	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Vinay M. Nadkarni	University of Pennsylvania, Children's Hospital of Philadelphia–Attending Physician, Pediatric Critical Care	†NIH RO1: Coinvestigator, Therapeutic Hypothermia After Pediatric Cardiac Arrest Center of Excellence Grant, PI, Laerdal Foundation for Acute Care Medicine AHRQ: Agency for Healthcare Research and Quality: PI, Tracheal Intubation Safety in Pediatric ICUs * NHTSA: Coinvestigator, Chest compression characteristics in children	None	None	None	None	*Volunteer (no salary or remuneration), World Federation of Pediatric Intensive and Critical Care Societies Volunteer (no salary), Data Safety and Monitoring Board, CIRC study
Lester T. Proctor	University of Wisconsin-Madison College of Medicine and Public Health–Profes	None	None	None	None	None	None
Faiqa A. Qureshi	Children's Specialty Group—Partner	None	None	None	None	None	None
Kennith Sartorelli	University of Vermont–Assc Professor of Surgery	None	None	None	None	None	None

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Ownership Interest	Consultant/ Advisory Board	Other
Alexis Topjian	University of Pennsylvania–Assistant Professor	*Site principal investigator at the Children's hospital of Philadelphia for the "Therpaeutic Hypothermia after Pediatric Cardiac Arrest" funded via an NIH U01	None	None	None	None	None
Elise W. van der Jagt	University of Rochester–Prof of Pediatrics and Critical Care	None	None	None	None	None	None
Arno L. Zaritsky	Childen's Hospital of The King's Daughters-Sr. VP for Clinical Services	None	None	None	None	*Data Safety Monitoring Board for NIH-funded pediatric hypothermia after cardiac arrest research project	None

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

8 Footnotes

The American Heart Association requests that this document be cited as follows:

American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and

Emergency Cardiovascular Care – Part 12: Pediatric Advanced Life Support. ECCguidelines.heart.org.

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