Part 10: Pediatric Basic and Advanced Life Support

2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations

Monica E. Kleinman, Co-Chair*; Allan R. de Caen, Co-Chair*; Leon Chameides; Dianne L. Atkins; Robert A. Berg; Marc D. Berg; Farhan Bhanji; Dominique Biarent; Robert Bingham; Ashraf H. Coovadia; Mary Fran Hazinski; Robert W. Hickey; Vinay M. Nadkarni; Amelia G. Reis; Antonio Rodriguez-Nunez; James Tibballs; Arno L. Zaritsky; David Zideman; on behalf of the Pediatric Basic and Advanced Life Support Chapter Collaborators

Note From the Writing Group: *Throughout this article, the reader will notice combinations of superscripted letters and numbers (eg, "Family Presence During Resuscitation*[Peds-003]*"). These callouts are hyperlinked to evidence-based worksheets, which were used in the development of this article. An appendix of worksheets, applicable to this article, is located at the end of the text. The worksheets are available in PDF format and are open access.*

The 2010 ILCOR Pediatric Task Force experts developed 55 questions related to pediatric resuscitation. Topics were selected based on the 2005 Consensus on Science and Treatment Recommendations (CoSTR) document,[1,2] emerging science, and newly identified issues. Not every topic reviewed for the 2005 International Consensus on Science was reviewed in the 2010 evidence evaluation process. In general, evidence-based worksheets were assigned to at least 2 authors for each topic. The literature search strategy was first reviewed by a "worksheet expert" for completeness. The expert also approved the final worksheet to ensure that the levels of evidence were correctly assigned according to the established criteria. Worksheet authors were requested to draft CoSTR statements (see Part 3: Evidence Evaluation Process). Each worksheet author or pair of authors presented their topic to the Task Force in person or via a webinar conference, and Task Force members discussed the available science and revised the CoSTR draft accordingly. These draft CoSTR summaries were recirculated to the International Liaison Committee on Resuscitation (ILCOR) Pediatric Task Force for further refinement until consensus was reached. Selected controversial and critical topics were presented at the 2010 ILCOR International Evidence Evaluation conference in Dallas, Texas, for further discussion to obtain additional input and feedback. This document presents the 2010 international consensus on the science, treatment, and knowledge gaps for each pediatric question.

The most important changes or points of emphasis in the recommendations for pediatric resuscitation since the publication of the *2005 ILCOR International Consensus on CPR and ECC Science With Treatment Recommendations*[1,2] are summarized in the following list. The scientific evidence supporting these changes is detailed in this document.

- Additional evidence shows that healthcare providers do not reliably determine the presence or absence of a pulse in infants or children.
- New evidence documents the important role of ventilations in CPR for infants and children. However, rescuers who are unable or unwilling to provide ventilations should be encouraged to perform compression-only CPR.
- To achieve effective chest compressions, rescuers should compress at least one third the anterior-posterior dimension of the chest. This corresponds to approximately 1½ inches (4 cm) in most infants and 2 inches (5 cm) in most children.
- When shocks are indicated for ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) in infants and children, an initial energy dose of 2 to 4 J/kg is reasonable; doses higher than 4 J/kg, especially if delivered with a biphasic defibrillator, may be safe and effective.
- More data support the safety and effectiveness of cuffed tracheal tubes in infants and young children, and the formula for selecting the appropriately sized cuffed tube was updated.
- The safety and value of using cricoid pressure during emergency intubation are not clear. Therefore, the application of cricoid pressure should be modified or discontinued if it impedes ventilation or the speed or ease of intubation.

The American Heart Association requests that this document be cited as follows: Kleinman ME, de Caen AR, Chameides L, Atkins DL, Berg RA, Berg MD, Bhanji F, Biarent D, Bingham R, Coovadia AH, Hazinski MF, Hickey RW, Nadkarni VM, Reis AG, Rodriguez-Nunez A, Tibballs J, Zaritsky AL, Zideman D; on behalf of the Pediatric Basic and Advanced Life Support Chapter Collaborators. Part 10: pediatric basic and advanced life support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. *Circulation.* 2010;122(suppl 2):S466–S515.

*Co-chairs and equal first co-authors.

(*Circulation*. 2010;122[suppl 2]:S466–S515.)

Circulation **is available at http://circ.ahajournals.org** **DOI: 10.1161/CIRCULATIONAHA.110.971093**

- Monitoring capnography/capnometry is recommended to confirm proper endotracheal tube position.
- Monitoring capnography/capnometry may be helpful during CPR to help assess and optimize quality of chest compressions.
- On the basis of increasing evidence of potential harm from exposure to high-concentration oxygen after cardiac arrest, once spontaneous circulation is restored, inspired oxygen concentration should be titrated to limit the risk of hyperoxemia.
- Use of a rapid response system in a pediatric inpatient setting may be beneficial to reduce rates of cardiac and respiratory arrest and in-hospital mortality.
- Use of a bundled approach to management of pediatric septic shock is recommended.
- The young victim of a sudden, unexpected cardiac arrest should have an unrestricted, complete autopsy, if possible, with special attention to the possibility of an underlying condition that predisposes to a fatal arrhythmia. Appropriate preservation and genetic analysis of tissue should be considered; detailed testing may reveal an inherited "channelopathy" that may also be present in surviving family members.

Systems

Medical emergency teams (METs) or rapid response teams (RRTs) have been shown to be effective in preventing respiratory and cardiac arrests in selected pediatric inpatient settings.

Family presence during resuscitations has been shown to be beneficial for the grieving process and in general was not found to be disruptive. Thus, family presence is supported if it does not interfere with the resuscitative effort.

Medical Emergency or Rapid Response Team[Peds-025A, Peds-025B]

Consensus on Science

The introduction of METs or RRTs was associated with a decrease in pediatric hospital mortality in 1 LOE 3 meta-analysis[3] and 3 pediatric LOE 3 studies with historic controls.[4–6] The introduction of a MET or RRT was associated with

- a decrease in respiratory but not cardiac arrest in 1 LOE 3[7] study with historic controls
- a decrease in preventable total number of arrests in 1 LOE 3 study compared with a retrospective chart review[8]
- a decrease in total number of arrests in 2 LOE 3[4,8] studies
- a decrease in preventable cardiac arrests in 1 LOE 3[6] study
- a decrease in cardiac arrest and non–pediatric intensive care unit (PICU) mortality in 1 LOE 3[9] pediatric cohort study using historical controls

Treatment Recommendations

Pediatric RRT or MET systems may be beneficial to reduce the risk of respiratory and/or cardiac arrest in hospitalized pediatric patients outside an intensively monitored environment.

Knowledge Gaps

Is it the team or the staff education associated with MET or RRT implementation that leads to improved patient outcomes? Is the team effectiveness due to validated team activation criteria or specific team composition? Do the benefits attributed to these teams extend to children in a community hospital setting?

Family Presence During Resuscitation[Peds-003]

Consensus on Science

Ten studies (LOE 2[10]; LOE 3[11]; LOE 4[12–19]) documented that parents wish to be given the option of being present during the resuscitation of their children. One LOE 2,[10] 1 LOE 3,[11] 2 LOE 4,[13,19] and 1 LOE 5[20] studies confirmed that most parents would recommend parent presence during resuscitation.

One LOE 2,[10] 1 LOE 3,[11] 6 LOE 4,[12,14,19,21–23] and 2 LOE 5[20,24] studies of relatives present during the resuscitation of a family member reported that they believed their presence was beneficial to the patient.

One LOE 2,[10] 1 LOE 3,[11] 6 LOE 4,[12,13,16–19] and 1 LOE 5[24] studies reported that most relatives present during the resuscitation of a family member benefited from the experience. One LOE 3,[11] 4 LOE 4,[12,13,20,21] and 2 LOE 5[24,25] studies reported that being present during the resuscitation helped their adjustment to the family member's death.

One LOE 2[10] and 2 LOE 4[12,13] studies observed that allowing family members to be present during a resuscitation in a hospital setting did them no harm, whereas 1 LOE 4[26] study suggested that some relatives present for the resuscitation of a family member experienced short-term emotional difficulty.

One LOE 2,[10] 1 LOE 3,[27] 3 LOE 4,[12,23,28] and 3 LOE 5[20,24,29] studies showed that family presence during resuscitation was not perceived as being stressful to staff or to have negatively affected staff performance. However, 1 survey (LOE 4[30]) found that 39% to 66% of emergency medical services (EMS) providers reported feeling threatened by family members during an out-of-hospital resuscitation and that family presence interfered with their ability to perform resuscitations.

Treatment Recommendations

In general, family members should be offered the opportunity to be present during the resuscitation of an infant or child. When deciding whether to allow family members to be present during an out-of-hospital resuscitation, the potential negative impact on EMS provider performance must be considered.

Knowledge Gaps

How does the presence of a dedicated support person help family members and, potentially, healthcare providers during the resuscitation of an infant or child? What training is appropriate for staff who may serve as support persons for family members during resuscitation of an infant or child? Why is family presence during resuscitation perceived more negatively by out-of-hospital care providers than by in-hospital staff?

Assessment

Many healthcare providers find it difficult to rapidly and accurately determine the presence or absence of a pulse. On the basis of available evidence, the Task Force decided to deemphasize but not eliminate the pulse check as part of the healthcare provider assessment. The Task Force members recognized that healthcare providers who work in specialized settings may have enhanced skills in accurate and rapid pulse checks, although this has not been studied.

There are considerable data regarding use of end-tidal carbon dioxide (P_{ETCO_2}) measurement, capnography and capnometry, during cardiopulmonary resuscitation (CPR) as an indicator of CPR quality and as a predictive measure of outcome. Although capnography/capnometry may reflect the quality of CPR, there is insufficient evidence of its reliability in predicting resuscitation success in infants and children.

Pulse Check Versus Check for Signs of Life[Peds-002A]

Consensus on Science

Thirteen LOE 5 studies[31–43] observed that neither laypersons nor healthcare providers are able to perform an accurate pulse check in healthy adults or infants within 10 seconds. In 2 LOE 5 studies in adults[44,45] and 2 LOE 3 studies in children with nonpulsatile circulation,[46,47] blinded healthcare providers commonly assessed pulse status inaccurately and their assessment often took >10 seconds. In the pediatric studies, healthcare professionals were able to accurately detect a pulse by palpation only 80% of the time. They mistakenly perceived a pulse when it was nonexistent 14% to 24% of the time and failed to detect a pulse when present in 21% to 36% of the assessments. The average time to detect an actual pulse was approximately 15 seconds, whereas the average time to confirm the absence of a pulse was 30 seconds. Because the pulseless patients were receiving extracorporeal membrane oxygenation (ECMO) support, one must be cautious in extrapolating these data to the arrest situation; all pulseless patients did have perfusion and therefore had signs of circulation as evidenced by warm skin temperature with brisk capillary refill. All patients evaluated were in an intensive care unit (ICU) setting without ongoing CPR.

Treatment Recommendations

Palpation of a pulse (or its absence) is not reliable as the sole determinant of cardiac arrest and need for chest compressions. If the victim is unresponsive, not breathing normally, and there are no signs of life, lay rescuers should begin CPR. In infants and children with no signs of life, healthcare providers should begin CPR unless they can definitely palpate a pulse within 10 seconds.

Knowledge Gaps

Is there an association between the time required to successfully detect a suspected cardiac arrest victim's pulse and resuscitation outcome? Is there a difference in outcome when the decision to start chest compressions is based on the absence of signs of life as opposed to absence of a pulse?

Focused Echocardiogram to Detect Reversible Causes of Cardiac Arrest[Peds-006B]

Consensus on Science

In 1 small LOE 4 pediatric case series[48] cardiac activity was rapidly visualized by echocardiography without prolonged interruption of chest compressions, and this cardiac activity correlated with the presence or absence of a central pulse. In 1 pediatric LOE 4 case report,[49] echocardiography was useful for diagnosing pericardial tamponade as the cause of cardiac arrest and was useful in guiding treatment.

In 8 LOE 5 adult case series,[50–57] echocardiographic findings correlated well with the presence or absence of cardiac activity in cardiac arrest. These reports also suggested that echocardiography may be useful in identifying patients with potentially reversible causes for the arrest.

Treatment Recommendations

There is insufficient evidence to recommend for or against the routine use of echocardiography during pediatric cardiac arrest. Echocardiography may be considered to identify potentially treatable causes of an arrest when appropriately skilled personnel are available, but the benefits must be carefully weighed against the known deleterious consequences of interrupting chest compressions.

Knowledge Gaps

Can echocardiography be performed during cardiac arrest in infants and children without significant interruptions in chest compressions? How often does echocardiography during cardiac arrest provide information that can affect treatment and outcome?

End-tidal CO_2 (P_{ETCO_2}) and Quality of CPR[Peds-005A, Peds-005B]

Consensus on Science

Three LOE 5 animal studies,[58–60] 4 LOE 5 adult,[61–64] and 1 LOE 5 pediatric series[65] showed a strong correlation between P_{ETCO_2} and interventions that increase cardiac output during resuscitation from shock or cardiac arrest. Similarly 3 LOE 5 animal models[66–68] showed that measures that markedly reduce cardiac output result in a fall in P_{ETCO_2}.

Two LOE 5 adult out-of-hospital studies[69,70] supported continuous P_{ETCO_2} monitoring during CPR as a way of determining return of spontaneous circulation (ROSC), particularly if the readings during CPR are >15 mm Hg (2.0 kPa). In 1 LOE 4[71] and 2 LOE 5 adult case series,[72,73] an abrupt and sustained rise in P_{ETCO_2} often preceded identification of ROSC.

Two LOE 4 pediatric cases series,[65,74] 8 LOE 5 adult,[70,75–81] and 1 LOE 5 animal study[59] showed that a low P_{ETCO_2} (<10 mm Hg [1.33 kPa] to <15 mm Hg [2.0 kPa]) despite 15 to 20 minutes of advanced life support (ALS) is strongly associated with failure to achieve ROSC. On the basis of 2 LOE 5 animal studies[71,82] and 2 adult LOE 5 case series,[70,78] P_{ETCO_2} after at least 1 minute of CPR may be more predictive of outcome than the initial value because the initial P_{ETCO_2} is often increased in patients with asphyxial cardiac arrest.

The wide variation for initial P_{ETCO_2} during resuscitation limits its reliability in predicting outcome of resuscitation and its value as a guide to limiting resuscitation efforts. Two LOE 5 animal studies[71,82] and 2 large LOE 5 adult trials[70,78] suggested that the initial P_{ETCO_2} is higher if the etiology of the cardiac arrest is asphyxial rather than if it is a primary cardiac arrest.

Interpretation of the end-tidal CO_2 during resuscitation is affected by the quality of the measurement, the minute ventilation delivered during resuscitation, the presence of lung disease that increases anatomic dead space, and the presence of right-to-left shunting.[83–85]

In 1 LOE 5 adult study,[86] sodium bicarbonate transiently increased end-tidal CO_2, and in 3 LOE 5 adult[87–89] and 2 LOE 5 animal[90,91] studies, epinephrine (and other systemic vasoconstrictive agents) transiently decreased P_{ETCO_2}.

Treatment Recommendations

Continuous capnography or capnometry monitoring, if available, may be beneficial by providing feedback on the effectiveness of chest compressions. Whereas a specific target number cannot be identified, if the P_{ETCO_2} is consistently $<$15 mm Hg, it is reasonable to focus efforts on improving the quality of chest compressions and avoiding excessive ventilation.

Although a threshold P_{ETCO_2} may predict a poor outcome from resuscitation and might be useful as a guide to termination of CPR, there are insufficient data to establish the threshold and the appropriate duration of ALS needed before such evaluation in children. The P_{ETCO_2} must be interpreted with caution for 1 to 2 minutes after administration of epinephrine or other vasoconstrictive medications because these medications may decrease the P_{ETCO_2}.

Knowledge Gaps

Does P_{ETCO_2} monitoring during CPR improve quality of chest compressions and/or outcome of pediatric resuscitation? During CPR, can P_{ETCO_2} be reliably measured via a laryngeal mask airway (LMA)? Is there a threshold P_{ETCO_2} that predicts ROSC or low likelihood of ROSC during resuscitation from pediatric cardiac arrest? Can the initial P_{ETCO_2} distinguish asphyxial from cardiac etiology of pediatric cardiac arrest? Is detection of ROSC using P_{ETCO_2} monitoring more accurate than palpation of a pulse? Are P_{ETCO_2} targets during CPR different for subgroups of infants and children with alterations in pulmonary blood flow or high airway resistance?

Airway and Ventilation

Opening and maintaining a patent airway and providing ventilations are fundamental elements of pediatric CPR, especially because cardiac arrest often results from, or is complicated by, asphyxia. There are no new data to change the 2005 ILCOR recommendation to use manual airway maneuvers (with or without an oropharyngeal airway) and bag-mask ventilation (BMV) for children requiring airway control or positive-pressure ventilation for short periods in the out-of-hospital setting. When airway control or BMV is not effective, supraglottic airways may be helpful when used by properly trained personnel.

When performing tracheal intubation, data suggest that the routine use of cricoid pressure may not protect against aspiration and may make intubation more difficult.

Routine confirmation of tracheal tube position with capnography/capnometry is recommended with the caveat that the P_{ETCO_2} in infants and children in cardiac arrest may be below detection limits for colorimetric devices.

Following ROSC, toxic oxygen byproducts (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). Although there are no clinical studies in children (outside the newborn period) comparing different concentrations of inspired oxygen during and immediately after resuscitation, animal data and data from newborn resuscitation studies suggest that it is prudent to titrate inspired oxygen after return of a perfusing rhythm to prevent hyperoxemia.

Supplementary Oxygen[Peds-015]

Consensus on Science

There are no studies comparing ventilation of infants and children in cardiac arrest with different inspired oxygen concentrations. Two LOE 5 meta-analyses of several randomized controlled trials comparing neonatal resuscitation initiated with room air versus 100% oxygen[92,93] showed increased survival when resuscitation was initiated with room air.

Seven LOE 5 animal studies[94–100] suggested that ventilation with room air or an F_{IO_2} of $<$1.0 during cardiac arrest may be associated with less neurologic deficit than ventilation with an F_{IO_2} of 1.0, whereas 1 LOE 5 animal study[101] showed no difference in outcome. In 5 LOE 5 animal studies[95,97–99,102] ventilation with 100% oxygen during and following resuscitation contributed to free radical–mediated reperfusion injury to the brain.

Treatment Recommendations

There is insufficient evidence to recommend any specific inspired oxygen concentration for ventilation *during* resuscitation from cardiac arrest in infants and children. Once circulation is restored, it is reasonable to titrate inspired oxygen to limit hyperoxemia.

Knowledge Gaps

Does the use of any specific concentration of supplementary oxygen during resuscitation from cardiac arrest in infants and children improve or worsen outcome? What is the appropriate target oxygen saturation for the pediatric patient after achieving ROSC?

Cuffed Versus Uncuffed Tracheal Tube[Peds-007]

Consensus on Science

There are no studies that compare the safety and efficacy of cuffed versus uncuffed tubes in infants and children who require emergency intubation.

Two LOE 5 randomized controlled studies[103,104] and 1 LOE 5 cohort-controlled study[105] in a pediatric anesthesia setting showed that the use of cuffed tracheal tubes was associated with a higher likelihood of selecting the correct tracheal tube size (and hence a lower reintubation rate) with no increased risk of perioperative or airway complications.

Cuff pressures in these 3 studies were maintained at <25 cm H_2O. Two perioperative LOE 5 cohort-controlled pediatric studies[105,106] similarly showed that cuffed tubes were not associated with an increased risk of perioperative airway complications.

One LOE 5 pediatric case series[107] observed that the use of cuffed tracheal tubes was not a risk factor for developing subglottic stenosis in patients having corrective surgery for congenital cardiac defects. In the intensive care setting, 2 LOE 5 prospective cohort-controlled studies[108,109] and 1 LOE 5 retrospective cohort-controlled study[110] documented no greater risk of complications for children. >8 years of age who were intubated with cuffed compared with uncuffed tracheal tubes.

One small LOE 5 case-controlled study[111] showed that cuffed tracheal tubes decreased the incidence of aspiration in the PICU, and 1 LOE 5 case series[105] of children with burns undergoing general anesthesia showed a significantly higher rate of excessive air leak requiring immediate reintubation in patients initially intubated with an uncuffed tracheal tube.

Treatment Recommendations

Both cuffed and uncuffed tracheal tubes are acceptable for infants and children undergoing emergency intubation. If cuffed tracheal tubes are used, avoid excessive cuff pressures.

Knowledge Gaps

What is the best technique to determine cuff pressure and/or the presence of an air leak when using cuffed tracheal tubes in infants and children? What is the optimal cuff or leak pressure for children of different ages? Does optimal cuff pressure vary based on the type of cuffed tube (eg, Microcuff®) used?

Are the data generated in elective operating room studies applicable to emergency resuscitation scenarios? Are there select populations of pediatric patients whose outcomes are improved by the use of cuffed tracheal tubes during resuscitation?

Tracheal Tube Size[Peds-057A, Peds-057B]

Consensus on Science

Evidence from 1 LOE 2 prospective randomized trial of elective intubation in a pediatric operating room[103] was used to support the existing formula for estimation of appropriate cuffed tracheal tube internal diameter (ID): ID (mm)=(age in years/4) + 3, also known as the Khine formula. Detailed analysis of this paper, however, reveals that the aggressive rounding up of age employed by the authors in their calculations commonly resulted in selection of a tube with an ID 0.5 mm larger than the size derived from the formula.

Evidence from 1 LOE 2 prospective randomized multicenter study,[104] 1 LOE 2,[112] and 3 LOE 4 prospective observational studies of elective intubation in the pediatric operating room[113–115] supported use of 3-mm ID cuffed tracheal tubes for newborns and infants (3.5 kg to 1 year of age) and 3.5-mm ID cuffed tracheal tubes for patients 1 to 2 years of age.

One LOE 2 prospective randomized multicenter study[104] and 3 LOE 4 prospective observational studies of elective intubation in the pediatric operating room[113–115] using Microcuff® tracheal tubes support the use of the following formula for cuffed endotracheal tubes in children: ID (mm)=(age/4) + 3.5. One LOE 2 prospective observational study of elective intubation in the pediatric operating room[112] found that formula acceptable but associated with a marginally greater reintubation rate than with the Khine formula (ID [mm]=[age in years/4] + 3).

Treatment Recommendations

If a cuffed tracheal tube is used in infants ≥3.5 kg and <1 year of age, it is reasonable to use a tube with an ID of 3.0 mm. If a cuffed tracheal tube is used in children between 1 and 2 years of age, it is reasonable to use a tube with an ID of 3.5 mm.

After the age of 2, it is reasonable to estimate the cuffed tracheal tube size with the formula ID (mm)=(age in years/4) + 3.5. If the tracheal tube meets resistance during insertion, a tube with an ID 0.5 mm smaller should be used. If there is no leak around the tube with the cuff deflated, reintubation with a tube ID 0.5 mm smaller may be beneficial when the patient is stable.

Knowledge Gaps

Are the formulas for estimation of tracheal tube size that are used for elective intubation in the operating room setting applicable during resuscitation? Is there an upper age limit for the validity of the formula to estimate tube size? Are length-based formulas more accurate compared with age- or weight-based formulas for estimating tracheal tube size in infants and children?

Bag-Mask Ventilation Versus Intubation[Peds-008]

Consensus on Science

One LOE 1 study[116] compared paramedic out-of-hospital BMV with intubation for children with cardiac arrest, respiratory arrest, or respiratory failure in an EMS system with short transport intervals and found equivalent rates of survival to hospital discharge and neurologic outcome. One LOE 1 systematic review that included this study[117] also reached the same conclusion.

One LOE 2 study of pediatric trauma patients[118] observed that out-of-hospital intubation is associated with a higher risk of mortality and postdischarge neurologic impairment compared with in-hospital intubation. These findings persisted even after stratification for severity of trauma and head trauma.

In 1 LOE 2 (nonrandomized) prehospital pediatric study,[119] if paramedics provided BMV while awaiting the arrival of a physician to intubate the patient, the risk of cardiac arrest and overall mortality was lower than if the patient was intubated by the paramedics. These findings persisted even after adjusting for Glasgow Coma Scale score.

Four LOE 4 studies[120–123] showed a significantly greater rate of failed intubations and complications in children compared with adults in the out-of-hospital and emergency department settings. Conversely 1 LOE 3 out-of-hospital study[124] and 1 LOE 4 out-of-hospital study[125] failed to demonstrate any difference in intubation failure rates between adults and children.

Treatment Recommendations
BMV is recommended over tracheal intubation in infants and children who require ventilatory support in the out-of-hospital setting when transport time is short.

Knowledge Gaps
For the experienced airway specialist, does tracheal intubation improve outcomes in comparison with BMV for pediatric resuscitation? Does the use of neuromuscular blocking drugs improve the outcome of children undergoing intubation during resuscitation? What is the minimal initial training and ongoing experience needed to improve success rate and reduce complications of emergent intubation of infants and children?

Bag-Mask Ventilation Versus Supraglottic Airway[Peds-009]

Consensus on Science
No studies have directly compared BMV to the use of supraglottic airway devices during pediatric resuscitation other than for the newly born in the delivery room. Nine LOE 5 case reports[126–134] demonstrated the effectiveness of supraglottic airway devices, primarily the LMA, for airway rescue of children with airway abnormalities.

One LOE 5 out-of-hospital adult study[135] supports the use of LMAs by first responders during CPR, but another LOE 5 out-of-hospital adult cardiac arrest study[136] of EMS personnel providing assisted ventilation by either bag-mask device or LMA failed to show any significant difference in ventilation ($Paco_2$). Six LOE 5 studies during anesthesia[137–142] demonstrated that complication rates with LMAs increase with decreasing patient age and size.

In 2 LOE 5 manikin studies[143,144] trained nonexpert providers successfully delivered positive-pressure ventilation using the LMA. Tracheal intubations resulted in a significant incidence of tube misplacement (esophageal or right mainstem bronchus), a problem not present with the LMA, but time to effective ventilation was shorter and tidal volumes were greater with BMV.

In 2 LOE 5 studies of anesthetized children[145,146] suitably trained ICU and ward nurses placed LMAs with a high success rate, although time to first breath was shorter in the BMV group. In a small number of cases ventilation was achieved with an LMA when it proved impossible with BMV.

Treatment Recommendations
BMV remains the preferred technique for emergency ventilation during the initial steps of pediatric resuscitation. In infants and children for whom BMV is unsuccessful, use of the LMA by appropriately trained providers may be considered for either airway rescue or support of ventilation.

Knowledge Gaps
Are the data regarding use of supraglottic airways for elective airway management in the operating room applicable to emergency resuscitation scenarios? With an LMA in place, is it necessary to pause chest compressions to provide effective ventilations? Is the combination of an oropharyngeal airway with BMV more or less effective than supraglottic airways?

Minute Ventilation[Peds-013A]

Consensus on Science
There are no data to identify the optimal minute ventilation (tidal volume or respiratory rate) for infants or children with an advanced airway during CPR, regardless of arrest etiology.

Three LOE 5 animal studies[147–149] showed that ventilation during CPR after VF or asphyxial arrest resulted in improved ROSC, survival, and/or neurologic outcome compared with no positive-pressure breaths.

Evidence from 4 LOE 5 adult studies[150–153] showed that excessive ventilation is common during resuscitation from cardiac arrest. In 1 LOE 5 animal study[150] excessive ventilation during resuscitation from cardiac arrest decreased cerebral perfusion pressure, ROSC, and survival compared with lower ventilation rates. One good LOE 5 animal study[149] found that increasing respiratory rate during conditions of reduced cardiac output improved alveolar ventilation but not oxygenation, and it reduced coronary perfusion pressure.

In 1 LOE 5 prospective, randomized adult study[154] constant-flow insufflation with oxygen compared with conventional mechanical ventilation during CPR did not change outcome (ROSC, survival to admission, and survival to ICU discharge). In another LOE 5 adult study,[155] adults with witnessed VF arrest had improved neurologically intact survival with passive oxygen insufflation compared with BMV, whereas there was no difference in survival if the VF arrest was unwitnessed.

Two LOE 5 animal studies showed that ventilation or continuous positive airway pressure (CPAP) with oxygen compared with no ventilation improved arterial blood gases[156] but did not change neurologically intact survival.[157] One good-quality LOE 5 animal study[158] showed that reducing tidal volume by 50% during CPR resulted in less excessive ventilation without affecting ROSC.

Treatment Recommendations
Following placement of a secure airway, avoid excess ventilation of infants and children during resuscitation from cardiac arrest, whether asphyxial or due to VF. A reduction in minute ventilation to less than baseline for age is reasonable to provide sufficient ventilation to maintain adequate ventilation-to-perfusion ratio during CPR while avoiding the harmful effects of excessive ventilation. There are insufficient data to identify the optimal tidal volume or respiratory rate.

Knowledge Gaps
What is the optimal minute ventilation to achieve ventilation-perfusion matching during pediatric CPR? Is it preferable to reduce tidal volume or respiratory rate to achieve optimal minute ventilation during pediatric CPR? Does hypoventilation (ie, hypercarbia) during resuscitation affect outcome from pediatric cardiac arrest? Does passive oxygen insufflation or CPAP during cardiac arrest in infants and children provide adequate gas exchange or improve outcome from resuscitation?

Devices to Verify Advanced Airway Placement[Peds-004]

Consensus on Science

No single assessment method accurately and consistently confirms tracheal tube position. Three LOE 4 studies[71,159,160] showed that when a perfusing cardiac rhythm is present in infants (>2 kg) and children, detection of exhaled CO_2 using a colorimetric detector or capnometer has a high sensitivity and specificity for confirming endotracheal tube placement. One of these studies[71] included infants and children in cardiac arrest. In the cardiac arrest population the sensitivity of exhaled CO_2 detection was only 85% (ie, false-negatives occurred), whereas the specificity remained at 100%.

One neonatal LOE 5 study[161] of delivery room intubation demonstrated that detection of exhaled CO_2 by capnography was 100% sensitive and specific for detecting esophageal intubation and took less time than clinical assessment to identify esophageal intubation. Two additional neonatal LOE 5 studies[162,163] showed that confirmation of tracheal tube position is faster with capnography than with clinical assessment.

Two pediatric LOE 4 studies[164,165] showed that in the presence of a perfusing rhythm, exhaled CO_2 detection or measurement can confirm tracheal tube position accurately during transport, while 2 LOE 5 animal studies[166,167] showed that tracheal tube displacement can be detected more rapidly by CO_2 detection than by pulse oximetry.

One LOE 2 operating room study[168] showed that the esophageal detector device (EDD) is highly sensitive and specific for correct tracheal tube placement in children >20 kg with a perfusing cardiac rhythm; there have been no studies of EDD use in children during cardiac arrest. An LOE 4 operating room (ie, non-arrest) study[169] showed that the EDD performed well but was less accurate in children <20 kg.

Treatment Recommendations

Confirmation of tracheal tube position using exhaled CO_2 detection (colorimetric detector or capnography) should be used for intubated infants and children with a perfusing cardiac rhythm in all settings (eg, out-of-hospital, emergency department, ICU, inpatient, operating room).

In infants and children with a perfusing rhythm, it may be beneficial to monitor continuous capnography or frequent intermittent detection of exhaled CO_2 during out-of-hospital and intra-/interhospital transport.

The EDD may be considered for confirmation of tracheal tube placement in children weighing >20 kg when a perfusing rhythm is present.

Knowledge Gaps

Which technique for CO_2 detection (colorimetric versus capnography) is more accurate during pediatric resuscitation? For infants and children in cardiac arrest, what is the most reliable way to achieve confirmation of tracheal tube position?

Cricoid Pressure[Peds-039A, Peds-039B]

Consensus on Science

There are no data to show that cricoid pressure prevents aspiration during rapid sequence or emergency tracheal intubation in infants or children. Two LOE 5 studies[170,171] showed that cricoid pressure may reduce gastric inflation in children. One LOE 5 study in children[172] and 1 LOE 5 study in adult cadavers[173] demonstrated that esophageal reflux is reduced with cricoid pressure.

In 1 LOE 5 adult systematic review[174] laryngeal manipulation enhanced BMV or intubation in some patients while impeding it in others. One LOE 5 study in anesthetized children[175] showed that cricoid pressure can distort the airway with a force of as low as 5 newtons.

Treatment Recommendations

If cricoid pressure is used during emergency intubations in infants and children it should be discontinued if it impedes ventilation or interferes with the speed or ease of intubation.

Knowledge Gaps

Can cricoid pressure reduce the incidence of aspiration during emergent intubation of infants or children? How much cricoid pressure should be applied, and what is the best technique to reduce gastric inflation during BMV?

Chest Compressions

The concept of chest compression-only CPR is appealing because it is easier to teach than conventional CPR, and immediate chest compressions may be beneficial for resuscitation from sudden cardiac arrest caused by VF or pulseless VT. Animal studies showed that conventional CPR, including ventilations and chest compressions, is best for resuscitation from asphyxial cardiac arrest. In a large study of out-of-hospital pediatric cardiac arrest,[176] few children with asphyxial arrest received compression-only CPR and their survival was no better than in children who received no CPR.

To be effective, chest compressions must be deep, but it is difficult to determine the optimal depth in infants and children; should recommended depth be expressed as a fraction of the depth of the chest or an absolute measurement? How can this be made practical and teachable?

Compression-Only CPR[Peds-012A]

Consensus on Science

Evidence from 1 LOE 2 large out-of-hospital pediatric prospective observational investigation[176] showed that children with cardiac arrest of noncardiac etiology (asphyxial arrest) had a higher 30-day survival with more favorable neurologic outcome if they received standard bystander CPR (chest compressions with rescue breathing) compared with chest compression-only CPR. Standard CPR and chest compression-only CPR were similarly effective and better than no bystander CPR for pediatric cardiac arrest from cardiac causes. Of note, the same study showed that more than 50% of children with out-of-hospital cardiac arrest did not receive any bystander CPR. Compression-only CPR was as ineffective as no CPR in the small number of infants and children with asphyxial arrest who did not receive ventilations.

Two LOE 5 animal studies[148,177] demonstrated improved survival rates and favorable neurologic outcome with standard CPR compared with no CPR. One LOE 5 animal study[178] showed that blood gases deteriorated with compression-only CPR compared with standard CPR in asphyxial arrests.

Data from 1 LOE 5 animal study[177] indicated that compression-only CPR is better than no CPR for asphyxial arrest but not as effective as standard CPR, and 7 LOE 5 clinical observational studies in adults[179–184] showed that compression-only CPR can result in successful resuscitation from an asphyxial arrest. Moreover, in 10 LOE 5 animal studies[185–194] and 7 LOE 5 adult clinical observational studies[179–184,195] compression-only bystander CPR was generally as effective as standard 1-rescuer bystander CPR for arrests from presumed cardiac causes.

Treatment Recommendations
Rescuers should provide conventional CPR (rescue breathing and chest compressions) for in-hospital and out-of-hospital pediatric cardiac arrests. Lay rescuers who cannot provide rescue breathing should at least perform chest compressions for infants and children in cardiac arrest.

Knowledge Gaps
Does teaching compression-only CPR to lay rescuers increase the likelihood that CPR will be performed during out-of-hospital pediatric cardiac arrest?

One- Versus 2-Hand Chest Compression in Children[Peds-033]

Consensus on Science
There are no outcome studies comparing 1- versus 2-hand chest compressions for children in cardiac arrest. Evidence from 1 LOE 5 randomized crossover child manikin study[196] showed that higher chest-compression pressures are generated by healthcare professionals using the 2-hand technique. Two LOE 5 studies[197,198] report no increase in rescuer fatigue comparing 1-hand with 2-hand chest compressions delivered by healthcare providers to a child-sized manikin.

Treatment Recommendations
Either a 1- or 2-hand technique can be used for performing chest compressions in children.

Knowledge Gaps
Does the use of 1-hand compared with 2-hand chest compressions during pediatric cardiac arrest affect quality of CPR or outcome?

Circumferential Chest Squeeze in Infants[Peds-034]

Consensus on Science
There are no studies that compare the 2-thumb chest compression technique with and without a "circumferential squeeze" in infants.

Treatment Recommendations
There are insufficient data for or against the need for a circumferential squeeze of the chest when performing the 2-thumb technique of external chest compression for infants.

Knowledge Gaps
Does the addition of a circumferential squeeze to 2-thumb compression in infants provide more effective chest compressions or improve resuscitation outcome?

Chest Compression Depth[Peds-040A, Peds-040B]

Consensus on Science
Evidence from anthropometric measurements in 3 good-quality LOE 5 case series[199–201] showed that in children the chest can be compressed to one third of the anterior-posterior chest diameter without causing damage to intrathoracic organs. One LOE 5 mathematical model based on neonatal chest computed tomography scans[202] suggests that one third anterior-posterior chest compression depth is more effective than one fourth compression depth and safer than one half anterior-posterior compression depth.

A good-quality LOE 5[152] adult study found that chest compressions are often inadequate, and a good-quality LOE 4 pediatric study[200] showed that during resuscitation of patients >8 years of age, compressions are often too shallow, especially following rescuer changeover. Evidence from 1 pediatric LOE 4 systematic review of the literature[203] showed that rib fractures are rarely associated with chest compressions.

Treatment Recommendations
In infants, rescuers should be taught to compress the chest by *at least* one third the anterior-posterior dimension or approximately 1½ inches (4 cm). In children, rescuers should be taught to compress the chest by *at least* one third the anterior-posterior dimension or approximately 2 inches (5 cm).

Knowledge Gaps
Can lay rescuers or healthcare providers reliably perform compressions to the recommended depth during pediatric cardiac arrest? Is there harm from compressions that are "too deep" in infants?

Compression-Ventilation Ratio

The ILCOR Neonatal Task Force continues to recommend a compression-ventilation ratio of 3:1 for resuscitation of the newly born in the delivery room, with a pause for ventilation whether or not the infant has an advanced airway. The Pediatric Task Force reaffirmed its recommendation for a 15:2 compression-ventilation ratio for 2-rescuer infant CPR, with a pause for ventilation in infants without an advanced airway, and continuous compressions without a pause for ventilation for infants with an advanced airway.

No previous recommendations were made for hospitalized newborns cared for in areas other than the delivery area or with primary cardiac rather than asphyxial arrest etiology. For example, consider the case of a 3-week-old infant who has a cardiac arrest following cardiac surgery. In the neonatal intensive care unit such an infant would be resuscitated according to the protocol for the newly born, but if the same infant is in the PICU, resuscitation would be performed according to the infant guidelines. A resolution to this dilemma is suggested on the basis of the arrest etiology and ease of training.

Optimal Compression-Ventilation Ratio for Infants and Children[Peds-011B]

Consensus on Science

There are insufficient data to identify an optimal compression-ventilation ratio for CPR in infants and children. In 4 LOE 5 manikin studies[204–207] examining the feasibility of compression-ventilation ratios of 15:2 and 5:1, lone rescuers could not deliver the desired number of chest compressions per minute at a ratio of 5:1. In 5 LOE 5 studies[208–212] using a variety of manikin sizes comparing compression-ventilation ratios of 15:2 with 30:2, a ratio of 30:2 yielded more chest compressions with no, or minimal, increase in rescuer fatigue. One LOE 5 study[213] of volunteers recruited in an airport to perform 1-rescuer layperson CPR on an adult-sized manikin observed less "no flow time" with the use of a 30:2 ratio compared with a 15:2 ratio.

One LOE 5 observational human study[214] comparing resuscitations by firefighters before and after the change from a recommended 15:2 to 30:2 compression-ventilation ratio reported more chest compressions per minute with the 30:2 ratio, but the rate of ROSC was unchanged. Three LOE 5 animal studies[192,215,216] showed that coronary perfusion pressure, a major determinant of success in resuscitation, rapidly declines when chest compressions are interrupted; once compressions are resumed, several chest compressions are needed to restore coronary perfusion pressure to preinterruption levels. Thus, frequent interruptions of chest compressions prolong the duration of low coronary perfusion pressure and flow and reduce the mean coronary perfusion pressure. Three LOE 5 manikin studies[213,217,218] and 3 LOE 5[151,152,219] in- and out-of-hospital adult human studies documented long interruptions in chest compressions during simulated or actual resuscitations. Three LOE 5 adult studies[220–222] demonstrated that these interruptions reduced ROSC.

In 5 LOE 5 animal studies[191,192,194,215–216] chest compressions without ventilations were sufficient to resuscitate animals with VF-induced cardiac arrest. Conversely in 2 LOE 5 animal studies[223,224] decreasing the frequency of ventilation was detrimental in the first 5 to 10 minutes of resuscitation of VF-induced cardiac arrest.

One LOE 5 mathematical model[225] suggested that the compression-ventilation ratio in children should be lower (more ventilations to compressions) than in adults and should decrease with decreasing weight. Two LOE 5 studies of asphyxial arrest in pigs[148,177] showed that ventilations added to chest compressions improved outcome compared with compressions alone. Thus, ventilations are more important during resuscitation from asphyxia-induced arrest than during resuscitation from VF. But even in asphyxial arrest, fewer ventilations are needed to maintain an adequate ventilation-perfusion ratio in the presence of the low cardiac output (and consequently low pulmonary blood flow) produced by chest compressions.

Treatment Recommendations

For ease of teaching and retention, a compression-ventilation ratio of 30:2 is recommended for the lone rescuer performing CPR in infants and children, as is used for adults. For healthcare providers performing 2-rescuer CPR in infants and children, a compression-ventilation ratio of 15:2 is recommended. When a tracheal tube is in place, compressions should not be interrupted for ventilations.

Knowledge Gaps

What is the optimal compression-ventilation ratio to improve outcome for neonates, infants, and children in cardiac arrest?

Newborns (Out of the Delivery Area) Without an Endotracheal Airway[Peds-027A]

Consensus on Science

There are insufficient data to identify an optimal compression-ventilation ratio for all infants in the first month of life. One LOE 5 animal study[192] showed that coronary perfusion pressure declined with interruptions in chest compressions; after each interruption, several chest compressions were required to restore coronary perfusion pressure to preinterruption levels. One LOE 5 adult human study[221] and 2 LOE 5 animal studies[215,222] showed that interruptions in chest compression reduced the likelihood of ROSC in VF cardiac arrest.

One LOE 5 1-rescuer manikin study[207] showed that more effective ventilation was achieved with a 3:1 ratio than with a 5:1, 10:2, or 15:2 ratio. One LOE 5 mathematical study of cardiovascular physiology[226] suggested that blood flow rates in neonates are best at compression rates of >120/min.

Treatment Recommendations

There are insufficient data to recommend an optimal compression-ventilation ratio during CPR for all infants in the first month of life (beyond the delivery room). The limited data available suggest that if the etiology of the arrest is cardiac, a 15:2 ratio (2 rescuers) may be more effective than a 3:1 ratio.

Knowledge Gaps

Do healthcare providers perform better CPR if they learn 1 rather than 2 compression-ventilation ratios based on etiology of the arrest (cardiac or asphyxial)?

Newborns (Out of Delivery Area) With a Tracheal Tube[Peds-026A]

Consensus on Science

There is insufficient evidence to determine if an intubated neonate has a better outcome from cardiac arrest using a 3:1 compression-ventilation ratio and interposed ventilations compared with continuous chest compressions without pause for ventilations (asynchronous compressions and ventilations).

Two LOE 5 adult[220,222] and 2 LOE 5 animal[191,192] studies demonstrated that interruptions in chest compressions reduced coronary perfusion pressure, a key determinant of successful resuscitation in adults, and decreased ROSC. There are no equivalent studies evaluating the impact of interrupted chest compressions in asphyxiated neonates or neonatal animal models.

In 1 LOE 5 piglet study[227] of VF arrest, myocardial blood flow increased using simultaneous chest compressions and high–airway pressure ventilations in a 1:1 ratio as compared

with conventional CPR at a 5:1 ratio. Another LOE 5 VF piglet study[228] demonstrated equivalent cardiac output but worsened gas exchange using a 1:1 compression-ventilation ratio (ie, simultaneous compressions and ventilations) with high airway pressures compared with conventional CPR at a 5:1 ratio.

One LOE 5[148] study in nonintubated asphyxiated piglets resuscitated with a 5:1 compression-ventilation ratio showed that ventilations are important for successful resuscitation. One LOE 5 study in intubated asphyxiated piglets[178] showed that the addition of ventilations resulted in lower arterial CO_2 tension ($Paco_2$) without compromising hemodynamics when compared with compressions alone. One LOE 5 manikin study[229] found that healthcare providers were unable to achieve the recommended rate of ventilations during infant CPR at a 3:1 compression-ventilation ratio, with 20% delivering a net rate of 40 breaths per minute after 5 minutes of resuscitation. There are no studies that evaluate the impact of continuous compressions on minute ventilation, gas exchange, or the outcome of resuscitation during CPR for intubated neonates.

Treatment Recommendations

For ease of training, providers should use the compression-ventilation ratio and resuscitation approach that is most commonly used in their practice environment for intubated term or near-term newborns within the first month of life. Intubated newborns (ie, those with an advanced airway) who require CPR in non-neonatal settings (eg, prehospital, emergency department, PICU, etc) or those with a cardiac etiology of cardiac arrest, regardless of location, should receive CPR according to infant guidelines (continuous chest compressions without pause for ventilations).

Knowledge Gaps

In intubated infants in cardiac arrest, can effective ventilations be performed during continuous chest compressions with asynchronous ventilations? Do pauses for ventilations during CPR affect the outcome from cardiac arrest in intubated infants?

Vascular Access and Drug Delivery

There is no new evidence to change the 2005 ILCOR recommendations on vascular access, including the early use of intraosseous (IO) access and deemphasis of the tracheal route of drug delivery. Epidemiological data, largely from the National Registry of CPR (NRCPR), reported an association between vasopressin, calcium, or sodium bicarbonate administration and an increased likelihood of death. These data, however, cannot be interpreted as a cause-and-effect relationship. The association may be due to more frequent use of these drugs in children who fail to respond to standard basic and advanced life support interventions. These and other data in adults question the benefit of intravenous (IV) medications during resuscitation and reemphasize the importance of high-quality CPR.

Intraosseous Access[Peds-035]

Consensus on Science

There are no studies comparing IO with IV access in children with cardiac arrest. In 1 LOE 5 study of children in shock[230] IO access was frequently more successful and achieved more rapidly than IV access. Eight LOE 4 case series[231–238] showed that providers with many levels of training could rapidly establish IO access with minimal complications for children with cardiac arrest.

Treatment Recommendations

IO cannulation is an acceptable route of vascular access in infants and children with cardiac arrest. It should be considered early in the care of critically ill children whenever venous access is not readily attainable.

Knowledge Gaps

Does the use of IO compared with IV vascular access improve outcome of pediatric cardiac arrest? Does the use of newer IO devices (eg, bone injection guns and drills) compared with conventional IO needles affect outcome in pediatric cardiac arrest?

Tracheal Drug Delivery[Peds-036]

Consensus on Science

One LOE 3 study of children with in-hospital cardiac arrest[239] demonstrated similar ROSC and survival rates, whereas 2 LOE 5 studies of adults in cardiac arrest[240,241] demonstrated reduced ROSC and survival to hospital discharge rates when tracheal instead of IV epinephrine was given. One LOE 5 case series of neonatal asphyxial bradycardia[242] demonstrated similar rates of ROSC whether IV or tracheal epinephrine was administered, whereas another LOE 5 study[243] demonstrated a lower rate of ROSC in neonates given tracheal as opposed to IV epinephrine. Many of the human studies used tracheal epinephrine doses of <0.1 mg/kg.

In some animal studies[244–249] lower doses of tracheal epinephrine (0.01 to 0.05 mg/kg) produced transient deleterious β-adrenergic vascular effects resulting in lower coronary artery perfusion. One LOE 5 study[250] of animals in VF cardiac arrest demonstrated a higher rate of ROSC in those treated with tracheal vasopressin compared with IV placebo.

Four LOE 5 studies of animals in cardiac arrest[251–254] demonstrated similar ROSC and survival rates when either tracheal or IV routes were used to deliver epinephrine. These studies also demonstrated that to reach an equivalent biological effect, the tracheal dose must be up to 10 times the IV dose.

Treatment Recommendations

The preferred routes of drug delivery for infants and children in cardiac arrest are IV and IO. If epinephrine is administered via a tracheal tube to infants and children (not including the newly born) in cardiac arrest, the recommended dose is 0.1 mg/kg.

Knowledge Gaps

What is the optimal dose of tracheal epinephrine during pediatric cardiac arrest?

Defibrillation

The Pediatric Task Force evaluated several issues related to defibrillation, including safe and effective energy dosing, stacked versus single shocks, use of automated external

defibrillators (AEDs) in infants <1 year of age and paddle/pad type, size, and position. There were a few new human and animal studies on these topics, and the level of evidence (LOE) was generally 3 to 5. No new data are available to support a change in drug treatment of recurrent or refractory VF/pulseless VT. There were several human and animal publications on defibrillation energy dose, but the data are contradictory and the optimal safe and effective energy dose remains unknown.

The new recommendation of an initial dose of 2 to 4 J/kg is based on cohort studies showing low success in termination of VF in children with 2 J/kg. However, these studies do not provide data on success or safety of higher energy doses. The reaffirmation of the recommendation for a single initial shock rather than stacked shocks (first made in 2005) is extrapolated from the ever-increasing adult data showing that long pauses in chest compressions required for stacked shocks are associated with worse resuscitation outcomes and that the initial shock success rate is relatively high with biphasic defibrillation.

No changes are recommended in pad/paddle size or position. Although the safety of AEDs in infants <1 year is unknown, case reports have documented successful defibrillation using AEDs in infants. A manual defibrillator or an AED with pediatric attenuation capabilities is preferred for use in infants and small children.

Paddle Size and Orientation[Peds-029]

Consensus on Science

One LOE 5 study in adults[255] demonstrated that shock success increased from 31% to 82% when pad size was increased from 8×8 cm to 12×12 cm. Three pediatric LOE 4,[256–258] 3 adult LOE 5,[255,259,260] and 3 LOE 5 animal[261–263] studies demonstrated that transthoracic impedance decreases with increasing pad size. Decreased transthoracic impedance increases transthoracic current and, thus, presumably, transmyocardial current.

Pad Position

Consensus on Science

One pediatric LOE 4 study[264] observed no difference in the rate of ROSC between antero-lateral and anterior-posterior electrode positions for shock delivery. One pediatric LOE 2 study,[256] 2 adult LOE 5 studies,[265,266] and 1 LOE 5 animal study[263] demonstrated that transthoracic impedance is not dependent on pad position. Transthoracic impedance was increased in 1 adult LOE 5[267] study by placing the pads too close together and in 1 LOE 5[260] study when the pads were placed over the female breast. Additionally, 1 adult LOE 5[268] study showed that placing the apical pad in a horizontal position lowers transthoracic impedance.

Treatment Recommendation

There is insufficient evidence to alter the current recommendations to use the largest size paddles/pads that fit on the infant or child's chest without touching each other or to recommend one paddle/pad position or type over another.

Self-Adhesive Pads Versus Paddles[Peds-043A, Peds-043B]

Consensus on Science

There are limited studies comparing self-adhesive defibrillation pads (SADPs) with paddles in pediatric cardiac arrest. One pediatric LOE 4[264] study demonstrated equivalent ROSC rates when paddles or SADPs were used. One LOE 5[269] adult out-of-hospital cardiac arrest study suggested improved survival to hospital admission when SADPs rather than paddles were used.

One adult LOE 5[270] study showed a lower rate of rhythm conversion, and 1 small adult LOE 5[271] study showed at least equivalent success with the use of SADPs in comparison with paddles in patients undergoing cardioversion for atrial fibrillation. Two adult LOE 5[272,273] studies showed equivalent transthoracic impedance with SADPs or paddles. One adult LOE 5[266] and 2 LOE 5 animal[274,275] studies showed that SADPs had a higher transthoracic impedance than paddles.

One LOE 4[276] study described difficulty with fitting self-adhesive pads onto the thorax of a premature infant without the pads touching. One LOE 5[277] study demonstrated the improved accuracy of cardiac rhythm monitoring following defibrillation using SADPs compared with the combination of paddles and gel pads.

Using standard resuscitation protocols in simulated clinical environments, 1 LOE 5[278] study found no significant difference in the time required to deliver shocks using either SADPs or paddles, and 1 LOE 5[279] study found no significant difference in time without compressions when SADPs or paddles were used.

Treatment Recommendations

Either self-adhesive defibrillation pads or paddles may be used in infants and children in cardiac arrest.

Knowledge Gaps

Is the use of hands-on defibrillation safe for rescuers and does it improve outcome for infants and children in cardiac arrest (eg, by presumably reducing interruptions in chest compressions)?

Number of Shocks[Peds-022A]

Consensus on Science

There are no randomized controlled studies examining a single versus sequential (stacked) shock strategy in children with VF/pulseless VT. Evidence from 7 LOE 5 studies in adults with VF[221,280–285] supported a single-shock strategy over stacked or sequential shocks because the relative efficacy of a single biphasic shock is high and the delivery of a single shock reduces duration of interruptions in chest compressions.

Treatment Recommendations

A single-shock strategy followed by immediate CPR (beginning with chest compressions) is recommended for children with out-of-hospital or in-hospital VF/pulseless VT.

Knowledge Gaps

Are there circumstances during which the use of stacked or multiple shocks can improve outcome from pediatric cardiac arrest?

Energy Dose[Peds-023A, Peds-023B]

Consensus on Science

Two LOE 4[264,286] studies reported no relationship between defibrillation dose and survival to hospital discharge or neurologic outcome from VF/pulseless VT. Evidence from 3 LOE 4 studies in children in out-of-hospital and in-hospital settings[264,287,288] observed that an initial dose of 2 J/kg was effective in terminating VF 18% to 50% of the time. Two LOE 4 studies[286,289] reported that children often received more than 2 J/kg during out-of hospital cardiac arrest, with many (69%) requiring ≥3 shocks of escalating energy doses. One in-hospital cardiac arrest LOE 4 study[264] reported that the need for multiple shocks with biphasic energy doses of 2.5 to 3.2 J/kg was associated with lack of ROSC.

Evidence from 2 LOE 5 animal studies[290,291] observed that 0% to 8% of episodes of long-duration VF were terminated by a 2 J/kg monophasic shock and up to 32% were terminated by biphasic shocks. Animals in these studies received both fixed and escalated doses, and most required 2 or more shocks to terminate VF. In 1 LOE 5 animal study[263] the defibrillation threshold for short-duration VF was 2.4 J/kg, whereas in another[291] it was 3.3 J/kg.

In 4 LOE 5 animal studies[290,292–294] of AED shocks delivered using a pediatric attenuator, 50 J and 50→76→86 J (2.5 to 4 J/ kg) escalating doses were effective at terminating long-duration VF but required multiple shocks. In 1 LOE 5 animal study[295] 10 J/kg shocks were more effective at terminating long-duration VF (6 minutes) with 1 shock than 4 J/kg shocks.

In 2 LOE 4 pediatric studies[264,286] and 4 LOE 5 animal studies,[290,292–294] energy doses of 2 to 10 J/kg for short- or long-duration VF resulted in equivalent rates of survival. Myocardial damage, as assessed by hemodynamic or biochemical measurements, was less when a pediatric attenuator was used with an adult energy dose compared with a full adult AED dose, but the degree of myocardial damage was not associated with any difference in 4- or 72-hour survival. An LOE 5 animal study[295] found no difference in hemodynamic parameters or biochemical measurements of myocardial damage comparing biphasic 150 J (4 J/kg) with monophasic 360 J/kg (10 J/kg) shocks.

In 2 LOE 5 animal studies[290,291] biphasic waveforms were more effective than monophasic waveforms for treatment of VF/pulseless VT. There are no human data that directly compare monophasic to biphasic waveforms for pediatric defibrillation.

Treatment Recommendations

An initial dose of 2 to 4 J/kg is reasonable for pediatric defibrillation. Higher subsequent energy doses may be safe and effective.

Knowledge Gaps

What is the minimum effective and maximum safe defibrillation energy dose for pediatric VF/pulseless VT? What is the optimal parameter (eg, weight or length) on which to base defibrillation energy doses for infants and children? Should the energy dose for defibrillation be escalated for shock-refractory VF?

Does the use of biphasic waveforms when compared to monophasic waveforms improve outcome from pediatric cardiac arrest?

Amiodarone Versus Lidocaine for Refractory VF/Pulseless VT[Peds-019]

Consensus on Science

In 2 LOE 5 prospective out-of-hospital adult trials IV amiodarone improved ROSC and survival to hospital admission but not hospital discharge when compared with placebo[296] or lidocaine[297] for treatment of shock-refractory VF/ pulseless VT. Evidence from 2 LOE 5 case series in children[298,299] supported the effectiveness of amiodarone for the treatment and acute conversion of life-threatening (non-arrest) ventricular arrhythmias. There are no pediatric data investigating the efficacy of lidocaine for shock refractory VF/ pulseless VT.

Treatment Recommendations

Amiodarone may be used for the treatment of shock-refractory or recurrent VF/pulseless VT in infants and children; if amiodarone is not available, lidocaine may be considered.

Knowledge Gaps

Does the use of amiodarone compared with lidocaine improve outcome from shock-refractory or recurrent VF/pulseless VT in infants and children? Is lidocaine effective for the treatment of VF/pulseless VT in children?

AED Use in Infants[Peds-001A, Peds-001B]

Consensus on Science

One LOE 4[300] and 2 LOE 5[288,301] studies showed that infants in cardiac arrest (in- and out-of-hospital) may have shockable rhythms. Evidence from 3 LOE 5[302–304] studies showed that many AED devices can safely and accurately distinguish between a shockable and nonshockable rhythm in infants and children.

The optimal energy dose for defibrillation in infants has not been established, but indirect data from 5 LOE 5 animal studies[287,294,305–307] showed that the young myocardium may be able to tolerate high-energy doses. In 3 LOE 5 animal studies a pediatric attenuator used with an adult-dose biphasic AED shock was as effective and less harmful than monophasic weight-based doses[290] or biphasic adult doses.[292,293]

Two LOE 4 case reports[308,309] described survival of infants with out-of-hospital cardiac arrest when AED use was coupled with bystander CPR and defibrillation using an AED. Two pediatric LOE 5 case reports[310,311] noted successful defibrillation with minimal myocardial damage and good neurologic outcome using an AED with adult energy doses.

Treatment Recommendations

For treatment of out-of-hospital VF/pulseless VT in infants, the recommended method of shock delivery by device is listed in order of preference below. If there is any delay in the availability of the preferred device, the device that is available should be used. The AED algorithm should have demonstrated high specificity and sensitivity for detecting

shockable rhythms in infants. The order of preference is as follows:

1. Manual defibrillator
2. AED with dose attenuator
3. AED without dose attenuator

Knowledge Gaps
Is there a lower limit of infant size or weight below which an AED should not be used?

Arrhythmia Therapy[Peds-030]

The evidence on emergency treatment of arrhythmias was reviewed and the only change was the addition of procainamide as possible therapy for refractory supraventricular tachycardia (SVT).

Unstable VT

Consensus on Science
There is insufficient evidence to support or refute the efficacy of electric therapy over drug therapy or the superiority of any drug for the emergency treatment of unstable VT in the pediatric age group. In 2 LOE 5 adult case series,[312,313] early electric cardioversion was effective for treatment of unstable VT.

In 4 small LOE 4 pediatric case series[298,299,314,315] amiodarone was effective in the management of VT. One prospective randomized multicenter safety and efficacy LOE 2 trial evaluating amiodarone for the treatment of pediatric tachyarrhythmias[316] found that 71% of children treated with amiodarone experienced cardiovascular side effects. Both efficacy and adverse events were dose-related.

Treatment Recommendations
It is reasonable to use synchronized electric cardioversion as the preferred first therapy for pediatric VT with hypotension or evidence of poor perfusion. If drug therapy is used to treat unstable VT, amiodarone may be a reasonable choice, with careful hemodynamic monitoring performed during its slow delivery.

Knowledge Gaps
What is the optimal dose of energy for synchronized cardioversion during treatment of unstable VT in pediatric patients?

Drugs for Supraventricular Tachycardia[Peds-031]

Consensus on Science
Twenty-two LOE 4 studies in infants and children[317–338] demonstrated the effectiveness of adenosine for the treatment of hemodynamically stable or unstable SVT. One LOE 4 study[339] and 4 larger LOE 5 studies involving teenagers and adults[340–343] also demonstrated the efficacy of adenosine, although frequent but transient side effects were reported.

One LOE 2 study[344] showed highly successful (approximately 90%) treatment of SVT in infants and children using adenosine or verapamil and superiority of these drugs to digitalis (61% to 71%). One LOE 5 randomized prospective adult study[345] and 1 LOE 5 meta-analysis, primarily involving adults but including some children,[346] demonstrated the effectiveness of verapamil and adenosine in treating SVT and highlighted the cost-effectiveness of verapamil over adenosine.

One LOE 4 randomized, prospective study[316] and 15 LOE 4 small case series and observational studies in infants and children[298,299,314,315,347–357] showed that amiodarone was effective in the treatment of supraventricular tachyarrhythmias. Generalization to pediatric SVT treatment with amiodarone may be limited, however, since most of these studies in children involved postoperative junctional tachycardia.

Rare but significant side effects have been reported in association with rapid administration of amiodarone. Bradycardia and hypotension were reported in 1 prospective LOE 4 study,[316] cardiovascular collapse was reported in 2 LOE 5 case reports,[358,359] and polymorphic VT was reported in 1 small LOE 4 case series.[360] Other LOE 5 case reports describe late side effects of pulmonary toxicity[359] and hypothyroidism.[362]

In 1 LOE 2 pediatric comparison control study[363] procainamide had a significantly higher success rate and an equal incidence of adverse effects when compared with amiodarone for treating refractory SVT. In 5 LOE 4 observational studies[364–368] and 5 LOE 5 case reports[369–373] procainamide effectively suppressed or slowed the rate in children with SVT. A wide variety of arrhythmias were studied, including ectopic atrial tachycardia, atrial flutter, and orthodromic reciprocating tachycardia.

In LOE 5 studies in children,[374] adults,[375,376] and animals,[377] hypotension from procainamide infusion resulted from vasodilation and not decreased myocardial contractility. Initial observational LOE 4 reports[378–380] and 1 LOE 4 case series[381] described successful treatment of pediatric SVT with verapamil. However, multiple small LOE 4 case series[344,382] and LOE 5 case reports[383,384] documented severe hypotension, bradycardia, and heart block causing hemodynamic collapse and death following IV administration of verapamil for SVT in infants. Two small LOE 4 pediatric case series[385,386] described esmolol and dexmedetomidine in the treatment of SVT.

Treatment Recommendations
For infants and children with SVT with a palpable pulse, adenosine should be considered the preferred medication. Verapamil may be considered as alternative therapy in older children but should not be routinely used in infants. Procainamide or amiodarone given by a slow IV infusion with careful hemodynamic monitoring may be considered for refractory SVT.

Knowledge Gaps
Does the use of alternate medications (eg, esmolol, dexmedetomidine) in the treatment of SVT in infants and children improve outcome? What is the role of vagal maneuvers in the treatment of SVT?

Shock

The Task Force reviewed evidence related to several key questions about the management of shock in children. There is ongoing uncertainty about the indications for using colloid versus crystalloid in shock resuscitation. One large adult trial suggested that normal saline (isotonic crystalloid) is equiva-

lent to albumin, although subgroup analysis suggested harm associated with the use of colloid in patients with traumatic brain injury. There were insufficient data to change the 2005 recommendations.

The optimal timing for intubation of children in shock remains unclear, although reports of children and adults with septic shock suggested potential beneficial effects of early intubation (before signs of respiratory failure develop) combined with a protocol-driven management approach. When children in septic shock were treated with a protocol that included therapy directed to normalizing central venous oxygen saturation, patient outcome appeared to improve.

Performing rapid sequence induction and tracheal intubation of a child with shock can cause acute cardiovascular collapse. Etomidate typically causes less hemodynamic compromise than other induction drugs and is therefore often used in this setting. However, data suggest that the use of this drug in children and adults with septic shock is associated with increased mortality that may be secondary to etomidate's inhibitory effects on corticosteroid synthesis. Administering stress-dose corticosteroids in septic shock remains controversial, with recent adult trials failing to show a beneficial effect.

Graded Volume Resuscitation for Hemorrhagic Shock[Peds-032]

Consensus on Science
There are no pediatric studies of the timing or extent of volume resuscitation in hemorrhagic shock with hypotension. Nine LOE 5 adult[387–395] studies reported conflicting results with regard to the effect of timing and extent of volume resuscitation on outcome of hemorrhagic shock with hypotension.

Treatment Recommendations
There is insufficient evidence as to the best timing or quantity for volume resuscitation in infants and children with hemorrhagic shock following trauma.

Knowledge Gaps
What is the appropriate clinical indicator for volume resuscitation during treatment of hemorrhagic shock in infants and children?

Early Ventilation in Shock[Peds-038B]

Consensus on Science
There are no studies investigating the role of intubation and assisted ventilation before the onset of respiratory failure in infants and children with shock. Two LOE 5 animal studies in septic shock[396,397] and 1 LOE 5 animal study in pericardial tamponade[398] showed improved hemodynamics and select organ perfusion with intubation before the onset of respiratory failure. One report of 2 adult patients[399] (LOE 5) described cardiac arrest following intubation of 1 adult patient with tamponade due to penetrating trauma and improvement in hemodynamics during spontaneous breathing in 1 mechanically ventilated adult patient with post–cardiac surgery tamponade.

One LOE 5 study of septic shock in adults[400] suggested a reduced mortality with early ventilation compared with historic controls who only received ventilation for respiratory failure. One LOE 5 study of animals in septic shock[401] showed that early assisted ventilation does not reduce oxygen extraction or prevent the development of lactic acidosis.

Treatment Recommendations
There is insufficient evidence to support or refute the use of endotracheal intubation of infants and children in shock before the onset of respiratory failure.

Knowledge Gaps
Does the timing of respiratory support in infants and children with shock affect outcome?

Colloid Versus Crystalloid Fluid Administration[Peds-044A, Peds-044B]

Consensus on Science
Evidence from 3 randomized blinded LOE 1 controlled trials in children with dengue shock syndrome[402–404] and 1 LOE 1 open randomized trial in children with septic shock[405] suggested no clinically important differences in survival from therapy with colloid versus therapy with isotonic crystalloid solutions for shock.

In 1 large LOE 5 randomized controlled trial of fluid therapy in adult ICU patients[406] and in 6 good-quality LOE 5 meta-analyses, predominantly of adults,[407–412] no mortality differences were noted when colloid was compared with hypertonic and isotonic crystalloid solutions, and no differences were noted between types of colloid solutions.

Three LOE 5 studies comparing the use of crystalloids and colloids for adults in shock suggested that crystalloid may have an associated survival benefit over colloid in subgroups of patients with shock, including general trauma,[409] traumatic brain injury,[413] and burns.[414] One randomized controlled LOE 5 study of children with severe malaria suggested better survival with colloid than with crystalloid infusion.[415]

Treatment Recommendations
Isotonic crystalloids are recommended as the *initial* resuscitation fluid for infants and children with any type of shock. There is insufficient evidence to identify the superiority of any specific isotonic crystalloid over others.

Knowledge Gaps
Does the use of any specific crystalloid solution (Ringer's lactate, normal saline, hypertonic saline) improve outcome for pediatric shock? Are there subgroups of children in shock whose outcome is improved with the use of colloid compared with crystalloid?

Vasoactive Agents in Distributive Shock[Peds-045A, Peds-045B]

Consensus on Science
One LOE 4 observational study[416] suggested that the course of pediatric septic shock physiology is dynamic and that serial assessments are required to titrate the type and dose of inotropes or vasopressor therapy to achieve optimal hemodynamic results. Evidence from 4 LOE 1 pediatric randomized controlled studies,[417–420] 3 LOE 5 adult randomized controlled studies,[421–423] and 1 LOE 5 adult systematic review[424]

showed that no inotrope or vasopressor is superior in reducing mortality from pediatric or adult distributive shock.

Two LOE 1 pediatric randomized controlled studies[417,418] showed that children with "cold" (ie, low cardiac index) septic shock improved hemodynamically with brief (4-hour) administration of milrinone (bolus and infusion). One LOE 1 pediatric randomized controlled study[420] of vasodilatory shock compared the addition of vasopressin versus placebo to standard vasoactive agents and showed no change in duration of vasopressor infusion but observed a trend toward increased mortality.

Eleven small LOE 4 pediatric case series[425–435] showed improved hemodynamics but not survival when vasopressin or terlipressin was administered to children with refractory, vasodilatory, septic shock.

Treatment Recommendations

There is insufficient evidence to recommend a specific inotrope or vasopressor to improve mortality in pediatric distributive shock. Selection of an inotrope or vasopressor to improve hemodynamics should be tailored to each patient's physiology and adjusted as clinical status changes.

Knowledge Gaps

Does the use of any specific vasoactive agent improve outcome for infants and children with distributive shock?

Vasoactive Agents in Cardiogenic Shock[Peds-046A]

Consensus on Science

One LOE 4 pediatric case series[436] showed that critically ill children requiring inotropic support have wide variability in hemodynamic responses to different infusion rates of dobutamine. One LOE 2 blinded crossover study[437] found dopamine and dobutamine had equal hemodynamic effects in infants and children requiring post–cardiac surgical inotropic support but that dopamine at an infusion rate of >7 mcg/kg per minute increased pulmonary vascular resistance.

Six LOE 3 studies[438–443] showed that both dopamine and dobutamine infusions improve hemodynamics in children with cardiogenic shock.

Evidence from 1 LOE 1 pediatric placebo-controlled trial[444] showed that milrinone is effective in preventing low cardiac output syndrome in infants and children following biventricular cardiac repair. One LOE 4 study[445] showed that milrinone improved cardiac index in neonates with low cardiac output following cardiac surgery.

One small LOE 1 study[446] showed that children had better hemodynamic parameters and shorter ICU stays if they received milrinone compared with low-dose epinephrine plus nitroglycerin for inotropic support following repair of tetralogy of Fallot.

In 2 LOE 4 small case series,[447,448] when children with heart failure secondary to myocardial dysfunction were given levosimendan, they demonstrated improved ejection fraction, required a shorter duration of catecholamine infusions,[447] and showed a trend toward improved hemodynamics and reduced arterial lactate levels.[448]

In subgroup analysis from 1 LOE 5 randomized controlled trial in adults,[449] patients with cardiogenic shock treated with norepinephrine versus dopamine had an improved survival at 28 days. When all causes of shock were included, patients treated with norepinephrine also had fewer arrhythmias than those treated with dopamine (12% versus 24%).

Treatment Recommendations

The catecholamine dose for inotropic support in cardiogenic shock must be individually titrated because there is a wide variability in clinical response. It is reasonable to use epinephrine, levosimendan, dopamine, or dobutamine for inotropic support in infants and children with cardiogenic shock. Milrinone may be beneficial for the prevention and treatment of low cardiac output following cardiac surgery.

There are insufficient data to support or refute the use of norepinephrine in pediatric cardiogenic shock.

Knowledge Gaps

Does the use of any specific vasoactive agent improve outcome for infants and children with cardiogenic shock who have not undergone cardiac surgery?

Etomidate for Intubation in Hypotensive Septic Shock[Peds-047A, Peds-047B]

Consensus on Science

One LOE 4 study of children with septic shock[450] showed that adrenal suppression occurred after the administration of a single dose of etomidate and persisted for at least 24 hours. Evidence from 2 LOE 4[451,452] studies and 1 LOE 5[453] study showed that etomidate can be used to facilitate tracheal intubation in infants and children with minimal hemodynamic effect, but very few of these reports included patients with hypotensive septic shock. One LOE 4 study[450] suggested an association with mortality when etomidate is used to facilitate the intubation of children with septic shock.

One adult LOE 5 study[454] observed an increased mortality associated with the use of etomidate for intubation of patients in septic shock, even with steroid supplementation. Conversely, 1 underpowered adult LOE 5 study[455] in septic patients did not show an increase in mortality.

One multicenter adult LOE 5 comparative trial of etomidate versus ketamine for intubation[456] found no difference in organ failure over the first 72 hours and no mortality difference, but this study included only a small number of patients with shock. Adrenal insufficiency was more common in etomidate-treated patients.

Treatment Recommendations

Etomidate should not be routinely used when intubating an infant or child with septic shock. If etomidate is used in infants and children with septic shock, the increased risk of adrenal insufficiency should be recognized.

Knowledge Gaps

If etomidate is used, does steroid administration improve outcome for infants and children with septic shock?

Corticosteroids in Hypotensive Shock[Peds-049A, Peds-049B]

Consensus on Science

In 6 LOE 5 randomized controlled trials in adults with septic shock[454,457–461] the addition of low-dose hydrocortisone de-

creased the time to shock reversal. Three LOE 5 randomized controlled trials in adults with vasopressor-dependent septic shock[457,462,463] showed that survival was improved when low-dose hydrocortisone was administered, while 1 small adult LOE 5 randomized controlled trial[464] showed a trend toward increased survival.

One fair-quality, small LOE 1 study in children with septic shock[465] found that low-dose hydrocortisone administration resulted in no survival benefit. One fair-quality LOE 1 study of administration of low-dose hydrocortisone to children with septic shock[466] demonstrated earlier shock reversal. Data from 1 LOE 4 hospital discharge database[467] noted the association between the use of steroids in children with severe sepsis and decreased survival.

In 1 LOE 5 study in adults with septic shock[457] survival improved significantly with the use of low-dose hydrocortisone and fludrocortisone compared with placebo. Conversely 4 LOE 5 adult trials in septic shock[454,459–461] showed no survival benefit with low-dose corticosteroid therapy. In 1 large LOE 5 randomized controlled trial of adults in septic shock,[454] corticosteroid administration was associated with an increased risk of secondary infection.

Treatment Recommendations

There is insufficient evidence to support or refute the routine use of stress-dose or low-dose hydrocortisone and/or other corticosteroids in infants and children with septic shock. Stress-dose corticosteroids may be considered in children with septic shock unresponsive to fluids and requiring vasoactive support.

Knowledge Gaps

What is the appropriate "stress dose" of hydrocortisone for hypotensive septic shock? Should the dose of hydrocortisone be titrated to the degree of shock? Should an adrenocorticotrophin (ACTH) stimulation test be performed to determine if an infant or child in septic shock has adrenal insufficiency?

Diagnostic Tests as Guide to Management of Shock[Peds-050A, Peds-050B]

Consensus on Science

In 1 LOE 1 randomized controlled trial in children with severe sepsis or fluid-refractory septic shock,[468] protocol-driven therapy that included targeting a superior vena caval oxygen saturation >70%, coupled with treating clinical signs of shock (prolonged capillary refill, reduced urine output, and reduced blood pressure), improved patient survival to hospital discharge in comparison to treatment guided by assessment of clinical signs alone.

Two LOE 5 studies of adults with septic shock, one a randomized controlled trial[469] and the other a cohort study,[470] documented improved survival to hospital discharge following implementation of protocol-driven early goal-directed therapy, including titration to a central venous oxygen saturation ($Svco_2$) ≥70%. In 1 large multicenter LOE 5 adult study[471] evaluating the "Surviving Sepsis" bundle, early goal-directed therapy to achieve an $Svco_2$ ≥70% was not associated with an improvement in survival, but venous oxygen saturations were measured in <25% of participants.

There are insufficient data on the utility of other diagnostic tests (eg, pH, lactate) to help guide the management of infants and children with shock.

Treatment Recommendations

A protocol-driven therapy, which includes titration to a superior vena caval oxygen saturation ≥70%, may be beneficial for infants and children (without cyanotic congenital heart disease) with fluid-refractory septic shock. No treatment recommendations can be made to target $Svco_2$ saturation in the management of fluid-refractory septic shock in pediatric patients with cyanotic congenital heart disease or for other forms of pediatric shock.

Knowledge Gaps

What is the optimal diagnostic test (ie, lactate, $Svco_2$) to guide management of pediatric shock? Does continuous versus intermittent $Svco_2$ monitoring affect outcome?

Medications in Cardiac Arrest and Bradycardia

The Task Force reviewed and updated evidence to support medications used during cardiac arrest and bradycardia, but no new recommendations were made. It was again emphasized that calcium and sodium bicarbonate should not be routinely used in pediatric cardiac arrest (ie, should not be used without specific indications).

Calculating Drug Dose[Peds-017B]

Consensus on Science

Eight LOE 5 studies[472–479] concluded that length-based methods are more accurate than age-based or observer (parent or provider) estimate-based methods in the prediction of body weight. Four LOE 5 studies[472,474,480,481] suggested that the addition of a category of body habitus to length may improve prediction of body weight.

Six LOE 5 studies[482–487] attempted to find a formula based on drug pharmacokinetics and physiology that would allow the calculation of a pediatric dose from the adult dose.

Treatment Recommendations

In nonobese pediatric patients, initial resuscitation drug doses should be based on actual body weight (which closely approximates ideal body weight). If necessary, body weight can be estimated from body length.

In obese patients the initial doses of resuscitation drugs should be based on ideal body weight that can be estimated from length. Administration of drug doses based on actual body weight in obese patients may result in drug toxicity.

Subsequent doses of resuscitation drugs in both nonobese and obese patients should take into account observed clinical effects and toxicities. It is reasonable to titrate the dose to the desired therapeutic effect, but it should not exceed the adult dose.

Knowledge Gaps

What is the most accurate method for calculating resuscitation drug doses for children? Does the accuracy of the estimated weight used to calculate drug dose affect patient outcome? Do specific resuscitation drugs require different

adjustments for estimated weight, maturity and/or body composition?

Are formulas for scaling drug doses with formulas from adult doses superior to existing weight-based methods?

Epinephrine Dose[Peds-018]

Consensus on Science

No studies have compared epinephrine versus placebo administration for pulseless cardiac arrest in infants and children. One LOE 5 randomized controlled adult study[488] of standard drug therapy compared with no drug therapy during out-of-hospital cardiac arrest showed improved survival to hospital admission with any drug delivery but no difference in survival to hospital discharge.

Evidence from 1 LOE 1 prospective, randomized, controlled trial,[489] 2 LOE 2 prospective trials,[490,491] and 2 LOE 2 case series with concurrent controls[492,493] showed no increase in survival to hospital discharge or improved neurologic outcome when epinephrine doses of >10 mcg/kg IV were used in out-of-hospital or in-hospital pediatric cardiac arrest. In 1 LOE 1 prospective trial[489] of pediatric in-hospital cardiac arrest comparing high-dose (100 mcg/kg) with standard-dose epinephrine administered if cardiac arrest persisted after 1 standard dose of epinephrine, 24-hour survival was reduced in the high-dose epinephrine group.

Evidence extrapolated from adult prehospital or in-hospital studies, including 9 LOE 1 randomized trials,[494–502] 3 LOE 2 trials,[503–505] and 3 LOE 3 studies,[506–508] showed no improvement in survival to hospital discharge or neurologic outcome when doses >1 mg of epinephrine were given.

Treatment Recommendations

In infants and children with out-of-hospital or in-hospital cardiac arrest, the appropriate dose of IV epinephrine is 10 mcg/kg per dose (0.01 mg/kg) for the first and for subsequent doses. The maximum single dose is 1 mg.

Knowledge Gaps

Does epinephrine administration improve outcome from cardiac arrest in infants and children? Are there specific patients or arrest types (eg, prolonged arrest, asphyxial arrest, VF arrest) for which epinephrine is more effective?

Sodium Bicarbonate During Cardiac Arrest[Peds-028]

Consensus on Science

There are no randomized controlled studies in infants and children examining the use of sodium bicarbonate as part of the management of pediatric cardiac arrest. One LOE 2 multicenter retrospective in-hospital pediatric study[509] found that sodium bicarbonate administered during cardiac arrest was associated with decreased survival, even after controlling for age, gender, and first documented cardiac rhythm.

Two LOE 5 randomized controlled studies have examined the value of sodium bicarbonate in the management of arrest in other populations: 1 adult out-of-hospital cardiac arrest study[510] and 1 study in neonates with respiratory arrest in the delivery room.[511] Both failed to show an improvement in overall survival.

Treatment Recommendations

Routine administration of sodium bicarbonate is not recommended in the management of pediatric cardiac arrest.

Knowledge Gaps

Are there circumstances under which sodium bicarbonate administration improves outcome from pediatric cardiac arrest?

Vasopressin[Peds-020A, Peds-020B]

Consensus on Science

In 1 pediatric LOE 3 study[512] vasopressin was associated with lower ROSC and a trend toward lower 24-hour and discharge survival. In 3 pediatric LOE 4[513–515] and 2 adult LOE 5[516,517] case series/reports (9 patients) vasopressin[513] or its long-acting analogue, terlipressin,[514,515] administration was associated with ROSC in patients with refractory cardiac arrest (ie, standard therapy failed).

Extrapolated evidence from 6 LOE 5 adult studies[518–523] and 1 LOE 1 adult meta-analysis[524] showed that vasopressin used either by itself or in combination with epinephrine during cardiac arrest does not improve ROSC, hospital discharge, or neurologic outcome. Evidence from 1 LOE 5 animal study[525] of an infant asphyxial arrest model showed no difference in ROSC when terlipressin was administered alone or in combination with epinephrine as compared with epinephrine alone.

Treatment Recommendations

There is insufficient evidence for or against the administration of vasopressin or its long-acting analogue, terlipressin, in pediatric cardiac arrest.

Knowledge Gaps

Are there patient subgroups who might benefit from vasopressin (with or without other vasopressors) for pediatric cardiac arrest? Does the use of "early" versus "late" (ie, rescue) vasopressin affect outcome in pediatric cardiac arrest? Is vasopressin effective when administered via a tracheal tube?

Calcium in Cardiac Arrest[Peds-021A, Peds-021B]

Consensus on Science

Evidence from 3 LOE 2[509,526,527] studies in children and 5 LOE 5 adult studies[528–532] failed to document an improvement in survival to hospital admission, hospital discharge, or favorable neurologic outcome when calcium was administered during cardiopulmonary arrest in the absence of documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia. Four LOE 5 animal studies[533–536] showed no improvement in ROSC when calcium, compared with epinephrine or placebo, was administered during cardiopulmonary arrest.

Two studies investigating calcium for in-hospital pediatric cardiac arrest suggested a potential for harm. One LOE 2 study examining data from the NRCPR[526] observed an adjusted odds ratio of survival to hospital discharge of 0.6 in children who received calcium, and 1 LOE 3 multicenter study[509] showed an odds ratio for increased hospital mortality

of 2.24 associated with the use of calcium. One LOE 2 study of cardiac arrest in the PICU setting[527] suggested a potential for harm with the administration of calcium during cardiac arrest; the administration of 1 or more boluses was an independent predictor of hospital mortality.

Treatment Recommendations
Routine use of calcium for infants and children with cardiopulmonary arrest is not recommended in the absence of hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia.

Knowledge Gaps
Are there indications for calcium administration that may be associated with improved outcome from pediatric cardiac arrest? Does the increased mortality risk associated with calcium administration reflect harm from calcium or does it simply identify patients who failed to respond to other ALS interventions and therefore were at a higher risk of death?

Atropine Versus Epinephrine for Bradycardia[Peds-052A]

Consensus on Science
Evidence from 1 LOE 3 study of in-hospital pediatric cardiac arrest[537] observed an improved odds of survival to discharge for those patients who received atropine based on multivariate analysis, whereas the use of epinephrine was associated with decreased odds of survival. Another large LOE 3 study[538] demonstrated no association between atropine administration and survival.

In 1 LOE 5 adult case series,[539] 6 of 8 patients in cardiac arrest who did not respond to epinephrine did respond to atropine with a change to a perfusing rhythm; 3 survived to hospital discharge. An LOE 5 retrospective adult review[540] observed that a small number of asystolic patients who failed to respond to epinephrine did respond to atropine, but none survived to hospital discharge.

Four LOE 5 adult studies[541–544] showed a benefit of atropine in vagally mediated bradycardia. One small LOE 4 pediatric case series[545] showed that atropine is more effective than epinephrine in increasing heart rate and blood pressure in children with post–cardiac surgical hypotension and bradycardia (Bezold-Jarisch reflex mediated bradycardia).

Four LOE 5 adult[542,546–548] and 4 LOE 5 animal[549–552] studies showed no benefit from atropine used to treat bradycardia or cardiac arrest. One LOE 5 animal study[553] did show a benefit of atropine when used with epinephrine in cardiac arrest.

Treatment Recommendations
Epinephrine may be used for infants and children with bradycardia and poor perfusion that is unresponsive to ventilation and oxygenation. It is reasonable to administer atropine for bradycardia caused by increased vagal tone or cholinergic drug toxicity. There is insufficient evidence to support or refute the routine use of atropine for pediatric cardiac arrest.

Knowledge Gaps
What is the optimal dose of epinephrine for pediatric bradycardia? Is there a role for titrated doses? Does the use of epinephrine versus atropine improve outcome from pediatric bradycardia? Are there circumstances under which atropine administration improves outcome from pediatric cardiac arrest?

Extracorporeal Cardiac Life Support[Peds-014, Peds-014B]

There is increasing evidence that extracorporeal cardiac life support (ECLS) can act as a bridge to maintain oxygenation and circulation in selected infants and children with cardiac arrest if they are transplant candidates or have a self-limited or treatable illness. When ECLS is initiated for the treatment of cardiac arrest, it is referred to as ECPR (extracorporeal CPR). ECPR can only be employed if the cardiac arrest occurs in a monitored environment with protocols and personnel for rapid initiation.

Consensus on Science
One LOE 2[554] and 26 LOE 4 studies[555–580] reported favorable early outcome after ECPR in children with primary cardiac disease who were located in an ICU or other highly supervised environment using ECPR protocols at the time of the arrest.

One LOE 2[554] and 2 LOE 4[555,564] studies indicated poor outcome from ECPR in children with noncardiac diseases.

In 1 LOE 4 study[556] survival following ECPR in children was associated with shorter time interval between arrest and ECPR team activation and shorter CPR duration. Two LOE 4 studies[560,581] found insignificant improvements in outcome after ECPR in children following protocol changes leading to shorter durations of CPR. One LOE 2[554] and 3 LOE 4[555,559,565] studies found no relationship between CPR duration and outcome after ECPR in children.

Three small LOE 4 studies,[582–584] including a total of 21 children, showed favorable outcome with ECPR following out-of-hospital cardiac arrest associated with environmentally induced severe hypothermia (temperature $<30°C$).

Treatment Recommendations
ECPR may be beneficial for infants and children with cardiac arrest if they have heart disease amenable to recovery or transplantation and the arrest occurs in a highly supervised environment such as an ICU with existing clinical protocols and available expertise and equipment to rapidly initiate ECPR. There is insufficient evidence for any specific threshold for CPR duration beyond which survival with ECPR is unlikely. ECPR may be considered in cases of environmentally induced severe hypothermia (temperature $<30°C$) for pediatric patients with out-of-hospital cardiac arrest if the appropriate expertise, equipment, and clinical protocols are in place.

Knowledge Gaps
What are the long-term neurologic outcomes of pediatric patients treated with ECPR? Is there an upper limit for the duration of standard CPR beyond which using ECPR will be of no benefit?

Post-Resuscitation Care

The Task Force reviewed evidence regarding hypothermia for pediatric patients who remain comatose following resuscita-

tion from cardiac arrest. There is clear benefit for adult patients who remain comatose after VF arrest, but there is little evidence regarding effectiveness for infants (ie, beyond the neonatal period) and young children who most commonly have asphyxial arrest.

Some patients with sudden death without an obvious cause have a genetic abnormality of myocardial ion channels (ie, a channelopathy), which presumably leads to a fatal arrhythmia. Because this is an inherited abnormality, family members might be affected, but special tests are required for the detection of this inherited genetic defect.

Hypothermia[Peds-010A, Peds-010B]

Consensus on Science
There are no randomized pediatric studies on induced therapeutic hypothermia following cardiac arrest.

Two prospective randomized LOE 5 studies of adults with VF arrest[585,586] and 2 prospective randomized LOE 5 studies of newborns with birth asphyxia[587,588] showed that therapeutic hypothermia (32° to 34°C) up to 72 hours after resuscitation has an acceptable safety profile and may be associated with better long-term neurologic outcome.

One LOE 2 observational study[589] neither supports nor refutes the use of therapeutic hypothermia after resuscitation from pediatric cardiac arrest. However, patients in this study were not randomized, and the cooled patients were much sicker and younger than those not cooled.

Treatment Recommendations
Therapeutic hypothermia (to 32°C to 34°C) may be beneficial for adolescents who remain comatose following resuscitation from sudden witnessed out-of-hospital VF cardiac arrest. Therapeutic hypothermia (to 32°C to 34°C) may be considered for infants and children who remain comatose following resuscitation from cardiac arrest.

Knowledge Gaps
Does therapeutic hypothermia improve outcome following pediatric cardiac arrest? Is there a difference in effectiveness for VF arrest versus asphyxial arrest? What is the optimal protocol for cooling after pediatric cardiac arrest (timing, duration, goal temperature, rate of rewarming)?

Vasoactive Drugs[Peds-024A, Peds-024B]

Consensus on Science
There are no studies evaluating the role of vasoactive medications after ROSC in children. Evidence from 2 LOE 3 studies in children,[590,591] 2 LOE 5 studies in adults,[592,593] and 2 LOE 5 animal studies[594,595] documented that myocardial dysfunction and vascular instability are common following resuscitation from cardiac arrest.

Evidence from 6 LOE 5 animal studies[594–599] documented hemodynamic improvement when vasoactive medications (dobutamine, milrinone, levosimendan) were given in the post–cardiac arrest period. Evidence from 1 large LOE 5 pediatric[444] and 4 LOE 5 adult[600–603] studies of patients with low cardiac output or at risk for low cardiac output following cardiac surgery documented consistent improvement in hemodynamics when vasoactive medications were administered.

Treatment Recommendations
It is reasonable to administer vasoactive medications to infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest. These vasoactive medications should be selected and titrated to improve myocardial function and/or organ perfusion while trying to limit adverse effects.

Knowledge Gaps
What is the optimal vasoactive drug regimen for postarrest myocardial dysfunction in infants and children?

Glucose[Peds-016]

Consensus on Science
There is insufficient evidence to support or refute any specific glucose management strategy in infants and children following cardiac arrest. Although there is an association of hyperglycemia and hypoglycemia with poor outcome following ROSC after cardiac arrest, there are no studies that show causation and no studies that show that the treatment of either hyperglycemia or hypoglycemia following ROSC improves outcome.

Two studies of adult survivors of cardiac arrest, including 1 LOE 5 prospective observational study[604] and 1 LOE 5 randomized controlled trial comparing tight with moderate glucose control[605] observed no survival benefit with tight glucose control. Two studies of tight glucose control in adult surgical ICU patients, including 1 LOE 1 prospective randomized controlled trial[606] and 1 LOE 1 meta-analysis[607] observed reduced mortality with tight glucose control. Two LOE 5 meta-analyses comparing tight with moderate glucose control in adult ICU patients[608,609] and 1 LOE 5 randomized controlled trial comparing tight with moderate glucose control in adult medical ICU patients[610] observed no differences in survival. Three LOE 5 studies of tight glucose control in adult ICU patients, including 1 randomized controlled trial in cardiac surgical patients,[611] 1 multicenter randomized controlled trial in medical and surgical ICU patients,[612] and 1 cohort-controlled study of medical and surgical ICU patients[613] demonstrated increased mortality with tight glucose control.

One LOE 5 randomized controlled trial of critically ill children[614] observed an improvement in inflammatory biochemical markers and reduced ICU length of stay with tight glucose control. One study of tight glucose control of critically ill neonates[615] was terminated early for reasons of futility. Significant rates of hypoglycemia are widely reported with the use of tight glucose control without explicit methodology or continuous glucose monitoring in critically ill neonates,[615] children,[614] and adults.[607,608,612]

Evidence from LOE 5 animal studies of neonatal cerebral ischemia[616] and critically ill adults[617,618] suggest that hypoglycemia combined with hypoxia and ischemia is harmful and associated with higher mortality. Evidence from 3 LOE 5 animal studies[619–621] showed that prolonged hyperglycemia after resuscitation is harmful to the brain. One LOE 5 animal

study[622] showed that glucose infusion with associated hyperglycemia after resuscitation worsened outcome, whereas another LOE 5 animal study[623] showed that moderate hyperglycemia managed with insulin improved neurologic outcome.

Treatment Recommendations
It is appropriate to monitor blood glucose levels and avoid hypoglycemia as well as sustained hyperglycemia following cardiac arrest. There is insufficient evidence to recommend specific strategies to manage hyperglycemia in infants and children with ROSC following cardiac arrest. If hyperglycemia is treated following ROSC in children, blood glucose concentrations should be carefully monitored to reduce episodes of hypoglycemia.

Knowledge Gaps
Does the use of "tight" glucose control improve outcome following pediatric cardiac arrest?

Channelopathy[Peds-048A, Peds-048B]

Consensus on Science
In 4 LOE 4 studies[624–627] 14% to 35% of young patients with sudden, unexpected death had no abnormalities found at autopsy.

In 7 LOE 3 studies[628–634] mutations causing channelopathies occurred in 2% to 10% of infants with sudden infant death syndrome noted as the cause of death. In 1 LOE 3[635] and 2 LOE 4[636,637] studies 14% to 20% of young adults with sudden, unexpected death had no abnormalities on autopsy but had genetic mutations causing channelopathies. In 4 LOE 4 studies,[638–641] using clinical and laboratory (electrocardiographic, molecular-genetic screening) investigations, 22% to 53% of first- and second-degree relatives of patients with sudden, unexplained death had inherited, arrhythmogenic disease.

Treatment Recommendations
When sudden unexplained cardiac arrest occurs in children and young adults, a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents/drownings, or sudden death) should be obtained and any available previous ECGs should be reviewed. All infants, children, and young adults with sudden, unexpected death should, if possible, have an unrestricted, complete autopsy, preferably performed by pathologists with training and expertise in cardiovascular pathology. Consideration should be given to preservation and genetic analysis of tissue to determine the presence of a channelopathy. It is recommended that families of patients whose cause of death is not found on autopsy be referred to a healthcare provider or center with expertise in cardiac rhythm disturbances.

Knowledge Gaps
What is the population-based incidence of inherited arrhythmic deaths in patients with sudden, unexpected death and a negative autopsy? What are the most effective strategies (eg, for emergency medicine physician, primary care provider, coroner, or others) to identify families at risk?

Special Situations

New topics introduced in this document include resuscitation of infants and children with certain congenital cardiac abnormalities, namely single ventricle following stage I procedure and following the Fontan or bidirectional Glenn procedures (BDGs) as well as resuscitation of infants and children with cardiac arrest and pulmonary hypertension.

Life Support for Trauma[Peds-041A, Peds-041B]

Consensus on Science
Cardiac arrest (out-of-hospital and in-hospital) due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) in children has a very high mortality rate.[642–645] In 1 LOE 4[645] and 1 LOE 5[117] study there was no survival advantage to intubating child victims of traumatic cardiac arrest in the out-of-hospital setting. One LOE 2[646] and 4 LOE 4[647–650] studies suggested that there is minimal survival advantage associated with resuscitative thoracotomy with or without internal cardiac massage for blunt trauma–induced cardiac arrest in children. Two LOE 4 studies[648,649] suggested that survival in children with cardiac arrest from penetrating trauma is improved by thoracotomy if time from event to hospital is short and signs of life are restored in the field.

Treatment Recommendations
There is insufficient evidence to make a recommendation for modification of standard resuscitation for infants and children suffering cardiac arrest due to major trauma, although consideration should be given to selectively performing a resuscitative thoracotomy in children with penetrating injuries who arrive at the hospital with a perfusing rhythm.

Knowledge Gaps
What is the role of open-chest CPR for nontraumatic etiologies of pediatric cardiac arrest?

Single-Ventricle Post Stage I Repair[Peds-059]

Consensus on Science
In 1 LOE 4 case series[651] cardiac arrest occurred frequently (in 20% of 112 patients) in infants following stage I repair for single-ventricle anatomy. Two LOE 5 case series of mechanically ventilated, chemically paralyzed patients with a single ventricle in the preoperative period[652,653] showed that excessive pulmonary blood flow may be attenuated in the short term by increasing the inspired fraction of CO_2 to achieve a $Paco_2$ of 50 to 60 mm Hg. In the same population, decreasing the fraction of inspired oxygen below 0.21 did not appear to improve systemic oxygen delivery. Three LOE 4 studies[654–656] showed that clinical identification of the prearrest state in patients with a single ventricle is difficult and may be aided by monitoring systemic oxygen extraction using superior vena caval oxygen saturation or near infrared spectroscopy of cerebral and splanchnic circulations.

One LOE 3 prospective, crossover design study[657] of infants following stage I repair showed that inspired carbon dioxide increased systemic oxygen delivery. Evidence from 3 LOE 4 studies of infants following stage I repair[658–660] showed that reducing systemic vascular resistance with agents such as phenoxybenzamine improved systemic oxygen

delivery,[659] reduced the risk for cardiovascular collapse,[658] and improved survival.[660]

There is no evidence for or against any specific modification of standard resuscitation practice for cardiac arrest in infants with single-ventricle anatomy following stage I repair.

Five LOE 4 pediatric studies[555,558,578,661,662] showed that survival to hospital discharge for patients with single-ventricle anatomy following ECPR (see ECPR above) is comparable to that of other neonates undergoing cardiac surgery. In 1 LOE 4 study[578] survival following ECPR initiated as a consequence of systemic-to-pulmonary artery shunt block after stage I repair was consistently higher than for other etiologies of cardiac arrest.

Treatment Recommendations

Standard resuscitation (prearrest and arrest) procedures should be followed for infants and children with single-ventricle anatomy following stage I repair. Neonates with a single ventricle before stage I repair who demonstrate shock caused by elevated pulmonary to systemic flow ratio (Qp-to-Qs ratio) might benefit from inducing mild hypercarbia ($Paco_2$ to 50 to 60 mm Hg); this can be achieved during mechanical ventilation by reducing minute ventilation, adding CO_2 to inspired air, or administering opioids with or without chemical paralysis.

Neonates in a prearrest state following stage I repair may benefit from α-adrenergic antagonists to treat or ameliorate excessive systemic vasoconstriction in order to improve systemic blood flow and oxygen delivery and reduce the likelihood of cardiac arrest. Assessment of systemic oxygen extraction by monitoring $Svco_2$ or near infrared spectroscopy monitoring of cerebral and splanchnic circulation may help identify evolving hemodynamic changes in infants following stage I procedures; such hemodynamic changes may herald impending cardiac arrest.

Knowledge Gaps

Is there benefit in using heparin or thrombolytics during cardiac arrest to open a potentially occluded systemic-to-pulmonary artery (PA) or right ventricle to pulmonary artery (RV-PA) shunt following stage I repair? What is the role of monitoring near infrared spectroscopy/$Svco_2$ to guide resuscitation following stage I repair? Is there a potential benefit from the administration of milrinone during the prearrest state in infants with a single ventricle? Is it better to use a pure β-adrenergic agonist (isoproterenol) or an α- and β-agonist (epinephrine) to achieve ROSC after cardiac arrest following stage I repair? Does $Petco_2$ reflect pulmonary blood flow in single-ventricle physiology and can it be used to guide resuscitative procedures? Should the inspired oxygen concentration (100% versus room air) be different in infants with single-ventricle physiology during resuscitation from cardiac arrest? How does the Sano modification of Stage I repair (RV-PA conduit instead of a systemic-pulmonary artery shunt) affect response to therapies for cardiac arrest?

Single-Ventricle Post-Fontan and Bidirectional Glenn Procedures[Peds-055B]

Consensus on Science

In 1 LOE 4 case series[663] ECLS was useful in resuscitating patients with Fontan circulation but was not successful in hemi-Fontan/BDG patients. One LOE 4 case report[664] described manual external abdominal compressions with closed chest cardiac compressions as an alternative for standard CPR following a modified Fontan procedure.

Evidence from 4 LOE 5 studies[665–668] of patients with BDG circulation who were not in cardiac arrest or shock supports increasing CO_2 tension and hypoventilation to improve cerebral, superior vena caval, and pulmonary blood flow in order to increase systemic oxygen delivery. In 2 LOE 5 studies[669,670] of patients with BDG circulation who were not in cardiac arrest or a prearrest state, excessive ventilation reduced cerebral oxygenation. In 2 LOE 5 studies[671,672] of patients following a Fontan procedure who were not in cardiac arrest or a prearrest state, negative-pressure ventilation improved stroke volume and cardiac output compared with intermittent positive-pressure ventilation.

One LOE 5 case series[673] of patients following a Fontan procedure who were not in cardiac arrest or a prearrest state showed that high-frequency jet ventilation improved pulmonary vascular resistance and cardiac index. However, another LOE 5 case series[674] found that high-frequency oscillation ventilation did not increase cardiac index or decrease pulmonary vascular resistance.

Changes in pulmonary blood flow typically reflect changes in cardiac output, but in infants and children with right-to-left shunts, an increase in right-to-left shunting that bypasses the lungs, as occurs in some infants and children with congenital heart disease or pulmonary hypertension, decreases the proportion of blood flowing through the pulmonary circulation, and as a result, the $Petco_2$ falls.[675] Conversely, increasing pulmonary blood flow, as happens following shunt insertion in infants with cyanotic heart disease, increases the $Petco_2$ and reduces the difference between the $Paco_2$ and end-tidal CO_2.[84,85] Likewise, if there are intrapulmonary shunts that bypass the alveoli, there will be a greater difference between the $Paco_2$ and $Petco_2$.[83]

Treatment Recommendations

In patients with Fontan or hemi-Fontan/BDG physiology who are in a prearrest state, hypercarbia achieved by hypoventilation may be beneficial to increase oxygenation and cardiac output, while negative-pressure ventilation, if available, may be beneficial by increasing cardiac output. During cardiopulmonary arrest it is reasonable to consider ECPR for patients with Fontan physiology. There is insufficient evidence to support or refute the use of ECPR in patients with hemi-Fontan/BDG physiology.

Knowledge Gaps

What is the optimal method for cannulation for ECPR in patients with hemi-Fontan/BDG or Fontan physiology? What is the optimal CPR strategy (eg, with or without manual external abdominal compression; with or without active chest decompression; with or without an impedance threshold device) for patients with hemi-Fontan/BDG or Fontan physiology? Is there an ideal compression-ventilation ratio during CPR for infants following hemi-Fontan/BDG or Fontan procedures? Are compression "boots" or a MAST (military antishock trouser) suit beneficial for patients in prearrest

states or cardiac arrest following hemi-Fontan/BDG or Fontan procedures?

Pulmonary Hypertension[Peds-056A]

Consensus on Science

Two LOE 5 observational pediatric studies[676,677] showed that children with pulmonary hypertension are at increased risk for cardiac arrest. There are no studies that demonstrate the superiority of any specific therapy for resuscitation from cardiac arrest in infants and children with a pulmonary hypertensive crisis.

In 1 LOE 5 retrospective study in adults[678] standard CPR techniques were often unsuccessful in victims with pulmonary hypertension and cardiac arrest. Those who were successfully resuscitated had a reversible cause and received a bolus of IV iloprost or inhaled nitric oxide (NO) during the resuscitation.

One LOE 5 study of adults after cardiac transplant[679] and 2 LOE 5 studies in children with congenital heart disease[680,681] observed that inhaled NO and aerosolized prostacyclin or analogues appear to be equally effective in reducing pulmonary vascular resistance. In 1 LOE 5 study in children with pulmonary hypertension after cardiac surgery[682] inhaled NO and alkalosis appeared to be equally effective in reducing pulmonary vascular resistance. There is no evidence of benefit or harm of excessive ventilation for infants and children in cardiac arrest with pulmonary hypertension.

Four LOE 5 studies in pulmonary hypertensive adults and children with crises or cardiac arrest[683–686] showed that mechanical right ventricular support improved survival.

Treatment Recommendations

Rescuers should provide conventional pediatric advanced life support, including oxygenation and ventilation for cardiac arrest associated with pulmonary hypertension. It may be beneficial to attempt to correct hypercarbia. If the administration of medications (IV or inhaled) to decrease pulmonary artery pressure has been interrupted, it may be advisable to reinstitute it.

Inhaled NO or aerosolized prostacyclin or analogue to reduce pulmonary vascular resistance should be considered. If unavailable, an IV bolus of prostacyclin may be considered.

Knowledge Gaps

Is epinephrine harmful for resuscitation of pediatric patients with pulmonary hypertension who are in prearrest states or cardiac arrest? Is excessive ventilation of infants and children in prearrest states or cardiac arrest in the setting of pulmonary hypertension helpful or harmful? Does vasopressin improve outcome for cardiac arrest in the setting of pulmonary hypertensive crisis? Is a pure β-agonist, such as isoproterenol, effective or harmful during prearrest states or cardiac arrest associated with pulmonary hypertension? If used early in resuscitation, does the use of ECLS improve the outcome of the infant or child with pulmonary hypertension?

Prognosis and Decision to Terminate CPR[Peds-060]

Consensus on Science

In 1 LOE 3[687] and 1 LOE 4[688] study, survival from in-hospital pediatric cardiac arrest in the 1980s was approximately 9%. One LOE 1[538] and 1 LOE 3 pediatric study[689] showed that survival from in-hospital cardiac arrest in the early 2000s was 16% to 18%. Three prognostic LOE 1 prospective observational pediatric studies from 2006[537,690,691] reported that survival from in-hospital cardiac arrest in 2006 was 26% to 27%.

One LOE 1 prospective study[300] showed that survival from all pediatric out-of-hospital cardiac arrest was 6% compared with 5% for adults. Survival in infants was 3%, and in children and adolescents survival was 9%. This study demonstrated that earlier poor survival rates were heavily influenced by poor infant survival (many of whom probably had sudden infant death syndrome and had probably been dead for some time).

Thirteen (LOE 1[300,301,537,538,690,692,693]; LOE 3[577,687,694]; LOE 4[688,695,696]) studies showed an association between several factors and survival from cardiac arrest. These factors include duration of CPR, number of doses of epinephrine, age, witnessed versus unwitnessed cardiac arrest, obesity,[697] and the first and subsequent cardiac rhythm. Thirteen studies (LOE 1[300,645]; LOE 2[698]; LOE 3[643,650,694,699–703]; LOE 4[704,705]) showed an association between mortality and causes of arrest such as submersion and trauma for out-of-hospital cardiac arrest. None of the associations reported in these studies allow prediction of outcome.

Treatment Recommendations

There is insufficient evidence to allow a reliable prediction of success or failure to achieve ROSC or survival from cardiac arrest in infants and children.

Knowledge Gaps

Are there reliable prognostic factors to guide decision making to terminate CPR in infants and children? Are there reliable clinical factors to predict neurologic outcome following resuscitation from cardiac arrest in infants and children?

Acknowledgments

We thank the following individuals (the Pediatric Basic and Advanced Life Support Chapter Collaborators) for their collaborations on the worksheets contained in this section: Ian Adatia; Richard P. Aickin; John Berger; Jeffrey M. Berman; Desmond Bohn; Kate L. Brown; Mark G. Coulthard; Douglas S. Diekema; Aaron Donoghue; Jonathan Duff; Jonathan R. Egan; Christoph B. Eich; Diana G. Fendya; Ericka L. Fink; Loh Tsee Foong; Eugene B. Freid; Susan Fuchs; Anne-Marie Guerguerian; Bradford D. Harris; George M. Hoffman; James S. Hutchison; Sharon B. Kinney; Sasa Kurosawa; Jesús Lopez-Herce; Sharon E. Mace; Ian Maconochie; Duncan Macrae; Mioara D. Manole; Bradley S. Marino; Felipe Martinez; Reylon A. Meeks; Alfredo Misraji; Marilyn Morris; Akira Nishisaki; Masahiko Nitta; Gabrielle Nuthall; Sergio Pesutic Perez; Lester T. Proctor; Faiqa A. Qureshi; Sergio Rendich; Ricardo A. Samson; Kennith Sartorelli; Stephen M. Schexnayder; William Scott; Vijay Srinivasan; Robert M. Sutton; Mark Terry; Shane Tibby; Alexis Topjian; Elise W. van der Jagt; and David Wessel.

Disclosures

CoSTR Part 10: 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: Non-profit organization—Senior Associate in Critical Care Medicine	None	None	None	None	None	None
Allan R. de Caen	Self-employed, Clinical Associate Professor Pediatric Critical Care Medicine, Stollery Children's Hospital/University of Alberta	None	None	None	None	None	*Medical Expert for the Canadian Medical Protective Association
Leon Chameides	Emeritus Director, Pediatric Cardiology, Connecticut Children's Medical Center; Clinical Professor, University of Connecticut	None	None	None	None	None	None
Dianne L. Atkins	University of Iowa: Medical School—Professor †Serving as a Worksheet editor for 2010 Guidelines Process. Compensation is paid partially to my institution (66%) and partially to me (34%). The salary from my institution is not altered by this	None	None	None	None	None	*Serving as a defense expert witness in a case of ventricular fibrillation in a 2 year old child. Compensation is paid directly to me
Robert A. Berg	U of Pennsylvania—Professor	None	None	None	None	None	None
Marc D. Berg	University of Arizona—Assoc. Prof. Clinical Pediatrics; Attending Physician's Healthcare (UPH): Also serve on the UPH Board of Directors—Intensivist, Pediatric Critical Care Medicine	None	None	None	None	None	None
Farhan Bhanji	Montreal Children's Hospital, McGill University—Assistant Professor of Pediatrics	None	None	None	None	None	None
Dominique Biarent	Hôpital Universitaire des Enfants reine Fabiola: PICU Director	None	None	None	None	None	*Medical expert consultant in one trial for an insurance co. (Fortis) Medical expert for legal proceedings †grant of 25000 euro from ″Fondation Roi Baudoin″ to the non profit organization ″sauvez mon enfant″ for a psychological research in the PICU (I am administrator of the non profit organization and promotor of research). The grant is not given to me but to the NPO Grant of 67500 euro from the Belgian ″Loterie Nationale″ to build a teaching lab to teach paediatric resuscitation to health care provider Grant given to the non profit organization ″sauvez mon enfant″ for psychological research in the PICU (I am administrator of the non profit organization and promotor of the research) the grant is not given to me but to the NPO Grant from Baxter Foundation to pay a psychologist in my PICU (44.540 $) the grant is paid to the NPO
Robert Bingham	National Health Service of England—Consultant paediatric anaesthetist	None	None	None	None	None	*Occasional expert witness reports on pediatric resuscitation related topics

(*Continued*)

CoSTR Part 10: Writing Group Disclosures, *Continued*

Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Ashraf Coovadia	Rahima Moosa Mother and Child Hospital: Paediatric Consultant (Attending) in Department of Paediatrics and Child Health—Adjunct Professor in Paediatrics	None	None	None	None	None	None
Mary Fran Hazinski	Vanderbilt University School of Nursing—Prof; AHA ECC Product Dev.—Senior Science Editor. I receive significant compensation† from the AHA as consultant/ SSE to provide protected time to serve as the co-editor of the 2010 ILCOR CoSTR and the 2010 AHA Guidelines for CPR and ECC	None	None	None	None	None	None
Robert W. Hickey	University of Pittsburgh—Associate Professor	†Salary support from NIH for examining cyclopentenone prostaglandin effects in ischemic brain injury	None	None	None	None	*1–2 X/year medical malpractice expert
Vinay M. Nadkarni	University of Pennsylvania School of Medicine, Children's Hospital of Philadelphia: Non-profit, Academic, University Hospital—Attending Physician, Anesthesia, Critical Care and Pediatrics	†NIH RO1: Therapeutic Hypothermia after Cardiac Arrest, Co-Investigator. †Center of Excellence Grant, Laerdal Foundation for Acute Care Medicine, PI: Understanding the mechanics and quality of CPR *NHTSA: Mechanics of Chest Compression in children, Co-Investigator	None	None	None	None	None
Amelia Reis	Hospital das Clinico Universidade de Sao Paulo, Pediatric Emergency Physician	None	None	None	None	None	None
Antonio Rodriguez-Nunez	Hospital Clinico Universitario de Santiago de Compostela—Pediatric Emergency and Critical Care Division; University of Santiago de Compostela—Associate Professor of Pediatrics	None	None	None	None	None	None
James Tibballs	Royal Children's Hospital, Melbourne Healthcare, Physician ICU	None	None	None	None	None	None
Arno L. Zaritsky	Children's Hospital of The King's Daughters—Sr. VP for Clinical Services	None	None	None	None	*Data Safety Monitoring Board for NIH-sponsored trial of therapeutic hypothermia after pediatric cardiac arrest	None
David Zideman	Imperial College NHS Trust: United Kingdom Healthcare provider—Consultant Anaesthetist; London Organizing Committee of the Olympic Games—Lead Clinician for EMS	None	None	None	None	None	*Expert testimony on Cardiac Arrest under General Anaesthesis to Her Majesty's Coroner for Surrey (Expert witness fee for case review and court appearance) less than 1500 US dollars

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.

CoSTR Part 10: Worksheet Collaborator Disclosures

Worksheet Collaborator	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Ian Adatia	University of Alberta and Alberta Health Services; Professor Pediatrics, Director Pediatric Cardiac Critical Care Program and Pulmonary Hypertension Clinic	None	None	None	None	None	None
Richard P. Aickin	Auckland District Health Board Government Funded Healthcare Provider (primary care through to tertiary hospital services) for Auckland population and for national tertiary services. Director of Child Health	None	None	None	None	*New Zealand Health and Disability Commission: occasional expert reports provided with respect to alleged breaches of healthcare standards. 1–2 reports per year. Small personal payment received	*Expert Witness: Occasional expert testimony for Coroner's Court and criminal (Child protection) cases. Approx 1 × year. No personal payment—small payment to Auckland District Health Board for my time
John Berger	Children's National Medical Center Non-profit children's hospital Medical Director, Cardiac Intensive Care and Pulmonary Hypertension	†5 U 10 HD 049981—DL Wessel (PI) 12/1/09/–11/30/14 Sponsor: NIH/NICHD/NCMRR Pediatric Critical Care Research Network The major aims of the network are to reduce morbidity and mortality in pediatric critical illness and injury, and to provide a framework for the development of the scientific basis of pediatric critical care practice. I am responsible for conduct of network approved studies at CNMC. As a member of the network steering committee, I am responsible for contributing to design of studies, analyzing results and disseminating research findings. Grant money comes to institution. Role: Co-Investigator *Therapeutic Hypothermia after Pediatric Cardiac Arrest (THAPCA) Trials. PI:JT Berger 2009 Sponsor: of Michigan I am the site PI for the conduct of a randomized trial of therapeutic hypothermia in children who have had a cardiac arrest funded by NHLBI. Money comes to the institution. Role: Consortium Site PI *Tracking Outcomes and Practice in Pediatric Pulmonary Hypertension. PI: JT Berger. 2008 Sponsor: Association in Pediatric Pulmonary Hypertension I am the site PI responsible for contributing subject data to a registry of pediatric pulmonary hypertension patients and their therapy Role: Site PI	None	None	None	None	None
Jeffrey M. Berman	University of North Carolina:Faculty member UNC School of Medicine—Professor of Anesthesiology	None	None	None	None	None	None
Desmond Bohn	The Hospital for Sick Children, Toronto—Chief, Department of Critical Care Medicine	None	None	None	None	None	None
Kate L. Brown	Great Ormond Street Hospital for Children NHS Trust Hospital consultant in paediatric intensive care Consultant paediatric cardiac intensive care	None	None	None	None	None	None
Mark G. Coulthard	Queensland Health: State Health Employer Organisation—Paediatric Intensive Care Specialist	None	None	None	None	None	None
Douglas S. Diekema	Children's University Medical Group: Delivery of medical care in Children's Hospital of Seattle and the University of Washington—Professor of Pediatrics, Attending Physician, Emergency Department	None	None	None	None	None	None

(Continued)

CoSTR Part 10: Worksheet Collaborator Disclosures, *Continued*

Worksheet Collaborator	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Aaron Donoghue	University of Pennsylvania—Assistant Professor	None	None	None	None	None	None
Jonathan Duff	Alberta Health Services: Pediatric Intensivist	None	None	None	None	None	None
Jonathan R. Egan	The Children's hospital at Westmead, Sydney—Pediatric Intensivist	None	None	None	None	None	None
Christoph B. Eich	University Medical Centre of Göttingen, Germany: Attending Anesthesiologist, Intensivist and Emergency Physician	None	None	None	None	None	None
Diana G. Fendya	Children's National Medical Center, EMSC National Resource Center; Trauma/Acute Care Nursing Specialist	None	None	None	None	None	None
Ericka L. Fink	Children's Hospital of Pittsburgh of UPMC—Assistant Professor	†P.I., K23 from NINDS Duration of Hypothermia for Neuroprotection after Pediatric Cardiac Arrest Institution P.I., Laerdal Foundation grant $21,365 Same topic Institution *Children's Hospital of Pittsburgh of UPMC Clinical and Translational Science Institute $6500 Same topic Institution	None	None	None	None	None
Loh Tsee Foong	KK Women's and Children's Hospital	None	None	None	None	None	None
Eugene B. Freid	Nemours Children's Clinics Health Care Organization Staff Anesthesiologist and Intensivist University of Florida Jacksonville Health Care Organization Pediatric Intensivist	None	None	*University of North Carolina—Speaker at Anesthesiology Refresher Course. 1000–1500/year honorarium sent to institution	None	None	None
Susan Fuchs	Children's Memorial Hospital-Assoc Director, Div Pediatric Emergency Medicine	None	None	None	None	None	*Currently on the American Academy of Pediatrics Advanced Pediatric Life Support Steering Committee and Currently Co-chairperson of the AAP Pediatric Education for Prehospital Professional (PEPP) steering committee
Anne-Marie Guerguerian	The Hospital for Sick Children; Staff Physician	None	None	None	None	None	None
Bradford D. Harris	UNC at Chapel Hill; Assist Prof	†5 P01 AT002620–02 (Peden) 09/30/04–06/30/09 5% NIH/NCCAM $1,660,813 Annual Direct Translational Research Center for CAM Therapy of Asthma The objective of this research is to identify antioxidant complementary and alternative medicine therapies for application in asthma. 5 RO1 ES012706–02 (Peden) 09/01/04–07/31/09 5% NIH/NIEHS $ 209,314 03 and LPS-Induced Airway Inflammation in Humans in vivo The objective is to test three hypotheses to define the ways that O3 and LPS interact to exacerbate airway disease. 5 R01 HL080337–02 (Peden) 05/06/05–04/30/09 5% NIH NHLBI/NIAID $350,000 Airway Biology of Acute Asthma: Translational Studies The major goal is to determine if asthma exacerbation and allergen challenge models allow for examination of innate/acquired immune interactions. R82952201 Cooperative Agreement (Bromberg) 11/01/01–10/31/06 5% U.S. Environmental Protection Agency $1,583,867 Health Effects of Exposure to Air Pollutants in Humans The major goal of this cooperative agreement is to examine the health effects of inhalation of environmental ambient air pollutants on human subjects	None	*Assoc Clinical Research Professor on peds pharm	None	None	None

(*Continued*)

CoSTR Part 10: Worksheet Collaborator Disclosures, *Continued*

Worksheet Collaborator	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
George M. Hoffman	Medical College of Wisconsin medical school Professor, Anesthesiology and Pediatrics [View] Children's Hospital of Wisconsin hospital Medical Director, Pediatric Anesthesiology	None	None	*Somanetics, Inc biomedical device manufacturer 1653 East Maple Road Troy, MI 48083-4208 i have informally served in consultant/advisory capacity and have received honoraria for speaking	None	*Edwards Life Sciences, Inc biomedical device manufacturer One Edwards Way Irvine, CA 92614 i have served informally in consultant/advisory capacity *Masimo, Inc biomedical device manufacturer 2852 Kelvin Ave. Irvine, CA, 92614 i have served informally in consultant/advisory capacity	None
James S. Hutchison	SickKlds Hospital Director Neurocritical Care	None	None	None	None	None	None
Sharon B. Kinney	University of Melbourne and Royal Children's Hospital Melbourne—Lecturer and MET Coordinator	None	None	None	None	None	None
Sasa Kurosawa	Shizuoka Children's Hospital Depertoment of Pediatric Emergency & General Pediatrics Doctor National Center for Child Health & Development Department of Health Policy, Research Institution researcher	None	None	None	None	None	None
Jesús López-Herce	Hospital General Universitario Gregorio Marañón—Pediatric Assistant	None	None	None	None	None	None
Sharon E. Mace	Clerveland Clinic—Physician employed fulltime by the hospital; Attending staff physician	None	None	*Baxter Healthcare Pharmaceutical Speaker Bureau	None	*Baxter Healthcare Pharmaceutical Consultant, Advisory Board	None
Ian Maconochie	Imperial Academic Health Sciences Centre, London: I run the pediatric emergency medicine department at St Mary's Hospital in Paddington, London—Lead Consultant in Pediatric Emergency medicine	None	*Postal for survey of UK departments about use of pain relief from Therakind, a company looking to obtain license for use of commonly used drugs from the medical regulatory authority in UK. Estimated payment was about 150 pounds sterling	None	None	*I am a deputy editor for *The Emergency Medicine Journal*, a commissioning editor for the *Archives of Diseases of Childhood* and sit on the editorial advisory panel for the *British Medical Journal*. I am editorial board member for *Current Pediatric Reviews* and *Pediatric Emergency Medical Journal*. The latter 2 I do not receive payment. I act as a consultant advisory to TSG associates in relation to major disaster management systems. I have advised Therakind on the licensing of drugs in the pediatric population., ie approaching the medical regulatory authority to obtain a license on the use of a commonly used drug in the treatment of fitting children in UK	*I have acted as an expert witness in cases relating to the management of children who may have had non accidental injury.
Duncan Macrae	The Royal Brompton and Harefield NHS Foundation Trust—Director of Children's Services	None	None	None	None	None	None
Mioara D. Manole	Univ of Pittsburgh: Ped Emerg. Medicine attending physician; assist Prof Ped	NIH K08HD58798-funds go to Univ Children's Hosp of Pitts RAC grant-funds to Univer.	None	None	None	None	None
Bradley S. Marino	Cincinnati Children's Hospital Medical Center Associate Professor of Pediatrics	None	None	None	None	None	None

(*Continued*)

CoSTR Part 10: Worksheet Collaborator Disclosures, *Continued*

Worksheet Collaborator	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Felipe Martinez	Universidad de Valparaíso—Professor	None	None	None	None	None	None
Reylon A. Meeks	Blank Children's Hospital, Pleasant Hill Fire Dept., N Clinical Specialist	None	None	None	None	None	None
Alfredo Misraji	Unidad Coronaria Movil	None	None	None	None	None	None
Marilyn Morris	Columbia Univ; assist Prof Ped	None	None	None	None	None	*Expert witness $900 for 3 hour case for defense of child that received ECPR
Akira Nishisaki	Children's Hosp of Philadelphia, non profit tertiary children's hospital; attending MD CCMedicine	None	None	None	None	None	None
Masahiko Nitta	Osaka Medical College—Assistant Professor	None	None	None	None	None	None
Gabrielle Nuthall	Auckland District Health Board: Pediatric Intensive Care Specialist	None	None	None	None	None	None
Sergio Pesutic Pérez	Centro de Formación en Apoyo Vital Director	None	None	None	None	None	None
Lester T. Proctor	University of Wisconsin-Madison College of Medicine and Public Health—Professor	None	None	None	None	None	None
Faiqa A. Qureshi	Children's Specialty Group—Physician	None	None	None	None	None	None
Sergio Rendich	Hospital Naval Almirante Nef—Pediatrician; Hospital Gustavo Fricke; Pediatrician—Intensive Care Unit; Universidad de Valparaíso Professor, Pediatrics Clínica Las Condes Critical Patient Unit Centro de Formación en Apoyo Vital; Instructor, NRP	None	None	None	None	None	None
Ricardo A. Samson	The University of Arizona: Faculty member within the Department of Pediatrics Chief of the Cardiology Section Provide clinical care, teaching and research related to the field of Pediatric Cardiology—Professor of Pediatrics	None	None	None	None	None	None
Kennith Sartorelli	University of Vermont Associate Professor of Surgery	None	None	None	None	None	None
Stephen M. Schexnayder	University of Arkansas for Medical Sciences–College of Medicine: Physician - Clinician Educator–Professor and Division Chief: AHA Consultant	*Pharmacokinetics of pantoperazole in pediatrics patients (Pediatric Pharmacology Research Unit) Pharmacokinetics of esomeprazole in pediatric patients (Astra Zeneca)	None	*Contemporary Forums (Nursing conference) Pediatric Clinics of North America (guest editor)	None	None	*Expert witness in various medicolegal cases involving pediatric critical care and emergency medicine
William Scott	UT Southwestern Medical Center—Professor	None	None	None	None	None	None
Vijay Srinivasan	Children's Hospital of Philadelphia—Attending Physician, Pediatric Intensive Care Unit	*A Reproducible Method for Blood Glucose Control in Critically Ill Children (RC1 sub contract with Inter Mountain Medical Center, PI: Alan Morris), site PI: Vijay Srinivasan—submitted for NIH Challenge Grants July 2009, approval pending	*PI: A Novel Application of Impedance Threshold Device technologies to optimize Fluid Removal during Hemodialysis in Children (unfunded research at CHOP—IRB Research Protocol No: 2007–12-5712)—have received impedance threshold devices for this study from Advanced Circulatory Systems, Inc, EdenPrairieMN	None	None	None	None
Robert M. Sutton	The Children's Hospital of Philadelphia Critical Care Attending	*Unrestricted research grant support from the Laerdal Foundation for Acute Care Medicine	None	None	None	None	None
Mark Terry	Johnson County Med-Act: County government ambulance service—Deputy Chief Operations	None	None	None	None	None	None

(*Continued*)

CoSTR Part 10: Worksheet Collaborator Disclosures, *Continued*

Worksheet Collaborator	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Ownership Interest	Consultant/Advisory Board	Other
Shane Tibby	Guy's and St Thomas' NHS Foundation Trust, London National Health Service Hospital trust in United Kingdom Consultant in Pediatric Intensive Care	None	None	None	None	None	None
Alexis Topjian	The Children's Hospital of Philadelphia—attending physician	*site PI for the Therapeutic hypothermia after cardiac arrest study. NIH funded study. Money goes to the institution	None	None	None	None	None
Elise W. van der Jagt	University of Rochester: Academic Institution including Medical School/Center—Professor of Pediatrics and Critical Care	†Project Title: Therapeutic Hypothermia After Pediatric Cardiac Arrest (THAPCA) Trials PI: Frank W. Moler, M.D. (University of Michigan) Proposed project period: 7/1/2009–6/30/2014 We are part of this multi-institutional grant but after the grant was funded, the initial institutions that would be involved were the higher volume/larger children's hospitals. At this time we are not receiving any funding from this grant.*PI andCo-Investigator/Site	None	None	None	None	None
David Wessel	Children's National Medical Center Senior Vice President	None	None	None	None	†IKARIA Holdings Inc. Pharmaceutical Consultant	None

This table represents the relationships of worksheet collaborators that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all worksheet collaborators 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.

Appendix

CoSTR Part 10: Worksheet Appendix

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-001A	In infants (<1 year, not including newly born) in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of AEDs (I) compared with standard management (which does not include use of AEDs) (C), improve outcomes (eg. termination of rhythm, ROSC, survival) (O)?	AEDs in children less than 1 yr	Reylon A. Meeks	http://circ.ahajournals.org/site/C2010/Peds-001A.pdf
Peds	Peds-001B	In infants (<1 year, not including newly born) in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of AEDs (I) compared with standard management (which does not include use of AEDs) (C), improve outcomes (eg. termination of rhythm, ROSC, survival) (O)?	AEDs in children less than 1 yr	Antonio Rodriguez-Nunez	http://circ.ahajournals.org/site/C2010/Peds-001B.pdf
Peds	Peds-002A	For infants and children in cardiac arrest, does the use of a pulse check (I) vs. assessment for signs of life (C) improve the accuracy of diagnosis of pediatric CPA (O)?	Pulse check accuracy	Aaron Donoghue, James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-002A.pdf
Peds	Peds-003	During cardiac arrest in infants or children (P), does the presence of family members during the resuscitation (I) compared to their absence (C) improve patient or family outcome measures (O)?	Family presence	Douglas S. Diekema	http://circ.ahajournals.org/site/C2010/Peds-003.pdf
Peds	Peds-004	In infants and children with respiratory failure who undergo endotracheal intubation (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of devices (eg. CO_2 detection device, CO_2 analyzer or esophageal detector device) (I) compared with usual management (C), improve the accuracy of diagnosis of airway placement (O)?	Verification of airway placement	Diana G. Fendya, Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-004.pdf
Peds	Peds-005A	In pediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of end-tidal CO_2 (I), compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?	End-tidal CO_2 to diagnose perfusing rhythm	Arno Zaritsky	http://circ.ahajournals.org/site/C2010/Peds-005A.pdf
Peds	Peds-005B	In pediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of end-tidal CO_2 (I), compared with clinical assessment (C), improve accuracy of diagnosis of a perfusing rhythm (O)?	End-tidal CO_2 to diagnose perfusing rhythm	Anne-Marie Guerguerian	http://circ.ahajournals.org/site/C2010/Peds-005B.pdf
Peds	Peds-006B	In pediatric patients in clinical cardiac arrest (prehospital [OHCA] or in hospital [IHCA]) (P), does the use of a focused echocardiogram (I) compared with standard assessment, assist in the diagnosis of reversible causes of cardiac arrest?	Methods to diagnose perfusing rhythm	Christoph B. Eich, Faiqa A. Qureshi	http://circ.ahajournals.org/site/C2010/Peds-006B.pdf
Peds	Peds-007	In children requiring emergent intubation (prehospital, in-hospital) (P), does the use of cuffed ETTs (I) compared with uncuffed ETTs (C) improve therapeutic endpoints (eg, oxygenation and ventilation) or reduce morbidity or risk of complications (eg, need for tube change, airway injury, aspiration) (O)?	Cuffed vs uncuffed ETTs	Ashraf Coovadia	http://circ.ahajournals.org/site/C2010/Peds-007.pdf

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CoSTR Part 10: Worksheet Appendix, *Continued*

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-008	In children requiring assisted ventilation (prehospital, in-hospital) (P), does the use of bag-valve-mask (I) compared with endotracheal intubation (C) improve therapeutic endpoints (oxygenation and ventilation), reduce morbidity or risk of complications (eg, aspiration), or improve survival (O)?	BVM vs intubation	Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-008.pdf
Peds	Peds-009	In pediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of supraglottic airway devices (I) compared with bag-valve-mask alone (C), improve therapeutic endpoints (eg, ventilation and oxygenation), improve quality of resuscitation (eg, reduce hands-off time, allow for continuous compressions), reduce morbidity or risk of complications (eg, aspiration) or improve survival (O)?	Supraglottic airway devices	Robert Bingham	http://circ.ahajournals.org/site/C2010/Peds-009.pdf
Peds	Peds-010A	For infants and children who have ROSC after cardiac arrest (P), does the use of induced hypothermia (I) compared with normothermia (C) improve outcome (survival to discharge, survival with good neurologic outcome) (O)?	Induced hypothermia after ROSC	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-010A.pdf
Peds	Peds-010B	For infants and children who have ROSC after cardiac arrest (P), does the use of induced hypothermia (I) compared with normothermia (C) improve outcome (survival to discharge, survival with good neurologic outcome) (O)?	Induced hypothermia after ROSC	James S. Hutchison	http://circ.ahajournals.org/site/C2010/Peds-010B.pdf
Peds	Peds-011B	In infants and children with cardiac arrest from a non-asphyxial or asphyxial cause (excluding newborns) (prehospital [OHCA] or in-hospital [IHCA]) (P), does the use of another specific C:V ratio by laypersons and HCPs (I) compared with standard care (15:2) (C), improve outcome (eg, ROSC, survival) (O)?	Compression ventilation ratio	Robert Bingham, Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-011B.pdf
Peds	Peds-012A	In infants and children (not including newborns) with cardiac arrest (out-of-hospital and in-hospital) (P), does the use of compression-only CPR (I) as opposed to standard CPR (ventilations and compressions) (C), improve outcome (O) (eg, ROSC, survival)?	Compression only CPR	Robert A. Berg, Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-012A.pdf
Peds	Peds-013A	In pediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) and a secure airway (P), does the use of a specific minute ventilation (combination of respiratory rate and tidal volume) depending on the etiology of the arrest (I) as opposed to standard care (8–10 asynchronous breaths per minute) (C), improve outcome (O) (eg. ROSC, survival)?	Etiology specific minute ventilation	Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-013A.pdf
Peds	Peds-013B	In pediatric patients with cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) and a secure airway (P), does the use of a specific minute ventilation (combination of respiratory rate and tidal volume) depending on the etiology of the arrest (I) as opposed to standard care (8–10 asynchronous breaths per minute) (C), improve outcome (O) (eg. ROSC, survival)?	Etiology specific minute ventilation	Naoki Shimizu	http://circ.ahajournals.org/site/C2010/Peds-013A.pdf
Peds	Peds-014	In pediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P) does the use of rapid deployment ECMO or emergency cardiopulmonary bypass (I), compared with standard treatment (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurologic outcomes) (O)?"	ECMO	Marilyn Morris	http://circ.ahajournals.org/site/C2010/Peds-014.pdf
Peds	Peds-014B	In pediatric patients in cardiac arrest (prehospital [OHCA] or in-hospital [IHCA]) (P) does the use of rapid deployment ECMO or emergency cardiopulmonary bypass (I), compared with standard treatment (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurologic outcomes) (O)?	ECMO	Kate L. Brown	http://circ.ahajournals.org/site/C2010/Peds-014B.pdf
Peds	Peds-015	In pediatric patients in cardiac arrest, associated with or without asphyxia (prehospital [OHCA] or in-hospital [IHCA]) (P) does ventilation with a specific oxygen concentration (room air or a titrated concentration between 0.21 and 1.0) (I), compared with standard treatment (100% oxygen) (C), improve outcome (ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Titrated oxygen vs 100% oxygen	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-015.pdf
Peds	Peds-016	In infants and children with ROSC after cardiac arrest (prehospital or in-hospital) (P), does the use of a specific strategy to manage blood glucose (eg. target range) (I) as opposed to standard care (C), improve outcome (O) (eg. survival)?	Glucose control following resuscitation	Duncan Macrae, Vijay Srinivasan	http://circ.ahajournals.org/site/C2010/Peds-016.pdf
Peds	Peds-017B	In pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of any specific alternative method for calculating drug dosages (I) compared with standard weight-based dosing (C), improve outcome (eg, achieving expected drug effect, ROSC, survival, avoidance of toxicity) (O)?	Methods for calculating drug dosages	Ian Maconochie, Vijay Srinivasan	http://circ.ahajournals.org/site/C2010/Peds-017B.pdf
Peds	Peds-018	In adult and pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA) (P), does the use of any specific alternative dosing regimen for epinephrine (I) compared with standard recommendations (C), improve outcome (eg. ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Epinephrine dose	Amelia Reis	http://circ.ahajournals.org/site/C2010/Peds-018.pdf
Peds	Peds-019	In pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) due to VF/pulseless VT (P), does the use of amiodarone (I) compared with lidocaine (C), improve outcome (eg, ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Amiodarone vs lidocaine for VF/VT	Dianne L. Atkins	http://circ.ahajournals.org/site/C2010/Peds-019.pdf
Peds	Peds-020A	In adult and pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of vasopressin or vasopressin + epinephrine (I) compared with standard treatment recommendations (C), improve outcome (eg, ROSC, survival to hospital discharge, or survival with favorable neurologic outcome) (O)?	Vasopressin	Elise W. van der Jagt	http://circ.ahajournals.org/site/C2010/Peds-020A.pdf

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CoSTR Part 10: Worksheet Appendix, *Continued*

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-020B	In adult and pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of vasopressin or vasopressin + epinephrine (I) compared with standard treatment recommendations (C), improve outcome (eg, ROSC, survival to hospital discharge, or survival with favorable neurologic outcome) (O)?	Vasopressin	Dominique Biarent	http://circ.ahajournals.org/site/C2010/Peds-020B.pdf
Peds	Peds-021A	In pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of calcium (I) compared with no calcium (C), improve outcome (O) (eg. ROSC, survival to hospital discharge, survival with favorable neurologic outcome)?	Calcium	Allan de Caen	http://circ.ahajournals.org/site/C2010/Peds-021A.pdf
Peds	Peds-021B	In pediatric patients with cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of calcium (I) compared with no calcium (C), improve outcome (O) (eg. ROSC, survival to hospital discharge, survival with favorable neurologic outcome)?	Calcium	Felipe Martinez, Sergio Pesutic, Sergio Rendich	http://circ.ahajournals.org/site/C2010/Peds-021B.pdf
Peds	Peds-022A	In pediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of more than one shock for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (eg. termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Single or stacked shocks	Marc Berg	http://circ.ahajournals.org/site/C2010/Peds-022A.pdf
Peds	Peds-023A	In pediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of a specific energy dose or regimen of energy doses for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (eg. termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Energy doses	Jonathan R. Egan	http://circ.ahajournals.org/site/C2010/Peds-023A.pdf
Peds	Peds-023B	In pediatric patients with cardiac arrest due to primary or secondary VF or pulseless VT (pre-hospital [OHCA] or in-hospital [IHCA]) (P), does the use of a specific energy dose or regimen of energy doses for the initial or subsequent defibrillation attempt(s) (I), compared with standard management (C), improve outcome (eg. termination of rhythm, ROSC, survival to hospital discharge, survival with favorable neurologic outcome) (O)?	Energy doses	Dianne L. Atkins	http://circ.ahajournals.org/site/C2010/Peds-023B.pdf
Peds	Peds-024A	In pediatric patients with ROSC after cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) who have signs of cardiovascular dysfunction (P), does the use of any specific cardioactive drugs (I) as opposed to standard care (or different cardioactive drugs) (C), improve physiologic endpoints (oxygen delivery, hemodynamics) or patient outcome (eg, survival to discharge or survival with favorable neurologic outcome) (O)?	Cardioactive drugs post resuscitation	Allan de Caen	http://circ.ahajournals.org/site/C2010/Peds-024A.pdf
Peds	Peds-024B	In pediatric patients with ROSC after cardiac arrest (pre-hospital [OHCA] or in-hospital [IHCA]) who have signs of cardiovascular dysfunction (P), does the use of any specific cardioactive drugs (I) as opposed to standard care (or different cardioactive drugs) (C), improve physiologic endpoints (oxygen delivery, hemodynamics) or patient outcome (eg, survival to discharge or survival with favorable neurologic outcome) (O)?	Cardioactive drugs post resuscitation	Mark G. Coulthard	http://circ.ahajournals.org/site/C2010/Peds-024B.pdf
Peds	Peds-025A	In pediatric patients with in-hospital cardiac or respiratory arrest (P), does use of EWSS/response teams/MET systems (I) compared with no such responses (C), improve outcome (eg, reduce rate of cardiac and respiratory arrests and in-hospital mortality) (O)?	METs	Elise W. van der Jagt	http://circ.ahajournals.org/site/C2010/Peds-025A.pdf
Peds	Peds-025B	In pediatric patients with in-hospital cardiac or respiratory arrest (P), does use of EWSS/response teams/MET systems (I) compared with no such responses (C), improve outcome (eg, reduce rate of cardiac and respiratory arrests and in-hospital mortality) (O)?	METs	James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-025B.pdf
Peds	Peds-026A	For intubated newborns within the first month of life (beyond the delivery room) who are receiving chest compressions (P), does the use of continuous chest compressions (without pause for ventilation) (I) vs. chest compressions with interruptions for ventilation (C) improve outcome (time to sustained heart rate >100, survival to ICU admission, survival to discharge, survival with favorable neurologic status) (O)?	Continuous chest compressions for intubated newborns outside of DR	Monica Kleinman	http://circ.ahajournals.org/site/C2010/Peds-026A.pdf
Peds	Peds-027A	For newborns within the first month of life (beyond the delivery room) who are not intubated and who are receiving CPR (P), does the use of a 3:1 compression to ventilation ratio (I), compared with a 15:2 compression to ventilation ratio (C) improve outcome (time to sustained heart rate >100, survival to ICU admission, survival to discharge, discharge with favorable neurologic status) (O)?	3:1 vs 15:2 ratio for neonates outside of DR	Leon Chameides	http://circ.ahajournals.org/site/C2010/Peds-027A.pdf
Peds	Peds-028	In pediatric patients with cardiac arrest (out-of-hospital and in-hospital) (including prolonged arrest states) (P), does the use of $NaHCO_3$ (I) compared with no $NaHCO_3$ (C), improve outcome (O) (eg. ROSC, survival)?	Sodium bicarbonate	Stephen M. Schexnayder	http://circ.ahajournals.org/site/C2010/Peds-028.pdf
Peds	Peds-029	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of any specific paddle/pad size/orientation and position (I) compared with standard resuscitation or other specific paddle/pad size/orientation and position) (C), improve outcomes (eg. successful defibrillation, ROSC, survival) (O)?	Paddle size and placement for defibrillation	Dianne L. Atkins	http://circ.ahajournals.org/site/C2010/Peds-029.pdf

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CoSTR Part 10: Worksheet Appendix, *Continued*

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-030	In infants and children with unstable ventricular tachycardia (pre-hospital and in-hospital) (P), does the use of any drug/ combination of drugs/ intervention (eg. cardioversion) (I) compared with no drugs/intervention (C) improve outcome (eg, termination of rhythm, survival) (O)?	Unstable VT	Jeffrey M. Berman, Bradford D. Harris	http://circ.ahajournals.org/site/C2010/Peds-030.pdf
Peds	Peds-031	In infants and children with supraventricular tachycardia with a pulse (P), does the use of any drug or combination of drugs (I), compared with adenosine (C), result in improved outcomes (termination of rhythm, survival)?	Drugs for SVT	Ricardo A. Samson	http://circ.ahajournals.org/site/C2010/Peds-031.pdf
Peds	Peds-032	In infants and children with hemorrhagic shock following trauma (P), does the use of graded volume resuscitation (I) as opposed to standard care (C), improve outcome (hemodynamics, survival) (O)?	Graded volume resuscitation for traumatic shock	Jesús Lopez-Herce	http://circ.ahajournals.org/site/C2010/Peds-032.pdf
Peds	Peds-033	In pediatric patients in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of one hand chest compressions (I) compared with two hand chest compressions (C) improve outcomes (eg. ROSC, rescuer performance) (O)?	One hand vs two hand compressions	Sharon B. Kinney	http://circ.ahajournals.org/site/C2010/Peds-033.pdf
Peds	Peds-034	In infants with cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of two-thumb chest compression without circumferential squeeze (I) compared to two-thumb chest compression with circumferential squeeze (C) improve outcome (eg. ROSC, rescuer performance (O)?	Circumferential squeeze for infant CPR	James Tibballs	http://circ.ahajournals.org/site/C2010/Peds-034.pdf
Peds	Peds-035	In infants and children with cardiac arrest (P), does establishing intraosseous access (I) compared to establishing conventional (non-intraosseous) venous access (C) improve patient outcome (eg. ROSC, survival to hospital discharge (O)?	IO vs IV	Jonathan Duff	http://circ.ahajournals.org/site/C2010/Peds-035.pdf
Peds	Peds-036	In infants and children with cardiac arrest (P), does the use of tracheal drug delivery (I) compared to intravenous drug delivery (C) worsen patient outcome (eg. ROSC, survival to hospital discharge (O)?	ET vs IV drugs	Mioara D. Manole	http://circ.ahajournals.org/site/C2010/Peds-036.pdf
Peds	Peds-038B	In infants and children in shock, does early intubation and assisted ventilation compared to the use of these interventions only for associated respiratory failure lead to improved patient outcome (hemodynamics, survival?)	Intubation for shock (timing)	Amelia Reis	http://circ.ahajournals.org/site/C2010/Peds-038B.pdf
Peds	Peds-039A	In infants and children with respiratory failure who require emergent endotracheal intubation (P), does the use of cricoid pressure or laryngeal manipulation (I), when compared with standard practice (C), improve or worsen outcome (eg. success of intubation, aspiration risk, side effects, etc) (O)?	Cricoid pressure and laryngeal manipulation	Lester T. Proctor	http://circ.ahajournals.org/site/C2010/Peds-039A.pdf
Peds	Peds-039B	In infants and children with respiratory failure who require emergent endotracheal intubation (P), does the use of cricoid pressure or laryngeal manipulation (I), when compared with standard practice (C), improve or worsen outcome (eg. success of intubation, aspiration risk, side effects, etc) (O)?	Cricoid pressure and laryngeal manipulation	Ian Maconochie	http://circ.ahajournals.org/site/C2010/Peds-039B.pdf
Peds	Peds-040A	In infants and children in cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (ie. depth specified in treatment algorithm) (C), improve outcome (O) (eg. Blood pressure, ROSC, survival)?	Compression depth	Robert M. Sutton	http://circ.ahajournals.org/site/C2010/Peds-040A.pdf
Peds	Peds-040B	In infants and children in cardiac arrest (out-of-hospital and in-hospital) (P), does any specific compression depth (I) as opposed to standard care (ie. depth specified in treatment algorithm) (C), improve outcome (O) (eg. Blood pressure, ROSC, survival)?	Compression depth	David Zideman	http://circ.ahajournals.org/site/C2010/Peds-040B.pdf
Peds	Peds-041A	In children and infants with cardiac arrest due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) (P), does the use of any specific modifications to standard resuscitation (I) compared with standard resuscitation (C), improve outcome (O) (eg. ROSC, survival)? eg. open vs closed chest CPR, other examples.	Traumatic arrest	Kennith Sartorelli	http://circ.ahajournals.org/site/C2010/Peds-041A.pdf
Peds	Peds-041B	In children and infants with cardiac arrest due to major (blunt or penetrating) injury (out-of-hospital and in-hospital) (P), does the use of any specific modifications to standard resuscitation (I) compared with standard resuscitation (C), improve outcome (O) (eg. ROSC, survival)? eg. open vs closed chest CPR, other examples.	Traumatic arrest	Jesús Lopez-Herce	http://circ.ahajournals.org/site/C2010/Peds-041B.pdf
Peds	Peds-043A	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of self-adhesive defibrillation pads (I) compared with paddles (C), improve outcomes (eg. successful defibrillation, ROSC, survival) (O)?	Hands off defibrillation vs paddles	Mark Terry	http://circ.ahajournals.org/site/C2010/Peds-043A.pdf
Peds	Peds-043B	In infants and children in cardiac arrest (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of self-adhesive defibrillation pads (I) compared with paddles (C), improve outcomes (eg. successful defibrillation, ROSC, survival) (O)?	Hands off defibrillation vs paddles	Farhan Bhanji	http://circ.ahajournals.org/site/C2010/Peds-043B.pdf
Peds	Peds-044A	In infants and children with any type of shock (P), does the use of any specific resuscitation fluid or combination of fluids [eg: isotonic crystalloid, colloid, hypertonic saline, blood products] (I) when compared with standard care (C) improve patient outcome (hemodynamics, survival) (O)?	Resuscitation fluids	Sharon E. Mace	http://circ.ahajournals.org/site/C2010/Peds-044A.pdf
Peds	Peds-044B	In infants and children with any type of shock (P), does the use of any specific resuscitation fluid or combination of fluids [eg: isotonic crystalloid, colloid, hypertonic saline, blood products] (I) when compared with standard care (C) improve patient outcome (hemodynamics, survival) (O)?	Resuscitation fluids	Richard P. Aickin	http://circ.ahajournals.org/site/C2010/Peds-044B.pdf

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CoSTR Part 10: Worksheet Appendix, *Continued*

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-045A	In infants and children with distributive shock with and without myocardial dysfunction (P), does the use of any specific inotropic agent (I) when compared to standard care (C), improve patient outcome (hemodynamics, survival) (O)?	Distributive shock and inotropes	Ericka L. Fink, Alfredo Misraji	http://circ.ahajournals.org/site/C2010/Peds-045A.pdf
Peds	Peds-045B	In infants and children with distributive shock with and without myocardial dysfunction (P), does the use of any specific inotropic agent (I) when compared to standard care (C), improve patient outcome (hemodynamics, survival) (O)?	Distributive shock and inotropes	Loh Tsee Foong	http://circ.ahajournals.org/site/C2010/Peds-045B.pdf
Peds	Peds-046A	In infants and children with cardiogenic shock (P), does the use of any specific inotropic agent (I) when compared with standard care (C), improve patient outcome (hemodynamics, survival) (O)?	Cardiogenic shock and inotropes	Akira Nishisaki	http://circ.ahajournals.org/site/C2010/Peds-046A.pdf
Peds	Peds-047A	In infants and children with hypotensive septic shock (P), does the use of etomidate as an induction agent to facilitate intubation (I) compared with a standard technique without etomidate (C) improve patient outcome (hemodynamics, survival) (O)?	Etomidate and septic shock	Stephen M. Schexnayder	http://circ.ahajournals.org/site/C2010/Peds-047A.pdf
Peds	Peds-047B	In infants and children with hypotensive septic shock (P), does the use of etomidate as an induction agent to facilitate intubation (I) compared with a standard technique without etomidate (C) improve patient outcome (hemodynamics, survival) (O)?	Etomidate and septic shock	Jonathan Duff	http://circ.ahajournals.org/site/C2010/Peds-047B.pdf
Peds	Peds-048A	In infants and children who are undergoing resuscitation from cardiac arrest (P), does consideration of a channelopathy as the etiology of the arrest (I), as compared with standard management (C), improve outcome (ROSC, survival to discharge, survival with favorable neurologic outcome) (O)?	Channelopathies	Robert Hickey	http://circ.ahajournals.org/site/C2010/Peds-048A.pdf
Peds	Peds-048B	In infants and children who are undergoing resuscitation from cardiac arrest (P), does consideration of a channelopathy as the etiology of the arrest (I), as compared with standard management (C), improve outcome (ROSC, survival to discharge, survival with favorable neurologic outcome) (O)?	Channelopathies	William Scott	http://circ.ahajournals.org/site/C2010/Peds-048B.pdf
Peds	Peds-049A	In infants and children with hypotensive septic shock (P), does the use of corticosteroids in addition to standard care (I) when compare with standard care without the use of corticosteroids (C), improve patient outcome (eg. Hemodynamics or survival) (O)?	Corticosteroids and septic shock	Arno Zaritsky	http://circ.ahajournals.org/site/C2010/Peds-049A.pdf
Peds	Peds-049B	In infants and children with hypotensive septic shock (P), does the use of corticosteroids in addition to standard care (I) when compare with standard care without the use of corticosteroids (C), improve patient outcome (eg. Hemodynamics or survival) (O)?	Corticosteroids and septic shock	Mark G. Coulthard	http://circ.ahajournals.org/site/C2010/Peds-049B.pdf
Peds	Peds-050A	In infants and children with acute illness or injury (P), do specific diagnostic tests (laboratory data [mixed venous oxygen saturation, pH, lactate], (I) as opposed to clinical data (vital signs, capillary refill, mental status, end-organ function [urine output]) (C), increase the accuracy of diagnosis of shock (O)?	Diagnostic tests for shock	Alexis Topjian	http://circ.ahajournals.org/site/C2010/Peds-050A.pdf
Peds	Peds-050B	In infants and children with acute illness or injury (P), do specific diagnostic tests (laboratory data [mixed venous oxygen saturation, pH, lactate], (I) as opposed to clinical data (vital signs, capillary refill, mental status, end-organ function [urine output]) (C), increase the accuracy of diagnosis of shock (O)?	Diagnostic tests for shock	Sharon B. Kinney	http://circ.ahajournals.org/site/C2010/Peds-050B.pdf
Peds	Peds-052A	In infants and children with cardiac arrest or symptomatic bradycardia that is unresponsive to oxygenation and/or ventilation (P), does the use of atropine (I), as compared with epinephrine or no atropine (C), improve patient outcome (return to age-appropriate heart rate, subsequent pulseless arrest, ROSC, survival) (O)?	Atropine vs epinephrine for bradycardia	Susan Fuchs, Sasa Kurosawa, Masahiko Nitta	http://circ.ahajournals.org/site/C2010/Peds-052A.pdf
Peds	Peds-055B	For infants and children with Fontan or hemi-Fontan circulation who require resuscitation from cardiac arrest or pre-arrest states (prehospital [OHCA] or in-hospital [IHCA]) (P), does any specific modification to standard practice (I) compared with standard resuscitation practice (C) improve outcome (eg. ROSC, survival to discharge, survival with good neurologic outcome (O)?	Resuscitation for hemi-Fontan/Fontan circulation	Desmond Bohn, Bradley S. Marino	http://circ.ahajournals.org/site/C2010/Peds-055B.pdf
Peds	Peds-056A	For infants and children in cardiac arrest with pulmonary hypertension (prehospital [OHCA] or in-hospital [IHCA]) (P), do any specific modifications to resuscitation techniques (I) compared with standard resuscitation techniques (C), improve outcome (ROSC, survival to discharge, favorable neurologic survival) (O)?	Resuscitation of the patient with pulmonary hypertension	Ian Adatia, John Berger, David Wessel	http://circ.ahajournals.org/site/C2010/Peds-056A.pdf
Peds	Peds-057A	For infants and children who require endotracheal intubation (prehospital or in hospital) (P) does the use of a specific formula to guide cuffed endotracheal tube size (I), as opposed to the use of the existing formula of 3 + age/4 (C), achieve better outcomes (eg. successful tube placement) (O)?	Formula for cuffed ET tube size	Robert Bingham	http://circ.ahajournals.org/site/C2010/Peds-057A.pdf
Peds	Peds-057B	For infants and children who require endotracheal intubation (prehospital or in hospital) (P) does the use of a specific formula to guide cuffed endotracheal tube size (I), as opposed to the use of the existing formula of 3 + age/4 (C), achieve better outcomes (eg. successful tube placement) (O)?	Formulas for predicting ET tube size	Eugene B. Freid	http://circ.ahajournals.org/site/C2010/Peds-057B.pdf

(*Continued*)

CoSTR Part 10: Worksheet Appendix, *Continued*

Task Force	WS ID	PICO Title	Short Title	Authors	URL
Peds	Peds-059	For infants and children with single ventricle, s/p stage I repair who require resuscitation from cardiac arrest or pre-arrest states (prehospital [OHCA] or in-hospital [IHCA]) (P), does any specific modification to standard practice (I) compared with standard resuscitation practice (C) improve outcome (eg. ROSC, survival to discharge, survival with good neurologic outcome) (O)?	Resuscitation of the patient with single ventricle	George M. Hoffman, Shane Tibby	http://circ.ahajournals.org/site/C2010/Peds-059.pdf
Peds	Peds-060	For pediatric patients (in any setting (P), is there a clinical decision rule (I) that enables reliable prediction of ROSC (or futile resuscitation efforts)? (PROGNOSIS)	Clinical decision rules to predict ROSC	Gabrielle Nuthall	http://circ.ahajournals.org/site/C2010/Peds-060.pdf

References

1. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Part 6: Paediatric Basic And Advanced Life Support. *Resuscitation.* 2005;67:271–291.
2. 2005 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations. Part 6: Pediatric Basic and Advanced Life Support. *Circulation.* 2005;112:III-73–III-90.
3. Chan PS, Jain R, Nallmothu BK, Berg RA, Sasson C. Rapid response teams: a systematic review and meta-analysis. *Arch Intern Med*. 2010; 170:18–26.
4. Sharek PJ, Parast LM, Leong K, Coombs J, Earnest K, Sullivan J, Frankel LR, Roth SJ. Effect of a rapid response team on hospital-wide mortality and code rates outside the ICU in a children's hospital. *JAMA*. 2007;298:2267–2274.
5. Tibballs J, Kinney S, Duke T, Oakley E, Hennessy M. Reduction of paediatric in-patient cardiac arrest and death with a medical emergency team: preliminary results. *Arch Dis Child*. 2005;90:1148–1152.
6. Tibballs J, Kinney S. Reduction of hospital mortality and of preventable cardiac arrest and death on introduction of a pediatric medical emergency team. *Pediatr Crit Care Med*. 2009;10:306–312.
7. Hunt EA, Zimmer KP, Rinke ML, Shilkofski NA, Matlin C, Garger C, Dickson C, Miller MR. Transition from a traditional code team to a medical emergency team and categorization of cardiopulmonary arrests in a children's center. *Arch Pediatr Adolesc Med*. 2008;162:117–122.
8. Brilli RJ, Gibson R, Luria JW, Wheeler TA, Shaw J, Linam M, Kheir J, McLain P, Lingsch T, Hall-Haering A, McBride M. Implementation of a medical emergency team in a large pediatric teaching hospital prevents respiratory and cardiopulmonary arrests outside the intensive care unit. *Pediatr Crit Care Med.* 2007;8:236–246.
9. Mistry KP, Turi J, Hueckel R, Mericle JM, Meliones JN. Pediatric rapid response teams in the academic medical center. *Clin Pediatr Emerg Med*. 2006;7:241–247.
10. Dudley NC, Hansen KW, Furnival RA, Donaldson AE, Van Wagenen KL, Scaife ER. The effect of family presence on the efficiency of pediatric trauma resuscitations. *Ann Emerg Med.* 2009;53:777–784 e773.
11. Tinsley C, Hill JB, Shah J, Zimmerman G, Wilson M, Freier K, Abd-Allah S, Tinsley C, Hill JB, Shah J, Zimmerman G, Wilson M, Freier K, Abd-Allah S. Experience of families during cardiopulmonary resuscitation in a pediatric intensive care unit. *Pediatrics*. 2008;122: e799–e804.
12. Mangurten J, Scott SH, Guzzetta CE, Clark AP, Vinson L, Sperry J, Hicks B, Voelmeck W. Effects of family presence during resuscitation and invasive procedures in a pediatric emergency department. *J Emerg Nurs*. 2006;32:225–233.
13. McGahey-Oakland PR, Lieder HS, Young A, Jefferson LS. Family experiences during resuscitation at a children's hospital emergency department. *J Pediatr Health Care*. 2007;21:217–225.
14. Jones M, Qazi M, Young KD. Ethnic differences in parent preference to be present for painful medical procedures. *Pediatrics*. 2005;116: e191–e197.
15. Boie ET, Moore GP, Brummett C, Nelson DR. Do parents want to be present during invasive procedures performed on their children in the emergency department? A survey of 400 parents. *Ann Emerg Med*. 1999;34:70–74.
16. Andrews R, Andrews R. Family presence during a failed major trauma resuscitation attempt of a 15-year-old boy: lessons learned. [see comment]. *J Emerg Nurs*. 2004;30:556–558.
17. Dill K, Gance-Cleveland B. With you until the end: family presence during failed resuscitation. *J Spec Pediatr Nurs*. 2005;10:204–207.
18. Gold KJ, Gorenflo DW, Schwenk TL, Bratton SL. Physician experience with family presence during cardiopulmonary resuscitation in children. [see comment]. *Pediatr Crit Care Med*. 2006;7:428–433.
19. Duran CR, Oman KS, Abel JJ, Koziel VM, Szymanski D. Attitudes toward and beliefs about family presence: a survey of healthcare providers, patients' families, and patients. *Am J Crit Care*. 2007;16: 270–279.
20. Doyle CJ, Post H, Burney RE, Maino J, Keefe M, Rhee KJ. Family participation during resuscitation: an option. *Ann Emerg Med*. 1987;16: 673–675.
21. Hanson C, Strawser D. Family presence during cardiopulmonary resuscitation: Foote Hospital Emergency Department's Nine-Year Perspective. *J Emerg Nurs*. 1992;18:104–106.
22. Meyers TA, Eichhorn DJ, Guzzetta CE. Do families want to be present during CPR? A retrospective survey. *J Emerg Nurs*. 1998;24:400–405.
23. Meyers TA, Eichhorn DJ, Guzzetta CE, Clark AP, Klein JD, Taliaferro E, Calvin A. Family presence during invasive procedures and resuscitation. *Am J Nurs.* 2000;100:32–42.
24. Holzhauser K, Finucane J, De Vries S. Family presence during resuscitation: a randomised controlled trial of the impact of family presence. *Australas. Emerg Nurs J*. 2005;8:139–147.
25. Robinson SM, Mackenzie-Ross S, Campbell Hewson GL, Egleston CV, Prevost AT. Psychological effect of witnessed resuscitation on bereaved relatives. *Lancet*. 1998;352:614–617.
26. van der Woning M. Relatives in the resuscitation area: a phenomenological study. *Nurs Crit Care*. 1999;4:186–192.
27. O'Connell KJ, Farah MM, Spandorfer P, Zorc JJ. Family presence during pediatric trauma team activation: an assessment of a structured program. *Pediatrics*. 2007;120:e565–574.
28. Engel KG, Barnosky AR, Berry-Bovia M, Desmond JS, Ubel PA. Provider experience and attitudes toward family presence during resuscitation procedures. *J Palliat Med*. 2007;10:1007–1009.
29. Boyd R, White S. Does witnessed cardiopulmonary resuscitation alter perceived stress in accident and emergency staff? *Eur J Emerg Med*. 2000;7:51–53.
30. Compton S, Madgy A, Goldstein M, Sandhu J, Dunne R, Swor R. Emergency medical service providers' experience with family presence during cardiopulmonary resuscitation. *Resuscitation*. 2006;70:223–228.
31. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. *Resuscitation*. 1997;35:23–26.
32. Brearley S, Shearman CP, Simms MH. Peripheral pulse palpation: an unreliable physical sign. *Ann R Coll Surg Engl*. 1992;74:169–171.
33. Cavallaro DL, Melker RJ. Comparison of two techniques for detecting cardiac activity in infants. *Crit Care Med*. 1983;11:189–190.
34. Inagawa G, Morimura N, Miwa T, Okuda K, Hirata M, Hiroki K. A comparison of five techniques for detecting cardiac activity in infants. *Paediatr Anaesth*. 2003;13:141–146.
35. Kamlin CO, O'Donnell CP, Everest NJ, Davis PG, Morley CJ. Accuracy of clinical assessment of infant heart rate in the delivery room. *Resuscitation*. 2006;71:319–321.
36. Lee CJ, Bullock LJ. Determining the pulse for infant CPR: time for a change? *Mil Med*. 1991;156:190–193.
37. Mather C, O'Kelly S. The palpation of pulses. *Anaesthesia*. 1996;51: 189–191.
38. Ochoa FJ, Ramalle-Gomara E, Carpintero JM, Garcia A, Saralegui I. Competence of health professionals to check the carotid pulse. *Resuscitation*. 1998;37:173–175.
39. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. *Resuscitation*. 2004;60:213–217.

40. Sarti A, Savron F, Casotto V, Cuttini M. Heartbeat assessment in infants: a comparison of four clinical methods. *Pediatr Crit Care Med*. 2005;6:212–215.
41. Sarti A, Savron F, Ronfani L, Pelizzo G, Barbi E. Comparison of three sites to check the pulse and count heart rate in hypotensive infants. *Paediatr Anaesth*. 2006;16:394–398.
42. Tanner M, Nagy S, Peat JK. Detection of infant's heart beat/pulse by caregivers: a comparison of 4 methods. *J Pediatr*. 2000;137:429–430.
43. Whitelaw CC, Goldsmith LJ. Comparison of two techniques for determining the presence of a pulse in an infant. *Acad Emerg Med*. 1997;4:153–154.
44. Dick WF, Eberle B, Wisser G, Schneider T. The carotid pulse check revisited: what if there is no pulse? *Crit Care Med*. 2000;28:N183–N185.
45. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation*. 1996;33:107–116.
46. Tibballs J, Russell P. Reliability of pulse palpation by healthcare personnel to diagnose paediatric cardiac arrest. *Resuscitation*. 2009;80:61–64.
47. Tibballs J, Weeranatna C. The influence of time on the accuracy of healthcare personnel to diagnose paediatric cardiac arrest by pulse palpation. *Resuscitation.* 2010;81:671–675.
48. Tsung JW, Blaivas M. Feasibility of correlating the pulse check with focused point-of-care echocardiography during pediatric cardiac arrest: a case series. *Resuscitation*. 2008;77:264–269.
49. Steiger HV, Rimbach K, Muller E, Breitkreutz R. Focused emergency echocardiography: lifesaving tool for a 14-year-old girl suffering out-of-hospital pulseless electrical activity arrest because of cardiac tamponade. *Eur J Emerg Med*. 2009;16:103–105.
50. Blaivas M, Fox JC. Outcome in cardiac arrest patients found to have cardiac standstill on the bedside emergency department echocardiogram. *Acad Emerg Med*. 2001;8:616–621.
51. Menaker J, Cushman J, Vermillion JM, Rosenthal RE, Scalea TM. Ultrasound-diagnosed cardiac tamponade after blunt abdominal trauma-treated with emergent thoracotomy. *J Emerg Med*. 2007;32:99–103.
52. Niendorff DF, Rassias AJ, Palac R, Beach ML, Costa S, Greenberg M. Rapid cardiac ultrasound of inpatients suffering PEA arrest performed by nonexpert sonographers. *Resuscitation*. 2005;67:81–87.
53. Querellou E, Meyran D, Petitjean F, Le Dreff P, Maurin O. Ventricular fibrillation diagnosed with trans-thoracic echocardiography. *Resuscitation*. 2009;80:1211–1213.
54. Salen P, Melniker L, Chooljian C, Rose JS, Alteveer J, Reed J, Heller M. Does the presence or absence of sonographically identified cardiac activity predict resuscitation outcomes of cardiac arrest patients? *Am J Emerg Med*. 2005;23:459–462.
55. Salen P, O'Connor R, Sierzenski P, Passarello B, Pancu D, Melanson S, Arcona S, Reed J, Heller M. Can cardiac sonography and capnography be used independently and in combination to predict resuscitation outcomes? *Acad Emerg Med*. 2001;8:610–615.
56. Tayal VS, Kline JA. Emergency echocardiography to detect pericardial effusion in patients in PEA and near-PEA states. *Resuscitation*. 2003;59:315–318.
57. Varriale P, Maldonado JM. Echocardiographic observations during inhospital cardiopulmonary resuscitation. *Crit Care Med*. 1997;25:1717–1720.
58. Li Y, Ristagno G, Bisera J, Tang W, Deng Q, Weil MH. Electrocardiogram waveforms for monitoring effectiveness of chest compression during cardiopulmonary resuscitation. *Crit Care Med*. 2008;36:211–215.
59. Ristagno G, Tang W, Chang YT, Jorgenson DB, Russell JK, Huang L, Wang T, Sun S, Weil MH. The quality of chest compressions during cardiopulmonary resuscitation overrides importance of timing of defibrillation. *Chest*. 2007;132:70–75.
60. Rubertsson S, Karlsten R. Increased cortical cerebral blood flow with LUCAS; a new device for mechanical chest compressions compared to standard external compressions during experimental cardiopulmonary resuscitation. *Resuscitation*. 2005;65:357–363.
61. Kern KB, Sanders AB, Raife J, Milander MM, Otto CW, Ewy GA. A study of chest compression rates during cardiopulmonary resuscitation in humans: the importance of rate-directed chest compressions. *Arch Intern Med*. 1992;152:145–149.
62. Ornato JP, Gonzalez ER, Garnett AR, Levine RL, McClung BK. Effect of cardiopulmonary resuscitation compression rate on end-tidal carbon dioxide concentration and arterial pressure in man. *Crit Care Med*. 1988;16:241–245.
63. Guly UM, Robertson CE. Active decompression improves the haemodynamic state during cardiopulmonary resuscitation. *Br Heart J*. 1995;73:372–376.
64. Wik L, Naess PA, Ilebekk A, Nicolaysen G, Steen PA. Effects of various degrees of compression and active decompression on haemodynamics, end-tidal CO2, and ventilation during cardiopulmonary resuscitation of pigs. *Resuscitation*. 1996;31:45–57.
65. Berg RA, Sanders AB, Milander M, Tellez D, Liu P, Beyda D. Efficacy of audio-prompted rate guidance in improving resuscitator performance of cardiopulmonary resuscitation on children. *Acad Emerg Med*. 1994;1:35–40.
66. Idris AH, Staples ED, O'Brien DJ, Melker RJ, Rush WJ, Del Duca KD, Falk JL. End-tidal carbon dioxide during extremely low cardiac output. *Ann Emerg Med*. 1994;23:568–572.
67. Jin X, Weil MH, Tang W, Povoas H, Pernat A, Xie J, Bisera J. End-tidal carbon dioxide as a noninvasive indicator of cardiac index during circulatory shock. *Crit Care Med*. 2000;28:2415–2419.
68. Ornato JP, Garnett AR, Glauser FL. Relationship between cardiac output and the end-tidal carbon dioxide tension. *Ann Emerg Med*. 1990;19:1104–1106.
69. Mauer D, Schneider T, Elich D, Dick W. Carbon dioxide levels during pre-hospital active compression–decompression versus standard cardiopulmonary resuscitation. *Resuscitation*. 1998;39:67–74.
70. Kolar M, Krizmaric M, Klemen P, Grmec S. Partial pressure of end-tidal carbon dioxide successful predicts cardiopulmonary resuscitation in the field: A prospective observational study. *Crit Care*. 2008;12:R115.
71. Bhende MS, Karasic DG, Karasic RB. End-tidal carbon dioxide changes during cardiopulmonary resuscitation after experimental asphyxial cardiac arrest. *Am J Emerg Med*. 1996;14:349–350.
72. Grmec S, Krizmaric M, Mally S, Kozelj A, Spindler M, Lesnik B. Utstein style analysis of out-of-hospital cardiac arrest–bystander CPR and end expired carbon dioxide. *Resuscitation*. 2007;72:404–414.
73. Pokorna M, Necas E, Kratochvil J, Skripsky R, Andrlik M, Franek O. A sudden increase in partial pressure end-tidal carbon dioxide (P(ET)CO(2)) at the moment of return of spontaneous circulation. *J Emerg Med.* 2009; Jun 30. Epub.
74. Bhende MS, Thompson AE. Evaluation of an end-tidal CO2 detector during pediatric cardiopulmonary resuscitation. *Pediatrics*. 1995;95:395–399.
75. Gomersall CD, Joynt GM, Morley AP. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med*. 1997;337:1694.
76. Grmec S, Klemen P. Does the end-tidal carbon dioxide (ETCO2) concentration have prognostic value during out-of-hospital cardiac arrest? *Eur J Emerg Med*. 2001;8:263–269.
77. Grmec S, Kupnik D. Does the Mainz emergency evaluation scoring (MEES) in combination with capnometry (MEESC) help in the prognosis of outcome from cardiopulmonary resuscitation in a prehospital setting? *Resuscitation*. 2003;58:89–96.
78. Grmec S, Lah K, Tusek-Bunc K. Difference in end-tidal CO2 between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. *Crit Care*. 2003;7:R139–R144.
79. Grmec S, Strnad M, Podgorsek D. Comparison of the characteristics and outcome among patients suffering from out-of-hospital primary cardiac arrest and drowning victims in cardiac arrest. *Int J Emerg Med*. 2009;2:7–12.
80. Levine RL, Wayne MA, Miller CC. End-tidal carbon dioxide and outcome of out-of-hospital cardiac arrest. *N Engl J Med*. 1997;337:301–306.
81. Mally S, Jelatancev A, Grmec S. Effects of epinephrine and vasopressin on end-tidal carbon dioxide tension and mean arterial blood pressure in out-of-hospital cardiopulmonary resuscitation: an observational study. *Crit Care*. 2007;11:R39.
82. Berg RA, Henry C, Otto CW, Sanders AB, Kern KB, Hilwig RW, Ewy GA. Initial end-tidal CO_2 is markedly elevated during cardiopulmonary resuscitation after asphyxial cardiac arrest. *Pediatr Emerg Care*. 1996;12:245–248.
83. Chuang ML, Chang HC, Lim KE, Vintch JR. Gas exchange detection of right-to-left shunt in dyspneic patients: report of three cases. *Int J Cardiol*. 2006;108:117–119.
84. Matthews IL, Bjornstad PG, Kaldestad RH, Heiberg L, Thaulow E, Gronn M. The impact of shunt size on lung function in infants with

univentricular heart physiology. *Pediatr Crit Care Med*. 2009;10:60–65.
85. Tugrul M, Camci E, Sungur Z, Pembeci K. The value of end-tidal carbon dioxide monitoring during systemic-to-pulmonary artery shunt insertion in cyanotic children. *J Cardiothorac Vasc Anesth*. 2004;18:152–155.
86. Falk JL, Rackow EC, Weil MH. End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. *N Engl J Med*. 1988;318:607–611.
87. Callaham M, Barton C, Matthay M. Effect of epinephrine on the ability of end-tidal carbon dioxide readings to predict initial resuscitation from cardiac arrest. *Crit Care Med*. 1992;20:337–343.
88. Cantineau JP, Merckx P, Lambert Y, Sorkine M, Bertrand C, Duvaldestin P. Effect of epinephrine on end-tidal carbon dioxide pressure during prehospital cardiopulmonary resuscitation. *Am J Emerg Med*. 1994;12:267–270.
89. Gonzalez ER, Ornato JP, Garnett AR, Levine RL, Young DS, Racht EM. Dose-dependent vasopressor response to epinephrine during CPR in human beings. *Ann Emerg Med*. 1989;18:920–926.
90. Chase PB, Kern KB, Sanders AB, Otto CW, Ewy GA. Effects of graded doses of epinephrine on both noninvasive and invasive measures of myocardial perfusion and blood flow during cardiopulmonary resuscitation. *Crit Care Med*. 1993;21:413–419.
91. Lindberg L, Liao Q, Steen S. The effects of epinephrine/norepinephrine on end-tidal carbon dioxide concentration, coronary perfusion pressure and pulmonary arterial blood flow during cardiopulmonary resuscitation. *Resuscitation*. 2000;43:129–140.
92. Davis PG, Tan A, O'Donnell CP, Schulze A. Resuscitation of newborn infants with 100% oxygen or air: a systematic review and meta-analysis. *Lancet*. 2004;364:1329–1333.
93. Rabi Y, Rabi D, Yee W. Room air resuscitation of the depressed newborn: a systematic review and meta-analysis. *Resuscitation*. 2007;72:353–363.
94. Balan IS, Fiskum G, Hazelton J, Cotto-Cumba C, Rosenthal RE. Oximetry-guided reoxygenation improves neurological outcome after experimental cardiac arrest. *Stroke*. 2006;37:3008–3013.
95. Liu Y, Rosenthal RE, Haywood Y, Miljkovic-Lolic M, Vanderhoek JY, Fiskum G. Normoxic ventilation after cardiac arrest reduces oxidation of brain lipids and improves neurological outcome. *Stroke*. 1998;29:1679–1686.
96. Marsala J, Marsala M, Vanicky I, Galik J, Orendacova J. Post cardiac arrest hyperoxic resuscitation enhances neuronal vulnerability of the respiratory rhythm generator and some brainstem and spinal cord neuronal pools in the dog. *Neurosci Lett*. 1992;146:121–124.
97. Richards EM, Rosenthal RE, Kristian T, Fiskum G. Postischemic hyperoxia reduces hippocampal pyruvate dehydrogenase activity. *Free Radic Biol Med*. 2006;40:1960–1970.
98. Richards EM, Fiskum G, Rosenthal RE, Hopkins I, McKenna MC. Hyperoxic reperfusion after global ischemia decreases hippocampal energy metabolism. *Stroke*. 2007;38:1578–1584.
99. Vereczki V, Martin E, Rosenthal RE, Hof PR, Hoffman GE, Fiskum G. Normoxic resuscitation after cardiac arrest protects against hippocampal oxidative stress, metabolic dysfunction, and neuronal death. *J Cereb Blood Flow Metab*. 2006;26:821–835.
100. Zwemer CF, Whitesall SE, D'Alecy LG. Cardiopulmonary-cerebral resuscitation with 100% oxygen exacerbates neurological dysfunction following nine minutes of normothermic cardiac arrest in dogs. *Resuscitation*. 1994;27:159–170.
101. Lipinski CA, Hicks SD, Callaway CW. Normoxic ventilation during resuscitation and outcome from asphyxial cardiac arrest in rats. *Resuscitation*. 1999;42:221–229.
102. Feet BA, Yu XQ, Rootwelt T, Oyasaeter S, Saugstad OD. Effects of hypoxemia and reoxygenation with 21% or 100% oxygen in newborn piglets: extracellular hypoxanthine in cerebral cortex and femoral muscle. *Crit Care Med*. 1997;25:1384–1391.
103. Khine HH, Corddry DH, Kettrick RG, Martin TM, McCloskey JJ, Rose JB, Theroux MC, Zagnoev M. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. *Anesthesiology*. 1997;86:627–631; discussion 627A.
104. Weiss M, Dullenkopf A, Fischer JE, Keller C, Gerber AC. Prospective randomized controlled multi-centre trial of cuffed or uncuffed endotracheal tubes in small children. *Br J Anaesth*. 2009;103:867–873.
105. Dorsey DP, Bowman SM, Klein MB, Archer D, Sharar SR. Perioperative use of cuffed endotracheal tubes is advantageous in young pediatric burn patients. *Burns*. 2010;36:856–860.
106. Bordet F, Allaouchiche B, Lansiaux S, Combet S, Pouyau A, Taylor P, Bonnard C, Chassard D. Risk factors for airway complications during general anaesthesia in paediatric patients. *Paediatr Anaesth*. 2002;12:762–769.
107. Mossad E, Youssef G. Subglottic stenosis in children undergoing repair of congenital heart defects. *J Cardiothorac Vasc Anesth*. 2009;23:658–662.
108. Newth CJ, Rachman B, Patel N, Hammer J. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. *J Pediatr*. 2004;144:333–337.
109. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. *J Pediatr*. 1994;125:57–62.
110. Mhanna MJ, Zamel YB, Tichy CM, Super DM. The "Air leak" Test around the endotracheal tube, as a predictor of postextubation stridor, is age dependent in children. *Crit Care Med*. 2002;30:2639–2643.
111. Browning DH, Graves SA. Incidence of aspiration with endotracheal tubes in children. *J Pediatr*. 1983;102:582–584.
112. Duracher C, Schmautz E, Martinon C, Faivre J, Carli P, Orliaguet G. Evaluation of cuffed tracheal tube size predicted using the Khine formula in children. *Paediatr Anaesth*. 2008;18:113–118.
113. Dullenkopf A, Gerber AC, Weiss M. Fit and seal characteristics of a new paediatric tracheal tube with high volume-low pressure polyurethane cuff. *Acta Anaesthesiol Scand*. 2005;49:232–237.
114. Dullenkopf A, Kretschmar O, Knirsch W, Tomaske M, Hug M, Stutz K, Berger F, Weiss M. Comparison of tracheal tube cuff diameters with internal transverse diameters of the trachea in children. *Acta Anaesthesiol Scand*. 2006;50:201–205.
115. Salgo B, Schmitz A, Henze G, Stutz K, Dullenkopf A, Neff S, Gerber AC, Weiss M. Evaluation of a new recommendation for improved cuffed tracheal tube size selection in infants and small children. *Acta Anaesthesiol Scand*. 2006;50:557–561.
116. Gausche M, Lewis RJ, Stratton SJ, Haynes BE, Gunter CS, Goodrich SM, Poore PD, McCollough MD, Henderson DP, Pratt FD, Seidel JS. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. *JAMA*. 2000;283:783–790.
117. Lecky F, Bryden D, Little R, Tong N, Moulton C. Emergency intubation for acutely ill and injured patients. *Cochrane Database Syst Rev*. 2008:CD001429.
118. DiRusso SM, Sullivan T, Risucci D, Nealon P, Slim M. Intubation of pediatric trauma patients in the field: predictor of negative outcome despite risk stratification. *J Trauma*. 2005;59:84–90; discussion 90–81.
119. Gerritse BM, Draaisma JM, Schalkwijk A, van Grunsven PM, Scheffer GJ. Should EMS-paramedics perform paediatric tracheal intubation in the field? *Resuscitation*. 2008;79:225–229.
120. A prospective multicenter evaluation of prehospital airway management performance in a large metropolitan region. *Prehosp Emerg Care*. 2009;13:304–310.
121. Garza AG, Algren DA, Gratton MC, Ma OJ. Populations at risk for intubation nonattempt and failure in the prehospital setting. *Prehosp Emerg Care*. 2005;9:163–166.
122. Hon KL, Olsen H, Totapally B, Leung TF. Hyperventilation at referring hospitals is common before transport in intubated children with neurological diseases. *Pediatr Emerg Care*. 2005;21:662–666.
123. Wang HE, Lave JR, Sirio CA, Yealy DM. Paramedic intubation errors: isolated events or symptoms of larger problems? *Health Aff (Millwood)*. 2006;25:501–509.
124. Tam RK, Maloney J, Gaboury I, Verdon JM, Trickett J, Leduc SD, Poirier P. Review of endotracheal intubations by Ottawa advanced care paramedics in Canada. *Prehosp Emerg Care*. 2009;13:311–315.
125. Warner KJ, Sharar SR, Copass MK, Bulger EM. Prehospital management of the difficult airway: a prospective cohort study. *J Emerg Med*. 2009;36:257–265.
126. Carenzi B, Corso RM, Stellino V, Carlino GD, Tonini C, Rossini L, Gentili G. Airway management in an infant with congenital centrofacial dysgenesia. *Br J Anaesth*. 2002;88:726–728.
127. Fraser J, Hill C, McDonald D, Jones C, Petros A. The use of the laryngeal mask airway for inter-hospital transport of infants with type 3 laryngotracheo-oesophageal clefts. *Intensive Care Med*. 1999;25:714–716.
128. Iohom G, Lyons B, Casey W. Airway management in a baby with femoral hypoplasia-unusual facies syndrome. *Paediatr Anaesth*. 2002;12:461–464.

129. Johr M, Berger TM, Ruppen W, Schlegel C. Congenital laryngotracheo-oesophageal cleft: successful ventilation with the laryngeal mask airway. *Paediatr Anaesth*. 2003;13:68–71.
130. Leal-Pavey YR. Use of the LMA classic to secure the airway of a premature neonate with Smith-Lemli-Opitz syndrome: a case report. *AANA J*. 2004;72:427–430.
131. Russell P, Chambers N, du Plessis J, Vijayasekeran S. Emergency use of a size 1 laryngeal mask airway in a ventilated neonate with an undiagnosed type IV laryngotracheo-oesophageal cleft. *Paediatr Anaesth*. 2008;18:658–662.
132. Scheller B, Schalk R, Byhahn C, Peter N, L'Allemand N, Kessler P, Meininger D. Laryngeal tube suction II for difficult airway management in neonates and small infants. *Resuscitation*. 2009;80:805–810.
133. Stocks RM, Egerman R, Thompson JW, Peery M. Airway management of the severely retrognathic child: use of the laryngeal mask airway. *Ear Nose Throat J*. 2002;81:223–226.
134. Yao CT, Wang JN, Tai YT, Tsai TY, Wu JM. Successful management of a neonate with Pierre-Robin syndrome and severe upper airway obstruction by long term placement of a laryngeal mask airway. *Resuscitation*. 2004;61:97–99.
135. Stone BJ, Chantler PJ, Baskett PJ. The incidence of regurgitation during cardiopulmonary resuscitation: a comparison between the bag valve mask and laryngeal mask airway. *Resuscitation*. 1998;38:3–6.
136. Comparison of arterial blood gases of laryngeal mask airway and bag-valve-mask ventilation in out-of-hospital cardiac arrests. *Circ J*. 2009; 73:490–496.
137. Lopez-Gil M, Brimacombe J, Alvarez M. Safety and efficacy of the laryngeal mask airway. A prospective survey of 1400 children. *Anaesthesia*. 1996;51:969–972.
138. Lopez-Gil M, Brimacombe J, Cebrian J, Arranz J. Laryngeal mask airway in pediatric practice: a prospective study of skill acquisition by anesthesia residents. *Anesthesiology*. 1996;84:807–811.
139. Park C, Bahk JH, Ahn WS, Do SH, Lee KH. The laryngeal mask airway in infants and children. *Can J Anaesth*. 2001;48:413–417.
140. Bagshaw O. The size 1.5 laryngeal mask airway (LMA) in paediatric anaesthetic practice. *Paediatr Anaesth*. 2002;12:420–423.
141. Harnett M, Kinirons B, Heffernan A, Motherway C, Casey W. Airway complications in infants: comparison of laryngeal mask airway and the facemask-oral airway. *Can J Anaesth*. 2000;47:315–318.
142. Flick RP, Wilder RT, Pieper SF, van Koeverden K, Ellison KM, Marienau ME, Hanson AC, Schroeder DR, Sprung J. Risk factors for laryngospasm in children during general anesthesia. *Paediatr Anaesth*. 2008;18:289–296.
143. Chen L, Hsiao AL. Randomized trial of endotracheal tube versus laryngeal mask airway in simulated prehospital pediatric arrest. *Pediatrics*. 2008;122:e294–e297.
144. Guyette FX, Roth KR, LaCovey DC, Rittenberger JC. Feasibility of laryngeal mask airway use by prehospital personnel in simulated pediatric respiratory arrest. *Prehosp Emerg Care*. 2007;11:245–249.
145. Rechner JA, Loach VJ, Ali MT, Barber VS, Young JD, Mason DG. A comparison of the laryngeal mask airway with facemask and oropharyngeal airway for manual ventilation by critical care nurses in children. *Anaesthesia*. 2007;62:790–795.
146. Blevin AE, McDouall SF, Rechner JA, Saunders TA, Barber VS, Young JD, Mason DG. A comparison of the laryngeal mask airway with the facemask and oropharyngeal airway for manual ventilation by first responders in children. *Anaesthesia*. 2009;64:1312–1316.
147. Yannopoulos D, Matsuura T, McKnite S, Goodman N, Idris A, Tang W, Aufderheide TP, Lurie KG. No assisted ventilation cardiopulmonary resuscitation and 24-hour neurological outcomes in a porcine model of cardiac arrest. *Crit Care Med*. 2010;38:254–260.
148. Berg RA, Hilwig RW, Kern KB, Babar I, Ewy GA. Simulated mouth-to-mouth ventilation and chest compressions (bystander cardiopulmonary resuscitation) improves outcome in a swine model of prehospital pediatric asphyxial cardiac arrest. *Crit Care Med*. 1999;27:1893–1899.
149. Idris AH, Becker LB, Fuerst RS, Wenzel V, Rush WJ, Melker RJ, Orban DJ. Effect of ventilation on resuscitation in an animal model of cardiac arrest. *Circulation*. 1994;90:3063–3069.
150. Aufderheide TP, Sigurdsson G, Pirrallo RG, Yannopoulos D, McKnite S, von Briesen C, Sparks CW, Conrad CJ, Provo TA, Lurie KG. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109:1960–1965.
151. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, Vanden Hoek TL, Becker LB. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. *JAMA*. 2005;293:305–310.
152. Wik L, Kramer-Johansen J, Myklebust H, Sorebo H, Svensson L, Fellows B, Steen PA. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. *JAMA*. 2005;293:299–304.
153. O'Neill JF, Deakin CD. Do we hyperventilate cardiac arrest patients? *Resuscitation*. 2007;73:82–85.
154. Bertrand C, Hemery F, Carli P, Goldstein P, Espesson C, Ruttimann M, Macher JM, Raffy B, Fuster P, Dolveck F, Rozenberg A, Lecarpentier E, Duvaldestin P, Saissy JM, Boussignac G, Brochard L. Constant flow insufflation of oxygen as the sole mode of ventilation during out-of-hospital cardiac arrest. *Intensive Care Med*. 2006;32:843–851.
155. Bobrow BJ, Ewy GA, Clark L, Chikani V, Berg RA, Sanders AB, Vadeboncoeur TF, Hilwig RW, Kern KB. Passive oxygen insufflation is superior to bag-valve-mask ventilation for witnessed ventricular fibrillation out-of-hospital cardiac arrest. *Ann Emerg Med*. 2009;54:656–662 e651.
156. Hevesi ZG, Thrush DN, Downs JB, Smith RA. Cardiopulmonary resuscitation: effect of CPAP on gas exchange during chest compressions. *Anesthesiology*. 1999;90:1078–1083.
157. Hayes MM, Ewy GA, Anavy ND, Hilwig RW, Sanders AB, Berg RA, Otto CW, Kern KB. Continuous passive oxygen insufflation results in a similar outcome to positive pressure ventilation in a swine model of out-of-hospital ventricular fibrillation. *Resuscitation*. 2007;74:357–365.
158. Winkler M, Mauritz W, Hackl W, Gilly H, Weindlmayr-Goettel M, Steinbereithner K, Schindler I. Effects of half the tidal volume during cardiopulmonary resuscitation on acid-base balance and haemodynamics in pigs. *Eur J Emerg Med*. 1998;5:201–206.
159. Bhende MS, Thompson AE, Cook DR, Saville AL. Validity of a disposable end-tidal CO2 detector in verifying endotracheal tube placement in infants and children. *Ann Emerg Med*. 1992;21:142–145.
160. Kelly JS, Wilhoit RD, Brown RE, James R. Efficacy of the FEF colorimetric end-tidal carbon dioxide detector in children. *Anesth Analg*. 1992;75:45–50.
161. Hosono S, Inami I, Fujita H, Minato M, Takahashi S, Mugishima H. A role of end-tidal CO(2) monitoring for assessment of tracheal intubations in very low birth weight infants during neonatal resuscitation at birth. *J Perinat Med*. 2009;37:79–84.
162. O'Donnell CP, Kamlin CO, Davis PG, Morley CJ. Endotracheal intubation attempts during neonatal resuscitation: Success rates, duration, and adverse effects. *Pediatrics*. 2006;117:e16–21.
163. Salthe J, Kristiansen SM, Sollid S, Oglaend B, Soreide E. Capnography rapidly confirmed correct endotracheal tube placement during resuscitation of extremely low birthweight babies (<1000 g). *Acta Anaesthesiol Scand*. 2006;50:1033–1036.
164. Bhende MS, Allen WD Jr. Evaluation of a capno-flo resuscitator during transport of critically ill children. *Pediatr Emerg Care*. 2002;18: 414–416.
165. Singh S, Allen WD Jr, Venkataraman ST, Bhende MS. Utility of a novel quantitative handheld microstream capnometer during transport of critically ill children. *Am J Emerg Med*. 2006;24:302–307.
166. Gonzalez del Rey JA, Poirier MP, Digiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO2 system in the detection of airway mishaps: an animal trial. *Pediatr Emerg Care*. 2000;16:121–123.
167. Poirier MP, Gonzalez Del-Rey JA, McAneney CM, DiGiulio GA. Utility of monitoring capnography, pulse oximetry, and vital signs in the detection of airway mishaps: a hyperoxemic animal model. *Am J Emerg Med*. 1998;16:350–352.
168. Sharieff GQ, Rodarte A, Wilton N, Silva PD, Bleyle D. The self-inflating bulb as an esophageal detector device in children weighing more than twenty kilograms: a comparison of two techniques. *Ann Emerg Med*. 2003;41:623–629.
169. Sharieff GQ, Rodarte A, Wilton N, Bleyle D. The self-inflating bulb as an airway adjunct: is it reliable in children weighing less than 20 kilograms? *Acad Emerg Med*. 2003;10:303–308.
170. Moynihan RJ, Brock-Utne JG, Archer JH, Feld LH, Kreitzman TR. The effect of cricoid pressure on preventing gastric insufflation in infants and children. *Anesthesiology*. 1993;78:652–656.
171. Salem MR, Wong AY, Mani M, Sellick BA. Efficacy of cricoid pressure in preventing gastric inflation during bag- mask ventilation in pediatric patients. *Anesthesiology*. 1974;40:96–98.
172. Salem MR, Wong AY, Fizzotti GF. Efficacy of cricoid pressure in preventing aspiration of gastric contents in paediatric patients. *Br J Anaesth*. 1972;44:401–404.

173. Salem MR, Joseph NJ, Heyman HJ, Belani B, Paulissian R, Ferrara TP. Cricoid compression is effective in obliterating the esophageal lumen in the presence of a nasogastric tube. *Anesthesiology*. 1985;63:443–446.
174. Ellis DY, Harris T, Zideman D. Cricoid pressure in emergency department rapid sequence tracheal intubations: a risk-benefit analysis. *Ann Emerg Med*. 2007;50:653–665.
175. Walker RW, Ravi R, Haylett K. Effect of cricoid force on airway calibre in children: a bronchoscopic assessment. *Br J Anaesth*. 2010;104:71–74.
176. Kitamura T, Iwami T, Kawamura T, Nagao K, Tanaka H, Nadkarni VM, Berg RA, Hiraide A. Conventional and chest-compression-only cardiopulmonary resuscitation by bystanders for children who have out-of-hospital cardiac arrests: a prospective, nationwide, population-based cohort study. *Lancet.* 2010;375:1347–1354.
177. Berg RA, Hilwig RW, Kern KB, Ewy GA. "Bystander" chest compressions and assisted ventilation independently improve outcome from piglet asphyxial pulseless "cardiac arrest." *Circulation*. 2000;101:1743–1748.
178. Iglesias JM, Lopez-Herce J, Urbano J, Solana MJ, Mencia S, Del Castillo J. Chest compressions versus ventilation plus chest compressions in a pediatric asphyxial cardiac arrest animal model. *Intensive Care Med*. 2010;36:712–716.
179. SOS-KANTO Study Group. Cardiopulmonary resuscitation by bystanders with chest compression only (SOS-Kanto): an observational study. *Lancet.* 2007;369:920–926.
180. Hallstrom A, Cobb L, Johnson E, Copass M. Cardiopulmonary resuscitation by chest compression alone or with mouth-to-mouth ventilation. *N Engl J Med*. 2000;342:1546–1553.
181. Iwami T, Kawamura T, Hiraide A, Berg RA, Hayashi Y, Nishiuchi T, Kajino K, Yonemoto N, Yukioka H, Sugimoto H, Kakuchi H, Sase K, Yokoyama H, Nonogi H. Effectiveness of bystander-initiated cardiac-only resuscitation for patients with out-of-hospital cardiac arrest. *Circulation*. 2007;116:2900–2907.
182. Ong ME, Ng FS, Anushia P, Tham LP, Leong BS, Ong VY, Tiah L, Lim SH, Anantharaman V. Comparison of chest compression only and standard cardiopulmonary resuscitation for out-of-hospital cardiac arrest in Singapore. *Resuscitation*. 2008;78:119–126.
183. Van Hoeyweghen RJ, Bossaert LL, Mullie A, Calle P, Martens P, Buylaert WA, Delooz H. Quality and efficiency of bystander CPR. Belgian cerebral resuscitation study group. *Resuscitation*. 1993;26:47–52.
184. Waalewijn RA, Tijssen JG, Koster RW. Bystander initiated actions in out-of-hospital cardiopulmonary resuscitation: results from the Amsterdam resuscitation study (ARRESUST). *Resuscitation*. 2001;50:273–279.
185. Berg RA, Kern KB, Sanders AB, Otto CW, Hilwig RW, Ewy GA. Bystander cardiopulmonary resuscitation. Is ventilation necessary? *Circulation*. 1993;88:1907–1915.
186. Chandra NC, Gruben KG, Tsitlik JE, Brower R, Guerci AD, Halperin HH, Weisfeldt ML, Permutt S. Observations of ventilation during resuscitation in a canine model. *Circulation*. 1994;90:3070–3075.
187. Berg RA, Wilcoxson D, Hilwig RW, Kern KB, Sanders AB, Otto CW, Eklund DK, Ewy GA. The need for ventilatory support during bystander CPR. *Ann Emerg Med*. 1995;26:342–350.
188. Engoren M, Plewa MC, Buderer NF, Hymel G, Brookfield L. Effects of simulated mouth-to-mouth ventilation during external cardiac compression or active compression-decompression in a swine model of witnessed cardiac arrest. *Ann Emerg Med*. 1997;29:607–615.
189. Berg RA, Kern KB, Hilwig RW, Ewy GA. Assisted ventilation during 'bystander' CPR in a swine acute myocardial infarction model does not improve outcome. *Circulation*. 1997;96:4364–4371.
190. Berg RA, Kern KB, Hilwig RW, Berg MD, Sanders AB, Otto CW, Ewy GA. Assisted ventilation does not improve outcome in a porcine model of single-rescuer bystander cardiopulmonary resuscitation. *Circulation*. 1997;95:1635–1641.
191. Kern KB, Hilwig RW, Berg RA, Ewy GA. Efficacy of chest compression-only BLS CPR in the presence of an occluded airway. *Resuscitation*. 1998;39:179–188.
192. Berg RA, Sanders AB, Kern KB, Hilwig RW, Heidenreich JW, Porter ME, Ewy GA. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. *Circulation*. 2001;104:2465–2470.
193. Sanders AB, Kern KB, Berg RA, Hilwig RW, Heidenrich J, Ewy GA. Survival and neurologic outcome after cardiopulmonary resuscitation with four different chest compression-ventilation ratios. *Ann Emerg Med*. 2002;40:553–562.
194. Dorph E, Wik L, Stromme TA, Eriksen M, Steen PA. Oxygen delivery and return of spontaneous circulation with ventilation:compression ratio 2:30 versus chest compressions only CPR in pigs. *Resuscitation*. 2004;60:309–318.
195. Bohm K, Rosenqvist M, Herlitz J, Hollenberg J, Svensson L. Survival is similar after standard treatment and chest compression only in out-of-hospital bystander cardiopulmonary resuscitation. *Circulation*. 2007;116:2908–2912.
196. Stevenson AG, McGowan J, Evans AL, Graham CA. CPR for children: one hand or two? *Resuscitation*. 2005;64:205–208.
197. Peska E, Kelly AM, Kerr D, Green D. One-handed versus two-handed chest compressions in paediatric cardio-pulmonary resuscitation. *Resuscitation*. 2006;71:65–69.
198. Udassi JP, Udassi S, Theriaque DW, Shuster JJ, Zaritsky AL, Haque IU. Effect of alternative chest compression techniques in infant and child on rescuer performance. *Pediatr Crit Care Med*. 2009;10:328–333.
199. Kao PC, Chiang WC, Yang CW, Chen SJ, Liu YP, Lee CC, Hsidh MJ, Ko PC, Chen SC, Ma MH. What is the correct depth of chest compression for infants and children? A radiological study. *Pediatrics*. 2009;124:49–55.
200. Sutton RM, Maltese MR, Niles D, French B, Nishisaki A, Arbogast KB, Donoghue A, Berg RA, Helfaer MA, Nadkarni V. Quantitative analysis of chest compression interruptions during in-hospital resuscitation of older children and adolescents. *Resuscitation*. 2009;80:1259–1263.
201. Braga MS, Dominguez TE, Pollock AN, Niles D, Meyer A, Myklebust H, Nysaether J, Nadkarni V. Estimation of optimal CPR chest compression depth in children by using computer tomography. *Pediatrics*. 2009;124:e69–e74.
202. Meyer A, Nadkarni V, Pollock A, Babbs C, Nishisaki A, Braga M, Berg RA, Ades A. Evaluation of the neonatal resuscitation program's recommended chest compression depth using computerized tomography imaging. *Resuscitation*. 2010;81:544–548.
203. Maguire S, Mann M, John N, Ellaway B, Sibert JR, Kemp AM. Does cardiopulmonary resuscitation cause rib fractures in children? A systematic review. *Child Abuse Negl*. 2006;30:739–751.
204. Dorph E, Wik L, Steen PA. Effectiveness of ventilation-compression ratios 1:5 and 2:15 in simulated single rescuer paediatric resuscitation. *Resuscitation*. 2002;54:259–264.
205. Greingor JL. Quality of cardiac massage with ratio compression-ventilation 5/1 and 15/2. *Resuscitation*. 2002;55:263–267.
206. Kinney SB, Tibballs J. An analysis of the efficacy of bag-valve-mask ventilation and chest compression during different compression-ventilation ratios in manikin-simulated paediatric resuscitation. *Resuscitation*. 2000;43:115–120.
207. Srikantan SK, Berg RA, Cox T, Tice L, Nadkarni VM. Effect of one-rescuer compression/ventilation ratios on cardiopulmonary resuscitation in infant, pediatric, and adult manikins. *Pediatr Crit Care Med*. 2005;6:293–297.
208. Betz AE, Callaway CW, Hostler D, Rittenberger JC. Work of CPR during two different compression to ventilation ratios with real-time feedback. *Resuscitation*. 2008;79:278–282.
209. Haque IU, Udassi JP, Udassi S, Theriaque DW, Shuster JJ, Zaritsky AL. Chest compression quality and rescuer fatigue with increased compression to ventilation ratio during single rescuer pediatric CPR. *Resuscitation*. 2008;79:82–89.
210. Bjorshol CA, Soreide E, Torsteinbo TH, Lexow K, Nilsen OB, Sunde K. Quality of chest compressions during 10 min of single-rescuer basic life support with different compression:ventilation ratios in a manikin model. *Resuscitation*. 2008;77:95–100.
211. Deschilder K, De Vos R, Stockman W. The effect on quality of chest compressions and exhaustion of a compression–ventilation ratio of 30:2 versus 15:2 during cardiopulmonary resuscitation–a randomised trial. *Resuscitation*. 2007;74:113–118.
212. Yannopoulos D, Aufderheide TP, Gabrielli A, Beiser DG, McKnite SH, Pirrallo RG, Wigginton J, Becker L, Vanden Hoek T, Tang W, Nadkarni VM, Klein JP, Idris AH, Lurie KG. Clinical and hemodynamic comparison of 15:2 and 30:2 compression-to-ventilation ratios for cardiopulmonary resuscitation. *Crit Care Med*. 2006;34:1444–1449.
213. Odegaard S, Saether E, Steen PA, Wik L. Quality of lay person CPR performance with compression:ventilation ratios 15:2, 30:2 or continuous chest compressions without ventilations on manikins. *Resuscitation*. 2006;71:335–340.

214. Hostler D, Rittenberger JC, Roth R, Callaway CW. Increased chest compression to ventilation ratio improves delivery of CPR. *Resuscitation*. 2007;74:446–452.
215. Kern KB, Hilwig RW, Berg RA, Sanders AB, Ewy GA. Importance of continuous chest compressions during cardiopulmonary resuscitation: improved outcome during a simulated single lay-rescuer scenario. *Circulation*. 2002;105:645–649.
216. Ewy GA, Zuercher M, Hilwig RW, Sanders AB, Berg RA, Otto CW, Hayes MM, Kern KB. Improved neurological outcome with continuous chest compressions compared with 30:2 compressions-to-ventilations cardiopulmonary resuscitation in a realistic swine model of out-of-hospital cardiac arrest. *Circulation*. 2007;116:2525–2530.
217. Assar D, Chamberlain D, Colquhoun M, Donnelly P, Handley AJ, Leaves S, Kern KB. Randomised controlled trials of staged teaching for basic life support, 1: skill acquisition at bronze stage. *Resuscitation*. 2000;45:7–15.
218. Heidenreich JW, Sanders AB, Higdon TA, Kern KB, Berg RA, Ewy GA. Uninterrupted chest compression CPR is easier to perform and remember than standard CPR. *Resuscitation*. 2004;63:123–130.
219. Valenzuela TD, Kern KB, Clark LL, Berg RA, Berg MD, Berg DD, Hilwig RW, Otto CW, Newburn D, Ewy GA. Interruptions of chest compressions during emergency medical systems resuscitation. *Circulation*. 2005;112:1259–1265.
220. Abella BS, Sandbo N, Vassilatos P, Alvarado JP, O'Hearn N, Wigder HN, Hoffman P, Tynus K, Vanden Hoek TL, Becker LB. Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest. *Circulation*. 2005;111:428–434.
221. Eftestol T, Sunde K, Steen PA. Effects of interrupting precordial compressions on the calculated probability of defibrillation success during out-of-hospital cardiac arrest. *Circulation*. 2002;105:2270–2273.
222. Yu T, Weil MH, Tang W, Sun S, Klouche K, Povoas H, Bisera J. Adverse outcomes of interrupted precordial compression during automated defibrillation. *Circulation*. 2002;106:368–372.
223. Kill C, Torossian A, Freisburger C, Dworok S, Massmann M, Nohl T, Henning R, Wallot P, Gockel A, Steinfeldt T, Graf J, Eberhart L, Wulf H. Basic life support with four different compression/ventilation ratios in a pig model: the need for ventilation. *Resuscitation*. 2009;80:1060–1065.
224. Lurie KG, Yannopoulos D, McKnite SH, Herman ML, Idris AH, Nadkarni VM, Tang W, Gabrielli A, Barnes TA, Metzger AK. Comparison of a 10-breaths-per-minute versus a 2-breaths-per-minute strategy during cardiopulmonary resuscitation in a porcine model of cardiac arrest. *Respir Care*. 2008;53:862–870.
225. Babbs CF, Nadkarni V. Optimizing chest compression to rescue ventilation ratios during one-rescuer CPR by professionals and lay persons: children are not just little adults. *Resuscitation*. 2004;61:173–181.
226. Babbs CF, Meyer A, Nadkarni V. Neonatal CPR: room at the top–a mathematical study of optimal chest compression frequency versus body size. *Resuscitation*. 2009;80:1280–1284.
227. Berkowitz ID, Chantarojanasiri T, Koehler RC, Schleien CL, Dean JM, Michael JR, Rogers MC, Traystman RJ. Blood flow during cardiopulmonary resuscitation with simultaneous compression and ventilation in infant pigs. *Pediatr Res*. 1989;26:558–564.
228. Hou SH, Lue HC, Chu SH. Comparison of conventional and simultaneous compression-ventilation cardiopulmonary resuscitation in piglets. *Jpn Circ J*. 1994;58:426–432.
229. Whyte SD, Sinha AK, Wyllie JP. Neonatal resuscitation–a practical assessment. *Resuscitation*. 1999;40:21–25.
230. Banerjee S, Singhi SC, Singh S, Singh M. The intraosseous route is a suitable alternative to intravenous route for fluid resuscitation in severely dehydrated children. *Indian Pediatr*. 1994;31:1511–1520.
231. Rosetti VA, Thompson BM, Miller J, Mateer JR, Aprahamian C. Intraosseous infusion: an alternative route of pediatric intravascular access. *Ann Emerg Med*. 1985;14:885–888.
232. Brunette DD, Fischer R. Intravascular access in pediatric cardiac arrest. *Am J Emerg Med*. 1988;6:577–579.
233. Seigler RS, Tecklenburg FW, Shealy R. Prehospital intraosseous infusion by emergency medical services personnel: a prospective study. *Pediatrics*. 1989;84:173–177.
234. Glaeser PW, Hellmich TR, Szewczuga D, Losek JD, Smith DS. Five-year experience in prehospital intraosseous infusions in children and adults. *Ann Emerg Med*. 1993;22:1119–1124.
235. Ellemunter H, Simma B, Trawoger R, Maurer H. Intraosseous lines in preterm and full term neonates. *Arch Dis Child Fetal Neonatal Ed*. 1999;80:F74–F75.
236. Claudet I, Baunin C, Laporte-Turpin E, Marcoux MO, Grouteau E, Cahuzac JP. Long-term effects on tibial growth after intraosseous infusion: a prospective, radiographic analysis. *Pediatr Emerg Care*. 2003;19:397–401.
237. Fiorito BA, Mirza F, Doran TM, Oberle AN, Cruz EC, Wendtland CL, Abd-Allah SA. Intraosseous access in the setting of pediatric critical care transport. *Pediatr Crit Care Med*. 2005;6:50–53.
238. Horton MA, Beamer C. Powered intraosseous insertion provides safe and effective vascular access for pediatric emergency patients. *Pediatr Emerg Care*. 2008;24:347–350.
239. Guay J, Lortie L. An evaluation of pediatric in-hospital advanced life support interventions using the pediatric Utstein guidelines: a review of 203 cardiorespiratory arrests. *Can J Anaesth*. 2004;51:373–378.
240. Niemann JT, Stratton SJ. Endotracheal versus intravenous epinephrine and atropine in out-of-hospital "primary" and postcountershock asystole. *Crit Care Med*. 2000;28:1815–1819.
241. Quinton DN, O'Byrne G, Aitkenhead AR. Comparison of endotracheal and peripheral intravenous adrenaline in cardiac arrest: is the endotracheal route reliable? *Lancet*. 1987;1:828–829.
242. Lindemann R. Resuscitation of the newborn. Endotracheal administration of epinephrine. *Acta Paediatr Scand*. 1984;73:210–212.
243. Barber CA, Wyckoff MH. Use and efficacy of endotracheal versus intravenous epinephrine during neonatal cardiopulmonary resuscitation in the delivery room. *Pediatrics*. 2006;118:1028–1034.
244. Efrati O, Barak A, Ben-Abraham R, Modan-Moses D, Berkovitch M, Manisterski Y, Lotan D, Barzilay Z, Paret G. Should vasopressin replace adrenaline for endotracheal drug administration? *Crit Care Med*. 2003;31:572–576.
245. Elizur A, Ben-Abraham R, Manisterski Y, Barak A, Efrati O, Lotan D, Barzilay Z, Paret G. Tracheal epinephrine or norepinephrine preceded by beta blockade in a dog model. Can beta blockade bestow any benefits? *Resuscitation*. 2003;59:271–276.
246. Manisterski Y, Vaknin Z, Ben-Abraham R, Efrati O, Lotan D, Berkovitch M, Barak A, Barzilay Z, Paret G. Endotracheal epinephrine: a call for larger doses. *Anesth Analg*. 2002;95:1037–1041.
247. Orlowski JP, Gallagher JM, Porembka DT. Endotracheal epinephrine is unreliable. *Resuscitation*. 1990;19:103–113.
248. Paret G, Vaknin Z, Ezra D, Peleg E, Rosenthal T, Vardi A, Mayan H, Barzilay Z. Epinephrine pharmacokinetics and pharmacodynamics following endotracheal administration in dogs: the role of volume of diluent. *Resuscitation*. 1997;35:77–82.
249. Vaknin Z, Manisterski Y, Ben-Abraham R, Efrati O, Lotan D, Barzilay Z, Paret G. Is endotracheal adrenaline deleterious because of the beta adrenergic effect? *Anesth Analg*. 2001;92:1408–1412.
250. Wenzel V, Lindner KH, Prengel AW, Lurie KG, Strohmenger HU. Endobronchial vasopressin improves survival during cardiopulmonary resuscitation in pigs. *Anesthesiology*. 1997;86:1375–1381.
251. Hornchen U, Schuttler J, Stoeckel H, Eichelkraut W, Hahn N. Endobronchial instillation of epinephrine during cardiopulmonary resuscitation. *Crit Care Med*. 1987;15:1037–1039.
252. Ralston SH, Tacker WA, Showen L, Carter A, Babbs CF. Endotracheal versus intravenous epinephrine during electromechanical dissociation with CPR in dogs. *Ann Emerg Med*. 1985;14:1044–1048.
253. Redding JS, Asuncion JS, Pearson JW. Effective routes of drug administration during cardiac arrest. *Anesth Analg*. 1967;46:253–258.
254. Yang LY, He CQ, Zhang ZG. Endotracheal administration of epinephrine during cardiopulmonary resuscitation. *Chin Med J (Engl)*. 1991;104:986–991.
255. Dalzell GW, Cunningham SR, Anderson J, Adgey AA. Electrode pad size, transthoracic impedance and success of external ventricular defibrillation. *Am J Cardiol*. 1989;64:741–744.
256. Atkins DL, Sirna S, Kieso R, Charbonnier F, Kerber RE. Pediatric defibrillation: importance of paddle size in determining transthoracic impedance. *Pediatrics*. 1988;82:914–918.
257. Atkins DL, Kerber RE. Pediatric defibrillation: current flow is improved by using "adult" electrode paddles. *Pediatrics*. 1994;94:90–93.
258. Samson RA, Atkins DL, Kerber RE. Optimal size of self-adhesive preapplied electrode pads in pediatric defibrillation. *Am J Cardiol*. 1995;75:544–545.
259. Kerber RE, Grayzel J, Hoyt R, Marcus M, Kennedy J. Transthoracic resistance in human defibrillation. Influence of body weight, chest size,

serial shocks, paddle size and paddle contact pressure. *Circulation*. 1981;63:676–682.
260. Pagan-Carlo LA, Spencer KT, Robertson CE, Dengler A, Birkett C, Kerber RE. Transthoracic defibrillation: importance of avoiding electrode placement directly on the female breast. *J Am Coll Cardiol*. 1996;27:449–452.
261. Hoyt R, Grayzel J, Kerber RE. Determinants of intracardiac current in defibrillation. Experimental studies in dogs. *Circulation*. 1981;64:818–823.
262. Pagan-Carlo LA, Birkett CL, Smith RA, Kerber RE. Is there an optimal electrode pad size to maximize intracardiac current in transthoracic defibrillation? *Pacing Clin Electrophysiol*. 1997;20:283–292.
263. Killingsworth CR, Melnick SB, Chapman FW, Walker RG, Smith WM, Ideker RE, Walcott GP. Defibrillation threshold and cardiac responses using an external biphasic defibrillator with pediatric and adult adhesive patches in pediatric-sized piglets. *Resuscitation*. 2002;55:177–185.
264. Tibballs J, Carter B, Kiraly NJ, Ragg P, Clifford M. External and internal biphasic direct current shock doses for pediatric ventricular fibrillation and pulseless ventricular tachycardia. *Pediatr Crit Care Med.* 2010.
265. Garcia LA, Kerber RE. Transthoracic defibrillation: does electrode adhesive pad position alter transthoracic impedance? *Resuscitation*. 1998;37:139–143.
266. Dodd TE, Deakin CD, Petley GW, Clewlow F. External defibrillation in the left lateral position–a comparison of manual paddles with self-adhesive pads. *Resuscitation*. 2004;63:283–286.
267. Caterine MR, Yoerger DM, Spencer KT, Miller SG, Kerber RE. Effect of electrode position and gel-application technique on predicted transcardiac current during transthoracic defibrillation. *Ann Emerg Med*. 1997;29:588–595.
268. Deakin CD, Sado DM, Petley GW, Clewlow F. Is the orientation of the apical defibrillation paddle of importance during manual external defibrillation? *Resuscitation*. 2003;56:15–18.
269. Stults KR, Brown DD, Cooley F, Kerber RE. Self-adhesive monitor/defibrillation pads improve prehospital defibrillation success. *Ann Emerg Med*. 1987;16:872–877.
270. Kirchhof P, Monnig G, Wasmer K, Heinecke A, Breithardt G, Eckardt L, Bocker D. A trial of self-adhesive patch electrodes and hand-held paddle electrodes for external cardioversion of atrial fibrillation (MOBIPAPA). *Eur Heart J*. 2005;26:1292–1297.
271. Jakobsson J, Odmansson I, Nordlander R. Comparison of two different electrodes for the delivery of DC-shocks. *Resuscitation*. 1990;20:25–29.
272. Deakin CD, McLaren RM, Petley GW, Clewlow F, Dalrymple-Hay MJ. A comparison of transthoracic impedance using standard defibrillation paddles and self-adhesive defibrillation pads. *Resuscitation*. 1998;39:43–46.
273. Kerber RE, Martins JB, Kelly KJ, Ferguson DW, Kouba C, Jensen SR, Newman B, Parke JD, Kieso R, Melton J. Self-adhesive preapplied electrode pads for defibrillation and cardioversion. *J Am Coll Cardiol*. 1984;3:815–820.
274. Ewy GA, Horan WJ, Ewy MD. Disposable defibrillator electrodes. *Heart Lung*. 1977;6:127–130.
275. Kerber RE, Martins JB, Ferguson DW, Jensen SR, Parke JD, Kieso R, Melton J. Experimental evaluation and initial clinical application of new self-adhesive defibrillation electrodes. *Int J Cardiol*. 1985;8:57–66.
276. Cornwell L, Mukherjee R, Kelsall AW. Problems with the use of self-adhesive electrode pads in neonates. *Resuscitation*. 2006;68:425–428.
277. Bradbury N, Hyde D, Nolan J. Reliability of ECG monitoring with a gel pad/paddle combination after defibrillation. *Resuscitation*. 2000;44:203–206.
278. Perkins GD, Roberts C, Gao F. Delays in defibrillation: influence of different monitoring techniques. *Br J Anaesth*. 2002;89:405–408.
279. Perkins GD, Davies RP, Soar J, Thickett DR. The impact of manual defibrillation technique on no-flow time during simulated cardiopulmonary resuscitation. *Resuscitation*. 2007;73:109–114.
280. Mittal S, Ayati S, Stein KM, Knight BP, Morady F, Schwartzman D, Cavlovich D, Platia EV, Calkins H, Tchou PJ, Miller JM, Wharton JM, Sung RJ, Slotwiner DJ, Markowitz SM, Lerman BB. Comparison of a novel rectilinear biphasic waveform with a damped sine wave monophasic waveform for transthoracic ventricular defibrillation. Zoll investigators. *J Am Coll Cardiol*. 1999;34:1595–1601.
281. van Alem AP, Chapman FW, Lank P, Hart AA, Koster RW. A prospective, randomised and blinded comparison of first shock success of monophasic and biphasic waveforms in out-of-hospital cardiac arrest. *Resuscitation*. 2003;58:17–24.
282. Rea TD, Helbock M, Perry S, Garcia M, Cloyd D, Becker L, Eisenberg M. Increasing use of cardiopulmonary resuscitation during out-of-hospital ventricular fibrillation arrest: survival implications of guideline changes. *Circulation*. 2006;114:2760–2765.
283. Menegazzi JJ, Hsieh M, Niemann JT, Swor RA. Derivation of clinical predictors of failed rescue shock during out-of-hospital ventricular fibrillation. *Prehosp Emerg Care*. 2008;12:347–351.
284. Rea TD, Shah S, Kudenchuk PJ, Copass MK, Cobb LA. Automated external defibrillators: to what extent does the algorithm delay CPR? *Ann Emerg Med*. 2005;46:132–141.
285. Becker L, Gold LS, Eisenberg M, White L, Hearne T, Rea T. Ventricular fibrillation in King County, Washington: a 30-year perspective. *Resuscitation*. 2008;79:22–27.
286. Rossano JW, Quan L, Kenney MA, Rea TD, Atkins DL. Energy doses for treatment of out-of-hospital pediatric ventricular fibrillation. *Resuscitation*. 2006;70:80–89.
287. Berg MD, Samson RA, Meyer RJ, Clark LL, Valenzuela TD, Berg RA. Pediatric defibrillation doses often fail to terminate prolonged out-of-hospital ventricular fibrillation in children. *Resuscitation*. 2005;67:63–67.
288. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Bellon JM. Pediatric defibrillation after cardiac arrest: initial response and outcome. *Crit Care*. 2006;10:R113.
289. Atkins DL, Hartley LL, York DK. Accurate recognition and effective treatment of ventricular fibrillation by automated external defibrillators in adolescents. *Pediatrics*. 1998;101:393–397.
290. Berg RA, Chapman FW, Berg MD, Hilwig RW, Banville I, Walker RG, Nova RC, Sherrill D, Kern KB. Attenuated adult biphasic shocks compared with weight-based monophasic shocks in a swine model of prolonged pediatric ventricular fibrillation. *Resuscitation*. 2004;61:189–197.
291. Clark CB, Zhang Y, Davies LR, Karlsson G, Kerber RE. Pediatric transthoracic defibrillation: biphasic versus monophasic waveforms in an experimental model. *Resuscitation*. 2001;51:159–163.
292. Berg MD, Banville IL, Chapman FW, Walker RG, Gaballa MA, Hilwig RW, Samson RA, Kern KB, Berg RA. Attenuating the defibrillation dosage decreases postresuscitation myocardial dysfunction in a swine model of pediatric ventricular fibrillation. *Pediatr Crit Care Med*. 2008;9:429–434.
293. Berg RA, Samson RA, Berg MD, Chapman FW, Hilwig RW, Banville I, Walker RG, Nova RC, Anavy N, Kern KB. Better outcome after pediatric defibrillation dosage than adult dosage in a swine model of pediatric ventricular fibrillation. *J Am Coll Cardiol*. 2005;45:786–789.
294. Tang W, Weil MH, Jorgenson D, Klouche K, Morgan C, Yu T, Sun S, Snyder D. Fixed-energy biphasic waveform defibrillation in a pediatric model of cardiac arrest and resuscitation. *Crit Care Med*. 2002;30:2736–2741.
295. Walcott GP, Melnick SB, Killingsworth CR, Ideker RE. Comparison of low-energy versus high-energy biphasic defibrillation shocks following prolonged ventricular fibrillation. *Prehosp Emerg Care*. 2010;14:62–70.
296. Kudenchuk PJ, Cobb LA, Copass MK, Cummins RO, Doherty AM, Fahrenbruch CE, Hallstrom AP, Murray WA, Olsufka M, Walsh T. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. *N Engl J Med*. 1999;341:871–878.
297. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. *N Engl J Med*. 2002;346:884–890.
298. Perry JC, Fenrich AL, Hulse JE, Triedman JK, Friedman RA, Lamberti JJ. Pediatric use of intravenous amiodarone: efficacy and safety in critically ill patients from a multicenter protocol. *J Am Coll Cardiol*. 1996;27:1246–1250.
299. Perry JC, Knilans TK, Marlow D, Denfield SW, Fenrich AL, Friedman RA. Intravenous amiodarone for life-threatening tachyarrhythmias in children and young adults. *J Am Coll Cardiol*. 1993;22:95–98.
300. Atkins DL, Everson-Stewart S, Sears GK, Daya M, Osmond MH, Warden CR, Berg RA. Epidemiology and outcomes from out-of-hospital cardiac arrest in children: the Resuscitation Outcomes Consortium Epistry-cardiac arrest. *Circulation*. 2009;119:1484–1491.
301. Samson RA, Nadkarni VM, Meaney PA, Carey SM, Berg MD, Berg RA. Outcomes of in-hospital ventricular fibrillation in children. *N Engl J Med*. 2006;354:2328–2339.
302. Cecchin F, Jorgenson DB, Berul CI, Perry JC, Zimmerman AA, Duncan BW, Lupinetti FM, Snyder D, Lyster TD, Rosenthal GL, Cross B,

Atkins DL. Is arrhythmia detection by automatic external defibrillator accurate for children? Sensitivity and specificity of an automatic external defibrillator algorithm in 696 pediatric arrhythmias. *Circulation*. 2001;103:2483–2488.
303. Atkins DL, Scott WA, Blaufox AD, Law IH, Dick M II, Geheb F, Sobh J, Brewer JE. Sensitivity and specificity of an automated external defibrillator algorithm designed for pediatric patients. *Resuscitation*. 2008; 76:168–174.
304. Atkinson E, Mikysa B, Conway JA, Parker M, Christian K, Deshpande J, Knilans TK, Smith J, Walker C, Stickney RE, Hampton DR, Hazinski MF. Specificity and sensitivity of automated external defibrillator rhythm analysis in infants and children. *Ann Emerg Med*. 2003;42: 185–196.
305. Babbs CF, Tacker WA, VanVleet JF, Bourland JD, Geddes LA. Therapeutic indices for transchest defibrillator shocks: effective, damaging, and lethal electrical doses. *Am Heart J*. 1980;99:734–738.
306. Gaba DM, Talner NS. Myocardial damage following transthoracic direct current countershock in newborn piglets. *Pediatr Cardiol*. 1982;2: 281–288.
307. Berg RA. Attenuated adult biphasic shocks for prolonged pediatric ventricular fibrillation: support for pediatric automated defibrillators. *Crit Care Med*. 2004;32:S352–355.
308. Bar-Cohen Y, Walsh EP, Love BA, Cecchin F. First appropriate use of automated external defibrillator in an infant. *Resuscitation*. 2005;67: 135–137.
309. Divekar A, Soni R. Successful parental use of an automated external defibrillator for an infant with long-QT syndrome. *Pediatrics*. 2006;118: e526–e529.
310. Gurnett CA, Atkins DL. Successful use of a biphasic waveform automated external defibrillator in a high-risk child. *Am J Cardiol*. 2000;86:1051–1053.
311. Konig B, Benger J, Goldsworthy L. Automatic external defibrillation in a 6 year old. *Arch Dis Child*. 2005;90:310–311.
312. Desanctis RW. Electrical conversion of ventricualar tachycardia. *JAMA*. 1965;191:632–636.
313. Domanovits H, Paulis M, Nikfardjam M, Holzer M, Stuhlinger HG, Hirschl MM, Laggner AN. Sustained ventricular tachycardia in the emergency department. *Resuscitation*. 1999;42:19–25.
314. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. *Eur J Pediatr*. 2003; 162:880–884.
315. Drago F, Mazza A, Guccione P, Mafrici A, Di Liso G, Ragonese P. Amiodarone used alone or in combination with propranolol: a very effective therapy for tachyarrhythmias in infants and children. *Pediatric Cardiology*. 1998;19:445–449.
316. Saul JP, Scott WA, Brown S, Marantz P, Acevedo V, Etheridge SP, Perry JC, Triedman JK, Burriss SW, Cargo P, Graepel J, Koskelo EK, Wang R. Intravenous amiodarone for incessant tachyarrhythmias in children: a randomized, double-blind, antiarrhythmic drug trial. *Circulation*. 2005;112:3470–3477.
317. Dilber E, Mutlu M, Dilber B, Aslan Y, Gedik Y, Celiker A. Intravenous amiodarone used alone or in combination with digoxin for life-threatening supraventricular tachyarrhythmia in neonates and small infants. *Pediatr Emerg Care*. 2010;26:82–84.
318. Balaguer Gargallo M, Jordan Garcia I, Caritg Bosch J, Cambra Lasaosa FJ, Prada Hermogenes F, Palomaque Rico A. [supraventricular tachycardia in infants and children]. *An Pediatr (Barc)*. 2007;67: 133–138.
319. Dixon J, Foster K, Wyllie J, Wren C. Guidelines and adenosine dosing in supraventricular tachycardia. *Arch Dis Child*. 2005;90:1190–1191.
320. Moghaddam M, Mohammad Dalili S, Emkanjoo Z. Efficacy of adenosine for acute treatment of supraventricular tachycardia in infants and children. *J Teh Univ Heart Ctr*. 2008;3:157–162.
321. Van der Merwe DM, Van der Merwe PL. Supraventricular tachycardia in children. *Cardiovasc J S Afr*. 2004;15:64–69.
322. Losek JD, Endom E, Dietrich A, Stewart G, Zempsky W, Smith K. Adenosine and pediatric supraventricular tachycardia in the emergency department: multicenter study and review. *Ann Emerg Med*. 1999;33: 185–191.
323. Koh E, Chan I, Wong KY. Five paediatric case reports of the use of adenosine in supraventricular tachycardia. *Ann Acad Med Singapore*. 1998;27:363–365.
324. Sherwood MC, Lau KC, Sholler GF. Adenosine in the management of supraventricular tachycardia in children. *J Paediatr Child Health*. 1998; 34:53–56.
325. Dimitriu AG, Nistor N, Russu G, Cristogel F, Streanga V, Varlam L. Value of intravenous ATP in the diagnosis and treatment of tachyarrhythmias in children. *Rev Med Chir Soc Med Nat Iasi*. 1998; 102:100–102.
326. Bakshi F, Barzilay Z, Paret G. Adenosine in the diagnosis and treatment of narrow complex tachycardia in the pediatric intensive care unit. *Heart Lung*. 1998;27:47–50.
327. Lenk M, Celiker A, Alehan D, Kocak G, Ozme S. Role of adenosine in the diagnosis and treatment of tachyarrhythmias in pediatric patients. *Acta Paediatr Jpn*. 1997;39:570–577.
328. Paret G, Steinmetz D, Kuint J, Hegesh J, Frand M, Barzilay Z. Adenosine for the treatment of paroxysmal supraventricular tachycardia in full-term and preterm newborn infants. *Am J Perinatol*. 1996;13: 343–346.
329. Pfammatter JP, Paul T, Bachmann D, Weber JW, Stocker FP, Kallfelz HC. [therapeutic efficacy and diagnostic potential of adenosine in infants and children]. *Z Kardiol*. 1995;84:243–249.
330. De Wolf D, Rondia G, Verhaaren H, Matthys D. Adenosine-triphosphate treatment for supraventricular tachycardia in infants. *Eur J Pediatr*. 1994;153:793–796.
331. Muller G, Deal BJ, Benson DW Jr. "Vagal maneuvers" and adenosine for termination of atrioventricular reentrant tachycardia. *Am J Cardiol*. 1994;74:500–503.
332. Crosson JE, Etheridge SP, Milstein S, Hesslein PS, Dunnigan A. Therapeutic and diagnostic utility of adenosine during tachycardia evaluation in children. *Am J Cardiol*. 1994;74:155–160.
333. Ralston MA, Knilans TK, Hannon DW, Daniels SR. Use of adenosine for diagnosis and treatment of tachyarrhythmias in pediatric patients. *J Pediatr*. 1994;124:139–143.
334. Reyes G, Stanton R, Galvis AG. Adenosine in the treatment of paroxysmal supraventricular tachycardia in children. *Ann Emerg Med*. 1992;21:1499–1501.
335. Rossi AF, Steinberg LG, Kipel G, Golinko RJ, Griepp RB. Use of adenosine in the management of perioperative arrhythmias in the pediatric cardiac intensive care unit. *Crit Care Med*. 1992;20: 1107–1111.
336. Till J, Shinebourne EA, Rigby ML, Clarke B, Ward DE, Rowland E. Efficacy and safety of adenosine in the treatment of supraventricular tachycardia in infants and children. *Br Heart J*. 1989;62:204–211.
337. Overholt ED, Rheuban KS, Gutgesell HP, Lerman BB, DiMarco JP. Usefulness of adenosine for arrhythmias in infants and children. *Am J Cardiol*. 1988;61:336–340.
338. Clarke B, Till J, Rowland E, Ward DE, Barnes PJ, Shinebourne EA. Rapid and safe termination of supraventricular tachycardia in children by adenosine. *Lancet*. 1987;1:299–301.
339. Jaeggi E, Chiu C, Hamilton R, Gilljam T, Gow R. Adenosine-induced atrial pro-arrhythmia in children. *Can J Cardiol*. 1999;15:169–172.
340. Riccardi A, Arboscello E, Ghinatti M, Minuto P, Lerza R. Adenosine in the treatment of supraventricular tachycardia: 5 years of experience (2002–2006). *Am J Emerg Med*. 2008;26:879–882.
341. Ertan C, Atar I, Gulmez O, Atar A, Ozgul A, Aydinalp A, Muderrisoglu H, Ozin B. Adenosine-induced ventricular arrhythmias in patients with supraventricular tachycardias. *Ann Noninvasive Electrocardiol*. 2008; 13:386–390.
342. Tan H, Spekhorst H, Peters R, Wilde A. Adenosine induced ventricular arrhythmias in the emergency room. *Pacing Clin Electrophysiol*. 2001; 24:450–455.
343. Glatter KA, Cheng J, Dorostkar P, Modin G, Talwar S, Al-Nimri M, Lee RJ, Saxon LA, Lesh MD, Scheinman MM. Electrophysiologic effects of adenosine in patients with supraventricular tachycardia. *Circulation*. 1999;99:1034–1040.
344. Greco R, Musto B, Arienzo V, Alborino A, Garofalo S, Marsico F. Treatment of paroxysmal supraventricular tachycardia in infancy with digitalis, adenosine-5′-triphosphate, and verapamil: a comparative study. *Circulation*. 1982;66:504–508.
345. Lim SH, Anantharaman V, Teo WS, Chan YH. Slow infusion of calcium channel blockers compared with intravenous adenosine in the emergency treatment of supraventricular tachycardia. *Resuscitation*. 2009;80:523–528.
346. Holdgate A, Foo A. Adenosine versus intravenous calcium channel antagonists for the treatment of supraventricular tachycardia in adults. *Cochrane Database Syst Rev*. 2006:CD005154.
347. Haas NA, Camphausen CK. Acute hemodynamic effects of intravenous amiodarone treatment in pediatric patients with cardiac surgery. *Clin Res Cardiol*. 2008;97:801–810.

348. Valsangiacomo E, Schmid ER, Schüpbach RW, Schmidlin D, Molinari L, Waldvogel K, Bauersfeld U. Early postoperative arrhythmias after cardiac operation in children. *Ann Thorac Surg*. 2002;74:792–796.
349. Laird WP, Snyder CS, Kertesz NJ, Friedman RA, Miller D, Fenrich AL. Use of intravenous amiodarone for postoperative junctional ectopic tachycardia in children. *Pediatr Cardiol*. 2003;24:133–137.
350. Hoffman TM, Bush DM, Wernovsky G, Cohen MI, Wieand TS, Gaynor JW, Spray TL, Rhodes LA. Postoperative junctional ectopic tachycardia in children: incidence, risk factors, and treatment. *Ann Thorac Surg*. 2002;74:1607–1611.
351. Juneja R, Shah S, Naik N, Kothari SS, Saxena A, Talwar KK. Management of cardiomyopathy resulting from incessant supraventricular tachycardia in infants and children. *Indian Heart J*. 2002;54:176–180.
352. Cabrera Duro A, Rodrigo Carbonero D, Galdeano Miranda J, Martinez Corrales P, Pastor Menchaca E, Macua Biurrun P, Pilar Orive J. [the treatment of postoperative junctional ectopic tachycardia]. *An Esp Pediatr*. 2002;56:505–509.
353. Dodge-Khatami A, Miller O, Anderson R, Gil-Jaurena J, Goldman A, de Leval M. Impact of junctional ectopic tachycardia on postoperative morbidity following repair of congenital heart defects. *Eur J Cardiothorac Surg*. 2002;21:255–259.
354. Michael JG, Wilson WR Jr, Tobias JD. Amiodarone in the treatment of junctional ectopic tachycardia after cardiac surgery in children: report of two cases and review of the literature. *Am J Ther*. 1999;6:223–227.
355. Celiker A, Ceviz N, Ozme S. Effectiveness and safety of intravenous amiodarone in drug-resistant tachyarrhythmias of children. *Acta Paediatr Jpn*. 1998;40:567–572.
356. Soult JA, Munoz M, Lopez JD, Romero A, Santos J, Tovaruela A. Efficacy and safety of intravenous amiodarone for short-term treatment of paroxysmal supraventricular tachycardia in children. *Pediatr Cardiol*. 1995;16:16–19.
357. Figa FH, Gow RM, Hamilton RM, Freedom RM. Clinical efficacy and safety of intravenous amiodarone in infants and children. *Am J Cardiol*. 1994;74:573–577.
358. Ng GY, Hampson Evans DC, Murdoch LJ. Cardiovascular collapse after amiodarone administration in neonatal supraventicular tachycardia. *Eur J Emerg Med*. 2003;10:323–325.
359. Daniels CJ, Schutte DA, Hammond S, Franklin WH. Acute pulmonary toxicity in an infant from intravenous amiodarone. *Am J Cardiol*. 1997;80:1113–1116.
360. Yap S-C, Hoomtje T, Sreeram N. Polymorphic ventricular tachycardia after use of intravenous amiodarone for postoperative junctional ectopic tachycardia. *Int J Cardiol*. 2000;76:245–247.
361. Deleted.
362. Gandy J, Wonko N, Kantoch MJ. Risks of intravenous amiodarone in neonates. *Can J Cardiol*. 1998;14:855–858.
363. Chang PM, Silka MJ, Moromisato DY, Bar-Cohen Y. Amiodarone versus procainamide for the acute treatment of recurrent supraventricular tachycardia in pediatric patients. *Circ Arrhythm Electrophysiol*. 2010;3:134–140.
364. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. *J Formos Med Assoc*. 2000;99:766–770.
365. Mandapati R, Byrum CJ, Kavey RE, Smith FC, Kveselis DA, Hannan WP, Brandt B III, Gaum WE. Procainamide for rate control of postsurgical junctional tachycardia. *Pediatr Cardiol*. 2000;21:123–128.
366. Walsh EP, Saul JP, Sholler GF, Triedman JK, Jonas RA, Mayer JE, Wessel DL. Evaluation of a staged treatment protocol for rapid automatic junctional tachycardia after operation for congenital heart disease. *J Am Coll Cardiol*. 1997;29:1046–1053.
367. Rhodes LA, Walsh EP, Saul JP. Conversion of atrial flutter in pediatric patients by transesophageal atrial pacing: a safe, effective, minimally invasive procedure. *Am Heart J*. 1995;130:323–327.
368. Benson DJ, Dunnigan A, Green T, Benditt D, Schneider S. Periodic procainamide for paroxysmal tachycardia. *Circulation*. 1985;72:147–152.
369. Gouin S, Ali S. A patient with chaotic atrial tachycardia. *Pediatr Emerg Care*. 2003;19:95–98.
370. Azzam F, Fiore A. Postoperative junctional ectopic tachycardia. *Can J Anaesth*. 1998;45:898–902.
371. Wu MH, Wang JK, Lin JL, Lai LP, Lue HC, Young ML, Hsieh FJ. Supraventricular tachycardia in patients with right atrial isomerism. *J Am Coll Cardiol*. 1998;32:773–779.
372. Dodo H, Gow RM, Hamilton RM, Freedom RM. Chaotic atrial rhythm in children. *Am Heart J*. 1995;129:990–995.
373. Cowan R, Waldo A, Harris H, Cassady G, Brans Y. Neonatal paroxysmal supraventricular tachycardia with hydrops. *Pediatrics*. 1975;55:428–430.
374. Karlsson E, Sonnhag C. Haemodynamic effects of procainamide and phenytoin at apparent therapeutic plasma levels. *Eur J Clin Pharmacol*. 1976;10:305–310.
375. Singh BN, Kehoe R, Woosley RL, Scheinman M, Quart B. Multicenter trial of sotalol compared with procainamide in the suppression of inducible ventricular tachycardia: a double-blind, randomized parallel evaluation. Sotalol Multicenter Study Group. *Am Heart J*. 1995;129:87–97.
376. Jawad-Kanber G, Sherrod TR. Effect of loading dose of procaine amide on left ventricular performance in man. *Chest*. 1974;66:269–272.
377. Shih JY, Gillette PC, Kugler JD, Garson A Jr, Fukushige J, Zinner A, Driscoll DJ. The electrophysiologic effects of procainamide in the immature heart. *Pediatr Pharmacol (New York)*. 1982;2:65–73.
378. Bein G, Wolf D. The treatment of supraventricular tachycardia in infants and children with verapamil. *Cardiol Pneumol*. 1971;9:151.
379. Soler-Soler J, Sagrista-Sauleda J, Cabrera A, Sauleda-Pares J, Iglesias-Berengue J, Permanyer-Miralda G, Roca-Llop J. Effect of verapamil in infants with paroxysmal supraventricular tachycardia. *Circulation*. 1979;59:876–879.
380. Leitner RP, Hawker RE, Celermajer JM. Intravenous verapamil in the treatment of paroxysmal supraventricular tachycardia in children. *Aust Paediatr J*. 1983;19:40–44.
381. Wu MH, Chang YC, Lin JL, Young ML, Wang JK, Lue HC. Probability of supraventricular tachycardia recurrence in pediatric patients. *Cardiology*. 1994;85:284–289.
382. Kirk CR, Gibbs JL, Thomas R, Radley-Smith R, Qureshi SA. Cardiovascular collapse after verapamil in supraventricular tachycardia. *Arch Dis Child*. 1987;62:1265–1266.
383. Garland JS, Berens RJ, Losek JD, Wilson AD. An infant fatality following verapamil therapy for supraventricular tachycardia: cardiovascular collapse following intravenous verapamil. *Pediatr Emerg Care*. 1985;1:198–200.
384. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. *Arch Dis Child*. 1990;65:127–129.
385. Adamson PC, Rhodes LA, Saul JP, Dick M II, Epstein MR, Moate P, Boston R, Schreiner MS. The pharmacokinetics of esmolol in pediatric subjects with supraventricular arrhythmias. *Pediatr Cardiol*. 2006;27:420–427.
386. Chrysostomou C, Beerman L, Shiderly D, Berry D, Morell VO, Munoz R. Dexmedetomidine: A novel drug for the treatment of atrial and junctional tachyarrhythmias during the perioperative period for congenital cardiac surgery: a preliminary study. *Anesth Analg*. 2008;107:1514–1522.
387. Bickell WH, Wall MJ Jr, Pepe PE, Martin RR, Ginger VF, Allen MK, Mattox KL. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. *N Engl J Med*. 1994;331:1105–1109.
388. Dunham CM, Belzberg H, Lyles R, Weireter L, Skurdal D, Sullivan G, Esposito T, Namini M. The rapid infusion system: a superior method for the resuscitation of hypovolemic trauma patients. *Resuscitation*. 1991;21:207–227.
389. Dutton RP, Mackenzie CF, Scalea TM. Hypotensive resuscitation during active hemorrhage: impact on in-hospital mortality. *J Trauma*. 2002;52:1141–1146.
390. Hambly PR, Dutton RP. Excess mortality associated with the use of a rapid infusion system at a level 1 trauma center. *Resuscitation*. 1996;31:127–133.
391. Kwan I, Bunn F, Roberts I. Timing and volume of fluid administration for patients with bleeding. *Cochrane Database Syst Rev*. 2003:CD002245.
392. Mattox KL, Maningas PA, Moore EE, Mateer JR, Marx JA, Aprahamian C, Burch JM, Pepe PE. Prehospital hypertonic saline/dextran infusion for post-traumatic hypotension. The U.S.A. Multicenter trial. *Ann Surg*. 1991;213:482–491.
393. Sampalis JS, Tamim H, Denis R, Boukas S, Ruest SA, Nikolis A, Lavoie A, Fleiszer D, Brown R, Mulder D, Williams JI. Ineffectiveness of on-site intravenous lines: is prehospital time the culprit? *J Trauma.* 1997;43:608–615; discussion 615–617.
394. Turner J, Nicholl J, Webber L, Cox H, Dixon S, Yates D. A randomised controlled trial of prehospital intravenous fluid replacement therapy in serious trauma. *Health Technol Assess*. 2000;4:1–57.

395. Wade CE, Grady JJ, Kramer GC. Efficacy of hypertonic saline dextran fluid resuscitation for patients with hypotension from penetrating trauma. *J Trauma*. 2003;54:S144–S148.
396. Hussain SN, Roussos C. Distribution of respiratory muscle and organ blood flow during endotoxic shock in dogs. *J Appl Physiol*. 1985;59: 1802–1808.
397. Tang W, Pakula JL, Weil MH, Noc M, Fukui M, Bisera J. Adrenergic vasopressor agents and mechanical ventilation for the treatment of experimental septic shock. *Crit Care Med*. 1996;24:125–130.
398. Viires N, Sillye G, Aubier M, Rassidakis A, Roussos C. Regional blood flow distribution in dog during induced hypotension and low cardiac output. Spontaneous breathing versus artificial ventilation. *J Clin Invest*. 1983;72:935–947.
399. Ho AM, Graham CA, Ng CS, Yeung JH, Dion PW, Critchley LA, Karmakar MK. Timing of tracheal intubation in traumatic cardiac tamponade: a word of caution. *Resuscitation*. 2009;80:272–274.
400. Ledingham IM, McArdle CS. Prospective study of the treatment of septic shock. *Lancet*. 1978;1:1194–1197.
401. Griffel MI, Astiz ME, Rackow EC, Weil MH. Effect of mechanical ventilation on systemic oxygen extraction and lactic acidosis during early septic shock in rats. *Crit Care Med*. 1990;18:72–76.
402. Dung NM, Day NPJ, Tam DTH, Loan HT, Chau HTT, Minh LN, Diet TV, Bethell DB, Kneen R, Hien TT, White NJ, Farrar JJ. Fluid replacement in dengue shock syndrome: a randomized, double-blind comparison of four intravenous-fluid regimens. *Clin Infect Dis*. 1999; 29:787–794.
403. Ngo NT, Cao XT, Kneen R, Wills B, Nguyen VM, Nguyen TQ, Chu VT, Nguyen TT, Simpson JA, Solomon T, White NJ, Farrar J. Acute management of dengue shock syndrome: a randomized double-blind comparison of 4 intravenous fluid regimens in the first hour. *Clin Infect Dis*. 2001;32:204–213.
404. Wills BA, Nguyen MD, Ha TL, Dong TH, Tran TN, Le TT, Tran VD, Nguyen TH, Nguyen VC, Stepniewska K, White NJ, Farrar JJ. Comparison of three fluid solutions for resuscitation in dengue shock syndrome. *N Engl J Med*. 2005;353:877–889.
405. Upadhyay M, Singhi S, Murlidharan J, Kaur N, Majumdar S. Randomized evaluation of fluid resuscitation with crystalloid (saline) and colloid (polymer from degraded gelatin in saline) in pediatric septic shock. *Indian Pediatr*. 2005;42:223–231.
406. Finfer S, Bellomo R, Boyce N, French J, Myburgh J, Norton R. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med*. 2004;350:2247–2256.
407. Alderson P, Bunn F, Lefebvre C, Li WP, Li L, Roberts I, Schierhout G. Human albumin solution for resuscitation and volume expansion in critically ill patients. *Cochrane Database Syst Rev*. 2004:CD001208.
408. Bulger EM, Jurkovich GJ, Nathens AB, Copass MK, Hanson S, Cooper C, Liu PY, Neff M, Awan AB, Warner K, Maier RV. Hypertonic resuscitation of hypovolemic shock after blunt trauma: a randomized controlled trial. *Arch Surg*. 2008;143:139–148; discussion 149.
409. Choi PT, Yip G, Quinonez LG, Cook DJ. Crystalloids vs. colloids in fluid resuscitation: a systematic review. *Crit Care Med*. 1999;27: 200–210.
410. Cooper DJ, Myles PS, McDermott FT, Murray LJ, Laidlaw J, Cooper G, Tremayne AB, Bernard SS, Ponsford J. Prehospital hypertonic saline resuscitation of patients with hypotension and severe traumatic brain injury: a randomized controlled trial. *JAMA*. 2004;291:1350–1357.
411. Perel P, Roberts I. Colloids versus crystalloids for fluid resuscitation in critically ill patients. *Cochrane Database Syst Rev*. 2007:CD000567.
412. Wilkes MM, Navickis RJ. Patient survival after human albumin administration. A meta-analysis of randomized, controlled trials. *Ann Intern Med*. 2001;135:149–164.
413. Myburgh J, Cooper DJ, Finfer S, Bellomo R, Norton R, Bishop N, Kai Lo S, Vallance S. Saline or albumin for fluid resuscitation in patients with traumatic brain injury. *N Engl J Med*. 2007;357:874–884.
414. Huang PP, Stucky FS, Dimick AR, Treat RC, Bessey PQ, Rue LW. Hypertonic sodium resuscitation is associated with renal failure and death. *Ann Surg*. 1995;221:543–554; discussion 554–547.
415. Maitland K, Pamba A, English M, Peshu N, Marsh K, Newton C, Levin M. Randomized trial of volume expansion with albumin or saline in children with severe malaria: preliminary evidence of albumin benefit. *Clin Infect Dis*. 2005;40:538–545.
416. Ceneviva G, Paschall JA, Maffei F, Carcillo JA. Hemodynamic support in fluid-refractory pediatric septic shock. *Pediatrics*. 1998;102:e19.
417. Barton P, Garcia J, Kouatli A, Kitchen L, Zorka A, Lindsay C, Lawless S, Giroir B. Hemodynamic effects of I.V. milrinone lactate in pediatric patients with septic shock: a prospective, double-blinded, randomized, placebo-controlled, interventional study. *Chest*. 1996;109:1302–1312.
418. Lindsay CA, Barton P, Lawless S, Kitchen L, Zorka A, Garcia J, Kouatli A, Giroir B. Pharmacokinetics and pharmacodynamics of milrinone lactate in pediatric patients with septic shock. *J Pediatr*. 1998;132: 329–334.
419. Yildizdas D, Yapicioglu H, Celik U, Sertdemir Y, Alhan E. Terlipressin as a rescue therapy for catecholamine-resistant septic shock in children. *Intensive Care Med*. 2008;34:511–517.
420. Choong K, Bohn D, Fraser DD, Gaboury I, Hutchison JS, Joffe AR, Litalien C, Menon K, McNamara P, Ward RE. Vasopressin in pediatric vasodilatory shock: a multicenter randomized controlled trial. *Am J Respir Crit Care Med*. 2009;180:632–639.
421. Annane D, Vignon P, Renault A, Bollaert PE, Charpentier C, Martin C, Troche G, Ricard JD, Nitenberg G, Papazian L, Azoulay E, Bellissant E. Norepinephrine plus dobutamine versus epinephrine alone for management of septic shock: a randomised trial. *Lancet*. 2007;370:676–684.
422. Russell JA, Walley KR, Singer J, Gordon AC, Hebert PC, Cooper DJ, Holmes CL, Mehta S, Granton JT, Storms MM, Cook DJ, Presneill JJ, Ayers D. Vasopressin versus norepinephrine infusion in patients with septic shock. *N Engl J Med*. 2008;358:877–887.
423. Staubach KH, Schroder J, Stuber F, Gehrke K, Traumann E, Zabel P. Effect of pentoxifylline in severe sepsis: results of a randomized, double-blind, placebo-controlled study. *Arch Surg*. 1998;133:94–100.
424. Mullner M, Urbanek B, Havel C, Losert H, Waechter F, Gamper G Vasopressors for shock. *Cochrane Database Syst Rev*. 2004:CD003709.
425. Masutani S, Senzaki H, Ishido H, Taketazu M, Matsunaga T, Kobayashi T, Sasaki N, Asano H, Kyo S, Yokote Y. Vasopressin in the treatment of vasodilatory shock in children. *Pediatr Int*. 2005;47:132–136.
426. Jerath N, Frndova H, McCrindle BW, Gurofsky R, Humpl T. Clinical impact of vasopressin infusion on hemodynamics, liver and renal function in pediatric patients. *Intensive Care Med*. 2008;34:1274–1280.
427. Vasudevan A, Lodha R, Kabra SK. Vasopressin infusion in children with catecholamine-resistant septic shock. *Acta Paediatr*. 2005;94: 380–383.
428. Tobias JD. Arginine vasopressin for retractory distributive shock in two adolescents. *J Intensive Care Med*. 2002;17:48–52.
429. Efrati O, Modan-Moses D, Vardi A, Matok I, Bazilay Z, Paret G. Intravenous arginine vasopressin in critically ill children: is it beneficial? *Shock*. 2004;22:213–217.
430. Zeballos G, Lopez-Herce J, Fernandez C, Brandstrup KB, Rodriguez-Nunez A. Rescue therapy with terlipressin by continuous infusion in a child with catecholamine-resistant septic shock. *Resuscitation*. 2006;68:151–153.
431. Michel F, Thomachot L, David M, Nicaise C, Vialet R, Di Marco JN, Lagier P, Martin C. Continuous low-dose infusion of terlipressin as a rescue therapy in meningococcal septic shock. *Am J Emerg Med*. 2007; 25:863 e861–e862.
432. Matok I, Vard A, Efrati O, Rubinshtein M, Vishne T, Leibovitch L, Adam M, Barzilay Z, Paret G. Terlipressin as rescue therapy for intractable hypotension due to septic shock in children. *Shock*. 2005;23: 305–310.
433. Peters MJ, Booth RA, Petros AJ. Terlipressin bolus induces systemic vasoconstriction in septic shock. *Pediatr Crit Care Med*. 2004;5: 112–115.
434. Rodriguez-Nunez A, Fernandez-Sanmartin M, Martinon-Torres F, Gonzalez-Alonso N, Martinon-Sanchez JM. Terlipressin for catecholamine-resistant septic shock in children. *Intensive Care Med*. 2004;30: 477–480.
435. Rodriguez-Nunez A, Lopez-Herce J, Gil-Anton J, Hernandez A, Rey C. Rescue treatment with terlipressin in children with refractory septic shock: a clinical study. *Crit Care*. 2006;10:R20.
436. Berg RA, Donnerstein RL, Padbury JF. Dobutamine infusions in stable, critically ill children: pharmacokinetics and hemodynamic actions. *Crit Care Med*. 1993;21:678–686.
437. Booker PD, Evans C, Franks R. Comparison of the haemodynamic effects of dopamine and dobutamine in young children undergoing cardiac surgery. *Br J Anaesth*. 1995;74:419–423.
438. Driscoll DJ, Gillette PC, McNamara DG. The use of dopamine in children. *J Pediatr*. 1978;92:309–314.
439. Lang P, Williams RG, Norwood WI, Castaneda AR. The hemodynamic effects of dopamine in infants after corrective cardiac surgery. *J Pediatr*. 1980;96:630–634.

440. Outwater KM, Treves ST, Lang P, Castaneda AR, Crone RK. Renal and hemodynamic effects of dopamine in infants following cardiac surgery. *J Clin Anesth*. 1990;2:253–257.
441. Williams DB, Kiernan PD, Schaff HV, Marsh HM, Danielson GK. The hemodynamic response to dopamine and nitroprusside following right atrium-pulmonary artery bypass (Fontan procedure). *Ann Thorac Surg*. 1982;34:51–57.
442. Bohn DJ, Poirier CS, Edmonds JF, Barker GA. Hemodynamic effects of dobutamine after cardiopulmonary bypass in children. *Crit Care Med*. 1980;8:367–371.
443. Perkin RM, Levin DL, Webb R, Aquino A, Reedy J. Dobutamine: a hemodynamic evaluation in children with shock. *J Pediatr*. 1982;100: 977–983.
444. Hoffman TM, Wernovsky G, Atz AM, Kulik TJ, Nelson DP, Chang AC, Bailey JM, Akbary A, Kocsis JF, Kaczmarek R, Spray TL, Wessel DL. Efficacy and safety of milrinone in preventing low cardiac output syndrome in infants and children after corrective surgery for congenital heart disease. *Circulation*. 2003;107:996–1002.
445. Chang AC, Atz AM, Wernovsky G, Burke RP, Wessel DL. Milrinone: systemic and pulmonary hemodynamic effects in neonates after cardiac surgery. *Crit Care Med*. 1995;23:1907–1914.
446. Abdallah I, Shawky H. A randomised controlled trial comparing milrinone and epinephrine as inotropes in paediatric patients undergoing total correction of tetralogy of fallot. *Egypt J Anaesth*. 2003;19:323–329.
447. Namachivayam P, Crossland DS, Butt WW, Shekerdemian LS. Early experience with levosimendan in children with ventricular dysfunction. *Pediatr Crit Care Med*. 2006;7:445–448.
448. Egan JR, Clarke AJ, Williams S, Cole AD, Ayer J, Jacobe S, Chard RB, Winlaw DS. Levosimendan for low cardiac output: a pediatric experience. *J Intensive Care Med*. 2006;21:183–187.
449. De Backer D, Biston P, Devriendt J, Madl C, Chochrad D, Aldecoa C, Brasseur A, Defrance P, Gottignies P, Vincent JL. Comparison of dopamine and norepinephrine in the treatment of shock. *N Engl J Med*. 2010;362:779–789.
450. den Brinker M, Hokken-Koelega AC, Hazelzet JA, de Jong FH, Hop WC, Joosten KF. One single dose of etomidate negatively influences adrenocortical performance for at least 24h in children with meningococcal sepsis. *Intensive Care Med*. 2008;34:163–168.
451. Zuckerbraun NS, Pitetti RD, Herr SM, Roth KR, Gaines BA, King C. Use of etomidate as an induction agent for rapid sequence intubation in a pediatric emergency department. *Acad Emerg Med*. 2006;13:602–609.
452. Sokolove PE, Price DD, Okada P. The safety of etomidate for emergency rapid sequence intubation of pediatric patients. *Pediatr Emerg Care*. 2000;16:18–21.
453. Guldner G, Schultz J, Sexton P, Fortner C, Richmond M. Etomidate for rapid-sequence intubation in young children: hemodynamic effects and adverse events. *Acad Emerg Med*. 2003;10:134–139.
454. Sprung CL, Annane D, Keh D, Moreno R, Singer M, Freivogel K, Weiss YG, Benbenishty J, Kalenka A, Forst H, Laterre PF, Reinhart K, Cuthbertson BH, Payen D, Briegel J. Hydrocortisone therapy for patients with septic shock. *N Engl J Med*. 2008;358:111–124.
455. Tekwani KL, Watts HF, Rzechula KH, Sweis RT, Kulstad EB. A prospective observational study of the effect of etomidate on septic patient mortality and length of stay. *Acad Emerg Med*. 2009;16:11–14.
456. Jabre P, Combes X, Lapostolle F, Dhaouadi M, Ricard-Hibon A, Vivien B, Bertrand L, Beltramini A, Gamand P, Albizzati S, Perdrizet D, Lebail G, Chollet-Xemard C, Maxime V, Brun-Buisson C, Lefrant JY, Bollaert PE, Megarbane B, Ricard JD, Anguel N, Vicaut E, Adnet F. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. *Lancet*. 2009;374:293–300.
457. Annane D, Sebille V, Charpentier C, Bollaert PE, Francois B, Korach JM, Capellier G, Cohen Y, Azoulay E, Troche G, Chaumet-Riffaud P, Bellissant E. Effect of treatment with low doses of hydrocortisone and fludrocortisone on mortality in patients with septic shock. *JAMA*. 2002; 288:862–871.
458. Bollaert PE, Charpentier C, Levy B, Debouverie M, Audibert G, Larcan A. Reversal of late septic shock with supraphysiologic doses of hydrocortisone. *Crit Care Med*. 1998;26:645–650.
459. Briegel J, Forst H, Haller M, Schelling G, Kilger E, Kuprat G, Hemmer B, Hummel T, Lenhart A, Heyduck M, Stoll C, Peter K. Stress doses of hydrocortisone reverse hyperdynamic septic shock: a prospective, randomized, double-blind, single-center study. *Crit Care Med*. 1999;27: 723–732.
460. Oppert M, Reinicke A, Graf KJ, Barckow D, Frei U, Eckardt KU. Plasma cortisol levels before and during "low-dose" hydrocortisone therapy and their relationship to hemodynamic improvement in patients with septic shock. *Intensive Care Med*. 2000;26:1747–1755.
461. Oppert M, Schindler R, Husung C, Offermann K, Graf KJ, Boenisch O, Barckow D, Frei U, Eckardt KU. Low-dose hydrocortisone improves shock reversal and reduces cytokine levels in early hyperdynamic septic shock. *Crit Care Med*. 2005;33:2457–2464.
462. Bollaert PE, Bauer P, Audibert G, Lambert H, Larcan A. Effects of epinephrine on hemodynamics and oxygen metabolism in dopamine-resistant septic shock. *Chest*. 1990;98:949–953.
463. Russell JA, Walley KR, Gordon AC, Cooper DJ, Hebert PC, Singer J, Holmes CL, Mehta S, Granton JT, Storms MM, Cook DJ, Presneill JJ. Interaction of vasopressin infusion, corticosteroid treatment, and mortality of septic shock. *Crit Care Med*. 2009;37:811–818.
464. Yildiz O, Doganay M, Aygen B, Guven M, Kelestimur F, Tutuu A. Physiological-dose steroid therapy in sepsis [isrctn36253388]. *Crit Care*. 2002;6:251–259.
465. Slusher T, Gbadero D, Howard C, Lewison L, Giroir B, Toro L, Levin D, Holt E, McCracken GH Jr. Randomized, placebo-controlled, double blinded trial of dexamethasone in african children with sepsis. *Pediatr Infect Dis J*. 1996;15:579–583.
466. Valoor HT, Singhi S, Jayashree M. Low-dose hydrocortisone in pediatric septic shock: an exploratory study in a third world setting. *Pediatr Crit Care Med*. 2009;10:121–125.
467. Markovitz BP, Goodman DM, Watson RS, Bertoch D, Zimmerman J. A retrospective cohort study of prognostic factors associated with outcome in pediatric severe sepsis: what is the role of steroids? *Pediatr Crit Care Med*. 2005;6:270–274.
468. de Oliveira CF, de Oliveira DS, Gottschald AF, Moura JD, Costa GA, Ventura AC, Fernandes JC, Vaz FA, Carcillo JA, Rivers EP, Troster EJ. ACCM/PALS haemodynamic support guidelines for paediatric septic shock: an outcomes comparison with and without monitoring central venous oxygen saturation. *Intensive Care Med*. 2008;34:1065–1075.
469. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345: 1368–1377.
470. Nguyen HB, Corbett SW, Steele R, Banta J, Clark RT, Hayes SR, Edwards J, Cho TW, Wittlake WA. Implementation of a bundle of quality indicators for the early management of severe sepsis and septic shock is associated with decreased mortality. *Crit Care Med*. 2007;35: 1105–1112.
471. Levy MM, Dellinger RP, Townsend SR, Linde-Zwirble WT, Marshall JC, Bion J, Schorr C, Artigas A, Ramsay G, Beale R, Parker MM, Gerlach H, Reinhart K, Silva E, Harvey M, Regan S, Angus DC. The surviving sepsis campaign: results of an international guideline-based performance improvement program targeting severe sepsis. *Crit Care Med*. 2010;38:367–374.
472. Black K, Barnett P, Wolfe R, Young S. Are methods used to estimate weight in children accurate? *Emerg Med (Fremantle)*. 2002;14: 160–165.
473. Chan GM, Moyer-Mileur L, Rallison L. An easy and accurate method of estimating newborn birthweight for resuscitation. *Am J Perinatol*. 1992; 9:371–373.
474. Garland JS, Kishaba RG, Nelson DB, Losek JD, Sobocinski KA. A rapid and accurate method of estimating body weight. *Am J Emerg Med*. 1986;4:390–393.
475. Krieser D, Nguyen K, Kerr D, Jolley D, Clooney M, Kelly AM. Parental weight estimation of their child's weight is more accurate than other weight estimation methods for determining children's weight in an emergency department? *Emerg Med J*. 2007;24:756–759.
476. Lubitz DS, Seidel JS, Chameides L, Luten RC, Zaritsky AL, Campbell FW. A rapid method for estimating weight and resuscitation drug dosages from length in the pediatric age group. *Ann Emerg Med*. 1988; 17:576–581.
477. Varghese A, Vasudevan VK, Lewin S, Indumathi CK, Dinakar C, Rao SD. Do the length-based (Broselow) tape, APLS, Argall and Nelson's formulae accurately estimate weight of Indian children? *Indian Pediatr*. 2006;43:889–894.
478. Vilke GM, Marino A, Fisher R, Chan TC. Estimation of pediatric patient weight by EMT-Ps. *J Emerg Med*. 2001;21:125–128.
479. Hofer CK, Ganter M, Tucci M, Klaghofer R, Zollinger A. How reliable is length-based determination of body weight and tracheal tube size in the paediatric age group? The Broselow tape reconsidered. *Br J Anaesth*. 2002;88:283–285.

480. DuBois D, Baldwin S, King WD. Accuracy of weight estimation methods for children. *Pediatr Emerg Care*. 2007;23:227–230.
481. Yamamoto LG, Inaba AS, Young LL, Anderson KM. Improving length-based weight estimates by adding a body habitus (obesity) icon. *Am J Emerg Med*. 2009;27:810–815.
482. Johnson TN. The problems in scaling adult drug doses to children. *Arch Dis Child*. 2008;93:207–211.
483. Mahmood I. Prediction of drug clearance in children: impact of allometric exponents, body weight, and age. *Ther Drug Monit*. 2007;29:271–278.
484. Edginton AN, Schmitt W, Willmann S. Development and evaluation of a generic physiologically based pharmacokinetic model for children. *Clin Pharmacokinet*. 2006;45:1013–1034.
485. Gill MA, Ueda CT. Novel method for the determination of pediatric dosages. *Am J Hosp Pharm*. 1976;33:389–392.
486. Rodriguez W, Selen A, Avant D, Chaurasia C, Crescenzi T, Gieser G, Di Giacinto J, Huang SM, Lee P, Mathis L, Murphy D, Murphy S, Roberts R, Sachs HC, Suarez S, Tandon V, Uppoor RS. Improving pediatric dosing through pediatric initiatives: what we have learned. *Pediatrics*. 2008;121:530–539.
487. Traub SL, Kichen L. Estimating ideal body mass in children. *Am J Hosp Pharm*. 1983;40:107–110.
488. Olasveengen TM, Sunde K, Brunborg C, Thowsen J, Steen PA, Wik L. Intravenous drug administration during out-of-hospital cardiac arrest: a randomized trial. *JAMA*. 2009;302:2222–2229.
489. Perondi MB, Reis AG, Paiva EF, Nadkarni VM, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. *N Engl J Med*. 2004;350:1722–1730.
490. Rodriguez Nunez A, Garcia C, Lopez-Herce Cid J. [Is high-dose epinephrine justified in cardiorespiratory arrest in children?] *An Pediatr (Barc)*. 2005;62:113–116.
491. Patterson MD, Boenning DA, Klein BL, Fuchs S, Smith KM, Hegenbarth MA, Carlson DW, Krug SE, Harris EM. The use of high-dose epinephrine for patients with out-of-hospital cardiopulmonary arrest refractory to prehospital interventions. *Pediatr Emerg Care*. 2005;21:227–237.
492. Dieckmann RA, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. *Pediatrics*. 1995;95:901–913.
493. Carpenter TC, Stenmark KR. High-dose epinephrine is not superior to standard-dose epinephrine in pediatric in-hospital cardiopulmonary arrest. *Pediatrics*. 1997;99:403–408.
494. Lindner KH, Ahnefeld FW, Bowdler IM. Comparison of different doses of epinephrine on myocardial perfusion and resuscitation success during cardiopulmonary resuscitation in a pig model. *Am J Emerg Med*. 1991;9:27–31.
495. Brown CG, Martin DR, Pepe PE, Stueven H, Cummins RO, Gonzalez E, Jastremski M. A comparison of standard-dose and high-dose epinephrine in cardiac arrest outside the hospital. The Multicenter High-Dose Epinephrine Study Group. *N Engl J Med*. 1992;327:1051–1055.
496. Callaham M, Madsen CD, Barton CW, Saunders CE, Pointer J. A randomized clinical trial of high-dose epinephrine and norepinephrine vs standard-dose epinephrine in prehospital cardiac arrest. *JAMA*. 1992;268:2667–2672.
497. Stiell IG, Hebert PC, Weitzman BN, Wells GA, Raman S, Stark RM, Higginson LA, Ahuja J, Dickinson GE. High-dose epinephrine in adult cardiac arrest. *N Engl J Med*. 1992;327:1045–1050.
498. Choux C, Gueugniaud PY, Barbieux A, Pham E, Lae C, Dubien PY, Petit P. Standard doses versus repeated high doses of epinephrine in cardiac arrest outside the hospital. *Resuscitation*. 1995;29:3–9.
499. Gueugniaud PY, Mols P, Goldstein P, Pham E, Dubien PY, Deweerdt C, Vergnion M, Petit P, Carli P. A comparison of repeated high doses and repeated standard doses of epinephrine for cardiac arrest outside the hospital. European Epinephrine Study Group. *N Engl J Med*. 1998;339:1595–1601.
500. Vandycke C, Martens P. High dose versus standard dose epinephrine in cardiac arrest — a meta-analysis. *Resuscitation*. 2000;45:161–166.
501. Lipman J, Wilson W, Kobilski S, Scribante J, Lee C, Kraus P, Cooper J, Barr J, Moyes D. High-dose adrenaline in adult in-hospital asystolic cardiopulmonary resuscitation: a double-blind randomised trial. *Anaesth Intensive Care*. 1993;21:192–196.
502. Sherman BW, Munger MA, Foulke GE, Rutherford WF, Panacek EA. High-dose versus standard-dose epinephrine treatment of cardiac arrest after failure of standard therapy. *Pharmacotherapy*. 1997;17:242–247.
503. Callaham M, Barton CW, Kayser S. Potential complications of high-dose epinephrine therapy in patients resuscitated from cardiac arrest. *JAMA*. 1991;265:1117–1122.
504. Rivers EP, Wortsman J, Rady MY, Blake HC, McGeorge FT, Buderer NM. The effect of the total cumulative epinephrine dose administered during human CPR on hemodynamic, oxygen transport, and utilization variables in the postresuscitation period. *Chest*. 1994;106:1499–1507.
505. Woodhouse SP, Cox S, Boyd P, Case C, Weber M. High dose and standard dose adrenaline do not alter survival, compared with placebo, in cardiac arrest. *Resuscitation*. 1995;30:243–249.
506. Marwick TH, Case C, Siskind V, Woodhouse SP. Adverse effect of early high-dose adrenaline on outcome of ventricular fibrillation. *Lancet*. 1988;2:66–68.
507. Carvolth RD, Hamilton AJ. Comparison of high-dose epinephrine versus standard-dose epinephrine in adult cardiac arrest in the prehospital setting. *Prehospital Disaster Med*. 1996;11:219–222.
508. Behringer W, Kittler H, Sterz F, Domanovits H, Schoerkhuber W, Holzer M, Mullner M, Laggner AN. Cumulative epinephrine dose during cardiopulmonary resuscitation and neurologic outcome. *Ann Intern Med*. 1998;129:450–456.
509. Meert KL, Donaldson A, Nadkarni V, Tieves KS, Schleien CL, Brilli RJ, Clark RS, Shaffner DH, Levy F, Statler K, Dalton HJ, van der Jagt EW, Hackbarth R, Pretzlaff R, Hernan L, Dean JM, Moler FW. Multicenter cohort study of in-hospital pediatric cardiac arrest. *Pediatr Crit Care Med*. 2009;10:544–553.
510. Vukmir RB, Katz L. Sodium bicarbonate improves outcome in prolonged prehospital cardiac arrest. *Am J Emerg Med*. 2006;24:156–161.
511. Lokesh L, Kumar P, Murki S, Narang A. A randomized controlled trial of sodium bicarbonate in neonatal resuscitation-effect on immediate outcome. *Resuscitation*. 2004;60:219–223.
512. Duncan JM, Meaney P, Simpson P, Berg RA, Nadkarni V, Schexnayder S. Vasopressin for in-hospital pediatric cardiac arrest: results from the American Heart Association National Registry of Cardiopulmonary Resuscitation. *Pediatr Crit Care Med*. 2009;10:191–195.
513. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. *Resuscitation*. 2002;52:149–156.
514. Matok I, Vardi A, Augarten A, Efrati O, Leibovitch L, Rubinshtein M, Paret G. Beneficial effects of terlipressin in prolonged pediatric cardiopulmonary resuscitation: a case series. *Crit Care Med*. 2007;35:1161–1164.
515. Gil-Anton J, Lopez-Herce J, Morteruel E, Carrillo A, Rodriguez-Nunez A. Pediatric cardiac arrest refractory to advanced life support: is there a role for terlipressin? *Pediatr Crit Care Med*. 2010;11:139–141.
516. Lindner KH, Prengel AW, Brinkmann A, Strohmenger HU, Lindner IM, Lurie KG. Vasopressin administration in refractory cardiac arrest. *Ann Intern Med*. 1996;124:1061–1064.
517. Lee CC, Kim GW, Kim SH, Crupi RS. Cases of aminophylline and vasopressin use after failed prehospital resuscitation of cardiac arrest. *Prehosp Emerg Care*. 2001;5:304–307.
518. Callaway CW, Hostler D, Doshi AA, Pinchalk M, Roth RN, Lubin J, Newman DH, Kelly LJ. Usefulness of vasopressin administered with epinephrine during out-of-hospital cardiac arrest. *Am J Cardiol*. 2006;98:1316–1321.
519. Gueugniaud PY, David JS, Chanzy E, Hubert H, Dubien PY, Mauriaucourt P, Braganca C, Billeres X, Clotteau-Lambert MP, Fuster P, Thiercelin D, Debaty G, Ricard-Hibon A, Roux P, Espesson C, Querellou E, Ducros L, Ecollan P, Halbout L, Savary D, Guillaumee F, Maupoint R, Capelle P, Bracq C, Dreyfus P, Nouguier P, Gache A, Meurisse C, Boulanger B, Lae C, Metzger J, Raphael V, Beruben A, Wenzel V, Guinhouya C, Vilhelm C, Marret E. Vasopressin and epinephrine vs. epinephrine alone in cardiopulmonary resuscitation. *N Engl J Med*. 2008;359:21–30.
520. Lindner KH, Dirks B, Strohmenger HU, Prengel AW, Lindner IM, Lurie KG. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. *Lancet*. 1997;349:535–537.
521. Mukoyama T, Kinoshita K, Nagao K, Tanjoh K. Reduced effectiveness of vasopressin in repeated doses for patients undergoing prolonged cardiopulmonary resuscitation. *Resuscitation*. 2009;80:755–761.
522. Stiell IG, Hebert PC, Wells GA, Vandemheen KL, Tang AS, Higginson LA, Dreyer JF, Clement C, Battram E, Watpool I, Mason S, Klassen T, Weitzman BN. Vasopressin versus epinephrine for inhospital cardiac arrest: a randomised controlled trial. *Lancet*. 2001;358:105–109.

523. Wenzel V, Krismer AC, Arntz HR, Sitter H, Stadlbauer KH, Lindner KH. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. *N Engl J Med*. 2004;350:105–113.
524. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. *Arch Intern Med*. 2005;165:17–24.
525. Lopez-Herce J, Fernandez B, Urbano J, Mencia S, Solana MJ, Del Castillo J, Rodriguez-Nunez A, Bellon JM. Terlipressin versus adrenaline in an infant animal model of asphyxial cardiac arrest. *Intensive Care Med.* 2010.
526. Srinivasan V, Morris MC, Helfaer MA, Berg RA, Nadkarni VM. Calcium use during in-hospital pediatric cardiopulmonary resuscitation: a report from the National Registry of Cardiopulmonary Resuscitation. *Pediatrics*. 2008;121:e1144–e1151.
527. de Mos N, van Litsenburg RR, McCrindle B, Bohn DJ, Parshuram CS. Pediatric in-intensive-care-unit cardiac arrest: incidence, survival, and predictive factors. *Crit Care Med*. 2006;34:1209–1215.
528. Harrison EE, Amey BD. The use of calcium in cardiac resuscitation. *Am J Emerg Med*. 1983;1:267–273.
529. Ornato JP, Gonzales ER, Morkunas AR, Coyne MR, Beck CL. Treatment of presumed asystole during pre-hospital cardiac arrest: superiority of electrical countershock. *Am J Emerg Med*. 1985;3:395–399.
530. Stueven H, Thompson BM, Aprahamian C, Darin JC. Use of calcium in prehospital cardiac arrest. *Ann Emerg Med*. 1983;12:136–139.
531. Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. The effectiveness of calcium chloride in refractory electromechanical dissociation. *Ann Emerg Med*. 1985;14:626–629.
532. Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. Lack of effectiveness of calcium chloride in refractory asystole. *Ann Emerg Med*. 1985;14:630–632.
533. Blecic S, De Backer D, Huynh CH, Deleuze M, Domb M, Luypaert P, Vincent JL. Calcium chloride in experimental electromechanical dissociation: a placebo-controlled trial in dogs. *Crit Care Med*. 1987;15:324–327.
534. Niemann JT, Adomian GE, Garner D, Rosborough JP. Endocardial and transcutaneous cardiac pacing, calcium chloride, and epinephrine in postcountershock asystole and bradycardias. *Crit Care Med*. 1985;13:699–704.
535. Redding JS, Haynes RR, Thomas JD. Drug therapy in resuscitation from electromechanical dissociation. *Crit Care Med*. 1983;11:681–684.
536. Redding JS, Pearson JW. Evaluation of drugs for cardiac resuscitation. *Anesthesiology*. 1963;24:203–207.
537. Meaney PA, Nadkarni VM, Cook EF, Testa M, Helfaer M, Kaye W, Larkin GL, Berg RA. Higher survival rates among younger patients after pediatric intensive care unit cardiac arrests. *Pediatrics*. 2006;118:2424–2433.
538. Reis AG, Nadkarni V, Perondi MB, Grisi S, Berg RA. A prospective investigation into the epidemiology of in-hospital pediatric cardiopulmonary resuscitation using the international utstein reporting style. *Pediatrics*. 2002;109:200–209.
539. Brown DC, Lewis AJ, Criley JM. Asystole and its treatment: the possible role of the parasympathetic nervous system in cardiac arrest. *JACEP*. 1979;8:448–452.
540. Stueven HA, Tonsfeldt DJ, Thompson BM, Whitcomb J, Kastenson E, Aprahamian C. Atropine in asystole: human studies. *Ann Emerg Med*. 1984;13:815–817.
541. Yilmaz O, Eser M, Sahiner A, Altintop L, Yesildag O. Hypotension, bradycardia and syncope caused by honey poisoning. *Resuscitation*. 2006;68:405–408.
542. Brady WJ, Swart G, DeBehnke DJ, Ma OJ, Aufderheide TP. The efficacy of atropine in the treatment of hemodynamically unstable bradycardia and atrioventricular block: prehospital and emergency department considerations. *Resuscitation*. 1999;41:47–55.
543. Smith I, Monk TG, White PF. Comparison of transesophageal atrial pacing with anticholinergic drugs for the treatment of intraoperative bradycardia. *Anesth Analg*. 1994;78:245–252.
544. Chadda KD, Lichstein E, Gupta PK, Kourtesis P. Effects of atropine in patients with bradyarrhythmia complicating myocardial infarction: usefulness of an optimum dose for overdrive. *Am J Med*. 1977;63:503–510.
545. Fullerton DA, St Cyr JA, Clarke DR, Campbell DN, Toews WH, See WM. Bezold-Jarisch reflex in postoperative pediatric cardiac surgical patients. *Ann Thorac Surg*. 1991;52:534–536.
546. Chow LT, Chow SS, Anderson RH, Gosling JA. Autonomic innervation of the human cardiac conduction system: changes from infancy to senility–an immunohistochemical and histochemical analysis. *Anat Rec*. 2001;264:169–182.
547. Coon GA, Clinton JE, Ruiz E. Use of atropine for brady-asystolic prehospital cardiac arrest. *Ann Emerg Med*. 1981;10:462–467.
548. Iseri LT, Humphrey SB, Siner EJ. Prehospital brady-asystolic cardiac arrest. *Ann Intern Med*. 1978;88:741–745.
549. Angelos MG, Butke RL, Panchal AR, Torres CA, Blumberg A, Schneider JE, Aune SE. Cardiovascular response to epinephrine varies with increasing duration of cardiac arrest. *Resuscitation*. 2008;77:101–110.
550. Kaplan JL, Gao E, De Garavilla L, Victain M, Minczak B, Dalsey WC. Adenosine a1 antagonism attenuates atropine-resistant hypoxic bradycardia in rats. *Acad Emerg Med*. 2003;10:923–930.
551. McCaul CL, McNamara PJ, Engelberts D, Wilson GJ, Romaschin A, Redington AN, Kavanagh BP. Epinephrine increases mortality after brief asphyxial cardiac arrest in an in vivo rat model. *Anesth Analg*. 2006;102:542–548.
552. DeBehnke DJ, Swart GL, Spreng D, Aufderheide TP. Standard and higher doses of atropine in a canine model of pulseless electrical activity. *Acad Emerg Med*. 1995;2:1034–1041.
553. Blecic S, Chaskis C, Vincent JL. Atropine administration in experimental electromechanical dissociation. *Am J Emerg Med*. 1992;10:515–518.
554. Morris MC, Wernovsky G, Nadkarni VM. Survival outcomes after extracorporeal cardiopulmonary resuscitation instituted during active chest compressions following refractory in-hospital pediatric cardiac arrest. *Pediatr Crit Care Med*. 2004;5:440–446.
555. Alsoufi B, Al-Radi OO, Nazer RI, Gruenwald C, Foreman C, Williams WG, Coles JG, Caldarone CA, Bohn DG, Van Arsdell GS. Survival outcomes after rescue extracorporeal cardiopulmonary resuscitation in pediatric patients with refractory cardiac arrest. *J Thorac Cardiovasc Surg*. 2007;134:952–959 e952.
556. Huang SC, Wu ET, Chen YS, Chang CI, Chiu IS, Wang SS, Lin FY, Ko WJ. Extracorporeal membrane oxygenation rescue for cardiopulmonary resuscitation in pediatric patients. *Crit Care Med*. 2008;36:1607–1613.
557. Allan CK, Thiagarajan RR, Armsby LR, del Nido PJ, Laussen PC. Emergent use of extracorporeal membrane oxygenation during pediatric cardiac catheterization. *Pediatr Crit Care Med*. 2006;7:212–219.
558. Chan T, Thiagarajan RR, Frank D, Bratton SL. Survival after extracorporeal cardiopulmonary resuscitation in infants and children with heart disease. *J Thorac Cardiovasc Surg*. 2008;136:984–992.
559. del Nido PJ, Dalton HJ, Thompson AE, Siewers RD. Extracorporeal membrane oxygenator rescue in children during cardiac arrest after cardiac surgery. *Circulation.* 1992;86:II300–304.
560. Duncan BW, Ibrahim AE, Hraska V, del Nido PJ, Laussen PC, Wessel DL, Mayer JE Jr, Bower LK, Jonas RA. Use of rapid-deployment extracorporeal membrane oxygenation for the resuscitation of pediatric patients with heart disease after cardiac arrest. *J Thorac Cardiovasc Surg*. 1998;116:305–311.
561. Hoskote A, Bohn D, Gruenwald C, Edgell D, Cai S, Adatia I, Van Arsdell G. Extracorporeal life support after staged palliation of a functional single ventricle: subsequent morbidity and survival. *J Thorac Cardiovasc Surg*. 2006;131:1114–1121.
562. Ibrahim AE, Duncan BW, Blume ED, Jonas RA. Long-term follow-up of pediatric cardiac patients requiring mechanical circulatory support. *Ann Thorac Surg*. 2000;69:186–192.
563. Prodhan P, Fiser RT, Dyamenahalli U, Gossett J, Imamura M, Jaquiss RD, Bhutta AT. Outcomes after extracorporeal cardiopulmonary resuscitation (ECPR) following refractory pediatric cardiac arrest in the intensive care unit. *Resuscitation*. 2009;80:1124–1129.
564. Thiagarajan RR, Laussen PC, Rycus PT, Bartlett RH, Bratton SL. Extracorporeal membrane oxygenation to aid cardiopulmonary resuscitation in infants and children. *Circulation*. 2007;116:1693–1700.
565. Lequier L, Joffe AR, Robertson CM, Dinu IA, Wongswadiwat Y, Anton NR, Ross DB, Rebeyka IM. Two-year survival, mental, and motor outcomes after cardiac extracorporeal life support at less than five years of age. *J Thorac Cardiovasc Surg.* 2008;136:976–983 e973.
566. Mahle WT, Forbess JM, Kirshbom PM, Cuadrado AR, Simsic JM, Kanter KR. Cost-utility analysis of salvage cardiac extracorporeal membrane oxygenation in children. *J Thorac Cardiovasc Surg*. 2005;129:1084–1090.
567. Aharon AS, Drinkwater DC Jr, Churchwell KB, Quisling SV, Reddy VS, Taylor M, Hix S, Christian KG, Pietsch JB, Deshpande JK, Kambam J, Graham TP, Chang PA. Extracorporeal membrane oxygenation in children after repair of congenital cardiac lesions. *Ann Thorac Surg.* 2001;72:2095–2101; discussion 2101–2092.

568. Barrett CS, Bratton SL, Salvin JW, Laussen PC, Rycus PT, Thiagarajan RR. Neurological injury after extracorporeal membrane oxygenation use to aid pediatric cardiopulmonary resuscitation. *Pediatr Crit Care Med*. 2009;10:445–451.
569. Baslaim G, Bashore J, Al-Malki F, Jamjoom A. Can the outcome of pediatric extracorporeal membrane oxygenation after cardiac surgery be predicted? *Ann Thorac Cardiovasc Surg*. 2006;12:21–27.
570. Ghez O, Feier H, Ughetto F, Fraisse A, Kreitmann B, Metras D. Postoperative extracorporeal life support in pediatric cardiac surgery: Recent results. *ASAIO J*. 2005;51:513–516.
571. Cochran JB, Tecklenburg FW, Lau YR, Habib DM. Emergency cardiopulmonary bypass for cardiac arrest refractory to pediatric advanced life support. *Pediatr Emerg Care*. 1999;15:30–32.
572. Dalton HJ, Siewers RD, Fuhrman BP, Del Nido P, Thompson AE, Shaver MG, Dowhy M. Extracorporeal membrane oxygenation for cardiac rescue in children with severe myocardial dysfunction. *Crit Care Med*. 1993;21:1020–1028.
573. del Nido PJ. Extracorporeal membrane oxygenation for cardiac support in children. *Ann Thorac Surg*. 1996;61:336–339; discussion 340–341.
574. Ghez O, Fouilloux V, Charpentier A, Fesquet P, Lion F, Lebrun L, Commandeur M, Fraisse A, Metras D, Kreitmann B. Absence of rapid deployment extracorporeal membrane oxygenation (ECMO) team does not preclude resuscitation ECMO in pediatric cardiac patients with good results. *ASAIO J*. 2007;53:692–695.
575. Jaggers JJ, Forbess JM, Shah AS, Meliones JN, Kirshbom PM, Miller CE, Ungerleider RM. Extracorporeal membrane oxygenation for infant postcardiotomy support: significance of shunt management. *Ann Thorac Surg*. 2000;69:1476–1483.
576. Kelly RB, Porter PA, Meier AH, Myers JL, Thomas NJ. Duration of cardiopulmonary resuscitation before extracorporeal rescue: how long is not long enough? *ASAIO J*. 2005;51:665–667.
577. Parra DA, Totapally BR, Zahn E, Jacobs J, Aldousany A, Burke RP, Chang AC. Outcome of cardiopulmonary resuscitation in a pediatric cardiac intensive care unit. *Crit Care Med*. 2000;28:3296–3300.
578. Ravishankar C, Dominguez TE, Kreutzer J, Wernovsky G, Marino BS, Godinez R, Priestley MA, Gruber PJ, Gaynor WJ, Nicolson SC, Spray TL, Tabbutt S. Extracorporeal membrane oxygenation after stage I reconstruction for hypoplastic left heart syndrome. *Pediatr Crit Care Med*. 2006;7:319–323.
579. Shah SA, Shankar V, Churchwell KB, Taylor MB, Scott BP, Bartilson R, Byrne DW, Christian KG, Drinkwater DC. Clinical outcomes of 84 children with congenital heart disease managed with extracorporeal membrane oxygenation after cardiac surgery. *ASAIO J*. 2005;51:504–507.
580. Thourani VH, Kirshbom PM, Kanter KR, Simsic J, Kogon BE, Wagoner S, Dykes F, Fortenberry J, Forbess JM. Venoarterial extracorporeal membrane oxygenation (VA-ECMO) in pediatric cardiac support. *Ann Thorac Surg*. 2006;82:138–144; discussion 144–135.
581. Yamasaki Y, Hayashi T, Nakatani T, Yotsuida H, Nishigaki T, Takahashi Y, Inamori S, Kagisaki K, Hagino H, Ishizaka T, Yagihara T. Early experience with low-prime (99 ml) extracorporeal membrane oxygenation support in children. *ASAIO J*. 2006;52:110–114.
582. Scaife ER, Connors RC, Morris SE, Nichol PF, Black RE, Matlak ME, Hansen K, Bolte RG. An established extracorporeal membrane oxygenation protocol promotes survival in extreme hypothermia. *J Pediatr Surg*. 2007;42:2012–2016.
583. Walpoth BH, Walpoth-Aslan BN, Mattle HP, Radanov BP, Schroth G, Schaeffler L, Fischer AP, von Segesser L, Althaus U. Outcome of survivors of accidental deep hypothermia and circulatory arrest treated with extracorporeal blood warming. *N Engl J Med*. 1997;337:1500–1505.
584. Wollenek G, Honarwar N, Golej J, Marx M. Cold water submersion and cardiac arrest in treatment of severe hypothermia with cardiopulmonary bypass. *Resuscitation*. 2002;52:255–263.
585. Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, Gutteridge G, Smith K. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med*. 2002;346:557–563.
586. Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med*. 2002;346:549–556.
587. Gluckman PD, Wyatt JS, Azzopardi D, Ballard R, Edwards AD, Ferriero DM, Polin RA, Robertson CM, Thoresen M, Whitelaw A, Gunn AJ. Selective head cooling with mild systemic hypothermia after neonatal encephalopathy: multicentre randomised trial. *Lancet*. 2005;365:663–670.
588. Shankaran S, Laptook AR, Ehrenkranz RA, Tyson JE, McDonald SA, Donovan EF, Fanaroff AA, Poole WK, Wright LL, Higgins RD, Finer NN, Carlo WA, Duara S, Oh W, Cotten CM, Stevenson DK, Stoll BJ, Lemons JA, Guillet R, Jobe AH. Whole-body hypothermia for neonates with hypoxic-ischemic encephalopathy. *N Engl J Med*. 2005;353:1574–1584.
589. Doherty DR, Parshuram CS, Gaboury I, Hoskote A, Lacroix J, Tucci M, Joffe A, Choong K, Farrell R, Bohn DJ, Hutchison JS. Hypothermia therapy after pediatric cardiac arrest. *Circulation*. 2009;119:1492–1500.
590. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. *Crit Care Med*. 1988;16:331–335.
591. Checchia PA, Sehra R, Moynihan J, Daher N, Tang W, Weil MH. Myocardial injury in children following resuscitation after cardiac arrest. *Resuscitation*. 2003;57:131–137.
592. Laurent I, Monchi M, Chiche JD, Joly LM, Spaulding C, Bourgeois B, Cariou A, Rozenberg A, Carli P, Weber S, Dhainaut JF. Reversible myocardial dysfunction in survivors of out-of-hospital cardiac arrest. *J Am Coll Cardiol*. 2002;40:2110–2116.
593. Mayr V, Luckner G, Jochberger S, Wenzel V, Ulmer H, Pajk W, Knotzer H, Friesenecker B, Lindner K, Hasibeder W, Dunser M. Arginine vasopressin in advanced cardiovascular failure during the post-resuscitation phase after cardiac arrest. *Resuscitation*. 2007;72:35–44.
594. Kern KB, Hilwig RW, Berg RA, Rhee KH, Sanders AB, Otto CW, Ewy GA. Postresuscitation left ventricular systolic and diastolic dysfunction: treatment with dobutamine. *Circulation*. 1997;95:2610–2613.
595. Meyer RJ, Kern KB, Berg RA, Hilwig RW, Ewy GA. Post-resuscitation right ventricular dysfunction: delineation and treatment with dobutamine. *Resuscitation*. 2002;55:187–191.
596. Huang L, Weil MH, Sun S, Cammarata G, Cao L, Tang W. Levosimendan improves postresuscitation outcomes in a rat model of CPR. *J Lab Clin Med*. 2005;146:256–261.
597. Huang L, Weil MH, Tang W, Sun S, Wang J. Comparison between dobutamine and levosimendan for management of postresuscitation myocardial dysfunction. *Crit Care Med*. 2005;33:487–491.
598. Studer W, Wu X, Siegemund M, Marsch S, Seeberger M, Filipovic M. Influence of dobutamine on the variables of systemic haemodynamics, metabolism, and intestinal perfusion after cardiopulmonary resuscitation in the rat. *Resuscitation*. 2005;64:227–232.
599. Vasquez A, Kern KB, Hilwig RW, Heidenreich J, Berg RA, Ewy GA. Optimal dosing of dobutamine for treating post-resuscitation left ventricular dysfunction. *Resuscitation*. 2004;61:199–207.
600. Alvarez J, Bouzada M, Fernandez AL, Caruezo V, Taboada M, Rodriguez J, Ginesta V, Rubio J, Garcia-Bengoechea JB, Gonzalez-Juanatey JR. [hemodynamic effects of levosimendan compared with dobutamine in patients with low cardiac output after cardiac surgery]. *Rev Esp Cardiol*. 2006;59:338–345.
601. Jorgensen K, Bech-Hanssen O, Houltz E, Ricksten SE. Effects of levosimendan on left ventricular relaxation and early filling at maintained preload and afterload conditions after aortic valve replacement for aortic stenosis. *Circulation*. 2008;117:1075–1081.
602. Lobato EB, Willert JL, Looke TD, Thomas J, Urdaneta F. Effects of milrinone versus epinephrine on left ventricular relaxation after cardiopulmonary bypass following myocardial revascularization: assessment by color m-mode and tissue Doppler. *J Cardiothorac Vasc Anesth*. 2005;19:334–339.
603. Nijhawan N, Nicolosi AC, Montgomery MW, Aggarwal A, Pagel PS, Warltier DC. Levosimendan enhances cardiac performance after cardiopulmonary bypass: a prospective, randomized placebo-controlled trial. *J Cardiovasc Pharmacol*. 1999;34:219–228.
604. Losert H, Sterz F, Roine RO, Holzer M, Martens P, Cerchiari E, Tiainen M, Mullner M, Laggner AN, Herkner H, Bischof MG. Strict normoglycaemic blood glucose levels in the therapeutic management of patients within 12h after cardiac arrest might not be necessary. *Resuscitation*. 2008;76:214–220.
605. Oksanen T, Skrifvars MB, Varpula T, Kuitunen A, Pettila V, Nurmi J, Castren M. Strict versus moderate glucose control after resuscitation from ventricular fibrillation. *Intensive Care Med*. 2007;33:2093–2100.
606. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in the critically ill patients. *N Engl J Med*. 2001;345:1359–1367.
607. Gandhi GY, Murad MH, Flynn DN, Erwin PJ, Cavalcante AB, Bay Nielsen H, Capes SE, Thorlund K, Montori VM, Devereaux PJ. Effect of perioperative insulin infusion on surgical morbidity and mortality:

systematic review and meta-analysis of randomized trials. *Mayo Clin Proc*. 2008;83:418–430.
608. Griesdale DE, de Souza RJ, van Dam RM, Heyland DK, Cook DJ, Malhotra A, Dhaliwal R, Henderson WR, Chittock DR, Finfer S, Talmor D. Intensive insulin therapy and mortality among critically ill patients: a meta-analysis including NICE-Sugar Study data. *CMAJ*. 2009;180:821–827.
609. Wiener RS, Wiener DC, Larson RJ. Benefits and risks of tight glucose control in critically ill adults: a meta-analysis. *JAMA*. 2008;300:933–944.
610. Van den Berghe G, Wilmer A, Hermans G, Meersseman W, Wouters PJ, Milants I, Van Wijngaerden E, Bobbaers H, Bouillon R. Intensive insulin therapy in the medical ICU. *N Engl J Med*. 2006;354:449–461.
611. Gandhi GY, Nuttall GA, Abel MD, Mullany CJ, Schaff HV, O'Brien PC, Johnson MG, Williams AR, Cutshall SM, Mundy LM, Rizza RA, McMahon MM. Intensive intraoperative insulin therapy versus conventional glucose management during cardiac surgery: a randomized trial. *Ann Intern Med*. 2007;146:233–243.
612. Finfer S, Chittock DR, Su SY, Blair D, Foster D, Dhingra V, Bellomo R, Cook D, Dodek P, Henderson WR, Hebert PC, Heritier S, Heyland DK, McArthur C, McDonald E, Mitchell I, Myburgh JA, Norton R, Potter J, Robinson BG, Ronco JJ. Intensive versus conventional glucose control in critically ill patients. *N Engl J Med*. 2009;360:1283–1297.
613. Treggiari MM, Karir V, Yanez ND, Weiss NS, Daniel S, Deem SA. Intensive insulin therapy and mortality in critically ill patients. *Crit Care*. 2008;12:R29.
614. Vlasselaers D, Milants I, Desmet L, Wouters PJ, Vanhorebeek I, van den Heuvel I, Mesotten D, Casaer MP, Meyfroidt G, Ingels C, Muller J, Van Cromphaut S, Schetz M, Van den Berghe G. Intensive insulin therapy for patients in paediatric intensive care: a prospective, randomised controlled study. *Lancet*. 2009;373:547–556.
615. Beardsall K, Vanhaesebrouck S, Ogilvy-Stuart AL, Vanhole C, Palmer CR, van Weissenbruch M, Midgley P, Thompson M, Thio M, Cornette L, Ossuetta I, Iglesias I, Theyskens C, de Jong M, Ahluwalia JS, de Zegher F, Dunger DB. Early insulin therapy in very-low-birth-weight infants. *N Engl J Med*. 2008;359:1873–1884.
616. Vannucci RC, Vannucci SJ. Hypoglycemic brain injury. *Semin Neonatol*. 2001;6:147–155.
617. Krinsley JS, Grover A. Severe hypoglycemia in critically ill patients: risk factors and outcomes. *Crit Care Med*. 2007;35:2262–2267.
618. Duning T, Ellger B. Is hypoglycaemia dangerous? *Best Pract Res Clin Anaesthesiol*. 2009;23:473–485.
619. Park WS, Chang YS, Lee M. Effects of hyperglycemia or hypoglycemia on brain cell membrane function and energy metabolism during the immediate reoxygenation-reperfusion period after acute transient global hypoxia-ischemia in the newborn piglet. *Brain Res*. 2001;901:102–108.
620. Siesjo BK. Cell damage in the brain: a speculative synthesis. *J Cereb Blood Flow Metab*. 1981;1:155–185.
621. Sieber FE, Traystman RJ. Special issues: glucose and the brain. *Crit Care Med*. 1992;20:104–114.
622. D'Alecy LG, Lundy EF, Barton KJ, Zelenock GB. Dextrose containing intravenous fluid impairs outcome and increases death after eight minutes of cardiac arrest and resuscitation in dogs. *Surgery*. 1986;100:505–511.
623. Katz LM, Wang Y, Ebmeyer U, Radovsky A, Safar P. Glucose plus insulin infusion improves cerebral outcome after asphyxial cardiac arrest. *Neuroreport*. 1998;9:3363–3367.
624. Doolan A, Langlois N, Semsarian C. Causes of sudden cardiac death in young Australians. *Med J Aust*. 2004;180:110–112.
625. Eckart RE, Scoville SL, Campbell CL, Shry EA, Stajduhar KC, Potter RN, Pearse LA, Virmani R. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med*. 2004;141:829–834.
626. Ong ME, Stiell I, Osmond MH, Nesbitt L, Gerein R, Campbell S, McLellan B. Etiology of pediatric out-of-hospital cardiac arrest by coroner's diagnosis. *Resuscitation*. 2006;68:335–342.
627. Puranik R, Chow CK, Duflou JA, Kilborn MJ, McGuire MA. Sudden death in the young. *Heart Rhythm*. 2005;2:1277–1282.
628. Ackerman MJ, Siu BL, Sturner WQ, Tester DJ, Valdivia CR, Makielski JC, Towbin JA. Postmortem molecular analysis of scn5a defects in sudden infant death syndrome. *JAMA*. 2001;286:2264–2269.
629. Arnestad M, Crotti L, Rognum TO, Insolia R, Pedrazzini M, Ferrandi C, Vege A, Wang DW, Rhodes TE, George AL, Jr, Schwartz PJ. Prevalence of long-QT syndrome gene variants in sudden infant death syndrome. *Circulation*. 2007;115:361–367.
630. Cronk LB, Ye B, Kaku T, Tester DJ, Vatta M, Makielski JC, Ackerman MJ. Novel mechanism for sudden infant death syndrome: persistent late sodium current secondary to mutations in caveolin-3. *Heart Rhythm*. 2007;4:161–166.
631. Millat G, Kugener B, Chevalier P, Chahine M, Huang H, Malicier D, Rodriguez-Lafrasse C, Rousson R. Contribution of long-QT syndrome genetic variants in sudden infant death syndrome. *Pediatr Cardiol*. 2009;30:502–509.
632. Otagiri T, Kijima K, Osawa M, Ishii K, Makita N, Matoba R, Umetsu K, Hayasaka K. Cardiac ion channel gene mutations in sudden infant death syndrome. *Pediatr Res*. 2008;64:482–487.
633. Plant LD, Bowers PN, Liu Q, Morgan T, Zhang T, State MW, Chen W, Kittles RA, Goldstein SA. A common cardiac sodium channel variant associated with sudden infant death in African Americans, scn5a s1103y. *J Clin Invest*. 2006;116:430–435.
634. Tester DJ, Dura M, Carturan E, Reiken S, Wronska A, Marks AR, Ackerman MJ. A mechanism for sudden infant death syndrome (SIDS): stress-induced leak via ryanodine receptors. *Heart Rhythm*. 2007;4:733–739.
635. Albert CM, Nam EG, Rimm EB, Jin HW, Hajjar RJ, Hunter DJ, MacRae CA, Ellinor PT. Cardiac sodium channel gene variants and sudden cardiac death in women. *Circulation*. 2008;117:16–23.
636. Chugh SS, Senashova O, Watts A, Tran PT, Zhou Z, Gong Q, Titus JL, Hayflick SJ. Postmortem molecular screening in unexplained sudden death. *J Am Coll Cardiol*. 2004;43:1625–1629.
637. Tester DJ, Spoon DB, Valdivia HH, Makielski JC, Ackerman MJ. Targeted mutational analysis of the ryr2-encoded cardiac ryanodine receptor in sudden unexplained death: a molecular autopsy of 49 medical examiner/coroner's cases. *Mayo Clin Proc*. 2004;79:1380–1384.
638. Behr E, Wood DA, Wright M, Syrris P, Sheppard MN, Casey A, Davies MJ, McKenna W. Cardiological assessment of first-degree relatives in sudden arrhythmic death syndrome. *Lancet*. 2003;362:1457–1459.
639. Behr ER, Dalageorgou C, Christiansen M, Syrris P, Hughes S, Tome Esteban MT, Rowland E, Jeffery S, McKenna WJ. Sudden arrhythmic death syndrome: familial evaluation identifies inheritable heart disease in the majority of families. *Eur Heart J*. 2008;29:1670–1680.
640. Hofman N, Tan HL, Clur SA, Alders M, van Langen IM, Wilde AA. Contribution of inherited heart disease to sudden cardiac death in childhood. *Pediatrics*. 2007;120:e967–e973.
641. Tan HL, Hofman N, van Langen IM, van der Wal AC, Wilde AA. Sudden unexplained death: heritability and diagnostic yield of cardiological and genetic examination in surviving relatives. *Circulation*. 2005;112:207–213.
642. Calkins CM, Bensard DD, Partrick DA, Karrer FM. A critical analysis of outcome for children sustaining cardiac arrest after blunt trauma. *J Pediatr Surg*. 2002;37:180–184.
643. Crewdson K, Lockey D, Davies G. Outcome from paediatric cardiac arrest associated with trauma. *Resuscitation*. 2007;75:29–34.
644. Lopez-Herce Cid J, Dominguez Sampedro P, Rodriguez Nunez A, Garcia Sanz C, Carrillo Alvarez A, Calvo Macias C, Bellon Cano JM. [cardiorespiratory arrest in children with trauma]. *An Pediatr (Barc)*. 2006;65:439–447.
645. Perron AD, Sing RF, Branas CC, Huynh T. Predicting survival in pediatric trauma patients receiving cardiopulmonary resuscitation in the prehospital setting. *Prehosp Emerg Care*. 2001;5:6–9.
646. Sheikh A, Brogan T. Outcome and cost of open- and closed-chest cardiopulmonary resuscitation in pediatric cardiac arrests. *Pediatrics*. 1994;93:392–398.
647. Beaver BL, Colombani PM, Buck JR, Dudgeon DL, Bohrer SL, Haller JA Jr. Efficacy of emergency room thoracotomy in pediatric trauma. *J Pediatr Surg*. 1987;22:19–23.
648. Powell RW, Gill EA, Jurkovich GJ, Ramenofsky ML. Resuscitative thoracotomy in children and adolescents. *Am Surg*. 1988;54:188–191.
649. Rothenberg SS, Moore EE, Moore FA, Baxter BT, Moore JB, Cleveland HC. Emergency department thoracotomy in children–a critical analysis. *J Trauma*. 1989;29:1322–1325.
650. Suominen P, Rasanen J, Kivioja A. Efficacy of cardiopulmonary resuscitation in pulseless paediatric trauma patients. *Resuscitation*. 1998;36:9–13.
651. Graham EM, Forbus GA, Bradley SM, Shirali GS, Atz AM. Incidence and outcome of cardiopulmonary resuscitation in patients with shunted single ventricle: advantage of right ventricle to pulmonary artery shunt. *J Thorac Cardiovasc Surg*. 2006;131:e7–e8.

652. Ramamoorthy C, Tabbutt S, Kurth CD, Steven JM, Montenegro LM, Durning S, Wernovsky G, Gaynor JW, Spray TL, Nicolson SC. Effects of inspired hypoxic and hypercapnic gas mixtures on cerebral oxygen saturation in neonates with univentricular heart defects. *Anesthesiology*. 2002;96:283–288.
653. Tabbutt S, Ramamoorthy C, Montenegro LM, Durning SM, Kurth CD, Steven JM, Godinez RI, Spray TL, Wernovsky G, Nicolson SC. Impact of inspired gas mixtures on preoperative infants with hypoplastic left heart syndrome during controlled ventilation. *Circulation*. 2001;104: I159–164.
654. Charpie JR, Dekeon MK, Goldberg CS, Mosca RS, Bove EL, Kulik TJ. Postoperative hemodynamics after Norwood palliation for hypoplastic left heart syndrome. *Am J Cardiol*. 2001;87:198–202.
655. Hoffman GM, Mussatto KA, Brosig CL, Ghanayem NS, Musa N, Fedderly RT, Jaquiss RD, Tweddell JS. Systemic venous oxygen saturation after the Norwood procedure and childhood neurodevelopmental outcome. *J Thorac Cardiovasc Surg*. 2005;130:1094–1100.
656. Johnson BA, Hoffman GM, Tweddell JS, Cava JR, Basir M, Mitchell ME, Scanlon MC, Mussatto KA, Ghanayem NS. Near-infrared spectroscopy in neonates before palliation of hypoplastic left heart syndrome. *Ann Thorac Surg*. 2009;87:571–577; discussion 577–579.
657. Bradley SM, Simsic JM, Atz AM. Hemodynamic effects of inspired carbon dioxide after the Norwood procedure. *Ann Thorac Surg*. 2001; 72:2088–2093; discussion 2093–2084.
658. De Oliveira NC, Van Arsdell GS. Practical use of alpha blockade strategy in the management of hypoplastic left heart syndrome following stage one palliation with a Blalock-Taussig shunt. *Semin Thorac Cardiovasc Surg Pediatr Card Surg Annu*. 2004;7:11–15.
659. Hoffman GM, Tweddell JS, Ghanayem NS, Mussatto KA, Stuth EA, Jaquis RD, Berger S. Alteration of the critical arteriovenous oxygen saturation relationship by sustained afterload reduction after the Norwood procedure. *J Thorac Cardiovasc Surg*. 2004;127:738–745.
660. Tweddell JS, Hoffman GM, Mussatto KA, Fedderly RT, Berger S, Jaquiss RD, Ghanayem NS, Frisbee SJ, Litwin SB. Improved survival of patients undergoing palliation of hypoplastic left heart syndrome: lessons learned from 115 consecutive patients. *Circulation*. 2002;106: I82–89.
661. Raymond TT, Cunnyngham CB, Thompson MT, Thomas JA, Dalton HJ, Nadkarni VM. Outcomes among neonates, infants, and children after extracorporeal cardiopulmonary resuscitation for refractory inhospital pediatric cardiac arrest: a report from the National Registry of Cardiopulmonary Resuscitation. *Pediatr Crit Care Med.* 2010;11: 362–371.
662. Tajik M, Cardarelli MG. Extracorporeal membrane oxygenation after cardiac arrest in children: what do we know? *Eur J Cardiothorac Surg*. 2008;33:409–417.
663. Booth KL, Roth SJ, Thiagarajan RR, Almodovar MC, del Nido PJ, Laussen PC. Extracorporeal membrane oxygenation support of the Fontan and bidirectional Glenn circulations. *Ann Thorac Surg*. 2004;77: 1341–1348.
664. Tewari P, Babu SG. Resuscitation after modified Fontan procedure. *Ann Thorac Surg*. 1994;58:880–882.
665. Hoskote A, Li J, Hickey C, Erickson S, Van Arsdell G, Stephens D, Holtby H, Bohn D, Adatia I. The effects of carbon dioxide on oxygenation and systemic, cerebral, and pulmonary vascular hemodynamics after the bidirectional superior cavopulmonary anastomosis. *J Am Coll Cardiol*. 2004;44:1501–1509.
666. Li J, Hoskote A, Hickey C, Stephens D, Bohn D, Holtby H, Van Arsdell G, Redington AN, Adatia I. Effect of carbon dioxide on systemic oxygenation, oxygen consumption, and blood lactate levels after bidirectional superior cavopulmonary anastomosis. *Crit Care Med*. 2005; 33:984–989.
667. Fogel MA, Durning S, Wernovsky G, Pollock AN, Gaynor JW, Nicolson S. Brain versus lung: hierarchy of feedback loops in single-ventricle patients with superior cavopulmonary connection. *Circulation.* 2004;110:II147–152.
668. Bradley SM, Simsic JM, Mulvihill DM. Hypoventilation improves oxygenation after bidirectional superior cavopulmonary connection. *J Thorac Cardiovasc Surg*. 2003;126:1033–1039.
669. Bradley SM, Simsic JM, Mulvihill DM. Hyperventilation impairs oxygenation after bidirectional superior cavopulmonary connection. *Circulation.* 1998;98:II372–376; discussion II376–377.
670. Mott AR, Alomrani A, Tortoriello TA, Perles Z, East DL, Stayer SA. Changes in cerebral saturation profile in response to mechanical ventilation alterations in infants with bidirectional superior cavopulmonary connection. *Pediatr Crit Care Med*. 2006;7:346–350.
671. Shekerdemian LS, Shore DF, Lincoln C, Bush A, Redington AN. Negative-pressure ventilation improves cardiac output after right heart surgery. *Circulation.* 1996;94:II49–55.
672. Shekerdemian LS, Bush A, Shore DF, Lincoln C, Redington AN. Cardiopulmonary interactions after Fontan operations: augmentation of cardiac output using negative pressure ventilation. *Circulation*. 1997; 96:3934–3942.
673. Meliones JN, Bove EL, Dekeon MK, Custer JR, Moler FW, Callow LR, Wilton NC, Rosen DB. High-frequency jet ventilation improves cardiac function after the Fontan procedure. *Circulation.* 1991;84:III364–368.
674. Kornecki A, Shekerdemian LS, Adatia I, Bohn D. High-frequency oscillation in children after Fontan operation. *Pediatr Crit Care Med*. 2002;3:144–147.
675. Burrows FA. Physiologic dead space, venous admixture, and the arterial to end-tidal carbon dioxide difference in infants and children undergoing cardiac surgery. *Anesthesiology*. 1989;70:219–225.
676. Polderman FN, Cohen J, Blom NA, Delhaas T, Helbing WA, Lam J, Sobotka-Plojhar MA, Temmerman AM, Sreeram N. Sudden unexpected death in children with a previously diagnosed cardiovascular disorder. *Int J Cardiol*. 2004;95:171–176.
677. Sanatani S, Wilson G, Smith CR, Hamilton RM, Williams WG, Adatia I. Sudden unexpected death in children with heart disease. *Congenit Heart Dis*. 2006;1:89–97.
678. Hoeper MM, Galie N, Murali S, Olschewski H, Rubenfire M, Robbins IM, Farber HW, McLaughlin V, Shapiro S, Pepke-Zaba J, Winkler J, Ewert R, Opitz C, Westerkamp V, Vachiery JL, Torbicki A, Behr J, Barst RJ. Outcome after cardiopulmonary resuscitation in patients with pulmonary arterial hypertension. *Am J Respir Crit Care Med*. 2002;165: 341–344.
679. Khan TA, Schnickel G, Ross D, Bastani S, Laks H, Esmailian F, Marelli D, Beygui R, Shemin R, Watson L, Vartapetian I, Ardehali A. A prospective, randomized, crossover pilot study of inhaled nitric oxide versus inhaled prostacyclin in heart transplant and lung transplant recipients. *J Thorac Cardiovasc Surg*. 2009;138:1417–1424.
680. Rimensberger PC, Spahr-Schopfer I, Berner M, Jaeggi E, Kalangos A, Friedli B, Beghetti M. Inhaled nitric oxide versus aerosolized iloprost in secondary pulmonary hypertension in children with congenital heart disease: vasodilator capacity and cellular mechanisms. *Circulation*. 2001;103:544–548.
681. Limsuwan A, Wanitkul S, Khosithset A, Attanavanich S, Samankatiwat P. Aerosolized iloprost for postoperative pulmonary hypertensive crisis in children with congenital heart disease. *Int J Cardiol*. 2008;129: 333–338.
682. Morris K, Beghetti M, Petros A, Adatia I, Bohn D. Comparison of hyperventilation and inhaled nitric oxide for pulmonary hypertension after repair of congenital heart disease. *Crit Care Med*. 2000;28: 2974–2978.
683. Strueber M, Hoeper MM, Fischer S, Cypel M, Warnecke G, Gottlieb J, Pierre A, Welte T, Haverich A, Simon AR, Keshavjee S. Bridge to thoracic organ transplantation in patients with pulmonary arterial hypertension using a pumpless lung assist device. *Am J Transplant*. 2009;9: 853–857.
684. Liu KS, Tsai FC, Huang YK, Wu MY, Chang YS, Chu JJ, Lin PJ. Extracorporeal life support: a simple and effective weapon for postcardiotomy right ventricular failure. *Artif Organs*. 2009;33:504–508.
685. Dhillon R, Pearson GA, Firmin RK, Chan KC, Leanage R. Extracorporeal membrane oxygenation and the treatment of critical pulmonary hypertension in congenital heart disease. *Eur J Cardiothorac Surg*. 1995;9:553–556.
686. Arpesella G, Loforte A, Mikus E, Mikus PM. Extracorporeal membrane oxygenation for primary allograft failure. *Transplant Proc*. 2008;40: 3596–3597.
687. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. *Ann Emerg Med*. 1987;16:1107–1111.
688. Gillis J, Dickson D, Rieder M, Steward D, Edmonds J. Results of inpatient pediatric resuscitation. *Crit Care Med*. 1986;14:469–471.
689. Suominen P, Olkkola KT, Voipio V, Korpela R, Palo R, Rasanen J. Utstein style reporting of in-hospital paediatric cardiopulmonary resuscitation. *Resuscitation*. 2000;45:17–25.
690. Nadkarni VM, Larkin GL, Peberdy MA, Carey SM, Kaye W, Mancini ME, Nichol G, Lane-Truitt T, Potts J, Ornato JP, Berg RA. First documented rhythm and clinical outcome from in-hospital cardiac arrest among children and adults. *JAMA*. 2006;295:50–57.

691. Tibballs J, Kinney S. A prospective study of outcome of in-patient paediatric cardiopulmonary arrest. *Resuscitation*. 2006;71:310–318.
692. Slonim AD, Patel KM, Ruttimann UE, Pollack MM. Cardiopulmonary resuscitation in pediatric intensive care units. *Crit Care Med*. 1997;25: 1951–1955.
693. Rodriguez-Nunez A, Lopez-Herce J, Garcia C, Carrillo A, Dominguez P, Calvo C, Delgado MA. Effectiveness and long-term outcome of cardiopulmonary resuscitation in paediatric intensive care units in Spain. *Resuscitation*. 2006;71:301–309.
694. Suominen P, Baillie C, Korpela R, Rautanen S, Ranta S, Olkkola KT. Impact of age, submersion time and water temperature on outcome in near-drowning. *Resuscitation*. 2002;52:247–254.
695. Innes PA, Summers CA, Boyd IM, Molyneux EM. Audit of paediatric cardiopulmonary resuscitation. *Arch Dis Child*. 1993;68:487–491.
696. Young KD, Gausche-Hill M, McClung CD, Lewis RJ. A prospective, population-based study of the epidemiology and outcome of out-of-hospital pediatric cardiopulmonary arrest. *Pediatrics*. 2004;114: 157–164.
697. Srinivasan V, Nadkarni VM, Helfaer MA, Carey SM, Berg RA. Childhood obesity and survival after in-hospital pediatric cardiopulmonary resuscitation. *Pediatrics*. 2010;125:e481–e488.
698. Donoghue AJ, Nadkarni V, Berg RA, Osmond MH, Wells G, Nesbitt L, Stiell IG. Out-of-hospital pediatric cardiac arrest: An epidemiologic review and assessment of current knowledge. *Ann Emerg Med*. 2005; 46:512–522.
699. Quan L, Kinder D. Pediatric submersions: prehospital predictors of outcome. *Pediatrics*. 1992;90:909–913.
700. Waugh JH, O'Callaghan MJ, Pitt WR. Prognostic factors and long-term outcomes for children who have nearly drowned. *Med J Aust.* 1994;161: 594–595, 598–599.
701. Hazinski MF, Chahine AA, Holcomb GW III, Morris JA Jr. Outcome of cardiovascular collapse in pediatric blunt trauma. *Ann Emerg Med*. 1994;23:1229–1235.
702. Fisher B, Worthen M. Cardiac arrest induced by blunt trauma in children. *Pediatr Emerg Care*. 1999;15:274–276.
703. Lin YR, Wu HP, Huang CY, Chang YJ, Lin CY, Chou CC. Significant factors in predicting sustained rosc in paediatric patients with traumatic out-of-hospital cardiac arrest admitted to the emergency department. *Resuscitation*. 2007;74:83–89.
704. Eich C, Brauer A, Timmermann A, Schwarz SK, Russo SG, Neubert K, Graf BM, Aleksic I. Outcome of 12 drowned children with attempted resuscitation on cardiopulmonary bypass: an analysis of variables based on the 'Utstein style for drowning.' *Resuscitation*. 2007;75:42–52.
705. Li G, Tang N, DiScala C, Meisel Z, Levick N, Kelen GD. Cardiopulmonary resuscitation in pediatric trauma patients: survival and functional outcome. *J Trauma*. 1999;47:1–7.

KEY WORDS: arrhythmia ■ cardiopulmonary resuscitation ■ pediatrics ■ resuscitation

Part 10: Pediatric Basic and Advanced Life Support: 2010 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations

Monica E. Kleinman, Allan R. de Caen, Leon Chameides, Dianne L. Atkins, Robert A. Berg, Marc D. Berg, Farhan Bhanji, Dominique Biarent, Robert Bingham, Ashraf H. Coovadia, Mary Fran Hazinski, Robert W. Hickey, Vinay M. Nadkarni, Amelia G. Reis, Antonio Rodriguez-Nunez, James Tibballs, Arno L. Zaritsky and David Zideman

Circulation. 2010;122:S466-S515
doi: 10.1161/CIRCULATIONAHA.110.971093
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539