Part 6: Pediatric Basic and Advanced Life Support

Circulation 2005;112;III-73-III-90
DOI: 10.1161/CIRCULATIONAHA.105.166476
Circulation is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0009-7322. Online ISSN: 1524-4539

Part 6: Pediatric Basic and Advanced Life Support

The ILCOR Pediatric Task Force included expert reviewers from Africa, Asia, Australia, Asia, Europe, North America, and South America. These experts reviewed 45 topics related to pediatric resuscitation. Topics were selected from previous recommendations (the *ECC Guidelines 2000*[1,2]), emerging science, and newly identified issues. Some well-established topics without controversies or new evidence (eg, adenosine for the treatment of supraventricular tachycardia [SVT]) are not included in this document.

Evidence-based worksheets on some topics were prepared and discussed but are not included here because there was insufficient evidence (eg, fibrinolytics in cardiac arrest,[W13] securing the endotracheal tube in children,[W1] use of impedance threshold device in children,[W2] sodium bicarbonate for prolonged resuscitation attempts[W34]) or because no new evidence was found (eg, evaluation of capillary refill,[W10] ventilation before naloxone,[W18] delayed volume resuscitation in trauma,[W17] use of hypertonic saline in shock[W16]).

The following is a summary of the most important changes in recommendations for pediatric resuscitation since the last ILCOR review in 2000.[1,2] The scientific evidence supporting these recommendations is summarized in this document:

- Emphasis on the quality of CPR is increased: "Push hard, push fast, minimize interruptions; allow full chest recoil, and don't hyperventilate"
  - —Recommended chest compression-ventilation ratio:
    - For 1 lay rescuer and lone healthcare provider: 30:2
    - For healthcare providers performing 2-rescuer CPR: 15:2
  - —Either the 2- or 1-hand technique is acceptable for chest compressions in children
  - —1 initial shock followed by immediate CPR for attempted defibrillation, instead of 3 stacked shocks
- Biphasic attenuated shocks with an automated external defibrillator (AED) are acceptable for children ≥1 year of age.
- Routine use of high-dose epinephrine is no longer recommended.
- Either cuffed or uncuffed tracheal tubes are acceptable in infants and children.
- Exhaled CO_2 monitoring is recommended for confirmation of tracheal tube placement and during transport.
- Consider induced hypothermia for patients who remain comatose following resuscitation.
- Emphasis is increased on intravascular (intravenous [IV] and intraosseous [IO]) rather than tracheal administration of drugs.

The ILCOR Pediatric Task Force reevaluated the definitions of newborn, infant, child, and adult. These definitions are somewhat arbitrary but are important because some recommendations for treatment differ according to patient size and the most likely etiology of arrest. The distinction between child and adult victims has been deemphasized by the recommendation of a universal compression-ventilation ratio for lay rescuers and the same chest compression technique for lay rescuers of children and adults. Some differences in treatment recommendations remain between the newborn and infant and between an infant and child, but those differences are chiefly linked to resuscitation training and practice. They are noted below.

Identified knowledge gaps in pediatric resuscitation include

- Sensitive and specific indicators of cardiac arrest that lay rescuers and healthcare providers can recognize reliably
- Effectiveness of etiology-based versus age-based resuscitation sequences
- The ideal ratio of chest compressions to ventilations during CPR
- Mechanisms to monitor and optimize quality of CPR during attempted resuscitation
- Best methods for securing a tracheal tube
- Clinical data on the safety and efficacy of automated external defibrillators (AEDs)
- Clinical data on the safety and efficacy of the laryngeal mask airway (LMA) during cardiac arrest
- The benefits and risks of supplementary oxygen during and after CPR
- Clinical data on antiarrhythmic and pressor medications during cardiac arrest
- Data on induced hypothermia in pediatric cardiac arrest
- The identification and treatment of postarrest myocardial dysfunction
- The use of fibrinolytics and anticoagulants in cardiac arrest
- Use of emerging technologies for assessment of tissue perfusion
- Predictors of outcome from cardiac arrest

Initial Steps of CPR

The *ECC Guidelines 2000*[1] recommended that lone rescuers of *adult* victims of cardiac arrest phone the emergency medical services (EMS) system and get an AED ("call first") before starting CPR. The lone rescuer of an unresponsive *infant or child* victim was instructed to provide a brief period

From the 2005 International Consensus Conference on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations, hosted by the American Heart Association in Dallas, Texas, January 23–30, 2005.

This article has been copublished in *Resuscitation*.

(*Circulation.* 2005;112:III-73-III-90.)

This special supplement to *Circulation* is freely available at http://www.circulationaha.org DOI: 10.1161/CIRCULATIONAHA.105.166476

of CPR before leaving the victim to phone for professional help and an AED ("call fast"). These sequence differences were based on the supposition that cardiac arrest in adults is due primarily to ventricular fibrillation (VF) and that a hypoxic-ischemic mechanism is more common in children. But this simplistic approach may be inaccurate and may not provide the ideal rescue sequence for many victims of cardiac arrest. Hypoxic-ischemic arrest may occur in adults, and VF may be the cause of cardiac arrest in up to 7% to 15% of infants and children. Resuscitation results might be improved if the sequence of lay rescuer CPR actions (ie, the priority of phoning for professional help, getting an AED, and providing CPR) is based on the etiology of cardiac arrest rather than age.

The pulse check was previously eliminated as an assessment for the lay rescuer. There is now evidence that healthcare professionals may take too long to check for a pulse and may not accurately determine the presence or absence of the pulse. This may lead to interruptions in chest compressions and affect the quality of CPR.

Experts reviewed the data on the technique of rescue breathing for infants and the 2-thumb–encircling hands versus 2-finger chest compression techniques for infants.

One of the most challenging topics debated during the 2005 Consensus Conference was the compression-ventilation ratio. The scientific evidence on which to base recommendations was sparse, and it was difficult to arrive at consensus. Evidence was presented that the ratio should be higher than 5:1, but the optimal ratio was not identified. The only data addressing a compression-ventilation ratio greater than 15:2 came from mathematical models. The experts acknowledged the educational benefit of simplifying training for lay rescuers (specifically 1-rescuer CPR) by adopting a single ratio for infants, children, and adults with the hope that simplification might increase the number of bystanders who will learn, remember, and perform CPR. On this basis experts agreed that this single compression-ventilation ratio should be 30:2. Healthcare providers will typically be experienced in CPR and practice it frequently. This group of experienced providers will learn 2-person CPR, and for them the recommended compression-ventilation ratio for 2 rescuers is 15:2.

Some laypeople are reluctant to perform mouth-to-mouth ventilation. For treatment of cardiac arrest in infants and children, chest compressions alone are better than no CPR but not as good as a combination of ventilations and compressions.

In the past 1-handed chest compressions were recommended for CPR in children. A review of the evidential basis for this recommendation was conducted. From an educational standpoint, we agree that it will simplify training to recommend a single technique for chest compressions for children and adults.

Activating Emergency Medical Services and Getting the AED[W4]

Consensus on Science

Most cardiac arrests in children are caused by asphyxia (LOE 4).[3–6] Observational studies of non-VF arrests in children show an association between bystander CPR and intact neurologic outcome (LOE 4).[6–8] Animal studies show that in asphyxial arrest, chest compressions plus ventilation CPR is superior to either chest compression-only CPR or ventilation-only CPR (LOE 6).[9]

Observational studies of children with VF report good (17% to 20%) rates of survival after early defibrillation (LOE 4).[5,6,10] The merits of "call first" versus "call fast" CPR sequences have not been adequately studied in adults or children with cardiac arrest of asphyxial or VF etiologies. Three animal studies (LOE 6)[9,11,12] show that even in prolonged VF, CPR increases the likelihood of successful defibrillation, and 7 adult human studies (LOE 7)[13–19] document improved survival with the combination of CPR with minimal interruptions in chest compression and early defibrillation.

Treatment Recommendation

A period of immediate CPR before phoning emergency medical services (EMS) and getting the AED ("call fast") is indicated for most pediatric arrests because they are presumed to be asphyxial or prolonged. In a witnessed sudden collapse (eg, during an athletic event), the cause is more likely to be VF, and the lone rescuer should phone for professional help and get the AED (when available) before starting CPR and using the AED, if appropriate. Rescuers should perform CPR with minimal interruptions in chest compressions until attempted defibrillation.

In summary, the priorities for *unwitnessed* or nonsudden collapse in children are as follows:

- Start CPR immediately.
- Activate EMS/get the AED.

The priorities for *witnessed* sudden collapse in children are as follows:

- Activate EMS/get the AED.
- Start CPR.
- Attempt defibrillation.

Pulse Check[W5A,W5B]

Consensus on Science

Ten studies (LOE 2[20,21]; LOE 4[22–26]; LOE 5[27]; LOE 6[28,29]) show that lay rescuers[23,25,30] and healthcare providers[20,21,24,26–29] are often unable to accurately determine the presence of a pulse within 10 seconds. Two studies in infants (LOE 5)[31,32] reported that rescuers rapidly detected cardiac activity by direct chest auscultation but were biased because they knew that the infants were healthy.

Treatment Recommendation

Lay rescuers should start chest compressions for an unresponsive infant or child who is not moving or breathing. Healthcare professionals may also check for a pulse but should proceed with CPR if they cannot feel a pulse within 10 seconds or are uncertain if a pulse is present.

Ventilations in Infants[W7A,W7B]

Consensus on Science

One LOE 5[33] study and 10 LOE 7[34–43] reports assessed a mouth-to-nose ventilation technique for infants. The LOE 5

study[33] is an anecdotal report of 3 infants ventilated with mouth-to-nose technique. The LOE 7 reports describe postmortem anatomy,[34] physiology of nasal breathing,[35–37] related breathing issues,[38,39] and measurements of infants' faces compared with the measurement of adult mouths.[40–43] There is great variation in these measurements, probably because of imprecise or inconsistent definitions.

Treatment Recommendation
There is no data to justify a change from the recommendation that the rescuer attempt mouth-to–mouth-and-nose ventilation for infants. Rescuers who have difficulty achieving a tight seal over the mouth and nose of an infant, however, may attempt either mouth-to-mouth or mouth-to-nose ventilation (LOE 5).[33]

Circumferential Versus 2-Finger Chest Compressions[W9A,W9B]

Consensus on Science
Two manikin (LOE 6)[44,45] and 2 animal (LOE 6)[46,47] studies showed that the 2 thumb–encircling hands technique of chest compressions with circumferential thoracic squeeze produces higher coronary perfusion pressures and more consistently correct depth and force of compression than the 2-finger technique.

Case reports (LOE 5)[48,49] of hemodynamic monitoring in infants receiving chest compressions showed higher systolic and diastolic arterial pressures in the 2-thumb–encircling hands technique compared with the 2-finger technique.

Treatment Recommendation
The 2 thumb–encircling hands chest compression technique with thoracic squeeze is the preferred technique for 2-rescuer infant CPR. The 2-finger technique is recommended for 1-rescuer infant CPR to facilitate rapid transition between compression and ventilation and to minimize interruptions in chest compressions. It remains an acceptable alternative method of chest compressions for 2 rescuers.

One- Versus 2-Hand Chest Compression Technique[W276]

Consensus on Science
There are no outcome studies that compare 1- versus 2-hand compressions of the chest in children. One (LOE 6)[50] study reported higher pressures generated in child manikins using the 2-hand technique to compress over the lower part of the sternum to a depth of approximately one third the anterior-posterior diameter of the chest. Rescuers reported that this technique was easy to perform.

Treatment Recommendation
Both the 1- and 2-hand techniques for chest compressions in children are acceptable provided that rescuers compress over the lower part of the sternum to a depth of approximately one third the anterior-posterior diameter of the chest. To simplify education, rescuers can be taught the same technique (ie, 2-hand) for adult and child compressions.

Compression-Ventilation Ratio[W3A,W3B,W3C]

Consensus on Science
There is insufficient data to identify an optimal compression-ventilation ratio for CPR in children. Manikin studies (LOE 6)[51–54] have examined the feasibility of compression-ventilation ratios of 15:2 and 5:1. Lone rescuers cannot deliver the desired number of chest compressions per minute at a ratio of 5:1. A mathematical model (LOE 7)[55] supports compression-ventilation ratios higher than 5:1 for infants and children.

Two animal (LOE 6)[56,57] studies show that coronary perfusion pressure, a major determinant of success in resuscitation, declines with interruptions in chest compressions. In addition, once compressions are interrupted, several chest compressions are needed to restore coronary perfusion pressure. Frequent interruptions of chest compressions (eg, with a 5:1 compression-ventilation ratio) prolongs the duration of low coronary perfusion pressure. Long interruptions in chest compressions have been documented in manikin studies (LOE 6)[58,59] and both in- and out-of-hospital adult CPR studies (LOE 7).[60,61] These interruptions reduce the likelihood of a return of spontaneous circulation (LOE 7).[62–64]

Five animal (LOE 6)[9,56,57,65,66] studies and one review (LOE 7)[67] suggest that ventilations are relatively less important in victims with VF or pulseless ventricular tachycardia (VT) cardiac arrest than in victims with asphyxia-induced arrest. But even in asphyxial arrest, few 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 Recommendation
For ease of teaching and retention, a universal compression-ventilation ratio of 30:2 is recommended for the lone rescuer responding to infants (for neonates see Part 7: "Neonatal Resuscitation"), children, and adults. For healthcare providers performing 2-rescuer CPR, a compression-ventilation ratio of 15:2 is recommended. When an advanced airway is established (eg, a tracheal tube, esophageal-tracheal combitube [Combitube], or laryngeal mask airway [LMA]), ventilations are given without interrupting chest compressions.

Some CPR Versus No CPR[W8]

Consensus on Science
Numerous reports (LOE 5)[4,5,8,68–70] document survival of children after cardiac arrest when bystander CPR was provided. Bystander CPR in these reports included rescue breathing alone, chest compressions alone, or a combination of compressions and ventilations.

One prospective and 3 retrospective studies of adult VF (LOE 7)[71–74] and numerous animal studies of VF cardiac arrest (LOE 6)[56,57,66,75–79] document comparable long-term survival after chest compressions alone or chest compressions plus ventilations, and both techniques result in better outcomes compared with no CPR. In animals with asphyxial arrest (LOE 6),[9] the more common mechanism of cardiac arrest in infants and children, best results are achieved with a combination of chest compressions and ventilations. But resuscitation with either ventilations only or chest compressions only is better than no CPR.

Treatment Recommendation
Bystander CPR is important for survival from cardiac arrest. Trained rescuers should be encouraged to provide both

ventilations and chest compressions. If rescuers are reluctant to provide rescue breaths, however, they should be encouraged to perform chest compressions alone without interruption.

Disturbances in Cardiac Rhythm

Evidence evaluation for the treatment of hemodynamically stable arrhythmias focused on vagal maneuvers, amiodarone, and procainamide. There was no new data to suggest a change in the indications for vagal maneuvers or procainamide. Several case series described the safe and effective use of amiodarone in children, but these studies involved selected patient populations (often with postoperative arrhythmias) treated by experienced providers in controlled settings. Although there is no change in the recommendation for amiodarone as a treatment option in children with stable arrhythmias, providers are encouraged to consult with an expert knowledgeable in pediatric arrhythmias before initiating drug therapy.

There is insufficient evidence to identify an optimal shock waveform, energy dose, and shock strategy (eg, fixed versus escalating shocks, 1 versus 3 stacked shocks) for defibrillation. The new recommendation for the sequence of defibrillation in children is based on extrapolated data from adult and animal studies with biphasic devices, data documenting the high rates of success for first shock conversion of VF with biphasic waveforms, and knowledge that interruption of chest compressions reduces coronary perfusion pressure. Thus, a 1-shock strategy may be preferable to the 3-shock sequence recommended in the *ECC Guidelines 2000*.[2] For further details, see Part 3: "Defibrillation."

Many but not all AED algorithms have been shown to be sensitive and specific for recognizing shockable arrhythmias in children. A standard AED ("adult" AED with adult pad-cable system) can be used for children older than about 8 years of age and weighing more than about 25 kg. Many manufacturers now provide a method for attenuating the energy delivered to make the AED suitable for smaller children (eg, use of a pad-cable system or an AED with a key or switch to select a smaller dose).

Management of Supraventricular Tachycardias

If the child with SVT is hemodynamically stable, we recommend early consultation with a pediatric cardiologist or other physician with appropriate expertise. This recommendation is common for all of the SVT topics below.

Vagal Maneuvers for SVT[W36]

Consensus on Science

One prospective (LOE 3)[80] and 9 observational studies (LOE 4[81]; LOE 5[82,83]; LOE 7[84–89]) show that vagal maneuvers are somewhat effective in terminating SVT in children. There are reports of complications from carotid sinus massage and application of ice to the face to stimulate the diving reflex (LOE 5),[90,91] but virtually none from the Valsalva maneuver.

Treatment Recommendation

The Valsalva maneuver and ice application to the face may be used to treat hemodynamically stable SVT in infants and children. When performed correctly, these maneuvers can be initiated quickly and safely and without altering subsequent therapies if they fail.

Amiodarone for Hemodynamically Stable SVT[W38]

Consensus on Science

One prospective (LOE 3)[92] and 10 observational (LOE 5)[93–102] studies show that amiodarone is effective for treating SVT in children. A limitation of this evidence is that most of the studies in children describe treatment for postoperative junctional ectopic tachycardia.

Treatment Recommendation

Amiodarone may be considered in the treatment of hemodynamically stable SVT refractory to vagal maneuvers and adenosine. Rare but significant acute side effects include bradycardia, hypotension, and polymorphic VT (LOE 5).[103–105]

Procainamide for Hemodynamically Stable SVT[W37]

Consensus on Science

Experience with procainamide in children is limited. Twelve LOE 5[106–117] and 4 LOE 6[118–121] observational studies show that procainamide can terminate SVT that is resistant to other drugs. Most of these reports include mixed adult-pediatric populations. Hypotension following procainamide infusion results from its vasodilator action rather than a negative inotropic effect.(LOE 5[122,123]; LOE 6[124]).

Treatment Recommendation

Procainamide may be considered in the treatment of hemodynamically stable SVT refractory to vagal maneuvers and adenosine.

Management of Stable Wide-QRS Tachycardia

If a child with wide-QRS tachycardia is hemodynamically stable, early consultation with a pediatric cardiologist or other physician with appropriate expertise is recommended. In general, amiodarone and procainamide should not be administered together because their combination may increase risk of hypotension and ventricular arrhythmias.

Amiodarone[W39A,W39B,W40]

Consensus on Science

One case series (LOE 5)[125] suggests that wide-QRS tachycardia in children is more likely to be supraventricular than ventricular in origin. Two prospective studies (LOE 3)[92,126] and 13 case series (LOE 5)[93–102,127–129] show that amiodarone is effective for a wide variety of tachyarrhythmias in children. None of these reports specifically evaluates the role of amiodarone in the setting of a stable, unknown wide-complex tachycardia.

Treatment Recommendation

Wide-QRS tachycardia in children who are stable may be treated as SVT. If the diagnosis of VT is confirmed, amiodarone should be considered.

Procainamide for Stable VT[W35]

Consensus on Science

Twenty (LOE 5)[106,115,123,130–146] and 2 LOE 6[118,124] observational studies primarily in adults but including some children

show that procainamide is effective in the treatment of stable VT.

Treatment Recommendation
Procainamide may be considered in the treatment of hemodynamically stable VT.

Management of Unstable VT

Amiodarone[W39A,W40]

Consensus on Science
In small pediatric case series (LOE 3[100]; LOE 5[93,95,97,99,147–149]) and extrapolation from animal (LOE 6)[150,151] and adult (LOE 7)[152–165] studies, amiodarone is safe and effective for hemodynamically unstable VT in children.

Treatment Recommendation
Synchronized cardioversion remains the treatment of choice for unstable VT. Amiodarone may be considered for treatment of hemodynamically unstable VT.

Pediatric Defibrillation

For additional information about consensus on science and treatment recommendations for defibrillation (eg, 1 versus 3 stacked shock sequences and sequence of CPR first versus defibrillation first), see Part 3: "Defibrillation."

Manual and Automated External Defibrillation[W41A,W41B]

Consensus on Science
The ideal energy dose for safe and effective defibrillation in children is unknown. Extrapolation from adult data (LOE 1[166,167]; LOE 2[168–170]) and pediatric animal studies (LOE 6)[171–173] suggests that biphasic shocks are at least as effective as monophasic shocks and produce less postshock myocardial dysfunction. One LOE 5[174] and one LOE 6[171] study show that an initial monophasic or biphasic shock dose of 2 J/kg generally terminates pediatric VF. Two pediatric case series (LOE 5)[171,175,176] report that doses >4 J/kg (up to 9 J/kg) have effectively defibrillated children <12 years of age, with negligible adverse effects.

In 5 animal studies (LOE 6)[172,173,177–179] large (per kilogram) energy doses caused less myocardial damage in young hearts than in adult hearts. In 3 animal studies (LOE 6)[173,179,180] and 1 small pediatric case series (LOE 5),[176] a 50-J biphasic dose delivered through a pediatric pad/cable system terminated VF and resulted in survival. One piglet (13 to 26 kg) study (LOE 6)[179] showed that pediatric biphasic AED shocks (50/75/86 J) terminated VF and caused less myocardial injury and better outcome than adult AED biphasic shocks (200/300/360 J).

Treatment Recommendation
The treatment of choice for pediatric VF/pulseless VT is prompt defibrillation, although the optimum dose is unknown. For manual defibrillation, we recommend an initial dose of 2 J/kg (biphasic or monophasic waveform). If this dose does not terminate VF, subsequent doses should be 4 J/kg.

For automated defibrillation, we recommend an initial pediatric attenuated dose for children 1 to 8 years of age and up to about 25 kg (55 pounds) and 127 cm (50 inches) in length. There is insufficient information to recommend for or against the use of an AED in infants <1 year of age. A variable dose manual defibrillator or an AED able to recognize pediatric shockable rhythms and equipped with dose attenuation are preferred; if such a defibrillator is not available, a standard AED with standard electrode pads may be used. A standard AED (without a dose attenuator) should be used for children ≥25 kg (about 8 years of age) and older adolescent and adult victims.

Management of Shock-Resistant VF/Pulseless VT

Amiodarone[W20,W21A,W21B]

Consensus on Science
Evidence extrapolated from 3 (LOE 1) studies in adults (LOE 7 when applied to pediatrics)[154,159,181] shows increased survival to hospital admission but not discharge when amiodarone is compared with placebo or lidocaine for shock-resistant VF. One study in children (LOE 3)[100] showed effectiveness of amiodarone for life-threatening ventricular arrhythmias.

Treatment Recommendation
IV amiodarone can be considered as part of the treatment of shock-refractory or recurrent VT/VF.

Airway and Ventilation

Maintaining a patent airway and ventilation are fundamental to resuscitation. Adult and animal studies during CPR suggest detrimental effects of hyperventilation and interruption of chest compressions. For children requiring airway control or ventilation for short periods in the out-of-hospital setting, bag-valve–mask (BVM) ventilation produces equivalent survival rates compared with ventilation with tracheal intubation.

The risks of tracheal tube misplacement, displacement, and obstruction are well recognized, and an evidence-based review led to a recommendation that proper tube placement and patency be monitored by exhaled CO_2 throughout transport. A review also found that cuffed tracheal tubes could be used safely even in infants.

Following the return of spontaneous circulation from cardiac arrest, toxic oxygen byproducts (reactive oxygen species, free radicals) are produced that may damage cell membranes, proteins, and DNA (reperfusion injury). There are no clinical studies in children outside the newborn period comparing different concentrations of inspired oxygen during and immediately after resuscitation, and it is difficult to differentiate "sufficient" from "excessive" oxygen therapy.

Bag-Valve–Mask Ventilation[W6]

Consensus on Science
One out-of-hospital pediatric prospective randomized controlled study (LOE 1)[182] in an EMS system with short transport times showed that BVM ventilation compared with tracheal intubation resulted in equivalent survival to hospital discharge rates and neurologic outcome in children requiring airway control, including children with cardiac arrest and trauma.

One study in pediatric cardiac arrest (LOE 4)[183] and 4 studies in children with trauma (LOE 3[184,185]; LOE 4[186,187])

found no advantage of tracheal intubation over BVM ventilation.

Treatment Recommendation
In the out-of-hospital setting with short transport times, BVM ventilation is the method of choice for children who require ventilatory support. When transport times are long, the relative benefit versus potential harm of tracheal intubation compared with BVM ventilation is uncertain. It is affected by the level of training and experience of the healthcare professional and the availability of exhaled CO_2 monitoring during intubation and transport.

Advanced Airways

Advanced airways include the tracheal tube, the Combitube, and the LMA. Experts at the 2005 Consensus Conference reviewed the available evidence on use of the tracheal tube and LMA in infants and children. There was no data on use of the Combitube in this age group.

Cuffed Versus Uncuffed Tracheal Tubes[W11A,W11B]

Consensus on Science
One randomized controlled trial (LOE 2),[188] 3 prospective cohort studies (LOE 3),[189–191] and 1 cohort study (LOE 4)[192] document no greater risk of complications for children <8 years of age when using cuffed tracheal tubes compared with uncuffed tubes in the operating room and intensive care unit.

Evidence from 1 randomized controlled trial (LOE 2)[188] and 1 small, prospective controlled study (LOE 3)[193] showed some advantage in cuffed over uncuffed tracheal tubes in children in the pediatric anesthesia and intensive care settings, respectively.

Treatment Recommendation
Cuffed tracheal tubes are as safe as uncuffed tubes for infants (except newborns) and children if rescuers use the correct tube size and cuff inflation pressure and verify tube position. Under certain circumstances (eg, poor lung compliance, high airway resistance, and large glottic air leak), cuffed tracheal tubes may be preferable.

Laryngeal Mask Airway[W26A,W26B]

Consensus on Science
There are no studies examining the use of the LMA in children during cardiac arrest. Evidence extrapolated from pediatric anesthesia shows a higher rate of complications with LMAs in smaller children compared with LMA experience in adults. The complication rate decreases with increasing operator experience (LOE 7).[194,195] Case reports document that the LMA can be helpful for management of the difficult airway.

Treatment Recommendation
There is insufficient data to support or refute a recommendation for the routine use of an LMA for children in cardiac arrest. The LMA may be an acceptable initial alternative airway adjunct for experienced providers during pediatric cardiac arrest when tracheal intubation is difficult to achieve.

Confirmation of Tube Placement

Exhaled CO_2[W25]

Consensus on Science
Misplaced, displaced, or obstructed tracheal tubes are associated with a high risk of death. No single method of tracheal tube confirmation is always accurate and reliable. One study (LOE 3)[196] showed that clinical assessment of tracheal tube position (observation of chest wall rise, mist in the tube, and auscultation of the chest) can be unreliable for distinguishing esophageal from tracheal intubation.

In 3 studies (LOE 5),[197–199] when a perfusing cardiac rhythm was present in infants >2 kg and children, detection of exhaled CO_2 using a colorimetric detector or capnometer had a high sensitivity and specificity for tracheal tube placement. In one study (LOE 5)[198] during cardiac arrest, the sensitivity of exhaled CO_2 detection for tracheal tube placement was 85% and specificity 100%. Both with a perfusing rhythm and during cardiac arrest, the presence of exhaled CO_2 reliably indicates tracheal tube placement, but the absence of exhaled CO_2 during cardiac arrest does not prove tracheal tube misplacement.

Treatment Recommendation
In all settings (ie, prehospital, emergency departments, intensive care units, operating rooms), confirmation of tracheal tube placement should be achieved using detection of exhaled CO_2 in intubated infants and children with a perfusing cardiac rhythm. This may be accomplished using a colorimetric detector or capnometry. During cardiac arrest, if exhaled CO_2 is not detected, tube position should be confirmed using direct laryngoscopy.

Esophageal Detector Device[W23]

Consensus on Science Statements
A study in the operating room (LOE 2)[200] showed that the esophageal detector device (EDD) was highly sensitive and specific for correct tracheal tube placement in children weighing >20 kg with a perfusing cardiac rhythm. There have been no studies of the EDD in children during cardiac arrest. A pediatric animal study (LOE 6)[201] showed only fair results with the EDD, but accuracy improved with use of a larger syringe device. The same animal study showed no difference when the tracheal tube cuff was either inflated or deflated.

Treatment Recommendation
The EDD may be considered for confirmation of tracheal tube placement in children weighing >20 kg.

Confirmation of Tracheal Tube Placement During Transport[W24]

Consensus on Science
Studies (LOE 1[202]; LOE 7[203]) have documented the high rate of inadvertent displacement of tracheal tubes during prehospital transport. There are no studies to evaluate the frequency of these events during intra- or interhospital transport.

Two studies (LOE 5)[204,205] show that in the presence of a perfusing rhythm, exhaled CO_2 detection or measurement can confirm tracheal tube position accurately during transport. In

2 animal studies (LOE 6),[206,207] loss of exhaled CO_2 detection indicated tracheal tube displacement more rapidly than pulse oximetry. On the basis of one case series (LOE 5),[204] continuous use of colorimetric exhaled CO_2 detectors may not be reliable for long (>30 minutes) transport duration.

Treatment Recommendation
We recommend monitoring tracheal tube placement and patency in infants and children with a perfusing rhythm by continuous measurement or frequent intermittent detection of exhaled CO_2 during prehospital and intra- and interhospital transport.

Oxygen

Oxygen During Resuscitation[W14A,W14B]

Consensus on Science
Meta-analyses of 4 human studies (LOE 1)[208,209] showed a reduction in mortality rates and no evidence of harm in newborns resuscitated with air compared with 100% oxygen (see Part 7: "Neonatal Resuscitation"). The 2 largest studies,[210,211] however, were not blinded, so results should be interpreted with caution. Two animal studies (LOE 6)[212,213] suggest that ventilation with room air may be superior to 100% oxygen during resuscitation from cardiac arrest, whereas one animal study (LOE 6)[214] showed no difference.

Treatment Recommendation
There is insufficient information to recommend for or against the use of any specific inspired oxygen concentration during and immediately after resuscitation from cardiac arrest. Until additional evidence is published, we support healthcare providers' use of 100% oxygen during resuscitation (when available). Once circulation is restored, providers should monitor oxygen saturation and wean inspired oxygen while ensuring adequate oxygen delivery.

Vascular Access and Drugs for Cardiac Arrest

Vascular access can be difficult to establish during resuscitation of children. Review of the evidence showed increasing experience with IO access and resulted in a deemphasis of the tracheal route for drug delivery. Evidence evaluation of resuscitation drugs was limited by a lack of reported experience in children. There was little experience with vasopressin in children in cardiac arrest and inconsistent results in adult patients. In contrast, there was a good study in children showing no benefit and possibly some harm in using high-dose epinephrine for cardiac arrest.

Routes of Drug Delivery

Intraosseous Access[W29]

Consensus on Science
Two prospective randomized trials in adults and children (LOE 3)[215,216] and 6 other studies (LOE 4[217]; LOE 5[218–220]; LOE 7[221,222]) document that IO access is safe and effective for fluid resuscitation, drug delivery, and blood sampling for laboratory evaluation.

Treatment Recommendation
We recommend establishing IO access if vascular access is not achieved rapidly in any infant or child for whom IV drugs or fluids are urgently required.

Drugs Given via Tracheal Tube[W32]

Consensus on Science
One study in children (LOE 2),[223] 5 studies in adults (LOE 2[224–226]; LOE 3[227,228]), and multiple animal studies (LOE 6)[229–231] indicate that atropine, epinephrine, naloxone, lidocaine, and vasopressin are absorbed via the trachea. Administration of resuscitation drugs into the trachea results in lower blood concentrations than the same dose given intravascularly. Furthermore, animal studies (LOE 6)[232–235] suggest that the lower epinephrine concentrations achieved when the drug is delivered by tracheal route may produce transient β-adrenergic effects. These effects can be detrimental, causing hypotension, lower coronary artery perfusion pressure and flow, and reduced potential for return of spontaneous circulation.

Treatment Recommendation
Intravascular, including IO, injection of drugs is preferable to administration by the tracheal route. The recommended tracheal dose of atropine, epinephrine, or lidocaine is higher than the vascular dose and is as follows:

- Epinephrine 0.1 mg/kg (multiple LOE 6 studies)
- Lidocaine 2 to 3 mg/kg (LOE 3)[228] and multiple LOE 6 studies
- Atropine 0.03mg/kg (LOE 2)[224]

The optimal tracheal doses of naloxone or vasopressin have not been determined.

Drugs in Cardiac Arrest

Dose of Epinephrine for Cardiac Arrest[W31A,W31B]

Consensus on Science
In 4 pediatric studies (LOE 2[236,237]; LOE 4[238,239]) there was no improvement in survival rates and a trend toward worse neurologic outcome after administration of high-dose epinephrine for cardiac arrest. A prospective, randomized, controlled trial (LOE 2)[236] comparing high-dose with standard-dose epinephrine for the second and subsequent ("rescue") doses in pediatric in-hospital cardiac arrest showed reduced 24-hour survival rates in the high-dose epinephrine group. In subgroup analysis, survival rates in asphyxia and sepsis were significantly worse with high-dose rescue epinephrine.

Treatment Recommendation
Children in cardiac arrest should be given 10 μg/kg of epinephrine as the first and subsequent intravascular doses. Routine use of high-dose (100 μg/kg) intravascular epinephrine is not recommended and may be harmful, particularly in asphyxia. High-dose epinephrine may be considered in exceptional circumstances (eg, β-blocker overdose).

Vasopressin in Cardiac Arrest[W19A,W19B]

Consensus on Science
Based on a small series of children (LOE 5),[240] vasopressin given after epinephrine may be associated with return of

spontaneous circulation after prolonged cardiac arrest. Animal data (LOE 6)[241,242] indicates that a combination of epinephrine and vasopressin may be beneficial. Adult data is inconsistent. Giving vasopressin after adult cardiac arrest (LOE 7)[243–247] has produced improved short-term outcomes (eg, return of spontaneous circulation or survival to hospital admission) but no improvement in neurologically intact survival to hospital discharge when compared with epinephrine.

Treatment Recommendation
There is insufficient evidence to recommend for or against the routine use of vasopressin during cardiac arrest in children.

Magnesium in Cardiac Arrest[W15]

Consensus on Science
The relationship between serum magnesium concentrations and outcome of CPR was analyzed in 2 studies in adults (LOE 3[248]; LOE 4[249]) and one animal study (LOE 6).[250] The first 2 studies indicated that a normal serum concentration of magnesium was associated with a higher rate of successful resuscitation, but it is unclear whether the association is causative. Six adult clinical studies (LOE 1[251]; LOE 2[252–255]; LOE 3[256]) and one study in an adult animal model (LOE 6)[257] indicated no significant difference in any survival end point in patients who received magnesium before, during, or after CPR.

Treatment Recommendation
Magnesium should be given for hypomagnesemia and torsades de pointes VT, but there is insufficient evidence to recommend for or against its routine use in cardiac arrest.

Postresuscitation Care

Postresuscitation care is critical to a favorable outcome. An evidence-based literature review was performed on the topics of brain preservation and myocardial function after resuscitation from cardiac arrest. It showed the potential benefits of induced hypothermia on brain preservation, the importance of preventing or aggressively treating hyperthermia, the importance of glucose control, and the role of vasoactive drugs in supporting hemodynamic function.

Ventilation

Hyperventilation[W27]

Consensus on Science
One study in cardiac arrest patients (LOE 3)[258] and extrapolation from 12 other studies (LOE 6[259]; LOE 2[260]; LOE 3[261–267]; LOE 4[268]; LOE 5[269,270]) suggest that hyperventilation may cause decreased venous return to the heart and cerebral ischemia and may be harmful in the comatose patient after cardiac arrest.

Treatment Recommendation
Hyperventilation after cardiac arrest may be harmful and should be avoided. The target of postresuscitation ventilation is normocapnia. Short periods of hyperventilation may be performed as a temporizing measure for the child with signs of impending cerebral herniation.

Temperature Control

Therapeutic Hypothermia[W22B,W22C]

Consensus on Science
Immediately after resuscitation from cardiac arrest, children often develop hypothermia followed by delayed hyperthermia (LOE 5).[271] Hypothermia (32°C to 34°C) may be beneficial to the injured brain. Although there are no pediatric studies of induced hypothermia after cardiac arrest, support for this treatment is extrapolated from

- Two prospective randomized studies of adults with VF arrest (LOE 1[272]; LOE 2[273])
- One study of newborns with birth asphyxia (LOE 2)[274]
- Numerous animal studies (LOE 6) of both asphyxial and VF arrest
- Acceptable safety profiles in adults (LOE 7)[272,273] and neonates (LOE 7)[275–278] treated with hypothermia (32°C to 34°C) for up to 72 hours

Treatment Recommendation
Induction of hypothermia (32°C to 34°C) for 12 to 24 hours should be considered in children who remain comatose after resuscitation from cardiac arrest.

Treatment of Hyperthermia[W22A,W22D]

Consensus on Science
Two studies (LOE 5)[271,279] show that fever is common after resuscitation from cardiac arrest, and 3 studies (LOE 7)[280–282] show that it is associated with worse outcome. Animal studies suggest that fever causes a worse outcome. One study (LOE 6)[283] shows that rats resuscitated from asphyxial cardiac arrest have a worse outcome if hyperthermia is induced within the first 24 hours of recovery. In rats with global ischemic brain injury (which produces endogenous fever), prevention of fever with a nonsteroidal anti-inflammatory drug (NSAID) class of antipyretic attenuated neuronal damage (LOE 6).[284,285]

Treatment Recommendation
Healthcare providers should prevent hyperthermia and treat it aggressively in infants and children resuscitated from cardiac arrest.

Hemodynamic Support

Vasoactive Drugs[W33A,W33B,W33C,W33D]

Consensus on Science
Two studies in children (LOE 5),[286,287] multiple studies in adults (LOE 7),[288–290] and animal studies (LOE 6)[291–293] indicate that myocardial dysfunction is common after resuscitation from cardiac arrest. Multiple animal studies (LOE 6)[294–296] document consistent improvement in hemodynamics when selected vasoactive drugs are given in the post–cardiac arrest period. Evidence extrapolated from multiple adult and pediatric studies (LOE 7)[297–302] of cardiovascular surgical patients with low cardiac output documents consistent improvement in hemodynamics when vasoactive drugs are titrated in the period after cardiopulmonary bypass.

Treatment Recommendation
Vasoactive drugs should be considered to improve hemodynamic status in the post–cardiac arrest phase. The choice, timing, and dose of specific vasoactive drugs must be individualized and guided by available monitoring data.

Blood Glucose Control

Treatment of Hypoglycemia and Hyperglycemia[W30A,W30B,W30C]

Consensus on Science
Adults with out-of-hospital cardiac arrest and elevated blood glucose on admission have poor neurologic and survival outcomes (LOE 7).[303–308] In critically ill children, hypoglycemia (LOE 5)[309] and hyperglycemia (LOE 5)[310] are associated with poor outcome. It is unknown if the association of hyperglycemia with poor outcome after cardiac arrest is causative or an epiphenomenon related to the stress response.

In critically ill adult surgical patients, (LOE 7)[311] strict glucose control improves outcome, but there is currently insufficient data in children showing that the benefit of tight glucose control outweighs the risk of inadvertent hypoglycemia.

Several animal studies (LOE 6)[312–316] and an adult clinical study (LOE 4)[317] show poor outcome when glucose is given immediately before or during cardiac arrest. It is unknown if there is harm in giving glucose-containing maintenance fluids to children after cardiac arrest.

Hypoglycemia is an important consideration in pediatric resuscitation because

- Critically ill children are hypermetabolic compared with baseline and have increased glucose requirement (6 to 8 mg/kg per minute) to prevent catabolism.
- The combined effects of hypoglycemia and hypoxia/ischemia on the immature brain (neonatal animals) appears more deleterious than the effect of either insult alone.[318]
- Four retrospective studies of human neonatal asphyxia show an association between hypoglycemia and subsequent brain injury (LOE 4[319,320]; LOE 5[321,322]).

Treatment Recommendation
Healthcare providers should check glucose concentration during cardiac arrest and monitor it closely afterward with the goal of maintaining normoglycemia. Glucose-containing fluids are not indicated during CPR unless hypoglycemia is present (LOE 7).[323]

Prognosis

One of the most difficult challenges in CPR is to decide the point at which further resuscitative efforts are futile. Unfortunately there are no simple guidelines. Certain characteristics suggest that resuscitation should be continued (eg, ice water drowning, witnessed VF arrest), and others suggest that further resuscitative efforts will be futile (eg, most cardiac arrests associated with blunt trauma or septic shock).

Predictors of Outcome in Children[W12B,W28]

Consensus on Science
Multiple studies in adults have linked characteristics of the patient or of the cardiac arrest with prognosis following in-hospital or out-of-hospital cardiac arrest. Experience in children is more limited. Six pediatric studies (LOE 5)[3,324–328] show that prolonged resuscitation is associated with a poor outcome. Although the likelihood of a good outcome is greater with short duration of CPR, 2 pediatric studies (LOE 3)[328,329] reported good outcomes in some patients following 30 to 60 minutes of CPR in the in-patient setting when the arrests were witnessed and prompt and presumably excellent CPR was provided. Children with cardiac arrest associated with environmental hypothermia or immersion in icy water can have excellent outcomes despite >30 minutes of cardiac arrest (LOE 5).[7,330]

One large pediatric study (LOE 4)[331] and several smaller studies (LOE 5)[332–336] show that good outcome can be achieved when extracorporeal CPR is started after 30 to 90 minutes of refractory standard CPR for in-hospital cardiac arrests. The good outcomes were reported primarily in patients with isolated heart disease. This data shows that 15 or 30 minutes of CPR does not define the limits of cardiac and cerebral viability.

Witnessed events, bystander CPR, and a short interval from collapse to arrival of EMS system personnel are important prognostic factors associated with improved outcome in adult resuscitation, and it seems reasonable to extrapolate these factors to children. At least one pediatric study (LOE 5)[328] showed that the interval from collapse to initiation of CPR is a significant prognostic factor.

Children with prehospital cardiac arrest caused by blunt trauma[337] and in-hospital cardiac arrest caused by septic shock[329] rarely survive.

Treatment Recommendation
The rescuer should consider whether to discontinue resuscitative efforts after 15 to 20 minutes of CPR. Relevant considerations include the cause of the arrest, preexisting conditions, whether the arrest was witnessed, duration of untreated cardiac arrest ("no flow"), effectiveness and duration of CPR ("low flow"), prompt availability of extracorporeal life support for a reversible disease process, and associated special circumstances (eg, icy water drowning, toxic drug exposure).

References

1. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science, Part 9: Pediatric Basic Life Support. *Circulation*. 2000;102(suppl I):I-253–I-290.
2. American Heart Association in collaboration with International Liaison Committee on Resuscitation. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care: International Consensus on Science, Part 10: Pediatric Advanced Life Support. *Circulation*. 2000;102(suppl I):I-291–I-342.
3. Zaritsky A, Nadkarni V, Getson P, Kuehl K. CPR in children. *Ann Emerg Med*. 1987;16:1107–1111.
4. Hickey RW, Cohen DM, Strausbaugh S, Dietrich AM. Pediatric patients requiring CPR in the prehospital setting. *Ann Emerg Med*. 1995;25:495–501.
5. Mogayzel C, Quan L, Graves JR, Tiedeman D, Fahrenbruch C, Herndon P. Out-of-hospital ventricular fibrillation in children and adolescents: causes and outcomes. *Ann Emerg Med*. 1995;25:484–491.
6. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. Characteristics and outcome among children suffering from out of hospital cardiac arrest in Sweden. *Resuscitation*. 2005;64:37–40.

7. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests: epidemiology and outcome. *Resuscitation.* 1995;30:141–150.
8. Kyriacou DN, Arcinue EL, Peek C, Kraus JF. Effect of immediate resuscitation on children with submersion injury. *Pediatrics*. 1994;94(pt 1):137–142.
9. 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.
10. Safranek DJ, Eisenberg MS, Larsen MP. The epidemiology of cardiac arrest in young adults. *Ann Emerg Med*. 1992;21:1102–1106.
11. Berg RA, Hilwig RW, Kern KB, Ewy GA. Precountershock cardiopulmonary resuscitation improves ventricular fibrillation median frequency and myocardial readiness for successful defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. *Ann Emerg Med*. 2002;40:563–570.
12. Berg RA, Hilwig RW, Kern KB, Sanders AB, Xavier LC, Ewy GA. Automated external defibrillation versus manual defibrillation for prolonged ventricular fibrillation: lethal delays of chest compressions before and after countershocks. *Ann Emerg Med*. 2003;42:458–467.
13. Cobb LA, Fahrenbruch CE, Walsh TR, Copass MK, Olsufka M, Breskin M, Hallstrom AP. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. *JAMA*. 1999;281:1182–1188.
14. Dowie R, Campbell H, Donohoe R, Clarke P. 'Event tree' analysis of out-of-hospital cardiac arrest data: confirming the importance of bystander CPR. *Resuscitation.* 2003;56:173–181.
15. Engdahl J, Bang A, Lindqvist J, Herlitz J. Factors affecting short- and long-term prognosis among 1069 patients with out-of-hospital cardiac arrest and pulseless electrical activity. *Resuscitation.* 2001;51:17–25.
16. Holmberg M, Holmberg S, Herlitz J. Effect of bystander cardiopulmonary resuscitation in out-of-hospital cardiac arrest patients in Sweden. *Resuscitation.* 2000;47:59–70.
17. Stiell IG, Nichol G, Wells G, De Maio V, Nesbitt L, Blackburn J, Spaite D. Health-related quality of life is better for cardiac arrest survivors who received citizen cardiopulmonary resuscitation. *Circulation*. 2003;108: 1939–1944.
18. Eftestol T, Wik L, Sunde K, Steen PA. Effects of cardiopulmonary resuscitation on predictors of ventricular fibrillation defibrillation success during out-of-hospital cardiac arrest. *Circulation*. 2004;110: 10–15.
19. Wik L, Hansen TB, Fylling F, Steen T, Vaagenes P, Auestad BH, Steen PA. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. *JAMA*. 2003;289:1389–1395.
20. 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.
21. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. *Resuscitation.* 2004;60:213–217.
22. Bahr J, Klingler H, Panzer W, Rode H, Kettler D. Skills of lay people in checking the carotid pulse. *Resuscitation.* 1997;35:23–26.
23. Cavallaro DL, Melker RJ. Comparison of two techniques for detecting cardiac activity in infants. *Crit Care Med*. 1983;11:189–190.
24. Graham CA, Lewis NF. Evaluation of a new method for the carotid pulse check in cardiopulmonary resuscitation. *Resuscitation.* 2002;53: 37–40.
25. Lee CJ, Bullock LJ. Determining the pulse for infant CPR: time for a change? *Mil Med*. 1991;156:190–193.
26. 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.
27. Mather C, O'Kelly S. The palpation of pulses. *Anaesthesia*. 1996;51: 189–191.
28. Lapostolle F, Le Toumelin P, Agostinucci JM, Catineau J, Adnet F. Basic cardiac life support providers checking the carotid pulse: performance, degree of conviction, and influencing factors. *Acad Emerg Med*. 2004;11:878–880.
29. Moule P. Checking the carotid pulse: diagnostic accuracy in students of the healthcare professions. *Resuscitation.* 2000;44:195–201.
30. Bahr J. CPR education in the community. *Eur J Emerg Med*. 1994;1: 190–192.
31. 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.
32. 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.
33. Tonkin SL, Gunn AJ. Failure of mouth-to-mouth resuscitation in cases of sudden infant death. *Resuscitation.* 2001;48:181–184.
34. Wilson-Davis SL, Tonkin SL, Gunn TR. Air entry in infant resuscitation: oral or nasal routes? *J Appl Physiol*. 1997;82:152–155.
35. Stocks J, Godfrey S. Nasal resistance during infancy. *Respir Physiol*. 1978;34:233–246.
36. Rodenstein DO, Perlmutter N, Stanescu DC. Infants are not obligatory nasal breathers. *Am Rev Respir Dis*. 1985;131:343–347.
37. James DS, Lambert WE, Mermier CM, Stidley CA, Chick TW, Samet JM. Oronasal distribution of ventilation at different ages. *Arch Environ Health*. 1997;52:118–123.
38. Berg MD, Idris AH, Berg RA. Severe ventilatory compromise due to gastric distention during pediatric cardiopulmonary resuscitation. *Resuscitation.* 1998;36:71–73.
39. Segedin E, Torrie J, Anderson B. Nasal airway versus oral route for infant resuscitation. *Lancet*. 1995;346:382.
40. Dembofsky CA, Gibson E, Nadkarni V, Rubin S, Greenspan JS. Assessment of infant cardiopulmonary resuscitation rescue breathing technique: relationship of infant and caregiver facial measurements. *Pediatrics*. 1999;103:E17.
41. Tonkin SL, Davis SL, Gunn TR. Nasal route for infant resuscitation by mothers. *Lancet*. 1995;345:1353–1354.
42. Sorribes del Castillo J, Carrion Perez C, Sanz Ribera J. Nasal route to ventilation during basic cardiopulmonary resuscitation in children under two months of age. *Resuscitation.* 1997;35:249–252.
43. Nowak AJ, Casamassimo PS. Oral opening and other selected facial dimensions of children 6 weeks to 36 months of age. *J Oral Maxillofac Surg*. 1994;52:845–847; discussion 848.
44. Whitelaw CC, Slywka B, Goldsmith LJ. Comparison of a two-finger versus two-thumb method for chest compressions by healthcare providers in an infant mechanical model. *Resuscitation.* 2000;43:213–216.
45. Dorfsman ML, Menegazzi JJ, Wadas RJ, Auble TE. Two-thumb vs two-finger chest compression in an infant model of prolonged cardiopulmonary resuscitation. *Acad Emerg Med*. 2000;7:1077–1082.
46. Menegazzi JJ, Auble TE, Nicklas KA, Hosack GM, Rack L, Goode JS. Two-thumb versus two-finger chest compression during CRP in a swine infant model of cardiac arrest. *Ann Emerg Med*. 1993;22:240–243.
47. Houri PK, Frank LR, Menegazzi JJ, Taylor R. A randomized, controlled trial of two-thumb vs two-finger chest compression in a swine infant model of cardiac arrest. *Prehosp Emerg Care*. 1997;1:65–67.
48. Todres ID, Rogers MC. Methods of external cardiac massage in the newborn infant. *J Pediatr*. 1975;86:781–782.
49. David R. Closed chest cardiac massage in the newborn infant. *Pediatrics*. 1988;81:552–554.
50. Stevenson AG, McGowan J, Evans AL, Graham CA. CPR for children: one hand or two? *Resuscitation.* 2005;64:205–208.
51. 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.
52. 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.
53. Greingor JL. Quality of cardiac massage with ratio compression-ventilation 5/1 and 15/2. *Resuscitation.* 2002;55:263–267.
54. Srikantan S, Berg RA, Cox T, Tice L, Nadkarni VM. Effect of 1-rescuer compression: ventilation ratios on cpr in infant, pediatric and adult manikins. *Crit Care Med*. In press.
55. 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.
56. 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.
57. 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.

58. 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.
59. Heidenreich JW, Higdon TA, Kern KB, Sanders AB, Berg RA, Niebler R, Hendrickson J, Ewy GA. Single-rescuer cardiopulmonary resuscitation: "two quick breaths"—an oxymoron. *Resuscitation.* 2004;62: 283–289.
60. 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.
61. 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.
62. 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.
63. 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.
64. 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.
65. 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.
66. 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.
67. Becker LB, Berg RA, Pepe PE, Idris AH, Aufderheide TP, Barnes TA, Stratton SJ, Chandra NC. A reappraisal of mouth-to-mouth ventilation during bystander-initiated cardiopulmonary resuscitation: a statement for healthcare professionals from the Ventilation Working Group of the Basic Life Support and Pediatric Life Support Subcommittees, American Heart Association. *Resuscitation.* 1997;35:189–201.
68. Suominen P, Rasanen J, Kivioja A. Efficacy of cardiopulmonary resuscitation in pulseless paediatric trauma patients. *Resuscitation.* 1998; 36:9–13.
69. Christensen DW, Jansen P, Perkin RM. Outcome and acute care hospital costs after warm water near drowning in children. *Pediatrics*. 1997;99: 715–721.
70. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: a collective review. *Ann Emerg Med*. 1999;33:195–205.
71. 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.
72. Holmberg M, Holmberg S, Herlitz J. Factors modifying the effect of bystander cardiopulmonary resuscitation on survival in out-of-hospital cardiac arrest patients in Sweden. *Eur Heart J*. 2001;22:511–519.
73. Bossaert L, Van Hoeyweghen R. Bystander cardiopulmonary resuscitation (CPR) in out-of-hospital cardiac arrest. The Cerebral Resuscitation Study Group. *Resuscitation.* 1989;17(suppl):S55–S69.
74. 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.
75. Berg RA, Kern KB, Sanders AB, Otto CW, Hilwig RW, Ewy GA. Bystander cardiopulmonary resuscitation. Is ventilation necessary? *Circulation*. 1993;88(pt 1):1907–1915.
76. 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.
77. 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.
78. 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.
79. 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.
80. Wen ZC, Chen SA, Tai CT, Chiang CE, Chiou CW, Chang MS. Electrophysiological mechanisms and determinants of vagal maneuvers for termination of paroxysmal supraventricular tachycardia. *Circulation*. 1998;98:2716–2723.
81. Bisset GSI, Gaum W, Kaplan S. The ice bag: a new technique for interruption of supraventricular tachycardia. *J Pediatr*. 1980;97: 593–595.
82. Sreeram N, Wren C. Supraventricular tachycardia in infants: response to initial treatment. *Arch Dis Child*. 1990;65:127–129.
83. Mehta D, Wafa S, Ward DE, Camm AJ. Relative efficacy of various physical manoeuvres in the termination of junctional tachycardia. *Lancet*. 1988;1:1181–1185.
84. Lim SH, Anantharaman V, Teo WS, Goh PP, Tan AT. Comparison of treatment of supraventricular tachycardia by Valsalva maneuver and carotid sinus massage. *Ann Emerg Med*. 1998;31:30–35.
85. Josephson ME, Seides SE, Batsford WB, Caracta AR, Damato AN, Kastor JA. The effects of carotid sinus pressure in re-entrant paroxysmal supraventricular tachycardia. *Am Heart J*. 1974;88:694–697.
86. Waxman MB, Wald RW, Sharma AD, Huerta F, Cameron DA. Vagal techniques for termination of paroxysmal supraventricular tachycardia. *Am J Cardiol*. 1980;46:655–664.
87. Wayne MA. Conversion of paroxysmal atrial tachycardia by facial immersion in ice water. *JACEP*. 1976;5:434–435.
88. Wildenthal K, Leshin SJ, Atkins JM, Skelton CL. The diving reflex used to treat paroxysmal atrial tachycardia. *Lancet*. 1975;1:12–14.
89. Hamilton J, Moodie D, Levy J. The use of the diving reflex to terminate supraventricular tachycardia in a 2-week-old infant. *Am Heart J*. 1979; 97:371–374.
90. Craig JE, Scholz TA, Vanderhooft SL, Etheridge SP. Fat necrosis after ice application for supraventricular tachycardia termination. *J Pediatr*. 1998;133:727.
91. Thomas MD, Torres A, Garcia-Polo J, Gavilan C. Life-threatening cervico-mediastinal haematoma after carotid sinus massage. *J Laryngol Otol*. 1991;105:381–383.
92. Bianconi L, Castro A, M. D, Alboni P, Pappalardo A, Richiardi E, Santini M. Comparison of intravenously administered dofetilide versus amiodarone in the acute termination of atrial fibrillation and flutter: a multicentre, randomized, double-blind, placebo-controlled study. *Eur Heart J*. 2000;21:1265–1273.
93. Burri S, Hug MI, Bauersfeld U. Efficacy and safety of intravenous amiodarone for incessant tachycardias in infants. *Eur J Pediatr*. 2003; 162:880–884.
94. 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]. [Spanish]. *An Esp Pediatr*. 2002;56:505–509.
95. 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.
96. 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.
97. 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.
98. 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.
99. 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.
100. 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.
101. 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.

102. Valsangiacomo E, Schmid ER, Schupbach RW, Schmidlin D, Molinari L, Waldvogel K, Bauersfeld U. Early postoperative arrhythmias after cardiac operation in children. *Ann Thorac Surg*. 2002;74:792–796.
103. 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.
104. Daniels CJ, Schutte DA, Hammond S, Franklin WH. Acute pulmonary toxicity in an infant from intravenous amiodarone. *Am J Cardiol*. 1998; 80:1113–1116.
105. Gandy J, Wonko N, Kantoch MJ. Risks of intravenous amiodarone in neonates. *Can J Cardiol*. 1998;14:855–858.
106. Benson DJ, Dunnigan A, Green T, Benditt D, Schneider S. Periodic procainamide for paroxysmal tachycardia. *Circulation*. 1985;72: 147–152.
107. Boahene KA, Klein GJ, Yee R, Sharma AD, Fujimura O. Termination of acute atrial fibrillation in the Wolff-Parkinson-White syndrome by procainamide and propafenone: importance of atrial fibrillatory cycle length. *J Am Coll Cardiol*. 1990;16:1408–1414.
108. Dodo H, Gow RM, Hamilton RM, Freedom RM. Chaotic atrial rhythm in children. *Am Heart J*. 1995;129:990–995.
109. Komatsu C, Ishinaga T, Tateishi O, Tokuhisa Y, Yoshimura S. Effects of four antiarrhythmic drugs on the induction and termination of paroxysmal supraventricular tachycardia. *Jpn Circ J*. 1986;50:961–972.
110. Mandapati R, Byrum CJ, Kavey RE, Smith FC, Kveselis DA, Hannan WP, Brandt B 3rd, Gaum WE. Procainamide for rate control of postsurgical junctional tachycardia. *Pediatr Cardiol*. 2000;21:123–128.
111. Mandel WJ, Laks MM, Obayashi K, Hayakawa H, Daley W. The Wolff-Parkinson-White syndrome: pharmacologic effects of procaine amide. *Am Heart J*. 1975;90:744–754.
112. Mehta AV, Sanchez GR, Sacks EJ, Casta A, Dunn JM, Donner RM. Ectopic automatic atrial tachycardia in children: clinical characteristics, management and follow-up. *J Am Coll Cardiol*. 1988;11:379–385.
113. 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.
114. Satake S, Hiejima K, Moroi Y, Hirao K, Kubo I, Suzuki F. Usefulness of invasive and non-invasive electrophysiologic studies in the selection of antiarrhythmic drugs for the patients with paroxysmal supraventricular tachyarrhythmia. *Jpn Circ J*. 1985;49:345–350.
115. Singh S, Gelband H, Mehta A, Kessler K, Casta A, Pickoff A. Procainamide elimination kinetics in pediatric patients. *Clin Pharmacol Ther*. 1982;32:607–611.
116. 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.
117. Wang JN, Wu JM, Tsai YC, Lin CS. Ectopic atrial tachycardia in children. *J Formos Med Assoc*. 2000;99:766–770.
118. Chen F, Wetzel G, TS. K. Acute effects of amiodarone on sodium currents in isolated neonatal ventricular myocytes: comparison with procainamide. *Dev Pharmacol Ther*. 1992;19:118–130.
119. Fujiki A, Tani M, Yoshida S, Inoue H. Electrophysiologic mechanisms of adverse effects of class I antiarrhythmic drugs (cibenzoline, pilsicainide, disopyramide, procainamide) in induction of atrioventricular re-entrant tachycardia. *Cardiovasc Drugs Ther*. 1996;10:159–166.
120. Bauernfeind RA, Swiryn S, Petropoulos AT, Coelho A, Gallastegui J, Rosen KM. Concordance and discordance of drug responses in atrioventricular reentrant tachycardia. *J Am Coll Cardiol*. 1983;2:345–350.
121. Hordof AJ, Edie R, Malm JR, Hoffman BF, Rosen MR. Electrophysiologic properties and response to pharmacologic agents of fibers from diseased human atria. *Circulation*. 1976;54:774–779.
122. Jawad-Kanber G, Sherrod TR. Effect of loading dose of procaine amide on left ventricular performance in man. *Chest.* 1974;66:269–272.
123. Karlsson E, Sonnhag C. Haemodynamic effects of procainamide and phenytoin at apparent therapeutic plasma levels. *Eur J Clin Pharmacol*. 1976;10:305–310.
124. 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.
125. Benson D Jr, Smith W, Dunnigan A, Sterba R, Gallagher J. Mechanisms of regular wide QRS tachycardia in infants and children. *Am J Cardiol*. 1982;49:1778–1788.
126. Kuga K, Yamaguchi I, Sugishita Y. Effect of intravenous amiodarone on electrophysiologic variables and on the modes of termination of atrioventricular reciprocating tachycardia in Wolff-Parkinson-White syndrome. *Jpn Circ J*. 1999;63:189–195.
127. 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.
128. 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.
129. 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.
130. 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.
131. Meldon SW, Brady WJ, Berger S, Mannenbach M. Pediatric ventricular tachycardia: a review with three illustrative cases. *Pediatr Emerg Care*. 1994;10:294–300.
132. Stanton MS, Prystowsky EN, Fineberg NS, Miles WM, Zipes DP, Heger JJ. Arrhythmogenic effects of antiarrhythmic drugs: a study of 506 patients treated for ventricular tachycardia or fibrillation. *J Am Coll Cardiol*. 1989;14:209–215; discussion 216–217.
133. Hasin Y, Kriwisky M, Gotsman MS. Verapamil in ventricular tachycardia. *Cardiology.* 1984;71:199–206.
134. Hernandez A, Strauss A, Kleiger RE, Goldring D. Idiopathic paroxysmal ventricular tachycardia in infants and children. *J Pediatr*. 1975; 86:182–188.
135. Horowitz LN, Josephson ME, Farshidi A, Spielman SR, Michelson EL, Greenspan AM. Recurrent sustained ventricular tachycardia, 3: role of the electrophysiologic study in selection of antiarrhythmic regimens. *Circulation*. 1978;58:986–997.
136. Mason JW, Winkle RA. Electrode-catheter arrhythmia induction in the selection and assessment of antiarrhythmic drug therapy for recurrent ventricular tachycardia. *Circulation*. 1978;58:971–985.
137. Cain M, Martin T, Marchlinski F, Josephson M. Changes in ventricular refractoriness after an extrastimulus: effects of prematurity, cycle length and procainamide. *Am J Cardiol.* 1983;52:996–1001.
138. Swiryn S, Bauernfeind RA, Strasberg B, Palileo E, Iverson N, Levy PS, Rosen KM. Prediction of response to class I antiarrhythmic drugs during electrophysiologic study of ventricular tachycardia. *Am Heart J*. 1982; 104:43–50.
139. Velebit V, Podrid P, Lown B, Cohen B, Graboys T. Aggravation and provocation of ventricular arrhythmias by antiarrhythmic drugs. *Circulation*. 1982;65:886–894.
140. Roden D, Reele S, Higgins S, Wilkinson G, Smith R, Oates J, Woosley R. Antiarrhythmic efficacy, pharmacokinetics and safety of N-acetylprocainamide in human subjects: comparison with procainamide. *Am J Cardiol*. 1980;46:463–468.
141. Naitoh N, Washizuka T, Takahashi K, Aizawa Y. Effects of class I and III antiarrhythmic drugs on ventricular tachycardia-interrupting critical paced cycle length with rapid pacing. *Jpn Circ J*. 1998;62:267–273.
142. Kasanuki H, Ohnishi S, Hosoda S. Differentiation and mechanisms of prevention and termination of verapamil-sensitive sustained ventricular tachycardia. *Am J Cardiol*. 1989;64:46J–49J.
143. Kasanuki H, Ohnishi S, Tanaka E, Hirosawa K. Clinical significance of Vanghan Williams classification for treatment of ventricular tachycardia: study of class IA and IB antiarrhythmic agents. *Jpn Circ J*. 1988;52:280–288.
144. Videbaek J, Andersen E, Jacobsen J, Sandoe E, Wennevold A. Paroxysmal tachycardia in infancy and childhood. II. Paroxysmal ventricular tachycardia and fibrillation. *Acta Paediatr Scand*. 1973;62: 349–357.
145. Sanchez J, Christie K, Cumming G. Treatment of ventricular tachycardia in an infant. *Can Med Assoc J*. 1972;107:136–138.
146. Gelband H, Steeg C, Bigger JJ. Use of massive doses of procaineamide in the treatment of ventricular tachycardia in infancy. *Pediatrics*. 1971; 48:110–115.
147. 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. *Pediatr Cardiol.* 1998;19:445–449.
148. Rokicki W, Durmala J, Nowakowska E. [Amiodarone for long term treatment of arrhythmia in children]. *Wiad Lek*. 2001;54:45–50.

149. Strasburger JF, Cuneo BF, Michon MM, Gotteiner NL, Deal BJ, McGregor SN, Oudijk MA, Meijboom EJ, Feinkind L, Hussey M, Parilla BV. Amiodarone therapy for drug-refractory fetal tachycardia. *Circulation*. 2004;109:375–379.
150. Beder SD, Cohen MH, BenShachar G. Time course of myocardial amiodarone uptake in the piglet heart using a chronic animal model. *Pediatr Cardiol*. 1998;19:204–211.
151. Paiva EF, Perondi MB, Kern KB, Berg RA, Timerman S, Cardoso LF, Ramirez JA. Effect of amiodarone on haemodynamics during cardiopulmonary resuscitation in a canine model of resistant ventricular fibrillation. *Resuscitation.* 2003;58:203–208.
152. Aiba T, Kurita T, Taguchi A, Shimizu W, Suyama K, Aihara N, Kamakura S. Long-term efficacy of empirical chronic amiodarone therapy in patients with sustained ventricular tachyarrhythmia and structural heart disease. *Circ J*. 2002;66:367–371.
153. Cairns JA, Connolly SJ, Gent M, Roberts R. Post-myocardial infarction mortality in patients with ventricular premature depolarizations. Canadian Amiodarone Myocardial Infarction Arrhythmia Trial Pilot Study. *Circulation*. 1991;84:550–557.
154. 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.
155. Fogel RI, Herre JM, Kopelman HA, Kowey PR, Trohman RG, Fineberg N, Prystowsky EN. Long-term follow-up of patients requiring intravenous amiodarone to suppress hemodynamically destabilizing ventricular arrhythmias. *Am Heart J*. 2000;139:690–695.
156. Heger JJ, Prystowsky EN, Jackman WM, Naccarelli GV, Warfel KA, Rinkenberger RL, Zipes DP. Clinical efficacy and electrophysiology during long-term therapy for recurrent ventricular tachycardia or ventricular fibrillation. *N Engl J Med*. 1981;305:539–545.
157. Kalbfleisch SJ, Williamson B, Man KC, Vorperian V, Hummel JD, Hasse C, Strickberger SA, Calkins H, Langberg JJ, Morady F. Prospective, randomized comparison of conventional and high dose loading regimens of amiodarone in the treatment of ventricular tachycardia. *J Am Coll Cardiol*. 1993;22:1723–1729.
158. Kowey PR, Levine JH, Herre JM, Pacifico A, Lindsay BD, Plumb VJ, Janosik DL, Kopelman HA, Scheinman MM. Randomized, double-blind comparison of intravenous amiodarone and bretylium in the treatment of patients with recurrent, hemodynamically destabilizing ventricular tachycardia or fibrillation. The Intravenous Amiodarone Multicenter Investigators Group. *Circulation*. 1995;92:3255–3263.
159. 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.
160. Lee KL, Tai YT. Long-term low-dose amiodarone therapy in the management of ventricular and supraventricular tachyarrhythmias: efficacy and safety. *Clin Cardiol*. 1997;20:372–377.
161. Levine JH, Massumi A, Scheinman MM, Winkle RA, Platia EV, Chilson DA, Gomes A, Woosley RL. Intravenous amiodarone for recurrent sustained hypotensive ventricular tachyarrhythmias. Intravenous Amiodarone Multicenter Trial Group. *J Am Coll Cardiol*. 1996;27:67–75.
162. Singh SN, Fletcher RD, Fisher SG, Singh BN, Lewis HD, Deedwania PC, Massie BM, Colling C, Lazzeri D. Amiodarone in patients with congestive heart failure and asymptomatic ventricular arrhythmia. Survival Trial of Antiarrhythmic Therapy in Congestive Heart Failure. *N Engl J Med*. 1995;333:77–82.
163. Somberg JC, Timar S, Bailin SJ, Lakatos F, Haffajee CI, Tarjan J, Paladino WP, Sarosi I, Kerin NZ, Borbola J, Bridges DE, Molnar J. Lack of a hypotensive effect with rapid administration of a new aqueous formulation of intravenous amiodarone. *Am J Cardiol*. 2004;93:576–581.
164. Sim I, McDonald KM, Lavori PW, Norbutas CM, Hlatky MA. Quantitative overview of randomized trials of amiodarone to prevent sudden cardiac death. *Circulation*. 1997;96:2823–2829.
165. Scheinman MM, Levine JH, Cannom DS, Friehling T, Kopelman HA, Chilson DA, Platia EV, Wilber DJ, Kowey PR. Dose-ranging study of intravenous amiodarone in patients with life-threatening ventricular tachyarrhythmias. The Intravenous Amiodarone Multicenter Investigators Group. *Circulation*. 1995;92:3264–3272.
166. Schneider T, Martens PR, Paschen H, Kuisma M, Wolcke B, Gliner BE, Russell JK, Weaver WD, Bossaert L, Chamberlain D. Multicenter, randomized, controlled trial of 150-J biphasic shocks compared with 200- to 360-J monophasic shocks in the resuscitation of out-of-hospital cardiac arrest victims. Optimized Response to Cardiac Arrest (ORCA) Investigators. *Circulation*. 2000;102:1780–1787.
167. 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.
168. Higgins SL, Herre JM, Epstein AE, Greer GS, Friedman PL, Gleva ML, Porterfield JG, Chapman FW, Finkel ES, Schmitt PW, Nova RC, Greene HL. A comparison of biphasic and monophasic shocks for external defibrillation. Physio-Control Biphasic Investigators. *Prehosp Emerg Care*. 2000;4:305–313.
169. Carpenter J, Rea TD, Murray JA, Kudenchuk PJ, Eisenberg MS. Defibrillation waveform and post-shock rhythm in out-of-hospital ventricular fibrillation cardiac arrest. *Resuscitation.* 2003;59:189–196.
170. Morrison LJ, Dorian P, Long J, Vermeulen M, Schwartz B, Sawadsky B, Frank J, Cameron B, Burgess R, Shield J, Bagley P, Mausz V, Brewer JE, Lerman BB. Out-of-hospital cardiac arrest rectilinear biphasic to monophasic damped sine defibrillation waveforms with advanced life support intervention trial (ORBIT). *Resuscitation.* 2005;66:149–157.
171. 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.
172. 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.
173. 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.
174. Gutgesell HP, Tacker WA, Geddes LA, Davis S, Lie JT, McNamara DG. Energy dose for ventricular defibrillation of children. *Pediatrics*. 1976;58:898–901.
175. 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.
176. Atkins D, Jorgenson D. Attenuated pediatric electrode pads for automated external defibrillator use in children. *Resuscitation.* In press.
177. Gaba DM, Talner NS. Myocardial damage following transthoracic direct current countershock in newborn piglets. *Pediatr Cardiol*. 1982;2:281–288.
178. 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.
179. 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.
180. Berg RA, Hilwig RW, Ewy GA, Kern KB. Precountershock cardiopulmonary resuscitation improves initial response to defibrillation from prolonged ventricular fibrillation: a randomized, controlled swine study. *Crit Care Med*. 2004;32:1352–1357.
181. Somberg JC, Bailin SJ, Haffajee CI, Paladino WP, Kerin NZ, Bridges D, Timar S, Molnar J. Intravenous lidocaine versus intravenous amiodarone (in a new aqueous formulation) for incessant ventricular tachycardia. *Am J Cardiol*. 2002;90:853–859.
182. 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.
183. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. *Prehosp Emerg Care*. 2002;6:283–290.
184. Cooper A, DiScala C, Foltin G, Tunik M, Markenson D, Welborn C. Prehospital endotracheal intubation for severe head injury in children: a reappraisal. *Semin Pediatr Surg*. 2001;10:3–6.
185. Eckstein M, Chan L, Schneir A, Palmer R. Effect of prehospital advanced life support on outcomes of major trauma patients. *J Trauma*. 2000;48:643–648.
186. Stockinger ZT, McSwain NE Jr. Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation. *J Trauma*. 2004;56:531–536.

187. 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.
188. 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 27A.
189. 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.
190. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. *J Pediatr*. 1994;125:57–62.
191. 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.
192. 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.
193. Browning DH, Graves SA. Incidence of aspiration with endotracheal tubes in children. *J Pediatr*. 1983;102:582–584.
194. 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.
195. 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.
196. Andersen KH, Hald A. Assessing the position of the tracheal tube: the reliability of different methods. *Anaesthesia*. 1989;44:984–985.
197. Bhende MS, Thompson AE, Cook DR, Saville AL. Validity of a disposable end-tidal CO_2 detector in verifying endotracheal tube placement in infants and children. *Ann Emerg Med*. 1992;21:142–145.
198. 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.
199. 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.
200. 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.
201. Bechtel K, Bhende M, Venkataraman S, Allen J. Use of esophageal detector device in a newborn-piglet model. *Ann Emerg Med*. 1998;31: 344–350.
202. Gausche-Hill M, Lewis RJ, Gunter CS, Henderson DP, Haynes BE, Stratton SJ. Design and implementation of a controlled trial of pediatric endotracheal intubation in the out-of-hospital setting. *Ann Emerg Med*. 2000;36:356–365.
203. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med*. 2001;37: 32–37.
204. Bhende MS, Allen WD Jr. Evaluation of a Capno-Flo resuscitator during transport of critically ill children. *Pediatr Emerg Care*. 2002;18: 414–416.
205. Campbell RC, Boyd CR, Shields RO, Odom JW, Corse KM. Evaluation of an end-tidal carbon dioxide detector in the aeromedical setting. *J Air Med Transp*. 1990;9:13–15.
206. Gonzalez del Rey JA, Poirier MP, Digiulio GA. Evaluation of an ambu-bag valve with a self-contained, colorimetric end-tidal CO_2 system in the detection of airway mishaps: an animal trial. *Pediatr Emerg Care*. 2000;16:121–123.
207. 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.
208. Tan A, Schulze A, O'Donnell CP, Davis PG. Air versus oxygen for resuscitation of infants at birth. *Cochrane Database Syst Rev*. 2004: CD002273.
209. 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.
210. Saugstad OD, Rootwelt T, Aalen O. Resuscitation of asphyxiated newborn infants with room air or oxygen: an international controlled trial: the Resair 2 study. *Pediatrics*. 1998;102:e1.
211. Ramji S, Rasaily R, Mishra PK, Narang A, Jayam S, Kapoor AN, Kambo I, Mathur A, Saxena BN. Resuscitation of asphyxiated newborns with room air or 100% oxygen at birth: a multicentric clinical trial. *Indian Pediatr*. 2003;40:510–517.
212. 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.
213. 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.
214. Lipinski CA, Hicks SD, Callaway CW. Normoxic ventilation during resuscitation and outcome from asphyxial cardiac arrest in rats. *Resuscitation.* 1999;42:221–229.
215. 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.
216. Brickman KR, Krupp K, Rega P, Alexander J, Guinness M. Typing and screening of blood from intraosseous access. *Ann Emerg Med*. 1992;21: 414–417.
217. Fiser RT, Walker WM, Seibert JJ, McCarthy R, Fiser DH. Tibial length following intraosseous infusion: a prospective, radiographic analysis. *Pediatr Emerg Care*. 1997;13:186–188.
218. Ummenhofer W, Frei FJ, Urwyler A, Drewe J. Are laboratory values in bone marrow aspirate predictable for venous blood in paediatric patients? *Resuscitation.* 1994;27:123–128.
219. 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.
220. Guy J, Haley K, Zuspan SJ. Use of intraosseous infusion in the pediatric trauma patient. *J Pediatr* Surg. 1993;28:158–161.
221. Macnab A, Christenson J, Findlay J, Horwood B, Johnson D, Jones L, Phillips K, Pollack C Jr, Robinson DJ, Rumball C, Stair T, Tiffany B, Whelan M. A new system for sternal intraosseous infusion in adults. *Prehosp Emerg Care*. 2000;4:173–177.
222. 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.
223. Howard RF, Bingham RM. Endotracheal compared with intravenous administration of atropine. *Arch Dis Child*. 1990;65:449–450.
224. Lee PL, Chung YT, Lee BY, Yeh CY, Lin SY, Chao CC. The optimal dose of atropine via the endotracheal route. *Ma Zui Xue Za Zhi*. 1989; 27:35–38.
225. Prengel AW, Lindner KH, Hahnel J, Ahnefeld FW. Endotracheal and endobronchial lidocaine administration: effects on plasma lidocaine concentration and blood gases. *Crit Care Med*. 1991;19:911–915.
226. Schmidbauer S, Kneifel HA, Hallfeldt KK. Endobronchial application of high dose epinephrine in out of hospital cardiopulmonary resuscitation. *Resuscitation.* 2000;47:89.
227. Raymondos K, Panning B, Leuwer M, Brechelt G, Korte T, Niehaus M, Tebbenjohanns J, Piepenbrock S. Absorption and hemodynamic effects of airway administration of adrenaline in patients with severe cardiac disease. *Ann Intern Med*. 2000;132:800–803.
228. Hahnel JH, Lindner KH, Schurmann C, Prengel A, Ahnefeld FW. Plasma lidocaine levels and Pao_2 with endobronchial administration: dilution with normal saline or distilled water? *Ann Emerg Med*. 1990; 19:1314–1317.
229. Brown LK, Diamond J. The efficacy of lidocaine in ventricular fibrillation due to coronary artery ligation: endotracheal vs intravenous use. *Proc West Pharmacol Soc*. 1982;25:43–45.
230. Jasani MS, Nadkarni VM, Finkelstein MS, Hofmann WT, Salzman SK. Inspiratory-cycle instillation of endotracheal epinephrine in porcine arrest. *Acad Emerg Med*. 1994;1:340–345.
231. Wenzel V, Lindner KH, Prengel AW, Lurie KG, Strohmenger HU. Endobronchial vasopressin improves survival during cardiopulmonary resuscitation in pigs. *Anesthesiology*. 1997;86:1375–1381.
232. 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.
233. 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, table of contents.
234. Efrati O, Ben-Abraham R, Barak A, Modan-Moses D, Augarten A, Manisterski Y, Barzilay Z, Paret G. Endobronchial adrenaline: should it

be reconsidered? Dose response and haemodynamic effect in dogs. *Resuscitation.* 2003;59:117–122.
235. 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.
236. 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.
237. 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.
238. 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.
239. Dieckmann R, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. *Pediatrics*. 1995;95:901–913.
240. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. *Resuscitation.* 2002;52: 149–156.
241. Voelckel WG, Lindner KH, Wenzel V, Bonatti J, Hangler H, Frimmel C, Kunszberg E, Lingnau W. Effects of vasopressin and epinephrine on splanchnic blood flow and renal function during and after cardiopulmonary resuscitation in pigs. *Crit Care Med*. 2000;28:1083–1088.
242. Voelckel WG, Lurie KG, McKnite S, Zielinski T, Lindstrom P, Peterson C, Wenzel V, Lindner KH, Benditt D. Effects of epinephrine and vasopressin in a piglet model of prolonged ventricular fibrillation and cardiopulmonary resuscitation. *Crit Care Med*. 2002;30:957–962.
243. 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.
244. 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.
245. 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.
246. Guyette FX, Guimond GE, Hostler D, Callaway CW. Vasopressin administered with epinephrine is associated with a return of a pulse in out-of-hospital cardiac arrest. *Resuscitation.* 2004;63:277–282.
247. Aung K, Htay T. Vasopressin for cardiac arrest: a systematic review and meta-analysis. *Arch Intern Med*. 2005;165:17–24.
248. Cannon LA, Heiselman DE, Dougherty JM, Jones J. Magnesium levels in cardiac arrest victims: relationship between magnesium levels and successful resuscitation. *Ann Emerg Med*. 1987;16:1195–1199.
249. Buylaert WA, Calle PA, Houbrechts HN. Serum electrolyte disturbances in the post-resuscitation period. *Resuscitation.* 1989;17:S189–S196.
250. Salerno DM, Elsperger KJ, Helseth P, Murakami M, Chepuri V. Serum potassium, calcium and magnesium after resuscitation from ventricular fibrillation: a canine study. *J Am Coll Cardiol*. 1987;10:178–185.
251. Allegra J, Lavery R, Cody R, Birnbaum G, Brennan J, Hartman A, Horowitz M, Nashed A, Yablonski M. Magnesium sulfate in the treatment of refractory ventricular fibrillation in the prehospital setting. *Resuscitation.* 2001;49:245–249.
252. Fatovich DM, Prentice DA, Dobb GJ. Magnesium in cardiac arrest (the magic trial). *Resuscitation.* 1997;35:237–241.
253. Hassan TB, Jagger C, Barnett DB. A randomised trial to investigate the efficacy of magnesium sulphate for refractory ventricular fibrillation. *Emerg Med J*. 2002;19:57–62.
254. Longstreth WT Jr, Fahrenbruch CE, Olsufka M, Walsh TR, Copass MK, Cobb LA. Randomized clinical trial of magnesium, diazepam, or both after out-of-hospital cardiac arrest. *Neurology*. 2002;59:506–514.
255. Thel MC, Armstrong AL, McNulty SE, Califf RM, O'Connor CM. Randomised trial of magnesium in in-hospital cardiac arrest. Duke Internal Medicine Housestaff. *Lancet*. 1997;350:1272–1276.
256. Miller B, Craddock L, Hoffenberg S, Heinz S, Lefkowitz D, Callender ML, Battaglia C, Maines C, Masick D. Pilot study of intravenous magnesium sulfate in refractory cardiac arrest: safety data and recommendations for future studies. *Resuscitation.* 1995;30:3–14.
257. Brown CG, Griffith RF, Neely D, Hobson J, Miller B. The effect of intravenous magnesium administration on aortic, right atrial and coronary perfusion pressures during CPR in swine. *Resuscitation.* 1993; 26:3–12.
258. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. *Stroke.* 1997;28: 1569–1573.
259. 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.
260. Muizelaar JP, Marmarou A, Ward JD, Kontos HA, Choi SC, Becker DP, Gruemer H, Young HF. Adverse effects of prolonged hyperventilation in patients with severe head injury: a randomized clinical trial. *J Neurosurg*. 1991;75:731–739.
261. Carmona Suazo JA, Maas AI, van den Brink WA, van Santbrink H, Steyerberg EW, Avezaat CJ. CO_2 reactivity and brain oxygen pressure monitoring in severe head injury. *Crit Care Med*. 2000;28:3268–3274.
262. Schneider GH, Sarrafzadeh AS, Kiening KL, Bardt TF, Unterberg AW, Lanksch WR. Influence of hyperventilation on brain tissue-Po_2, Pco_2, and pH in patients with intracranial hypertension. *Acta Neurochir Suppl*. 1998;71:62–65.
263. Unterberg AW, Kiening KL, Hartl R, Bardt T, Sarrafzadeh AS, Lanksch WR. Multimodal monitoring in patients with head injury: evaluation of the effects of treatment on cerebral oxygenation. *J Trauma*. 1997;42: S32–S37.
264. Skippen P, Seear M, Poskitt K, Kestle J, Cochrane D, Annich G, Handel J. Effect of hyperventilation on regional cerebral blood flow in head-injured children. *Crit Care Med*. 1997;25:1402–1409.
265. Hutchinson PJ, Gupta AK, Fryer TF, Al-Rawi PG, Chatfield DA, Coles JP, O'Connell MT, Kett-White R, Minhas PS, Aigbirhio FI, Clark JC, Kirkpatrick PJ, Menon DK, Pickard JD. Correlation between cerebral blood flow, substrate delivery, and metabolism in head injury: a combined microdialysis and triple oxygen positron emission tomography study. *J Cereb Blood Flow Metab*. 2002;22:735–745.
266. Coles JP, Minhas PS, Fryer TD, Smielewski P, Aigbirihio F, Donovan T, Downey SP, Williams G, Chatfield D, Matthews JC, Gupta AK, Carpenter TA, Clark JC, Pickard JD, Menon DK. Effect of hyperventilation on cerebral blood flow in traumatic head injury: clinical relevance and monitoring correlates. *Crit Care Med*. 2002;30:1950–1959.
267. Dings J, Meixensberger J, Amschler J, Roosen K. Continuous monitoring of brain tissue Po_2: a new tool to minimize the risk of ischemia caused by hyperventilation therapy. *Zentralbl Neurochir*. 1996;57: 177–183.
268. Cold GE. Measurements of CO_2 reactivity and barbiturate reactivity in patients with severe head injury. *Acta Neurochir (Wien)*. 1989;98: 153–163.
269. Zhang S, Zhi D, Lin X, Shang Y, Niu Y. Effect of mild hypothermia on partial pressure of oxygen in brain tissue and brain temperature in patients with severe head injury. *Chin J Traumatol*. 2002;5:43–45.
270. Obrist WD, Langfitt TW, Jaggi JL, Cruz J, Gennarelli TA. Cerebral blood flow and metabolism in comatose patients with acute head injury. Relationship to intracranial hypertension. *J Neurosurg*. 1984;61: 241–253.
271. Hickey RW, Kochanek PM, Ferimer H, Graham SH, Safar P. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. *Pediatrics*. 2000;106(pt 1):118–122.
272. 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.
273. 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.
274. 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.
275. Battin MR, Penrice J, Gunn TR, Gunn AJ. Treatment of term infants with head cooling and mild systemic hypothermia (35.0 degrees C and 34.5 degrees C) after perinatal asphyxia. *Pediatrics*. 2003;111:244–251.
276. Compagnoni G, Pogliani L, Lista G, Castoldi F, Fontana P, Mosca F. Hypothermia reduces neurological damage in asphyxiated newborn infants. *Biol Neonate*. 2002;82:222–227.
277. Debillon T, Daoud P, Durand P, Cantagrel S, Jouvet P, Saizou C, Zupan V. Whole-body cooling after perinatal asphyxia: a pilot study in term neonates. *Dev Med Child Neurol*. 2003;45:17–23.

278. Gunn AJ, Gluckman PD, Gunn TR. Selective head cooling in newborn infants after perinatal asphyxia: a safety study. *Pediatrics*. 1998;102: 885–892.
279. Albrecht RF 2nd, Wass CT, Lanier WL. Occurrence of potentially detrimental temperature alterations in hospitalized patients at risk for brain injury. *Mayo Clin Proc*. 1998;73:629–635.
280. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. *Intensive Care Med*. 1991;17:419–420.
281. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? *Resuscitation.* 2001;49: 273–277.
282. Zeiner A, Holzer M, Sterz F, Schorkhuber W, Eisenburger P, Havel C, Kliegel A, Laggner AN. Hyperthermia after cardiac arrest is associated with an unfavorable neurologic outcome. *Arch Intern Med*. 2001;161: 2007–2012.
283. Hickey RW, Kochanek PM, Ferimer H, Alexander HL, Garman RH, Graham SH. Induced hyperthermia exacerbates neurologic neuronal histologic damage after asphyxial cardiac arrest in rats. *Crit Care Med*. 2003;31:531–535.
284. Coimbra C, Boris-Moller F, Drake M, Wieloch T. Diminished neuronal damage in the rat brain by late treatment with the antipyretic drug dipyrone or cooling following cerebral ischemia. *Acta Neuropathol (Berl)*. 1996;92:447–453.
285. Coimbra C, Drake M, Boris-Moller F, Wieloch T. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an anti-inflammatory/antipyretic drug: evidence for chronic encephalopathic processes following ischemia. *Stroke.* 1996;27:1578–1585.
286. Hildebrand CA, Hartmann AG, Arcinue EL, Gomez RJ, Bing RJ. Cardiac performance in pediatric near-drowning. *Crit Care Med*. 1988;16:331–335.
287. 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.
288. 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.
289. Mullner M, Domanovits H, Sterz F, Herkner H, Gamper G, Kurkciyan I, Laggner AN. Measurement of myocardial contractility following successful resuscitation: quantitated left ventricular systolic function utilising non-invasive wall stress analysis. *Resuscitation.* 1998;39: 51–59.
290. 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.
291. Kamohara T, Weil MH, Tang W, Sun S, Yamaguchi H, Klouche K, Bisera J. A comparison of myocardial function after primary cardiac and primary asphyxial cardiac arrest. *Am J Respir Crit Care Med*. 2001;164: 1221–1224.
292. Gazmuri RJ, Weil MH, Bisera J, Tang W, Fukui M, McKee D. Myocardial dysfunction after successful resuscitation from cardiac arrest. *Crit Care Med*. 1996;24:992–1000.
293. Lobato EB, Gravenstein N, Martin TD. Milrinone, not epinephrine, improves left ventricular compliance after cardiopulmonary bypass. *J Cardiothorac Vasc Anesth*. 2000;14:374–377.
294. 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.
295. 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.
296. Niemann JT, Garner D, Khaleeli E, Lewis RJ. Milrinone facilitates resuscitation from cardiac arrest and attenuates postresuscitation myocardial dysfunction. *Circulation*. 2003;108:3031–3035.
297. 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.
298. Innes PA, Frazer RS, Booker PD, Allsop E, Kirton C, Lockie J, Franks R. Comparison of the haemodynamic effects of dobutamine with enoximone after open heart surgery in small children. *Br J Anaesth*. 1994;72:77–81.
299. Laitinen P, Happonen JM, Sairanen H, Peltola K, Rautiainen P. Amrinone versus dopamine and nitroglycerin in neonates after arterial switch operation for transposition of the great arteries. *J Cardiothorac Vasc Anesth*. 1999;13:186–190.
300. 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(4): 323–329.
301. Butterworth JFt, Royster RL, Prielipp RC, Lawless ST, Wallenhaupt SL. Amrinone in cardiac surgical patients with left-ventricular dysfunction: a prospective, randomized placebo-controlled trial. *Chest.* 1993;104: 1660–1667.
302. Kikura M, Sato S. The efficacy of preemptive Milrinone or Amrinone therapy in patients undergoing coronary artery bypass grafting. *Anesth Analg*. 2002;94:22–30, table of contents.
303. Calle PA, Buylaert WA, Vanhaute OA. Glycemia in the post-resuscitation period. The Cerebral Resuscitation Study Group. *Resuscitation.* 1989;17 Suppl:S181–S188; discussion S199–S206.
304. Langhelle A, Tyvold SS, Lexow K, Hapnes SA, Sunde K, Steen PA. In-hospital factors associated with improved outcome after out-of-hospital cardiac arrest: a comparison between four regions in Norway. *Resuscitation.* 2003;56:247–263.
305. Longstreth WT Jr, Diehr P, Inui TS. Prediction of awakening after out-of-hospital cardiac arrest. *N Engl J Med*. 1983;308:1378–1382.
306. Longstreth WT Jr, Inui TS. High blood glucose level on hospital admission and poor neurological recovery after cardiac arrest. *Ann Neurol*. 1984;15:59–63.
307. Mullner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. *J Cereb Blood Flow Metab*. 1997;17:430–436.
308. Skrifvars MB, Pettila V, Rosenberg PH, Castren M. A multiple logistic regression analysis of in-hospital factors related to survival at six months in patients resuscitated from out-of-hospital ventricular fibrillation. *Resuscitation.* 2003;59:319–328.
309. Losek JD. Hypoglycemia and the ABC'S (sugar) of pediatric resuscitation. *Ann Emerg Med*. 2000;35:43–46.
310. Srinivasan V, Spinella PC, Drott HR, Roth CL, Helfaer MA, Nadkarni V. Association of timing, duration, and intensity of hyperglycemia with intensive care unit mortality in critically ill children. *Pediatr Crit Care Med*. 2004;5:329–336.
311. 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.
312. Hoxworth JM, Xu K, Zhou Y, Lust WD, LaManna JC. Cerebral metabolic profile, selective neuron loss, and survival of acute and chronic hyperglycemic rats following cardiac arrest and resuscitation. *Brain Res*. 1999;821:467–479.
313. 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.
314. Nakakimura K, Fleischer JE, Drummond JC, Scheller MS, Zornow MH, Grafe MR, Shapiro HM. Glucose administration before cardiac arrest worsens neurologic outcome in cats. *Anesthesiology*. 1990;72: 1005–1011.
315. Farias LA, Willis M, Gregory GA. Effects of fructose-1,6-diphosphate, glucose, and saline on cardiac resuscitation. *Anesthesiology.* 1986;65: 595–601.
316. Natale JE, Stante SM, D'Alecy LG. Elevated brain lactate accumulation and increased neurologic deficit are associated with modest hyperglycemia in global brain ischemia. *Resuscitation.* 1990;19:271–289.
317. Longstreth WT Jr, Copass MK, Dennis LK, Rauch-Matthews ME, Stark MS, Cobb LA. Intravenous glucose after out-of-hospital cardiopulmonary arrest: a community-based randomized trial. *Neurology*. 1993;43: 2534–2541.
318. Vannucci RC, Vannucci SJ. Hypoglycemic brain injury. *Semin Neonatol*. 2001;6:147–155.
319. Salhab WA, Wyckoff MH, Laptook AR, Perlman JM. Initial hypoglycemia and neonatal brain injury in term infants with severe fetal acidemia. *Pediatrics*. 2004;114:361–366.
320. Lin Y, Greisen G. Analysis of the risk of brain damage in asphyxiated infants. *J Perinat Med*. 1996;24:581–589.

321. Mir NA, Faquih AM, Legnain M. Perinatal risk factors in birth asphyxia: relationship of obstetric and neonatal complications to neonatal mortality in 16,365 consecutive live births. *Asia Oceania J Obstet Gynaecol*. 1989;15:351–357.
322. Ondoa-Onama C, Tumwine JK. Immediate outcome of babies with low Apgar score in Mulago Hospital, Uganda. *East Afr Med J*. 2003;80:22–29.
323. Longstreth WT Jr, Diehr P, Cobb LA, Hanson RW, Blair AD. Neurologic outcome and blood glucose levels during out-of-hospital cardiopulmonary resuscitation. *Neurology*. 1986;36:1186–1191.
324. Gillis J, Dickson D, Rieder M, Steward D, Edmonds J. Results of inpatient pediatric resuscitation. *Crit Care Med*. 1986;14:469–471.
325. Schindler MB, Bohn D, Cox PN, McCrindle BW, Jarvis A, Edmonds J, Barker G. Outcome of out-of-hospital cardiac or respiratory arrest in children. *N Engl J Med*. 1996;335:1473–1479.
326. Suominen P, Korpela R, Kuisma M, Silfvast T, Olkkola KT. Paediatric cardiac arrest and resuscitation provided by physician-staffed emergency care units. *Acta Anaesthesiol Scand*. 1997;41:260–265.
327. 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.
328. Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Rodriguez-Nunez A, Calvo C, Delgado MA. Characteristics and outcome of cardiorespiratory arrest in children. *Resuscitation*. 2004;63:311–320.
329. 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.
330. Idris AH, Berg RA, Bierens J, Bossaert L, Branche CM, Gabrielli A, Graves SA, Handley AJ, Hoelle R, Morley PT, Papa L, Pepe PE, Quan L, Szpilman D, Wigginton JG, Modell JH. Recommended guidelines for uniform reporting of data from drowning: The "Utstein style". *Resuscitation*. 2003;59:45–57.
331. 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.
332. 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.
333. del Nido PJ. Extracorporeal membrane oxygenation for cardiac support in children. *Ann Thorac Surg*. 1996;61:336–339; discussion 340–341.
334. 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.
335. 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.
336. 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 2101–2102.
337. Hazinski MF, Chahine AA, Holcomb GW 3rd, Morris JA Jr. Outcome of cardiovascular collapse in pediatric blunt trauma. *Ann Emerg Med*. 1994;23:1229–1235.

Worksheets Cited

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W2. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC2*
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W9A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC13*
W9B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC14*
W10. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC15*
W11A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC16*
W11B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC17*
W12B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC19*
W13. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC20*
W14A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC21*
W14B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC22*
W15. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC23*
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W18. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC26*
W19A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC27*
W19B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC28*
W20. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC29*
W21A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC30*
W21B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC31*
W22A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC32*
W22B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC33*
W22C. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC34*
W22D. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC35*
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W25. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC38*
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W26B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC40*
W27. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC41*
W28. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC42*
W29. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC43*
W30A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC44*
W30B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC45*
W30C. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC46*
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W33C. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC52*
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W39A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC59*
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W41A. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC62*
W41B. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC63*
W276. *http://circ.ahajournals.org/cgi/content/full/CIRCULATIONAHA.105.170522/DC449*