AMERICAN HEALTH CONSULTANTS®

Rapid Access Card Enclosed

Enclosed in this issue: Trauma Reports

PEDIATRIC Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Volume 8, Number 1 January 2003

Pediatric Cardiopulmonary Arrest: Current Concepts and Future Directions

Authors: **Robert W. Hickey, MD,** and **Noel S. Zuckerbraun, MD,** Department of Pediatrics, Division of Emergency Medicine, Children's Hospital of Pittsburgh, PA.

Peer Reviewer: **David R. Kroning, MD**, Associate Chairman, Department of Emergency Medicine, St. Joseph's Regional Medical Center, St. Joseph's Children's Hospital, Paterson, NJ.

American Heart Association (AHA)'s Guidelines 2000 for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care: International Consensus on Science[1] was the culmination of a process involving approximately 500 international experts reviewing more than 25,000 journal articles with the objective of updating the previous Guidelines published by the AHA in 1992. This review article will discuss selected topics appearing in Guidelines 2000 with an emphasis on topics of interest to clinicians caring for critically ill/injured children. Topics were chosen because they either represent changes to current clinical management or contain data derived in innovative ways that have the potential to alter clinical care in the future. Following a brief background section, topics are organized using the chain of survival concept (prevention, CPR, emergency medical services [EMS], advanced life support). The review will end with a discussion of future directions and a brief summary.

Background

The causes of pediatric cardiopulmonary arrest are heterogeneous. The most common causes of arrest in the pre-hospital setting are sudden infant death syndrome (SIDS), trauma, poisoning, and respiratory distress secondary to drowning, choking, severe asthma or pneumonia. *(See Figure 1.)* Trauma remains the leading cause of death in childhood and young adulthood.[2] Traumatic death can be caused by exsanguination, massive head injury, or airway compromise. Non-traumatic cardiac arrest in children typically occurs as a consequence of hypoxia and hypercarbia leading to respiratory arrest, bradycardia, and then, finally, asystolic cardiac arrest.[3-5] Ventricular tachycardia (VT) or fibrillation (VF) occurs less commonly in children than in adults, but it is not rare; approximately 5-15% of children with pre-hospital arrest have VF/VT.[6-8] Overall, the rate of survival from pre-hospital pediatric cardiopulmonary arrest is approximately 10%.[5] Patients in asystolic arrest have the lowest rate of survival (approximately 5%), whereas patients with VF/VT have higher rates of survival (approximately 30%).[5] Patients presenting with isolated respiratory arrest without cardiovascular collapse have the highest rate of survival (approximately 75%).[9,10] Unfortunately, survivors of pediatric arrest often suffer from severe neurologic deficits.

The epidemiology of pediatric cardiac arrest directs attention to several targets for intervention. Specifically:

- Trauma is the leading killer of children; therefore, the development of successful injury control interventions is a high priority;[11,12]
- Most non-traumatic causes of arrest in children are secondary to airway compromise; thus, evaluation and support of the airway is a priority for rescuers;[3]
- VF/VT is a highly salvageable rhythm and should be actively sought early in resuscitation;[5,8] and
- Survival with intact neurologic recovery, rather than survival alone, is the ultimate goal of pediatric resuscitation.[5]

Achieving these goals requires coordination of a community-wide "chain of survival." The pediatric chain of survival includes prevention, early CPR, early EMS activation, and early advanced life support. Each of these links is a critical component in pediatric arrest.

Chain of Survival: Prevention

Injuries are responsible for more childhood deaths in the United States than all other causes combined.[11,12] The majority of traumatic deaths in children are secondary to motor vehicle injuries, drownings, fires, and suffocations. These, and other mechanisms, have become the target for important work done by those in the injury prevention field. For example, the use of child restraints in motor vehicles, bicycle helmets, pool fences, and fire alarms have contributed to important reductions in morbidity and mortality.[13-18]

A decade ago, few would have predicted that prevention strategies could decrease the risk of SIDS. Fortunately, the simple observation that sleeping in the prone position increased the risk of SIDS led to a successful prevention program. Key epidemiologic evidence in the United States was developed by investigators comparing the rate of SIDS before and after an article about the risk of prone sleeping appeared in the *Seattle Times.*[19] The investigators reported that subsequent to the newspaper article, the rate of deaths from SIDS was reduced by 52% in King County (Seattle) and 20% in an adjacent county where the paper was delivered, whereas there was no reduction in the remainder of the state. This helped to prompt the initiation of a nationwide public health education campaign using the slogan "Back to Sleep" in 1992. The number of SIDS cases has decreased in the United States from approximately 4900 infants in 1992 to 2600 infants in 1999 (rates of 120 and 67 per 100,000 live births, respectively).[20,21] The mortality rate from SIDS has declined by approximately 50% in various countries following similar intervention efforts.[22] The mechanism underlying the association between SIDS and prone sleeping is not known; however, one hypothesis is that prone sleeping contributes to rebreathing asphyxia.[23]

Chain of Survival: Early CPR

Early bystander CPR clearly improves survival and neurologic recovery in adults[24-28] and children with cardiac arrest.[4,6,29] Sirbaugh et al. have published the only prospective, population-based study of pediatric arrest.[4] They describe interventions and outcomes of 300 children who were in cardiac arrest upon EMS arrival. Consistent with other studies, 2% survived.[6] Of the six survivors, five were discharged from the hospital with significant neurologic deficits. However, in addition to the 300 patients with continued cardiac arrest upon EMS arrival, there were 41 children who received bystander CPR for apparent lifelessness following submersion and all had a pulse and respiratory efforts upon EMS arrival. All were discharged from the hospital with full neurologic recovery. It is possible that some of these children had good outcomes because they had isolated respiratory arrest or perhaps had no arrest at all, but were incorrectly perceived as lifeless by overzealous bystander rescuers. That being said, a detailed description of similar "bystander saves" reported in a different study supports the interpretation that bystander

Pediatric Emergency Medicine Reports™ (ISSN 1082-3344) is published monthly by American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
Marketing Manager: Schandale Kornegay

GST Registration No.: R128870672

Periodicals Postage Paid at Atlanta, GA 30304.

POSTMASTER: Send address changes to ***Pediatric Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $57.
Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail Address: customerservice@ahcpub.com

Editorial E-Mail Address: allison.mechem@ahcpub.com

World-Wide Web page: http://www.ahcpub.com

Subscription Prices

1 year *with* 30 ACEP, AMA, or AAP Category 1 credits: $389;
1 year without credit: $339;

Multiple copies:
One to nine additional copies: **$305 each;**
10 or more additional copies: **$271 each.**

Resident's Rate: **$169.50**

All prices U.S. only. U.S. possessions and Canada, add $30 postage plus applicable GST.
Other international orders, add $30.

Accreditation

Pediatric Emergency Medicine Reports'™ continuing education materials are sponsored and supervised by American Health Consultants. American Health Consultants designates this continuing education activity as meeting the criteria for 30 credit hours in Category 1 for Education Materials for the Physician's Recognition Award of the American Medical Association, provided it has been completed according to instructions.

This CME activity was planned and produced in accordance with the ACCME Essentials.

Pediatric Emergency Medicine Reports is also approved by the American College of Emergency Physicians for 30 hours of ACEP Category 1 credit. This continuing medical education activity has been reviewed by the American Academy of Pediatrics and is acceptable for 30 AAP Credit hours. These credits can be applied toward the PREP Education Award available to Fellows and Candidate Fellows of the American Academy of Pediatrics.

American Health Consultants is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

THOMSON
AMERICAN HEALTH CONSULTANTS ™

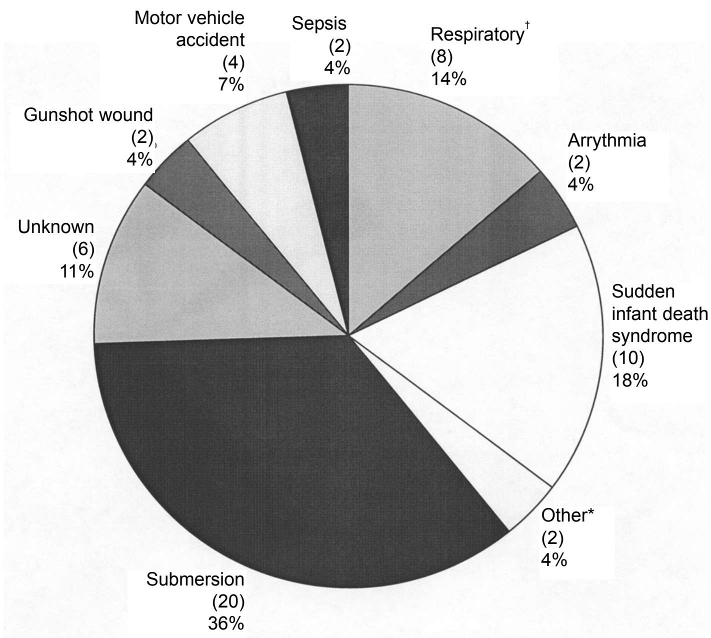
Questions & Comments

Please call **Allison Mechem,** Managing Editor at (404) 262-5589 between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

Statement of Financial Disclosure

American Health Consultants does not receive material commercial support for any of its continuing medical education publications. In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Hickey and Zuckerbraun (authors), and Kroning (peer reviewer) report no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study. Dr. Dietrich, editor-in-chief, also reports no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

Figure 1. Causes of Cardiac Arrest in 56 Pediatric Patients Requiring CPR in the Prehospital Setting



† Includes four episodes of aspiration (three emesis, one foreign body), one epiglottitis, one tracheostomy occlusion, one patient wedged between a wall and a mattress, and one lying over.
* Includes one case of child abuse and one ventriculoperitoneal shunt malfunction.

Percentages were rounded and do not equal 100%.

Adapted with permission from Hickey R, Cohen D, Strausbaugh R, et al. Pediatric patients requirirng CPR in the prehospital setting. *Ann Emerg Med* 1995;25:496.

CPR alone is both necessary and sufficient to rescue at least some children.[6] In this study, children resuscitated prior to EMS arrival were included only if they had persistent altered mental status or metabolic acidosis upon arrival to the emergency department (ED). Eleven children, all resuscitated from near drowning, fulfilled the criteria. Many of the bystander rescuers would be expected to have reasonable skill in determining the need to provide CPR (two police officers, two lifeguards, one off-duty EMT, and one physician). Three children were intubated upon EMS arrival because of persistent respiratory distress and one subsequently died of acute respiratory distress syndrome. Neurologists were consulted to evaluate three children with neurologic sequelae. Although these 11 children no longer required CPR upon EMS arrival, it is clear they suffered significant events and that the bystander CPR was more likely prudent and life-saving than overzealous and unnecessary.

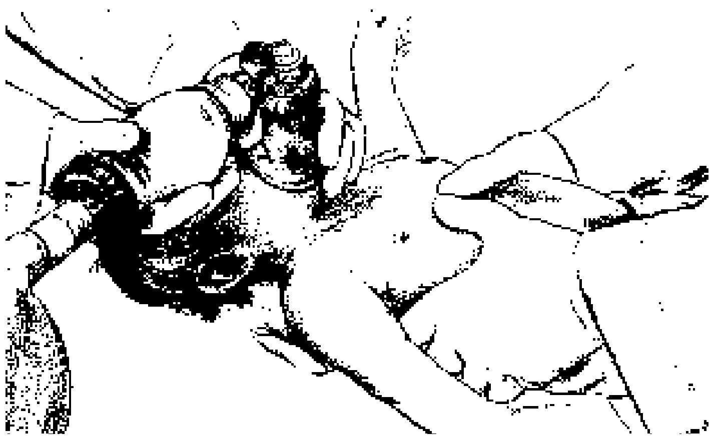
The evidence discussed above suggests that CPR saves lives and saves more lives than is acknowledged in the literature. CPR alone will rescue some patients, and those patients requiring additional advanced life support will have improved outcome if CPR is provided early. Increasing the rate of bystander CPR is an important public health measure that should be advocated strongly by health care professionals.

One barrier to bystander CPR is the layperson's anxiety about improperly performing CPR. Thus, any modifications that can simplify CPR without significantly decreasing efficacy are desirable. A recent example of such a modification is deletion of the "pulse check" by the lay rescuer. Traditionally, pulselessness is the trigger for starting CPR and is verified by palpating the carotid pulse (in adults) for 5-10 seconds. Strong evidence that this is unrealistic for lay rescuers was provided in a study by Eberle et al.[30] "Lay persons" (many of them EMT students) were escorted into an operating suite during open heart surgery. The lay persons were asked to assess for a carotid pulse while being shielded from monitor readouts and kept unaware of the patients' bypass status. During the cardiopulmonary bypass portions of surgery, blood flow is laminar, and no pulse should be detected, whereas a pulse should be palpable prior to and following bypass. Only 15% of lay persons were able to make the correct diagnosis within 10 seconds. Given additional time, a pulse eventually was palpated in only 45% of instances when a pulse was present. However, the most concerning finding was that in 10% of evaluations when a carotid pulse was absent, the provider believed a pulse was present. Extrapolated to the clinical scenario, 10% of pulseless patients might not receive CPR because a bystander incorrectly perceives a pulse to be present. On the basis of this elegant study, the AHA no longer recommends that lay providers check for a pulse.[1] Instead, rescuers should perform CPR whenever a victim has "no signs of circulation." Although the data is largely confined to adult studies, the same recommendation has been made for instructing lay providers in pediatric basic life support (BLS). The AHA continues to recommend that health care providers confirm pulselessness prior to starting CPR.

Two methods exist for delivering chest compressions to infants. Compressions can be supplied with two fingers from one hand or with the thumbs from both hands encircling the chest. *(See Figure 2.)* Based upon both animal and human data, the two-thumb method is now preferred when two or more health care providers are available.[31] When only one rescuer is present, the two-thumb method is not recommended because one hand is needed to maintain the head tilt. The two-thumb method is not taught to lay providers because it unnecessarily complicates CPR training.

Several alternative methods for performing CPR now are recognized by the AHA as acceptable options in adults, but have not been tested in or recommended for children. However, because these alternative methods are designed to manipulate the mechanical physiology of CPR, which is similar in children and adults, they may eventually prove to be of benefit in children. Two representative alternative methods will be discussed briefly. The first, active compression-decompression CPR (ACDC-CPR), was developed after a family reported successful resuscitation of their father using a toilet plunger for chest compressions.[32] The family, impressed by their success, recommended to the receiving doctor that a toilet plunger be placed at every bedside in the hospital. To the receiving doctor's credit, he kept an open mind and developed a portable suction device that delivers

Figure 2. Two-thumb Encircling Hands Chest Compression Technique in Infant (2 Rescuers)



Reprinted with permission: American Heart Association. *Pediatric Advanced Life Support* 1997;70:1091.

chest compressions using a push/pull motion. The active decompression (pull) phase creates a negative pressure in the chest, facilitating blood return to the heart and lungs. The increase in blood return during active decompression augments the amount of blood returned to the systemic circulation during the compression phase.[32-34] Results from animal studies are favorable, but results from clinical trials are conflicting.[35-37] The second technique is interposed abdominal compression CPR (IAC-CPR). To perform IAC-CPR, a second rescuer compresses the abdomen during the relaxation phase of chest compressions.[38,39] The interposed abdominal compression increases the diastolic pressure in the aorta and improves coronary artery perfusion pressure. Clinical studies of IAC-CPR during in-hospital resuscitations have demonstrated improved return of spontaneous circulation and improved survival to hospital discharge.[40,41] Recently, a device has been developed to facilitate IAC-CPR. The device is attached to the chest and abdomen using adhesive pads and is rocked back and forth in a seesaw motion. In addition to their potential clinical applications, these methods/devices are noteworthy because they demonstrate the power of clinical-investigator ingenuity and display the potential for improving the mechanics of CPR with "low-tech" modifications.

Chain of Survival: Early EMS Activation

Activation of EMS is a critical link in resuscitation from cardiac arrest. EMS personnel can ventilate with supplemental oxygen, administer medications, defibrillate VF/VT, and provide expeditious transport to the hospital.

The initial priority of the EMS system is to manage the airway. Traditionally, it has been assumed that pre-hospital intubation would provide the most secure and effective means of ventilation. Gausche et al. questioned this assumption in a study published in the February 2000 edition of the *Journal of the American Medical Association*.[42] The study was a prospective, clinical trial comparing survival, neurologic outcome, and adverse events in pediatric patients randomized to treatment with bag valve mask (BVM) ventilation vs. endotracheal (ET) intubation. There were 115 separate institutions, six funding agencies, and 3000 paramedics involved in the study. Patients determined by EMS to require supplemental airway support (because of seizures, trauma, cardiac arrest, etc.) were assigned to BVM on odd days or BVM followed by ET on even days. A total of 830 patients younger than 12 years of age were enrolled during a two-year period. There was no significant difference in survival or neurologic outcome between groups. There also was no significant difference in complications, including gastric distention, vomiting, and aspiration. However, time from EMS dispatch to arrival in the ED was longer in the ET intubation group. Furthermore, of 186 patients in whom intubation was believed to have been successful by the intubating medic, there were 3 (2%) with esophageal intubation and 12 (6%) with unrecognized dislodgement of the ET tube en route to the ED. The hard work and diligence incorporated into this study was astonishing. However, like most good research, this study has generated vigorous debate.[43] One of the limitations acknowledged by the study authors is the applicability of this study to other pre-hospital systems. This study was performed in a region with short transport times (six minutes from scene back to hospital). Thus, it is possible that a potential benefit of ET ventilation was masked by the short transport time. However, the counterargument is that systems with longer transport times still would have esophageal intubations and, possibly, more frequent dislodgements. It also is possible that Gausche's results reflect the local EMS training curriculum rather than the mode of ventilation (e.g., over-training BVM and inadequately teaching ET intubation). Regardless of the legitimacy of these limitations, they do not detract from the powerful message of this study: In an established EMS system, patients potentially were harmed by pre-hospital ET intubation. It is not adequate, therefore, to argue that "our system is different." The burden of proof now is shifted and prudent EMS medical directors will establish methods to document adequate acquisition and retention of skills if pre-hospital intubation remains part of their system. An important lesson for instructors teaching advanced airway skills is that BVM no longer should be "glossed over" in an effort to spend time practicing intubation. Rather, we believe BVM should be emphasized and ET intubation instruction should highlight methods for confirming and monitoring correct tube placement.

Early access to defibrillation has become a priority in resuscitation of adults in cardiac arrest. In adults, the time from collapse to defibrillation is the single greatest determinant of survival with survival declining 7-10% for each minute without defibrillation.[44] The development and distribution of automatic external defibrillators (AEDs) for use by first responders (and perhaps lay public) has greatly enhanced the ability to provide early defibrillation. For example, in 1997, American Airlines began placing AEDs on selected airplanes, and within two years, shocks were administered to 15 patients with VF. Remarkably, six (40%) were discharged alive from the hospital, each with full neurologic and functional recovery.[45]

Despite the dramatic results of AEDs in adults, their use in young children has not been a priority because most children suffering cardiopulmonary arrest are suffering from respiratory disease and are most likely to benefit from immediate attention to the airway. In addition, the widespread belief that children rarely have VF/VT also has lessened the enthusiasm for AED use in children. However, recent studies have documented that VF occurs in up to 15% of pediatric victims of pre-hospital arrest.[6-8] Furthermore, the survival rate is greater for VF than asystole (30% vs 5%).[5] Thus, VF is uncommon, but not rare, and it is a salvageable rhythm that should be sought and treated aggressively when found.

AEDs currently are approved by the AHA for use in children older than 8 years or weighing more than 55 pounds.[1] For younger/smaller children, there are two major concerns with AED usage. First, AED computer algorithms are designed to differentiate shockable vs. nonshockable dysrhythmias using decision rules applicable to adults. Because the definitions for tachyarrhythmias differ in children vs. adults, there is concern that a pediatric recording with a high-normal heart rate could be mischaracterized as a shockable rhythm using "adult criteria." The second concern is that most AEDs deliver an initial shock of 150-200 joules with subsequent doses escalating as high as 360 joules. Thus, a standard 200-joule shock delivered to an average size (10 kg) 1-year-old is a 20-joule per kilogram (j/kg) dosage. A study examining escalating doses in animals found evidence of myocardial injury at doses greater than 10 j/kg.[46] Two new positive developments in AED technology have begun to address both of these concerns. First, researchers recently have reported that a commercially available AED is able to successfully categorize normal and abnormal (shockable) rhythms when tested against a sample of pediatric ECGs.[47] Second, the FDA recently has granted marketing clearance for a "pediatric AED" that contains specially modified pads that deliver approximately 50 joules. Although these developments are promising, the AHA is awaiting reports on the initial clinical experience with AEDs prior to reconsidering their recommendation on their use in young children.

Chain of Survival: Early Advanced Life Support

Following bystander CPR and activation of EMS, the next link in the chain of survival is Advanced Life Support (ALS).

As discussed above, a life-threatening complication of intubation is the potential for misplacing the ET tube into the esophagus or having a correctly placed tube become dislodged without immediate recognition. The risk for this unacceptable error must be eliminated. There is perhaps no greater potential for iatrogenic harm during resuscitation than a misplaced ET tube. Accordingly, the AHA now recommends routine secondary confirmation of tracheal tube position.[1]

Pre-hospital intubation of pediatric patients is a difficult task. Studies generally report success rates approaching 50%.[42,48,49] In Gausche's previously mentioned report of 186 patients in whom intubation was believed successful, there were three patients with esophageal intubation, 12 with unrecognized dislodgement of the ET tube enroute to the ED, 15 with recognized dislodgement of the ET tube, 33 with mainstem intubation, and 44 with incorrect ET tube size placement.[42] CO_2 detectors were used to confirm initial tube placement in only 77% of intubated patients. A recently published study examining pre-hospital intubation of adults further supports the need for tube placement confirmation.[50] The study was a prospective observational study of patients intubated in the field by paramedics over an eight-month period. The ED physicians assessed tube position upon patient arrival to the ED. Pre-hospital CO_2 detection was "not consistently used." Of the 108 intubated patients, 27 (25%) were found to have improperly placed ET tubes, including 18 in the esophagus and nine in the hypopharynx above the vocal cords. An accompanying editorial strongly argues that this level of failure cannot go undetected or be tolerated in a modern health care system.[51]

End-tidal CO_2 detection is the method most commonly utilized for secondary confirmation of ET tube placement in children.[52] However, a false negative reading can occur when circulatory collapse is so severe that CO_2 is not delivered to the alveolar space. Thus, an ET tube can be placed correctly during CPR, but CO_2 is not detected. If CO_2 is not detected during CPR, an alternative method to confirm tube placement would be to "take a second look" with a laryngoscope. Alternatively, an esophageal detector device can be used in children,[53] but is unreliable in children younger than 1 year of age.[54] Although no single confirmation technique is 100% reliable in all circumstances, some effort of secondary confirmation of tube placement should be performed following intubation of all children.

Following intubation of a critically ill patient, two important questions arise. First, what is the appropriate amount of oxygen to deliver and, second, what is the appropriate target PCO_2?

Traditionally, patients are resuscitated using 100% oxygen. The rationale is that hypoxia often causes or contributes to the development of cardiac arrest, and an oxygen debt accumulates during cardiac arrest. However, although oxygen is necessary for life, it also is a potential toxin. Oxygen is most dangerous when it forms free radicals (molecules with highly reactive unpaired electrons). Free radicals are very reactive and can generate cascades of biochemical reactions damaging key cellular proteins, lipids, and DNA. During ischemia and reperfusion, there is increased production of free radicals and a depletion of endogenous antioxidants.[55,56] Furthermore, mitochondria, which are responsible for "handling" oxygen and electrons to generate energy, are severely impaired. Accordingly, some investigators have speculated that delivery of 100% oxygen during resuscitation can contribute to post-ischemic cellular injury.[57,58] There is a great deal of interest in this topic among neonatologists, and trials comparing resuscitation with room air vs. 100% oxygen in asphyxiated newborns recently have appeared in the literature.[59-61] Neonatologists participating in the international guidelines consensus process reviewed the literature and concluded that there currently is "insufficient evidence as to whether use of room air is superior to use of 100% oxygen in the resuscitation of newborns."[62]

In the end, too little or too much oxygen can be injurious, and getting it "just right" is problematic. If oxygen delivery is aggressively weaned to provide systemic normoxia (as measured by pulse-oximetry or blood gas analysis), isolated vascular beds with continued post-ischemic hypoperfusion will be exposed to additional hypoxia. Furthermore, if hemodynamic compromise recurs, the low oxygen reserve will facilitate rapid development of hypoxia. Yet delivery of "luxuriant" oxygen can contribute to the generation of reactive oxygen species and cause widespread cellular damage. Although the recommendation to resuscitate with 100% oxygen is unchanged, it is prudent for clinicians to recognize that there is a theoretic potential for harm, and prolonged delivery of unnecessarily high concentrations of oxygen should be avoided.

The 1997 Pediatric Advanced Life Support text recommended hyperventilating patients to a target $PaCO_2$ of 22-29 mmHg following resuscitation from trauma to correct respiratory acidosis and decrease "excessive cerebral blood flow." Cerebral blood vessels constrict in response to hypocarbia (cerebrovascular reactivity) and the reduction in blood flow decreases the volume and pressure of intracranial contents. Cerebrovascular reactivity remains intact in infants and young children following traumatic brain injury, and hyperventilation remains an important temporizing measure for children with elevated intracranial pressure and signs of impending herniation.[63] However, the decreased blood flow accompanying hyperventilation may come at a price. Specifically, cerebral vascular response may be so robust that the resulting vasoconstriction causes secondary ischemia. In a study using xenon blood flow in children with traumatic brain injury, researchers reported cerebral blood flows consistent with ischemia in approximately 30% of studies during normocapnia, and approximately 75% of studies during hyperventilation to a $PaCO_2$ of less than 25 torr.[64] Investigations in children following cardiac arrest have not been reported, but adults resuscitated from cardiac arrest demonstrate intact cerebrovascular reactivity with evidence of hyperventilation-associated ischemia.[65] Although an injured brain has a decreased metabolism that may offset the decrease in blood flow,[66] it seems prudent to avoid decreasing cerebral blood flow to an injured brain. Thus, hyperventilation should be reserved for patients with signs of cerebral herniation syndrome or suspected pulmonary hypertension.

In addition to avoiding purposeful hyperventilation, it is prudent to guard against inadvertent hyperventilation. Caregivers under stressful circumstances unintentionally (but predictably) hyperventilate patients. This was demonstrated convincingly in a study examining end-tidal CO_2 values on intubated patients during intrahospital transport (e.g., from the ICU to radiology).[67] Providers blinded to end-tidal CO_2 readings hand ventilated patients to values less than 20 torr in 23% of measurements. As discussed above, data in brain-injured patients and patients resuscitated from cardiac arrest suggest that this level of hypocarbia may exacerbate ischemic brain injury. Increased use of quantitative continuous CO_2 monitors throughout the health care system would decrease the potential from harm secondary to inadvertent hyperventilation.

Two drugs, amiodarone and vasopressin, have been added to the AHA pediatric algorithms based upon extrapolations from studies performed in adults. In two recent double-blind, randomized studies comparing intravenous amiodarone to placebo and lidocaine, respectively, for adult patients with out-of-hospital, shock-resistant VF/pulseless VT, amiodarone was more likely to result in admission to hospital (44% vs 34%, p = 0.03 in the placebo study and 22.8% vs 12%, p = 0.009 in the lidocaine study).[68,69] Neither study was able to show improved survival to hospital discharge. There are no published controlled trials for any antiarrhythmic treatments of pediatric arrest, and thus, guideline recommendations necessarily were extrapolated from animal data and studies performed in adults. Amiodarone (bolus) now is listed as a therapeutic option in the pediatric algorithms for pulseless arrest. Amiodarone (slow infusion) is an option for VT with a pulse, but should be used with extreme caution because of the risk for profound hypotension.

There is continuing controversy over the dose and choice of vasopressor for pulseless pediatric arrest. Initial enthusiasm for "high-dose" epinephrine (0.2 mg/kg) was replaced by caution after large, prospective, multicenter studies in adults failed to show a benefit.[70-73] Smaller, retrospective pediatric studies also failed to show a benefit from high-dose epinephrine.[74,75] Moreover, there was a concern that high-dose epinephrine could cause a post-arrest hyperadrenergic state characterized by tachycardia, hypertension, and myocardial dysfunction.[76-79] It is possible that a hyperadrenergic state is less of a concern in children because they generally have healthier cardiovascular systems. After deliberation, the AHA continued to support the option of high-dose epinephrine for children, but recognized that the accumulating data merited a weaker recommendation than previously assigned.

Vasopressin has emerged as a potential alternative vasopressor. Vasopressin causes potent peripheral vasoconstriction with relatively less constriction of coronary, renal, and cerebral vascular beds and thus enjoys a theoretic advantage compared to epinephrine. However, clinical data in adults with cardiac arrest is contradictory.[80-82] Regardless, there currently are insufficient data in children to recommend for or against vasopressin in the setting of cardiac arrest.

Two additional advanced life support issues that merit brief comment are: 1) the age limit for intraosseous (IO) use has been expanded; and 2) vagal maneuvers are recognized as a therapeutic option for treatment of SVT. IO access no longer is restricted to young children, but now is recommended for the entire spectrum of pediatric care. This change recognizes that venous access may be difficult to obtain in patients of all ages and that IO access has been a well-accepted historical technique in adults for a variety of circumstances, including resuscitation, blood typing, and fluid administration.[83,84] Vagal maneuvers are acceptable for children who are hemodynamically stable with SVT. They also are acceptable in less stable patients during preparation for cardioversion/drug therapy as long as cardioversion and/or drug therapy are not delayed by attempting vagal maneuvers. Vagal maneuvers seem to have fallen out of favor with the advent of adenosine, but merit reconsideration because they are inexpensive, easy to perform, and can be effective.

Post-Resuscitation Care

Most victims of pediatric cardiopulmonary arrest will not be successfully resuscitated. The difficulty of accepting this reality often results in prolonged attempts at resuscitation. In one study examining children resuscitated at outlying facilities and then transferred to a tertiary care facility, the duration of CPR ranged from 10-206 minutes, with a median of 55 minutes.[6] Thus, it is possible to restart the heart with 55 minutes (or 206 minutes) of CPR, but what are the consequences? With increasing ischemic time, the chance of survival with normal neurologic recovery diminishes rapidly, and at 55 minutes is virtually non-existent. The Guidelines 2000 state, "(R)esuscitative efforts in infants or children with asystole or pulseless electrical activity despite 30 minutes of cardiopulmonary resuscitation and elimination of reversible causes, including profound hypothermia, are unlikely to be effective." Additionally, "if a child fails to respond to at least two doses of epinephrine with the return of spontaneous circulation, the child is unlikely to survive. In the absence of recurring or refractory VF or ventricular tachycardia, history of a toxic drug exposure, or a primary hypothermic insult, resuscitative efforts may be discontinued if there is no return of spontaneous circulation despite advanced life support interventions. In general, this requires no more than 30 minutes."[85] This guideline acknowledges the futility of prolonged resuscitative efforts and empowers clinicians to feel permitted to stop resuscitative efforts. The guideline does not mandate stopping at a determined duration of CPR, but clinicians should recognize that the chance of survival with lifelong severe disabilities correlates with the duration of CPR.

For management of patients who successfully are resuscitated from cardiac arrest, monitoring and manipulation of brain/body temperature is an area of intense scientific and clinical interest. Post-resuscitation temperature management can be broken down into three related questions: 1) Should hyperthermia be treated? 2) Should hypothermia be treated? and, 3) Should hypothermia be purposely induced?

Treatment of hyperthermia appears to be the least contentious of these questions. Consensus that hyperthermia ought to be treated is based upon the observation that fever is associated with worse outcome in adults suffering from ischemic brain injury and extrapolation from ischemic animal experiments where hyperthermia is either induced or suppressed, yielding greater or lesser injury, respectively.[86-90] Additionally, clinicians are familiar with, and comfortable, treating fever.

In contrast, clinicians have not been in the practice of allowing critically ill patients to remain hypothermic. After resuscitation, children typically develop an initial hypothermia followed by delayed hyperthermia.[91] The initial period of hypothermia frequently is "treated" with warming lights and heated blankets. Although this practice appears nurturing, the evidence from animal experiments and clinical trials in adults suggests that it is potentially harmful. The mechanisms by which active rewarming has the potential to cause harm are two-fold: first, hypothermia is neuroprotective; and second, active rewarming may facilitate the development of hyperthermia, which can exacerbate brain injury.[91-95] Accumulating evidence from published animal studies and preliminary reports from human trials suggests that mild to moderate hypothermia is well tolerated and neuroprotective. Thus, the AHA now recommends "hemodynamically stable patients after resuscitation in whom mild hypothermia (34°-37°) spontaneously develops can be allowed to remain hypothermic."[96]

Purposeful induction of hypothermia was the most controversial of the three temperature-related questions during the AHA guideline deliberations. At the time, there was limited data from Austria and Australia suggesting that induced hypothermia following cardiac arrest was both feasible and well tolerated.[97,98] Although these preliminary reports, in addition to laboratory evidence, generated optimism, they were insufficient to generate a formal recommendation in favor of induced hypothermia. However, the Austrian and Australian studies are now completed and recently were published in the *New England Journal of Medicine* with an accompanying editorial by Peter Safar.[99-101] In both studies, neurologic outcome was improved significantly in cooled patients. Thus, a readily available and inexpensive therapy for reducing brain injury following cardiac arrest is likely to become an important part of our clinical armamentarium. Although the clinical trials were performed in adults remaining comatose after resuscitation from VF, it is likely that patients with other etiologies of ischemic brain injury also can benefit from therapeutic hypothermia. Preliminary experiments examining induced hypothermia for newborns with asphyxia have been published, and a large, multi-center trial is ongoing.[102-104]

Hypothermia is not without side effects. Specifically, hypothermia can suppress immune function, depress myocardial function, and cause coagulopathy. These effects are dependent upon the depth and/or duration of hypothermia. Additional work is necessary to identify patients most likely to benefit from induced hypothermia and to develop safe and effective paradigms for hypothermic treatment. Nonetheless, based upon animal studies and clinical trials, hypothermia has great potential as a robust neuroprotective treatment.

Future Directions

Medical advances in every field from biomedical engineering to molecular biology have the potential to impact clinical care of children suffering from cardiac arrest. This section briefly will discuss selected examples of possible future directions of research and discovery.

Recent developments suggest that it may become possible to screen patients for risk of sudden cardiac death. The first clue was provided by a 1998 *New England Journal of Medicine* paper.[105] The authors collected electrocardiograms (ECGs) from 34,442 neonates born between 1976 and 1994. During one-year follow up, they found 24 deaths from SIDS and reported that the infants dying of SIDS had a longer corrected QT interval than did survivors. The conclusion that there is an association between QT interval and SIDS provoked a controversy that was expressed in a series of editorials.[106-113] Authors of the editorials expressed several concerns, including the labeling of children "at risk for SIDS" when there is no proof that identifying infants with long QT intervals can prevent sudden death and there is the potential for harm by identifying "normal" infants with prolonged QT (false positives).

Additional evidence that prolonged QT may be an unrecognized cause of sudden cardiac death in children and young adults was provided in two papers by Ackerman et al. The first paper described a 10-year-old healthy boy who drowned. The patient successfully was defibrillated but had persistent prolongation of the QT interval in-hospital. Further history revealed an extensive family history of syncopal episodes and ECGs from several symptomatic family members demonstrated prolonged QT interval.[114] The second paper described a 19-year-old healthy woman who was found underwater in a health club pool. She was resuscitated pre-hospital, but subsequently died in the hospital. In a fascinating piece of medical detective work, the investigators obtained a sample of myocardium from autopsy material and used molecular genetic techniques to screen for the known mutations associated with the long QT syndrome. After failing to find any of the known mutations, they then began to sequence the patient's DNA within each of the genes already associated with long QT and discovered a novel mutation in an ion channel gene associated with long QT syndrome. This same mutation was then confirmed in the patient's 18-year-old sister, 49-year-old mother, and 81-year-old grandfather.[115] To bring this story full-circle back to the author first reporting an association between SIDS and long QT, one group recently described an infant who nearly died of SIDS and in whom the long QT syndrome was diagnosed and a spontaneous mutation of a sodium channel gene was identified.[116] Additional work is necessary to determine the exact frequency of long QT in association with sudden cardiac death/SIDS and to determine the appropriate role for screening. In the interim, these interesting reports demonstrate the power of modern molecular techniques and forecast the tremendous advances that are expected as a result of the human genome project.

The ultimate target of resuscitation is the brain. Research into the mechanisms of brain injury and repair by scientists interested in stroke, trauma, global ischemia (as occurs with cardiac arrest), and developmental injury have yielded discoveries relevant to pediatric resuscitation. For example, it is now known that injured neurons can die by necrosis (characterized by cell swelling with eventual lysis and release of toxic cellular contents into the surrounding milieu) or apoptosis (characterized by cellular contraction with disassembly into membrane-bound fragments that are recycled into surrounding cells by phagocytosis). Apoptosis is the "friendly" form of cell death utilized during normal development to prune unnecessary neurons without causing inflammation and collateral damage to surrounding tissue. Apoptotic cell death also occurs after mild-moderate hypoxic ischemia (severe ischemia results in necrosis). Apoptosis typically involves a "decision" by the cell to commit suicide through activation of a specific combination of genes (programmed cell death). The pro-death genes are held in check, under healthy conditions, by pro-survival genes. The fate of cells during development and injury depends upon the net effect of a shifting balance of pro-death and pro-survival genes. Although programmed cell death is adaptive during normal neurodevelopment and for removal of irreversibly injured cells, it can be maladaptive if it is unnecessarily triggered in cells with reversible injury. Researchers are hopeful that manipulating the balance of pro-death and pro-survival genes can rescue neurons with reversible hypoxic-ischemic injury.

Apoptosis may be an especially important mechanism in pediatric brain injury. For instance, laboratory studies have demonstrated that equivalent injury paradigms are more likely to cause apoptotic neuronal death in immature vs. mature brains. One theory is that the physiologic developmental process of pruning superfluous neuronal connections maintains the neuronal "death/survival rheostat" closer to the threshold for apoptosis in an immature brain.

In addition to an age-dependent susceptibility to injury, there is also an age-dependent capacity for repair (plasticity). For example, young children undergoing hemispherectomy for treatment of intractable seizures will reallocate functions previously housed in the removed hemisphere to the remaining hemisphere, an ability that is lost with age. In a related fashion, acquisition of language skills peaks in early childhood and deteriorates over time. The impact of global brain ischemia upon functional outcome during "developmentally sensitive" periods of skill acquisition and the role of plasticity in recovery from global ischemia, are poorly understood. A greater understanding of this dynamic has considerable potential benefit for infants and young children. As an example of this, it has been demonstrated that immature animals placed in enriched environments (environments with multiple objects that provided motor, olfactory, tactile, and visual stimulation) after traumatic brain injury have improved recovery of cognitive function.[117,118] Thus, the deliberative enhancement of endogenous repair mechanisms may prove to be beneficial.

Similar to the discovery of genes related to risk for sudden cardiac death, genes have been identified that are associated with risk for neuronal degeneration and impaired recovery from brain injury. One gene, the apolipoprotein E (APOE) gene, is involved in neuronal maintenance and repair. Individuals inheriting the APOE epsilon4 allele have increased risk for early development of Alzheimer's disease and increased risk for early development of dementia after exposure to traumatic brain injury.[119,120] Identifying additional genes associated with worse outcome from traumatic and ischemic brain injury and unlocking their mechanisms may lead to novel treatment strategies.

Another therapeutic strategy targeting the brain is manipulation of cerebral blood flow following resuscitation. Immediately after resuscitation, there is a brief period of hyperemia followed by a prolonged period of small discrete (multifocal) regions receiving very low blood flow. One theory suggests the immediate hyperemia is caused by endogenous/exogenous catecholamines and the subsequent hypoperfusion is caused by a hypercoagulable state promoting "sludging" of blood. Accordingly, immediate interventions to promote the hypertensive "blush" and decrease blood viscosity should be beneficial.[121] Similarly, strategies to combat hypercoagulability might be beneficial. Investigators have begun to explore the role of anticoagulant and thrombolytic therapy following resuscitation from cardiac arrest and have reported favorable preliminary data.[122]

Additional areas of active investigation in resuscitation research include the use of alternative methods of CPR, a few of which were described above. Most of these methods are untested in young children. An exception is the use of ECMO (extracorporeal membrane oxygenation) for resuscitation of children suffering from cardiovascular collapse.[123] Most of the experience comes from a highly selected population (often post-op cardiac patients) cared for in specialized units with extensive experience in ECMO. Thus, it would be unwise to generalize the results to other populations. Even so, ECMO "salvage" has resulted in remarkable recoveries in the units employing this treatment. Additional research is necessary to identify the subgroup of children most likely to benefit from this technology and to explore methods for extending its use outside of the ICU setting. Investigators in Japan have used cardiopulmonary bypass for treatment of pre-hospital arrest in adults. Although bypass was applied following prolonged arrest in patients with an expected dismal outcome, the preliminary data reports remarkable recoveries.[124]

Perhaps the most encouraging example of aggressive contemporary treatment of cardiac arrest comes from the laboratory at the Safar Center for Resuscitation Research in Pittsburgh, where ongoing research is broaching futuristic horizons. The Safar group has used profound hypothermia with a combination of "brain-oriented" treatments to place dogs in a state of "suspended animation" during hemorrhagic cardiac arrest.[125] The clinical correlate would be purposeful induction of suspended animation in patients (or soldiers) with otherwise fatal traumatic injuries until they can be transported to medical facilities capable of delivering definitive intraoperative care. Using this approach, Safar Center researchers have documented normal neurologic recovery in dogs treated with suspended animation for up to two hours of no cerebral blood flow.[126]

Summary

In conclusion:

- The best treatment of cardiac arrest is prevention of cardiac arrest. Preventive strategies include environmental safety measures, supine sleeping in infants, etc. In the future, it may become possible to screen for inherited causes of sudden cardiac death.
- CPR saves lives. CPR is an important public health issue and should be learned by everyone. CPR should be simple—dropping the pulse check for lay providers simplifies CPR without jeopardizing effectiveness. The two-thumb method is the preferred method in infants when two or more health care providers are available. IAC-CPR and ACD-CPR are acceptable alternatives to standard CPR for adult patients.
- The airway is important. BVM is an effective and safe method for pre-hospital airway management in children. Secondary confirmation of ET intubation should be performed routinely. Prolonged delivery of unnecessarily high concentrations of oxygen should be avoided. Hyperventilation should be reserved for selected indications such as impending cerebral herniation or suspected pulmonary hypertension.
- VF is an uncommon, but salvageable, rhythm in children that should be sought and treated aggressively when found. An AED for use in children has received FDA marketing clearance, but clinical experience is lacking. Current "adult" AEDs are approved for use in children older than 8 years or weighing more than 55 pounds.
- Amiodarone is a treatment option for pulseless arrest (bolus) and VT with a pulse (slow administration).
- IO lines can be placed in patients of any age.
- Vagal maneuvers for treatment of SVT are inexpensive, easy to perform, and often effective.
- Performing CPR for more than 30 minutes, except during specific circumstances, is futile.
- Hypothermia is neuroprotective.
- Hyperthermia exacerbates ischemic brain injury.
- Research focusing on the relationships between brain injury, developmentally sensitive periods of skill acquisition, and plasticity has considerable potential benefit for children.
- Aggressive technological support of cardiopulmonary function, manipulation of cerebral blood flow, and manipulation of gene expression are examples of futuristic resuscitation.

References

1. American Heart Association. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. 2000.
2. Fingerhut LA, Cox CS, Warner M. International comparative analysis of injury mortality. Findings from the ICE on injury statistics. International Collaborative Effort on Injury Statistics. *Adv Data* 1998;303:1-20.
3. Kuisma M, Suominen P, Korpela R. Paediatric out-of-hospital cardiac arrests—epidemiology and outcome. *Resuscitation* 1995; 30:141-150.
4. Sirbaugh PE, Pepe PE, Shook JE, et al. A prospective, population-based study of the demographics, epidemiology, management, and outcome of out-of-hospital pediatric cardiopulmonary arrest. (Published erratum appears in *Ann Emerg Med* 1999;33:358.) *Ann Emerg Med* 1999;33:174-184.
5. Young KD, Seidel JS. Pediatric cardiopulmonary resuscitation: A collective review. *Ann Emerg Med* 1999;33:195-205.
6. Hickey RW, Cohen DM, Strausbaugh S, et al. Pediatric patients requiring CPR in the prehospital setting. *Ann Emerg Med* 1995;25:495-501.
7. Appleton GO, Cummins RO, Larson MP, et al. CPR and the single rescuer: At what age should you "call first" rather than "call fast?" *Ann Emerg Med* 1995; 25:492-494.
8. Mogayzel C, Quan L, Graves JR, et al. Out-of-hospital ventricular fibrillation in children and adolescents: Causes and outcomes. *Ann Emerg Med* 1995;25: 484-491.
9. Friesen RM, Duncan P, Tweed WA. Appraisal of pediatric cardiopulmonary resuscitation. *Can Med Assoc J* 1982;16:1055.
10. Zaritsky A, Nadkarni V, Getson P. CPR in children. *Ann Emerg Med* 1987; 16:1107.
11. Rosenberg ML, Rodriguez JG, Chorba TL. Childhood injuries: Where we are. *Pediatrics* 1990;86:1084-1091.
12. Danseco ER, Miller TR, Spicer RS. Incidence and costs of 1987-1994 childhood injuries: Demographic breakdowns. *Pediatrics* 2000;105:E27.
13. U.S. Department of Transportation NHTSA. Occupant protection traffic safety facts, 1997. U.S. Department of Transportation, National Highway Traffic Safety Administration:1997.
14. Thompson DC, Rivara FP, Thompson RS. Effectiveness of bicycle safety helmets in preventing head injuries. A case-control study. *JAMA* 1996;276: 1968-1973.
15. Thompson RS, Rivara FP, Thompson DC. A case-control study of the effectiveness of bicycle safety helmets. *N Engl J Med* 1989;320:1361-1367.
16. Pitt WR, Balanda KP. Childhood drowning and near-drowning in Brisbane: The contribution of domestic pools. *Med J Aust* 1991;154:661-665.
17. Pitt W, Balanda K. Toddler drownings in domestic swimming pools in Queens-

land since uniform fencing requirements. *Med J Aust* 1998;169:557-558.
18. Runyan CW, Bangdiwala SI, Linzer MA, et al. Risk factors for fatal residential fires. *N Engl J Med* 1992;327:859-863.
19. Spiers PS, Guntheroth WG. Recommendations to avoid the prone sleeping position and recent statistics for sudden infant death syndrome in the United States. *Arch Pediatr Adolesc Med* 1994;148:141-146.
20. Sudden Infant Death Syndrome—United States, 1983-1994. *MMWR Morb Mortal Wkly Rep* 1996;45:859-863.
21. Matthews T. Infant Mortality Statistics for the 1999 Period Linked Birth/Infant Death Data Set. *National Vital Statistics Reports* 2002;50:20-21.
22. Willinger M, Hoffman HJ, Hartford RB. Infant sleep position and risk for sudden infant death syndrome: Report of meeting held January 13-14, 1994, National Institutes of Health, Bethesda, MD. *Pediatrics* 1994;93:814-819.
23. Altemeier IW. Crib death and managed care. *Pediatr Ann 1995;*7:345-383.
24. Swor RA, Jackson RE, Cynar M, et al. Bystander CPR, ventricular fibrillation, and survival in witnessed, unmonitored, out-of-hospital cardiac arrest. *Ann Emerg Med* 1995;25:780-784.
25. Guzy PM, Pearce ML, Greenfield S. The survival benefit of bystander cardiopulmonary resuscitation in a paramedic served metropolitan area. *Am J Public Health* 1983;73:766-769.
26. Roth R, Stewart RD, Rogers K, et al. Out-of-hospital cardiac arrest: Factors associated with survival. *Ann Emerg Med* 1984;13:237-243.
27. Cummins RO, Eisenberg MS. Prehospital cardiopulmonary resuscitation: Is it effective? *JAMA* 1985;253:2408.
28. Ritter G, Wolfe RA, Goldstein S, et al. The effect of bystander CPR on survival of out-of-hospital cardiac arrest victims. *Am Heart J* 1985;110:932-937.
29. Kyriacou DN, Arcinue EL, Peek C, et al. Effect of immediate resuscitation on children with submersion injury. *Pediatrics* 1994;94:137.
30. Eberle B, Dick WF, Schneider T, et al. Checking the carotid pulse check: Diagnostic accuracy of first responders in patients with and without a pulse. *Resuscitation* 1996;33:107-116.
31. Berg RA, Cobb LA, Doherty A, et al. Chest compressions and basic life support-defibrillation. *Ann Emerg Med* 2001;37:S26-S35.
32. Lurie KG, Lindo C, Chin J. CPR: The P stands for plumber's helper. *JAMA* 1990;264:1661.
33. Kern KB, Morley PT, Babbs CF, et al. Use of adjunctive devices in cardiopulmonary resuscitation. *Ann Emerg Med* 2001;37:S68-S77.
34. Halperin HR, Weisfeldt ML. New approaches to CPR: Four hands, a plunger, or a vest. *JAMA* 1992;267:2940.
35. Plaisance P, Adnet F, Vicaut E, et al. Benefit of active compression-decompression cardiopulmonary resuscitation as a prehospital advanced cardiac life support. A randomized multicenter study. *Circulation* 1997;95:955-961.
36. Plaisance P, Lurie KG, Vicaut E, et al. A comparison of standard cardiopulmonary resuscitation and active compression-decompression resuscitation for out-of-hospital cardiac arrest. French Active Compression-Decompression Cardiopulmonary Resuscitation Study Group. *N Engl J Med* 1999;341:569-575.
37. Stiell IG, Hebert PC, Wells GA, et al. The Ontario trial of active compression-decompression cardiopulmonary resuscitation for in-hospital and prehospital cardiac arrest. *JAMA* 1996;275:1417-1423.
38. Berryman CR, Phillips GM. Interposed abdominal compression CPR in human subjects. *Ann Emerg Med* 1984;13:226-229.
39. Babbs CF. The evolution of abdominal compression in cardiopulmonary resuscitation. *Acad Emerg Med* 1994;1:469-477.
40. Sack JB, Kesselbrenner MB, Bregman D. Survival from in-hospital cardiac arrest with interposed abdominal counterpulsation during cardiopulmonary resuscitation. *JAMA* 1992;267:379.
41. Sack JB, Kesselbrenner MB, Jarrad A. Interposed abdominal compression-cardiopulmonary resuscitation and resuscitation outcome during asystole and electromechanical dissociation. *Circulation* 1992;86:1692-1700.
42. Gausche M, Lewis RJ, Stratton SJ, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: A controlled clinical trial. *JAMA* 2000;283:783-790.
43. Glaeser P. Out-of-hospital intubation of children. *JAMA* 2000;283:797-798.
44. Cummins R. From concept to standard-of-care? Review of the clinical experience with automated external defibrillators. *Ann Emerg Med* 1989;18: 1269-1275.
45. Page RL, Joglar JA, Kowal RC, et al. Use of automated external defibrillators by a U.S. airline. *N Engl J Med* 2000;343:1210-1216.
46. Babbs CF, Tacker WA, VanVleet JF, et al. Therapeutic indices for transchest defibrillator shocks: Effective, damaging, and lethal electrical doses. *Am Heart J* 1980;99:734-738.
47. Cecchin F, Jorgenson DB, Berul CI, et al. Is arrhythmia detection by automatic external defibrillator accurate for children? Sensitivity and specificity of an automatic external defibrillator algorithm in 696 pediatric arrhythmias. *Circulation* 2001;103:2483-2488.
48. Aijian P, Tsai A, Knopp R, et al. Endotracheal intubation of pediatric patients by paramedics. *Ann Emerg Med* 1989;18:489-494.
49. Losek JD, Bonadio WA, Walsh-Kelly C, et al. Prehospital pediatric endotracheal intubation performance review. *Pediatr Emerg Care* 1989;5:1-4.
50. Katz SH, Falk JL. Misplaced endotracheal tubes by paramedics in an urban emergency medical services system. *Ann Emerg Med* 2001; 37:32-37.
51. Delbridge TR, Yealy DM. "A" is for airway...also for action. *Ann Emerg Med* 2001;37:62-64.
52. Bhende MS, LaCovey DC. End-tidal carbon dioxide monitoring in the prehospital setting. *Prehosp Emerg Care* 2001;5:208-213.
53. Wee MY, Walker AK. The oesophageal detector device. An assessment with uncuffed tubes in children. *Anaesthesia* 1991;46:869-871.
54. Haynes SR, Morton NS. Use of the oesophageal detector device in children under one year of age. *Anaesthesia* 1990;45:1067-1069.
55. Katz LM, Callaway CW, Kagan VE, et al. Electron spin resonance measure of brain antioxidant activity during ischemia/reperfusion. *Neuroreport* 1998;9: 1587-1593.
56. Ernster L. Biochemistry of reoxygenation injury. *Crit Care Med* 1988;16: 947-953.
57. Lefkowitz W. Oxygen and resuscitation: Beyond the myth. *Pediatrics* 2002; 109:517-519.
58. Thomson AJ, Webb DJ, Maxwell SR, et al. Oxygen therapy in acute medical care. *BMJ* 2002;324:1406-1407.
59. Vento M, Asensi M, Sastre J, et al. Resuscitation with room air instead of 100% oxygen prevents oxidative stress in moderately asphyxiated term neonates. *Pediatrics* 2001;107:642-647.
60. Ramji S, Ahuja S, Thirupuram S, et al. Resuscitation of asphyxic newborn infants with room air of 100% oxygen. *Pediatric Res* 1993;34:809.
61. 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.
62. Niermeyer S, Van Reempts P, Kattwinkel J, et al. Resuscitation of newborns. *Ann Emerg Med* 2001; 37:S110-S125.
63. Adelson PD, Clyde B, Kochanek PM, et al. Cerebrovascular response in infants and young children following severe traumatic brain injury: A preliminary report. *Pediatr Neurosurg* 1997;26:200-207.
64. Skippen P, Seear M, Poskitt K, et al. Effect of hyperventilation on regional cerebral blood flow in head- injured children. *Crit Care Med* 1997;25: 1402-1409.
65. Buunk G, van der Hoeven JG, Meinders AE. Cerebrovascular reactivity in comatose patients resuscitated from a cardiac arrest. *Stroke* 1997;28: 1569-1573.
66. Diringer MN, Videen TO, Yundt K, et al. Regional cerebrovascular and metabolic effects of hyperventilation after severe traumatic brain injury. *J Neurosurg* 2002;96:103-108.
67. Tobias JD, Lynch A, Garrett J. Alterations of end-tidal carbon dioxide during the intrahospital transport of children. *Pediatr Emerg Care* 1996;12:249-251.
68. Dorian P, Cass D, Schwartz B, et al. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. *N Engl J Med* 2002;346:884-890.
69. Kudenchuk PJ, Cobb LA, Copass MK, et al. Amiodarone for resuscitation after out-of-hospital cardiac arrest due to ventricular fibrillation. *N Engl J Med* 1999;341:871-878.
70. Brown CG, Martin DR, Pepe PE. A comparison of standard-dose and high-dose epinephrine in cardiac arrest outside the hospital. *N Engl J Med* 1992;327:1051.
71. Lipman J, Wilson W, Kobilski S, et al. High-dose adrenaline in adult in-hospital asystolic cardiopulmonary resuscitation: A double-blind, randomised trial. *Anaesth Intensive Care* 1993;21:192-196.
72. Lindner KH, Ahnefeld FW, Prengel AW. Comparison of standard and high-dose adrenaline in the resuscitation of asystole and electromechanical dissociation. *Acta Anaesthesiol Scand* 1991;35:253-256.
73. Sherman BW, Munger MA, Foulke GE, et al. High-dose versus standard-dose epinephrine treatment of cardiac arrest after failure of standard therapy. *Pharmacotherapy* 1997;17:242-247.
74. Dieckmann RA, Vardis R. High-dose epinephrine in pediatric out-of-hospital cardiopulmonary arrest. *Pediatrics* 1995;95:901-913.
75. Carpenter TC, Stenmark KR. High-dose epinephrine is not superior to stan-

dard-dose epinephrine in pediatric in-hospital cardiopulmonary arrest. *Pediatrics* 1997;99:403-408.
76. Berg RA, Otto CW, Kern KB, et al. High-dose epinephrine results in greater early mortality after resuscitation from prolonged cardiac arrest in pigs: A prospective, randomized study. *Crit Care Med* 1994;22:282-290.
77. Berg RA, Otto CW, Kern KB, et al. A randomized, blinded trial of high-dose epinephrine versus standard- dose epinephrine in a swine model of pediatric asphyxial cardiac arrest. *Crit Care Med* 1996;24:1695-1700.
78. Rivers EP, Wortsman J, Rady MY, et al. 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.
79. Tang W, Weil MH, Sun S, et al. Epinephrine increases the severity of postresuscitation myocardial dysfunction. *Circulation* 1995;92:3089-3093.
80. Lindner KH, Dirks B, Strohmenger HU, et al. Randomised comparison of epinephrine and vasopressin in patients with out-of-hospital ventricular fibrillation. *Lancet* 1997;349:535-537.
81. Stiell IG, Hebert PC, Wells GA, et al. Vasopressin versus epinephrine for inhospital cardiac arrest: a randomised, controlled trial. *Lancet* 2001;358:105-109.
82. Morley P. Vasopressin or epinephrine: Which initial vasopressor for cardiac arrests? *Lancet* 2001;358:85-86.
83. Iserson KV. Intraosseous infusions in adults. *J Emerg Med* 1989;7:587-591.
84. Waisman M, Waisman D. Bone marrow infusion in adults. *J Trauma* 1997;42: 288-293.
85. Atkins DL, Chameides L, Fallat ME, et al. Resuscitation science of pediatrics. *Ann Emerg Med* 2001; 37:S41-S48.
86. Coimbra C, Boris MF, Drake M, et al. 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.
87. Coimbra C, Drake M, Boris-Moller F, et al. Long-lasting neuroprotective effect of postischemic hypothermia and treatment with an antiinflammatory/antipyretic drug. Evidence for chronic encephalopathic processes following ischemia. *Stroke* 1996; 27:1578-1585.
88. Baena RC, Busto R, Dietrich WD, et al. Hyperthermia delayed by 24 hours aggravates neuronal damage in rat hippocampus following global ischemia. *Neurology* 1997;48:768-773.
89. Ginsberg MD, Busto R. Combating hyperthermia in acute stroke: A significant clinical concern. *Stroke* 1998;29:529-534.
90. Hickey RW. Iatrogenic fever worsens neurologic injury in rats resuscitated from asphyxial cardiac arrest. *Crit Care Med.* In press.
91. Hickey RW, Kochanek PM, Ferimer H, et al. Hypothermia and hyperthermia in children after resuscitation from cardiac arrest. *Pediatrics* 2000;106:118-122.
92. Hickey RW, Ferimer H, Alexander HL, et al. Delayed, spontaneous hypothermia reduces neuronal damage after asphyxial cardiac arrest in rats. *Crit Care Med* 2000;28:3511-3516.
93. Takino M, Okada Y. Hyperthermia following cardiopulmonary resuscitation. *Intens Care Med* 1991;17:419-420.
94. Lieberman E, Lang J, Richardson DK, et al. Intrapartum maternal fever and neonatal outcome. *Pediatrics* 2000;105:8-13.
95. Perlman JM. Maternal fever and neonatal depression: Preliminary observations. *Clin Pediatr* 1999;38:287-291.
96. Schleien CL, Osmond MH, Hickey R, et al. Postresuscitation management. *Ann Emerg Med* 2001;37:S182-S195.
97. Holzer M, Behringer W, Schorkhuber W, et al. Mild hypothermia and outcome after CPR. Hypothermia for Cardiac Arrest (HACA) Study Group. *Acta Anaesthesiol Scand* 1997;111:55-58.
98. Bernard SA, Jones BM, Horne MK. Clinical trial of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest. *Ann Emerg Med* 1997; 30:146-153.
99. Bernard SA, Gray TW, Buist MD, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *N Engl J Med* 2002; 346:557-563.
100. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. *N Engl J Med* 2002;346:549-556.
101. Safar PJ, Kochanek PM. Therapeutic hypothermia after cardiac arrest. *N Engl J Med* 2002;346:612-613.
102. Azzopardi D, Robertson NJ, Cowan FM, et al. Pilot study of treatment with whole body hypothermia for neonatal encephalopathy. *Pediatrics* 2000;106: 684-694.
103. Battin MR, Dezoete JA, Gunn TR, et al. Neurodevelopmental outcome of infants treated with head cooling and mild hypothermia after perinatal asphyxia. *Pediatrics* 2001;107:480-484.
104. Gunn AJ, Gluckman PD, Gunn TR. Selective head cooling in newborn infants after perinatal asphyxia: a safety study. *Pediatrics* 1998;102:885-892.
105. Schwartz P, Stramba-Badiale M, Segantini A, et al. Prolongation of the QT interval and sudden infant death syndrome. *N Engl J Med* 1998;338:1709-1714.
106. Lucey JF. Comments on a sudden infant death article in another journal. *Pediatrics* 1999;103:812.
107. Martin RJ, Miller MJ, Redline S. Screening for SIDS: A neonatal perspective. *Pediatrics* 1999;103:812-813.
108. Guntheroth WG, Spiers PS. Prolongation of the QT interval and the sudden infant death syndrome. *Pediatrics* 1999;103:813-814.
109. Hodgman JE, Siassi B. Prolonged QTc as a risk factor for SIDS. *Pediatrics* 1999;103:814-815.
110. Hoffman JI, Lister G. The implications of a relationship between prolonged QT interval and the sudden infant death syndrome. *Pediatrics* 1999;103:815-817.
111. Shannon DC. Method of analyzing QT interval can't support conclusions. *Pediatrics* 1999;103:819.
112. Tonkin SL, Clarkson PM. A view from New Zealand: Comments on the prolonged QT theory of SIDS causation. *Pediatrics* 1999;103:818-819.
113. Southall DP. Examine data in Schwartz article with extreme care. *Pediatrics* 1999;103:819-820.
114. Ackerman MJ, Porter CJ. Identification of a family with inherited long QT syndrome after a pediatric near-drowning. *Pediatrics* 1998;101:306-308.
115. Ackerman MJ, Tester DJ, Porter CJ, et al. Molecular diagnosis of the inherited long-QT syndrome in a woman who died after near-drowning. *N Engl J Med* 1999;341:1121-1125.
116. Schwartz PJ, Priori SG, Dumaine R, et al. A molecular link between the sudden infant death syndrome and the long-QT syndrome. *N Engl J Med* 2000;343: 262-267.
117. Passineau MJ, Green EJ, Dietrich WD. Therapeutic effects of environmental enrichment on cognitive function and tissue integrity following severe traumatic brain injury in rats. *Exp Neurol* 2001;168:373-384.
118. Hamm RJ, Temple MD, O'Dell DM, et al. Exposure to environmental complexity promotes recovery of cognitive function after traumatic brain injury. *J Neurotrauma* 1996;13:41-47.
119. Jordan BD, Relkin NR, Ravdin LD, et al. Apolipoprotein E epsilon4 associated with chronic traumatic brain injury in boxing. *JAMA* 1997;278:136-140.
120. Friedman G, Froom P, Sazbon L, et al. Apolipoprotein E-epsilon4 genotype predicts a poor outcome in survivors of traumatic brain injury. *Neurology* 1999;52:244-248.
121. Leonov Y, Sterz F, Safar P, et al. Hypertension with hemodilution prevents multifocal cerebral hypoperfusion after cardiac arrest in dogs. *Stroke* 1992;23: 45-53.
122. Bottiger BW, Bode C, Kern S, et al. Efficacy and safety of thrombolytic therapy after initially unsuccessful cardiopulmonary resuscitation: A prospective clinical trial. *Lancet* 2001;357:1583-1585.
123. Cochran JB, Tecklenburg FW, Lau YR, et al. Emergency cardiopulmonary

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

a.) Quickly recognize or increase index of suspicion for specific conditions;

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions

bypass for cardiac arrest refractory to pediatric advanced life support. *Pediatr Emerg Care* 1999;15:30-32.
124. Nagao K, Hayashi N, Kanmatsuse K, et al. Cardiopulmonary cerebral resuscitation using emergency cardiopulmonary bypass, coronary reperfusion therapy and mild hypothermia in patients with cardiac arrest outside the hospital. *J Am Coll Cardiol* 2000;36:776-783.
125. Behringer W, Prueckner S, Kentner R, et al. Rapid hypothermic aortic flush can achieve survival without brain damage after 30 minutes cardiac arrest in dogs. *Anesthesiology* 2000;93:1491-1499.
126. Behringer W, Safar P, Nozari A, et al. Intact survival of 120-minute cardiac arrest at 10°C in dogs. *Crit Care Med* 2001;29: A71.

Physician CME Questions

1. Leading causes of childhood injury fatalities include which of the following?
   A. Drowning
   B. Motor vehicle injuries
   C. Suffocation
   D. Fires
   E. All of the above

2. Which of the following is/are true regarding bystander CPR?
   A. The pulse check has been dropped to simplify lay provider CPR.
   B. A barrier to bystander CPR is anxiety over improperly performing CPR.
   C. Bystander CPR can save lives.
   D. Increasing the rate of bystander CPR is an important public health measure.
   E. All of the above

3. Which of the following statements regarding pediatric intubation is *false*?
   A. Secondary confirmation of ET placement should be performed routinely.
   B. Hyperventilation should be performed routinely.
   C. End-tidal CO_2 detection is the most common secondary confirmation method.
   D. BVM is an acceptable prehospital technique for managing the pediatric airway.

4. All of the following statements regarding VF/VT are true *except*:
   A. VF/VT occurs less commonly in children vs. adults, but is not rare.
   B. VF/VT arrest has a higher rate of survival than asystolic arrest.
   C. Approximately 5-15% of children in prehospital arrest have VF/VT.
   D. VF/VT is not a highly salvageable rhythm in children.

5. Pediatric arrest algorithms include which of the following current drug therapy options?
   A. Amiodorone bolus for pulseless arrest
   B. High dose epinephrine (0.2mg/kg) for pulseless arrest
   C. Amiodorone slow infusion for stable VT
   D. Standard dose epinephrine (0.01mg/kg) for pulseless arrest
   E. All of the above

6. All of the following statements are true *except*:
   A. IO lines should be attempted only in children younger than 6 years of age.
   B. Vagal maneuvers are an acceptable first-line treatment option for children with hemodynamically stable SVT.
   C. The two-thumb method of infant CPR is preferred over the two-finger method when two or more health care providers are present.
   D. The one hand, two-finger method of infant CPR is the method taught to lay providers.

7. Which of the following statements is/are true regarding pediatric cardiopulmonary resuscitation?
   A. As ischemic time increases, the chance for intact neurologic survival decreases.
   B. After 30 minutes of pulseless arrest, further resuscitative efforts are unlikely to be to be effective.
   C. Most family members would like to be present during the resuscitation.
   D. Allowing family presence during resuscitation requires multidisciplinary planning and efforts.
   E. All of the above

8. After resuscitation:
   A. Children typically develop an initial hypothermia followed by delayed hyperthermia.
   B. Children with hyperthermia should be treated with temperature reduction measures.
   C. Children with mild hypothermia (34-37°C) who are hemodynamically stable can be allowed to remain hypothermic without active rewarming.
   D. All of the above

9. Regarding temperature control post-resuscitation, fever is associated with worse outcomes.
   A. True
   B. False

10. Prevention strategies include which of the following?
    A. "Back to sleep" parental education to reduce the incidence of SIDS
    B. Protective environmental measures, such as pool fences and fire alarms
    C. Protective equipment, such as seat belts and bicycle helmets
    D. Continuation of research to screen for inherited causes of sudden death
    E. All of the above

In Future Issues: **Bronchiolitis**