Pediatric Out-of-Hospital Cardiac Arrest
Alexis A. Topjian, MD, MSCE; Robert A. Berg, MD

Case Presentation 1: A 9-year-old boy suddenly collapsed during a basketball game. Emergency medical services was called, but bystander cardiopulmonary resuscitation (CPR) was not provided. Emergency medical services personnel arrived 14 minutes later. After 30 minutes of CPR, successful defibrillation, and several doses of epinephrine, he had return of spontaneous circulation. On arrival in the pediatric intensive care unit, left ventricular ejection fraction was 30% by echocardiography and improved to 50% after hemodynamic support with a dobutamine infusion. To minimize further neurological injury, his temperature was maintained at 36°C to 37°C with a cooling blanket and an explicit normothermia protocol. An electroencephalogram (EEG) 2 hours after admission showed a burst-suppression pattern consistent with severe neurological injury, and continuous EEG monitoring revealed nonconvulsive status epilepticus on the following day.

Case Presentation 2: A 2-year-old boy was found at the bottom of a swimming pool by his mother. Because he was blue and lifeless when removed from the pool, his mother started chest compressions and mouth-to-mouth rescue breathing. His sister called 9-1-1, and emergency medical services arrived within 6 minutes. They continued CPR another 1 to 2 minutes until they saw the boy cough and move his arms and feet. When he arrived in the emergency department, he was breathing spontaneously and had return of spontaneous circulation with good pulses and poor perfusion. His initial arterial pH was 6.95 with a pCO2 of 35 mm Hg and lactate of 5.4 mmol/dL. Supportive care was provided, including mechanical ventilation and a dobutamine infusion for postarrest myocardial dysfunction, and neurological status was monitored closely.

Pediatric Cardiac Arrest Epidemiology
More than 5000 children experience a nontraumatic pediatric out-of-hospital cardiac arrest (OHCA) each year in the United States. Critical factors that influence survival include the environment in which the arrest occurs, the child’s preexisting condition, the duration of no flow before resuscitation, the initial ECG rhythm detected, and the quality of the basic and advanced life support interventions. Although most adult OHCA are caused by primary cardiac disease, pediatric OHCA are more than twice as likely to be attributable to noncardiac causes than to primary cardiac disease (Table). Outcomes after OHCA are better among children than adults.

Bystander CPR
The chain of survival from the American Heart Association for pediatric cardiac arrest highlights 5 elements of survival: Prevention, early CPR, call for help, rapid implementation of pediatric advance life support, and aggressive postresuscitation care (Figure 1). Bystander CPR is one of the key elements to increase survival from out-of-hospital cardiac arrest, yet only one third to one half of children are provided with bystander CPR (similar to the rates for adults). Not surprisingly, outcomes are worse without bystander CPR because of the prolonged no-flow period during the typical 6 to 15 minutes before emergency medical services personnel arrive.

What is the role for hands-only bystander CPR in children? For children with sudden collapse cardiac arrests, hands-only bystander CPR is as effective as chest compression plus rescue breathing because (1) the reservoir of oxygen in the lungs is adequate to oxygenate blood perfusing through the lungs during the low-flow state of CPR.
CPR Quality

High-quality CPR is critical for achieving return of spontaneous circulation. Focus should be on the basic life support mantra of, “Push hard, push fast, allow full chest recoil, minimize interruptions, avoid excessive ventilation.” Compression depth should be at least one third of the chest diameter, a minimum of 1.5 inches in infants and a minimum of 2 inches in children. Compression depth is often inadequate, even by pediatric healthcare providers. The goal rate should be >100 compressions per minute. Slower rates are common and are associated with lower rates of return of spontaneous circulation. Incomplete chest recoil (leaning on the chest during the relaxation phase) impedes venous return, thereby decreasing cardiac output and coronary perfusion. Interruptions during CPR result in no flow and are associated with worse outcomes. Finally, attention to ventilation rate is important, because high ventilation rates can increase intrathoracic pressure and decrease venous return.

Pediatric Defibrillation

The optimal energy dose for pediatric defibrillation is not known. The initial manual defibrillator dosing energy for ventricular fibrillation/ventricular tachycardia should be 2 to 4 J/kg. Fixed-dose automated external defibrillators are highly sensitive and specific for the detection of shockable versus non-shockable rhythms in children and adults. Therefore, automated external defibrillators are now recommended for analysis of rhythm and defibrillation for children between 1 and 8 years. Although American Heart Association guidelines note that an automated external defibrillator with a pediatric dose attenuator is preferred to avoid very high defibrillation doses for these 1- to 8-year-old children, defibrillation with an adult automated external defibrillator dose is preferable to no defibrillation at all. For children <1 year of age, manual defibrillation is preferred.

Post–Cardiac Arrest Care

After successful resuscitation from cardiac arrest, the clinician should focus on preserving end-organ function and minimizing secondary injury. Post–cardiac arrest hyperoxemia may worsen survival and neurological outcome. Therefore, American Heart Association 2010 guidelines recommend that children with postarrest arterial oxygen saturations of 100% should have their supplemental oxygen weaned while the saturation is maintained at ≥94%. Cardiovascular management of these patients should focus on continuous assessment and treatment to achieve and maintain adequate cardiac output and end-organ perfusion. Post–cardiac arrest myocardial dysfunction is characterized by left and right ventricular systolic and diastolic dysfunction and can be associated with life-threatening hemodynamic instability. Myocardial stunning occurs soon after resuscitation and can persist for 72 hours. Although post–cardiac arrest myocardial dysfunction is typically reversible, postresuscitation deaths during the first day of hospital admission are often attributed to this phenomenon. Postarrest myocardial dysfunction can be ameliorated successfully with inotropic support. The sepsis-like pathophysiology associated with postarrest myocardial dysfunction may also manifest as vasodilatory shock and can be treated successfully with vasopressor infusions. Although there are no comparative studies

Table. Common Causes of Pediatric Cardiac Arrest

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<th>Cause</th>
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<td>Asphyxia</td>
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<td>SIDS</td>
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<td>Drowning</td>
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<td>Acute respiratory illness</td>
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<td>Central apnea</td>
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<td>Smoke inhalation</td>
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<td>Hanging</td>
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<td>Circulatory shock</td>
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<td>Sepsis</td>
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<td>Trauma</td>
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<td>Congenital heart disease</td>
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<td>Neurological dysfunction</td>
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<tr>
<td>Seizures</td>
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<tr>
<td>Intracranial hypertension</td>
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<td>Toxin ingestion</td>
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SIDS indicates sudden infant death syndrome.

Figure 1. The pediatric chain of survival. Reproduced with permission from Berg et al.
regarding optimal hemodynamic support, clinicians should ensure adequate preload and use appropriate inotropic and vasoactive agents with the goals of maintaining adequate oxygen delivery and avoiding hypotension.\textsuperscript{10,11}

Prevention of secondary neurological injury after cardiac arrest includes (1) maintenance of adequate oxygen delivery to the brain (normoxia and adequate perfusion), (2) avoidance of hypocarbia and hypercarbia, (3) active prevention of hyperthermia (controlled normothermia or controlled hypothermia), (4) clinical and EEG monitoring for seizures, and (5) prompt treatment of seizures. Although therapeutic hypothermia is established for treatment of comatose adults after witnessed out-of-hospital ventricular fibrillation, results from 2 retrospective pediatric studies do not support the efficacy of therapeutic hypothermia after pediatric cardiac arrest.\textsuperscript{12,13} As with adults, most

Figure 2. The pediatric basic life support algorithm. Reproduced with permission from Berg et al.\textsuperscript{7} AED indicates automated external defibrillator; CPR, cardiopulmonary resuscitation; and ALS, advanced life support.
children admitted to a hospital after an out-of-hospital cardiac arrest are not provided with therapeutic hypothermia. The American Heart Association recommends that induced hypothermia may be considered after return of spontaneous circulation from pediatric cardiac arrest. A large, ongoing, multicenter, National Institutes of Health–funded pediatric clinical trial will provide further clarity to this issue.

Seizures after pediatric cardiac arrest are common. In one study, 42% of children treated with hypothermia after cardiac arrest had seizures and 32% had status epilepticus. Two thirds of these patients had nonconvulsive seizures that would have been undetected without continuous EEG. Furthermore, continuous and reactive background patterns on EEG are associated with good outcome, whereas burst-suppression and discontinuous backgrounds are associated with poor outcome. Continuous EEG monitoring can provide useful information to minimize brain injury and enhance prognosis.

**Evaluation of Sudden Death Channelopathies**

Genetic mutations that lead to channelopathies are relatively common among infants and children with OHCAs. The evaluation of these patients and their families for channelopathies is especially important because 25% to 53% of first- and second-degree relatives have these inherited arrhythmogenic diseases. Therefore, when a child or young adult has an unexplained sudden death, a full past medical history and family history should be obtained, and family members should be referred for evaluation regarding potential channelopathies. An autopsy is recommended and, if possible, genetic tissue evaluation.

**Hypertrophic Cardiomyopathy**

Hypertrophic cardiomyopathy occurs among 1 in 500 in the general population. The annual risk of death is 1% for affected patients, and such deaths are often caused by ventricular arrhythmias. Because hypertrophic cardiomyopathy is the most common cause of sudden cardiac death among young athletes and often has no preceding symptoms, evaluation for hypertrophic cardiomyopathy is appropriate for young athletes with sudden cardiac arrests either by echocardiography or postmortem examination.

**Coronary Artery Abnormalities**

Coronary artery abnormalities (generally aberrant coronary arteries with extrinsic obstruction) are the second-leading cause of sudden death in athletes. Up to 17% of sudden deaths among young athletes have been attributed to anomalous coronary arteries. Diagnoses of these anomalies may require postmortem examination by pathologists with special expertise.

**Conclusions**

Pediatric OHCA is less common than adult OHCA but remains a major public health problem. The chain of survival focuses on bystander intervention, high-quality CPR, and aggressive postresuscitation care. Post–cardiac arrest care should focus on maintenance of adequate oxygen delivery, provision of support for post–cardiac arrest myocardial dysfunction, avoidance of secondary neurological injury, and monitoring for and treatment of post–arrest seizures. It is important to determine the cause of arrest because of the possibility of genetic diseases among surviving family members.

**Case Outcomes**

Case No. 1 did not receive any bystander CPR (ie, had a prolonged no-flow period) and progressed to irreversible cessation of neurological function (brain death). Although his parents initially said there was no heart disease in the family, they later remembered that a cousin had collapsed suddenly and died. Case No. 2 was able to localize noxious stimuli within the first several hours in the hospital, was extubated on the second hospital day, and returned to his usual baseline neurobehavioral functioning over the ensuing 4 weeks.

**Disclosures**

Dr Topjian is The Children’s Hospital of Philadelphia site primary investigator for the Therapeutic Hypothermia After Pediatric Cardiac Arrest trial, for which she receives National Institutes of Health funding (grant #U01HL094345). Dr Berg is the past chair of the American Heart Association Emergency Cardiovascular Care Basic Life Support Committee (Chair during formulation of the 2010 guidelines).

**References**


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