In-Hospital Pediatric Cardiac Arrest

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Cardiac arrest is not rare in pediatric patients, occurring in 2% to 6% of children admitted to pediatric intensive care units (PICUs) [1,2]. The estimated frequency of pediatric out-of-hospital arrests is approximately 8 to 20 per 100,000 children per year [3,4]. Therefore, the rate of pediatric in-hospital cardiac arrests is approximately 100-fold higher than pediatric out-of-hospital arrests [5]. Although outcomes from pediatric cardiac arrests once were considered dismal [6,7], more recent data indicate that pediatric cardiopulmonary resuscitation (CPR) and advanced life support are saving many lives (Table 1). As many as two thirds of in-hospital pediatric cardiac arrest patients can be initially and successfully resuscitated [8–12] and more than 25% survive to hospital discharge.

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Epidemiology of pediatric in-hospital cardiac arrest

The true incidence of pediatric pulseless arrest is difficult to estimate, because of inconsistent definitions and difficulty assessing pulselessness in children. The international Utstein guidelines for reporting cardiac arrest and CPR data were developed to provide consistent definitions and encourage standardized data acquisition. According to these guidelines, pulseless cardiac arrest is defined as the cessation of cardiac mechanical activity, determined by the absence of a palpable central pulse, unresponsiveness, and apnea.

Distinguishing severe hypoxic-ischemic shock with poor perfusion from the nonpulsatile state of cardiac arrest can be challenging in patients of any age. A rescuer’s ability to make this determination by pulse check

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Setting</th>
<th>No. of patients</th>
<th>Return of spontaneous circulation</th>
<th>Survival to discharge</th>
<th>Good neurologic survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiagarajan 2007 [69]</td>
<td>In-hospital, ECMO</td>
<td>682</td>
<td>N/A</td>
<td>(38%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Tibballs 2006 [70]</td>
<td>In-hospital</td>
<td>111</td>
<td>(73%)</td>
<td>(36%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Meaney 2006 [8]</td>
<td>All ICU patients &lt;21</td>
<td>464</td>
<td>(50%)</td>
<td>(22%)</td>
<td>(14%)</td>
</tr>
<tr>
<td>Samson 2006 [12]</td>
<td>In-hospital CA (initial VF/VT rhythm)</td>
<td>272 (104)</td>
<td>(70%)</td>
<td>(35%)</td>
<td>(33%)</td>
</tr>
<tr>
<td>Nadkarni 2006 [9]</td>
<td>In-hospital CA</td>
<td>880</td>
<td>459 (52%)</td>
<td>236 (27%)</td>
<td>154 (18%)</td>
</tr>
<tr>
<td>Reis 2002 [11]</td>
<td>In-hospital CA</td>
<td>129</td>
<td>83 (64%)</td>
<td>21 (16%)</td>
<td>19 (15%)</td>
</tr>
<tr>
<td>Suominen 2000 [2]</td>
<td>In-hospital CA</td>
<td>118</td>
<td>74 (63%)</td>
<td>1-Year survival 21 (18%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Parra 2000 [10]</td>
<td>Ped CICU CA</td>
<td>32</td>
<td>24 (63%)</td>
<td>14 (44%)</td>
<td>8 (25%)</td>
</tr>
<tr>
<td>Rhodes 1999 [19]</td>
<td>In-hospital CA</td>
<td>34</td>
<td>23 (4%)</td>
<td>14 (2%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Young 1999 [3]</td>
<td>Meta-analysis in-hospital CA</td>
<td>544</td>
<td>Not reported</td>
<td>129 (24%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Slonim 1997 [1]</td>
<td>In-hospital PICU CA</td>
<td>205</td>
<td>Not reported</td>
<td>28 (14%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Torres 1997 [71]</td>
<td>In-hospital CA</td>
<td>92</td>
<td>Not reported</td>
<td>1-Year survival 9 (10%)</td>
<td>7 (8%)</td>
</tr>
<tr>
<td>Zaritsky 1987 [61]</td>
<td>In-hospital CA</td>
<td>CA 53</td>
<td>Not reported</td>
<td>CA 5 (9%)</td>
<td>Not reported</td>
</tr>
<tr>
<td>Hintz 2005 [72]</td>
<td>In-hospital CA resuscitation by ECMO</td>
<td>232</td>
<td>N/A all needed ECMO</td>
<td>88 (38%)</td>
<td>Not reported</td>
</tr>
</tbody>
</table>

**Abbreviations:** CA, cardiac arrest; N/A, not applicable; Ped CICU, pediatric cardiac intensive care unit.
is neither sensitive nor specific in adults [13,14] and is more problematic in infants and children because of their smaller size and lower normal blood pressure [15–17]. In adults, pulses typically can be palpated until the systolic pressure is less than 50 mm Hg. Because the normal systolic blood pressure in neonates generally is in the 60s, a decrease in blood pressure to “nonpalpable pulse” may occur earlier in the continuum from hypotensive shock to nonpulsatile cardiac arrest. Nevertheless, children who are unresponsive, apneic, and pulseless as a result of severe hypotensive shock or cardiac arrest should be treated with prompt CPR.

Cardiac arrests are reported in 2% to 6% of all children admitted to PICUs [1,2,18] and in approximately 4% to 6% of children admitted to a cardiac PICU after cardiac surgery [10,19]. The most common causes of in-hospital cardiac arrests are respiratory failure (asphyxia) and circulatory shock (ischemia) [2,8,9,11,12,20,21]. Treatment of in-hospital pediatric cardiac arrest with CPR resulted in return of spontaneous circulation (ROSC) in approximately 43% to 64% of patients, and approximately 25% to 33% survived to hospital discharge (see Table 1) [2,8,9,11,12]. Almost three quarters of those who survived to hospital discharge have favorable neurologic outcomes.

More recently published reports of in-hospital pediatric cardiac arrests are derived from the American Heart Association’s multicenter National Registry of Cardiopulmonary Resuscitation (NRCPR) [8,9,12,20]. The NRCPR is a prospective, multicenter observational registry of in-hospital cardiac arrests and resuscitations. The large size, scope, and quality of the NRCPR distinguish this North American data, which characterize the process and outcome of pediatric in-hospital CPR events. Summaries of these important characteristics are presented in Tables 2 and 3. Of NRCPR pediatric cardiac arrests, 95% were witnessed or monitored, 83% were monitored, and only 14% occurred on a general pediatric ward [9].

Outcomes after pediatric in-hospital cardiac arrests are superior to those after adult in-hospital cardiac arrests. Even though pediatric arrests less commonly are arrhythmogenic arrests resulting from ventricular tachycardia (VT)/ventricular fibrillation (VF) (14% of pediatric arrests versus 23% of adult arrests), 27% of the children survived to hospital discharge compared with 18% of the adults (odds ratio [OR] 2.29; 95% CI, 1.95–2.68) [9]. The superior overall pediatric survival rate reflected a substantially superior survival rate among children who had asystole or pulseless electrical activity (PEA) compared with adults who had asystole or PEA (24% versus 11%). Further investigations have shown that the superior survival rate among children predominantly is the result of better survival among infants and preschool children compared with older children or adults [8]. Perhaps the superior outcomes in the younger children in part are related to CPR resulting in better myocardial and cerebral blood flows in small children, who have compliant chest walls. In addition, survival from pediatric in-hospital CPR is more likely in hospitals staffed with pediatric physicians [20].
Etiologic and pathophysiologic categories of cardiac arrest

Cardiac arrests can result from a multitude of pathophysiologic processes. The three most common causes and pathophysiologic categories of cardiac arrests are asphyxial arrests, ischemic arrests, and arrhythmogenic arrests. Asphyxial cardiac arrests are precipitated by acute hypoxia or hypercarbia. Ischemic arrests are precipitated by inadequate myocardial blood flow. For children, ischemic cardiac arrests most commonly are the result of systemic circulatory shock from hypovolemia, sepsis, or myocardial dysfunction (cardiogenic shock). Although coronary artery problems, such as aberrant left coronary artery, can lead to myocardial ischemia in children, they are less common causes of pediatric ischemic cardiac arrests than

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Characteristics of pediatric in-hospital cardiac arrests (N = 880)</th>
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<tbody>
<tr>
<td>Age, year</td>
<td>Mean (SD) 5.6 (6.4)  Median (range) 1.8 (0–17.0)</td>
</tr>
<tr>
<td>Gender</td>
<td>Male 473 (54)  Female 407 (46)</td>
</tr>
<tr>
<td>Race/ethnicity (%)</td>
<td>White 447 (51)  Black 226 (26)  Hispanic 105 (12)  Other/unknown 102 (12)</td>
</tr>
<tr>
<td>Patient type (%)</td>
<td>In-patient 750 (85)  Emergency department 121 (14)  Other 9 (1)</td>
</tr>
<tr>
<td>Illness category</td>
<td>Medical, noncardiac 402 (46)  Medical, cardiac 158 (18)  Surgical, cardiac 150 (17)  Trauma 91 (10)  Surgical, noncardiac 62 (7)  Other 17 (2)</td>
</tr>
</tbody>
</table>
Pediatric ventricular fibrillation and ventricular tachycardia

Although the rhythms during most in-hospital cardiac arrests (in children and adults) are asystole and PEA, in many arrests the rhythms are VF or pulseless VT [9]. Recent studies indicate that VF and VT (shockable rhythms) occur in 27% of in-hospital cardiac arrests at some time during the arrest and resuscitation [12]. Among pediatric cardiac ICU patients, as many as 41% of the arrests were associated with VF or VT [19]. Among
the first 1005 pediatric in-hospital cardiac arrests in the American Heart Association’s NRCPR, 10% had an initial rhythm of VF/VT, an additional 15% had subsequent VF/VT (ie, some time later during the resuscitation effort), and another 2% had VF/VT but the timing of the arrhythmia was not clear [12].

Arrhythmogenic (“electrical”) cardiac arrests resulting from VF or pulseless VT occur most commonly in the setting of pre-existing heart disease, especially in the postoperative setting. Arrhythmogenic arrests can result from a multitude of congenital cardiac abnormalities and channelopathies associated with prolonged QT syndrome, familial cardiomyopathies (eg, hypertrophic, dilated, or arrhythmogenic right ventricle dysplasia), and mitochondrial diseases. In addition, VF and VT occur in the setting of acquired cardiomyopathies from drugs, toxins (eg, doxorubin cardiomyopathy), infections/myocarditis, electrolyte disturbances (eg, hyperkalemia), and commotio cordis or other mechanically induced VF.

In contrast to arrhythmogenic arrests, VF or VT can occur during resuscitation as subsequent VF/VT. For example, piglet animal models of asphyxial arrests demonstrate that VF often occurs during resuscitation even though the arrest initially was not arrhythmogenic and the first rhythm almost always is asystole or PEA. Asphyxia-associated VF also is well documented among pediatric drowning patients [22].

Traditionally, VF and VT have been considered “good” cardiac arrest rhythms, resulting in better outcomes than asystole and PEA. NRCPR data established, however, that survival to discharge was more common among children who had initial VF/VT than among children who had subsequent VF/VT (35% versus 11%; OR 2.6, 95% CI, 1.2–5.8) [12]. Surprisingly, the subsequent VF/VT group had worse outcomes than children who had asystole/PEA who never developed VF/VT during the resuscitation: 11% of children who had subsequent VF/VT (initial asystole/PEA followed by VF/VT during resuscitation) survived to hospital discharge versus 27% who had asystole/PEA alone. These data suggest that outcomes after initial VF/VT (an arrhythmogenic arrest) are “good,” but outcomes after subsequent VF/VT (ie, VF/VT in the setting of an asphyxial or ischemic arrest) are substantially worse when compared with asystole/PEA rhythms.

Why was the outcome so poor in the subsequent VF/VT group? We do not yet know. Plausible explanations include (1) a delay in the diagnosis of subsequent VF/VT during the resuscitative effort, (2) adverse effects of resuscitative interventions (eg, too much epinephrine), or (3) subsequent VF/VT is an epiphenomenon (eg, a marker of severe underlying myocardial pathology).

Defibrillation (defined as termination of VF) is necessary for successful resuscitation from VF cardiac arrest. The goal of defibrillation is return of an organized electrical rhythm with a palpable pulse. When prompt defibrillation is provided soon after the induction of VF in a cardiac
catheterization laboratory, the rates of successful defibrillation and survival approach 100%. When automated external defibrillators are used within 3 minutes of adult-witnessed VF, long-term survival can occur in more than 70% [23,24]. In general, mortality increases by 5% to 10% per minute of delay to defibrillation [25]. Early and effective chest compressions with minimal interruptions can maintain adequate coronary perfusion and slow the incremental increase in mortality with delayed defibrillation. Provision of high-quality CPR can improve outcome and save lives. Because pediatric cardiac arrests are commonly the result of progressive asphyxia or shock, the initial treatment of choice is prompt CPR, not defibrillation. Therefore, rhythm recognition has been de-emphasized compared with adult cardiac arrests. This historical emphasis must be counterbalanced by evidence that VF is not rare, outcomes from arrhythmogenic VF arrests are superior to other types of cardiac arrests, and early rhythm diagnosis is necessary for optimal care.

The four phases of cardiac arrest

Cardiac arrest may be categorized into four phases, each with unique physiology and treatment strategies: (1) prearrest, (2) no flow (untreated cardiac arrest), (3) low flow (CPR), and (4) post resuscitation.

The prearrest phase

The prearrest phase is the period before the arrest. The BRESUS study in the United Kingdom and the NRCPR data demonstrate that most in-hospital cardiac arrests are asphyxial or ischemic rather than sudden arrhythmia [9,26,27]. Many of these arrests could have been prevented by early recognition and treatment of respiratory failure and shock. This information has fueled interest in the development of medical emergency teams (or rapid response teams) to recognize and treat respiratory failure and circulatory shock before progression to cardiac arrest [28]. These issues were appreciated by the founders of the pediatric advanced life support course, designed to prevent cardiac arrests by early recognition and treatment of respiratory failure and shock in children [7]. In the prearrest phase, hospitalized children at high risk for a cardiac arrest should be in a monitored unit, where prompt diagnosis and treatment is available for respiratory failure, circulatory shock, and life-threatening arrhythmias.

The no-flow phase (untreated cardiac arrest)

Interventions during the no-flow phase of untreated pulseless cardiac arrest focus on early recognition of cardiac arrest and initiation of basic and advanced life support. To assure prompt diagnosis of cardiac arrest, children must be in a monitored unit with immediate health care provider availability. According to NRCPR data, 83% of pediatric in-hospital
arrests were witnessed and the children were on monitors [9]. It is becoming increasingly clear that any in-hospital pediatric cardiac arrest that does not occur in a monitored unit should be evaluated as a potentially avoidable death.

**The low-flow phase (cardiopulmonary resuscitation)**

The goal of effective CPR is to optimize coronary and cerebral perfusion pressure and blood flow to critical organs during the low flow phase. Excellent quality basic life support with continuous effective chest compressions (ie, push hard, push fast, allow full chest recoil, and minimize interruptions) is the emphasis in this low-flow phase. During this phase, the only source of coronary and cerebral perfusion comes from the blood pressure generated by good chest compressions. Providing adequate coronary and cerebral perfusion pressure is critical for successful resuscitation. Any interruption, to perform procedures, analyze rhythms, check for pulses, or change rescuer position for ventilation, is potentially harmful [29]. For VF and pulseless VT, rapid determination of electrocardiographic rhythm and prompt defibrillation when appropriate are important. For cardiac arrests resulting from asphyxia or ischemia, provision of adequate myocardial perfusion and myocardial oxygen delivery with ventilation to match blood flow is important.

Despite evidence-based guidelines, extensive provider training, and provider credentialing in resuscitation medicine, the quality of CPR typically is poor. Slow compression rates, inadequate depth of compression, and substantial pauses are the norm [30–32]. Moreover, observed ventilation rates during professional rescuer CPR often are too high, potentially leading to deleterious effects on venous return and outcome [32,33]. The mantra must be, “Push hard, push fast, minimize interruptions, allow full chest recoil, and do not over-ventilate.” This approach can markedly improve myocardial, cerebral, and systemic perfusion and likely will improve outcomes [34].

Although animal studies indicate that epinephrine can improve initial resuscitation success after asphyxial and VF cardiac arrests, no single medication is shown to improve survival to hospital discharge outcome from pediatric cardiac arrests. Medications commonly used for CPR in children are vasopressors (epinephrine or vasopressin), calcium chloride, sodium bicarbonate, and antiarrhythmics (amiodarone or lidocaine). During CPR, epinephrine’s \( \alpha \)-adrenergic effect increases systemic vascular resistance, increasing diastolic blood pressure, which in turn increases coronary perfusion pressure and blood flow and the likelihood of ROSC. Epinephrine also increases cerebral blood flow during CPR because peripheral vasoconstriction directs a greater proportion of flow to the cerebral circulation. The \( \beta \)-adrenergic effect increases myocardial contractility and heart rate and relaxes smooth muscle in the skeletal muscle vascular bed and bronchi.
although this effect is of less importance. Epinephrine also changes the character of VF (ie, higher amplitude, more “coarse”), increasing the likelihood of successful defibrillation.

High-dose epinephrine (0.05–0.2 mg/kg) improves myocardial and cerebral blood flow during CPR more than standard-dose epinephrine (0.01–0.02 mg/kg) and may increase the incidence of initial ROSC [35,36]. Prospective and retrospective studies indicate, however, that use of high-dose epinephrine in adults or children does not improve survival and may be associated with a worse neurologic outcome [37,38]. A randomized, blinded, controlled trial of rescue high-dose epinephrine after failed initial standard-dose epinephrine versus standard dose epinephrine for pediatric in-hospital cardiac arrest demonstrated a worse 24-hour survival in the high-dose epinephrine group (1/27 versus 6/23, \( P < .05 \)) [39]. High-dose epinephrine cannot be recommended for routine use during CPR.

The postresuscitation phase

The post–cardiac arrest syndrome is a unique and complex combination of pathophysiologic processes that occurs after successful resuscitation. This post–cardiac arrest syndrome includes (1) post–cardiac arrest brain injury, (2) post–cardiac arrest myocardial dysfunction, (3) systemic ischemia/reperfusion response, and (4) the unresolved pathologic process that caused the cardiac arrest.

Clinical manifestations of post–cardiac arrest brain injury include coma, seizures, myoclonus, varying degrees of neurocognitive dysfunction (ranging from memory deficits to persistent vegetative state), and brain death. Mild induced hypothermia is the most well-established postresuscitation therapy for adult post–cardiac arrest brain injury. Two seminal articles established that induced hypothermia (32°C–34°C) could improve outcome for comatose adults after resuscitation from VF cardiac arrest [40,41]. In both randomized controlled trials, the inclusion criteria were patients older than 18 years who were persistently comatose after successful resuscitation from nontraumatic VF. Interpretation and extrapolation of these studies to children is difficult. Fever after cardiac arrest, brain trauma, stroke, and other ischemic conditions is associated with poor neurologic outcome. Hyperthermia after cardiac arrest is common in children [42]. It is reasonable to believe that mild induced systemic hypothermia may benefit children resuscitated from nontraumatic cardiac arrest. Benefit from this treatment, however, has not been rigorously studied and reported in children or in any patients who have had non-VF arrests. Emerging neonatal trials of selective brain cooling and systemic cooling show promise for this therapy in neonatal hypoxic-ischemic encephalopathy, suggesting that induced hypothermia may improve outcomes [43].

Post–cardiac arrest myocardial dysfunction and hypotensive shock are common among human survivors of cardiac arrest. For example, Laurent
and colleagues [44] reported that 90 of 165 consecutive patients admitted to an ICU after successful resuscitation after an out-of-hospital cardiac arrest needed vasoactive infusions for hypotensive shock. Other studies have similarly demonstrated that LV dysfunction and hypotension are common among adult and pediatric survivors after cardiac arrests and generally are reversible among long-term survivors [40,41,45–49]. Postarrest myocardial dysfunction seems pathophysiologically similar to sepsis-related myocardial dysfunction, including increases in inflammatory mediators and nitric oxide production [44,46,47,50]. Although the optimal management of post–cardiac arrest hypotension and myocardial dysfunction are not defined, data suggest that aggressive hemodynamic support may improve outcomes. Controlled trials in animal models show that dobutamine, milrinone, or levosimendan can effectively ameliorate post–cardiac arrest myocardial dysfunction [51–55]. In clinical observational studies, fluid resuscitation has been provided for patients who have hypotension and concomitant low central venous pressure, and various vasoactive infusions, including epinephrine, dobutamine, and dopamine, have been provided for the myocardial dysfunction [40,41,45–49].

How should patients be managed in the postarrest setting? An organized multidisciplinary postresuscitation protocol that includes hemodynamic support, induced hypothermia, and percutaneous coronary intervention where indicated seems to improve outcomes in adults [49]. Post–cardiac arrest myocardial dysfunction and hemodynamic instability are common and should be anticipated. Therefore, continuous electrocardiographic and hemodynamic monitoring should be provided for all patients after successful resuscitation from a cardiac arrest. Furthermore, postarrest echocardiography should be considered for monitoring myocardial function. General critical care principles suggest that appropriate therapeutic goals are adequate blood pressure and adequate myocardial, cerebral, and systemic blood flows and oxygen delivery. The definition of “adequate” is elusive, however. Reasonable interventions for vasodilatory shock with low central venous pressure include fluid resuscitation and vasoactive infusions. Appropriate considerations for left ventricular myocardial dysfunction include euvolemia, inotropic infusions, and afterload reduction. The available evidence for many of these recommendations is taken from the adult critical care literature; the entire field of postresuscitation care, in adult and pediatric patients, is ripe for further study.

**Extracorporeal membrane oxygenation cardiopulmonary resuscitation**

The use of venoarterial extracorporeal membrane oxygenation (ECMO) to re-establish circulation and provide controlled reperfusion after cardiac arrest has been published, but prospective, controlled studies are lacking. Nevertheless, these series have reported extraordinary results with the use of ECMO as a rescue therapy for pediatric cardiac arrests, especially from
potentially reversible acute postoperative myocardial dysfunction or arrhythmia [10,56–59]. In one study, 11 children who suffered cardiac arrest in the PICU after cardiac surgery were placed on ECMO during CPR after 20 to 110 minutes of CPR. Prolonged CPR was continued until ECMO cannulae, circuits, and personnel were available. Six of these 11 children were long-term survivors who had no apparent neurologic sequelae. Most remarkably, Morris and coworkers reported 66 children who were placed on ECMO during CPR over 7 years [59]. In this single institution study, the median duration of CPR before establishment of ECMO was 50 minutes, and 35% (23/66) of these children survived to hospital discharge. These children had brief periods of “no flow,” excellent CPR during the “low flow” period, and a well-controlled postresuscitation phase. CPR and ECMO are not curative treatments. They simply are cardiopulmonary supportive measures that may allow tissue perfusion and viability until recovery from the precipitating disease process.

**When should cardiopulmonary resuscitation be discontinued?**

Several factors determine the likelihood of survival after cardiac arrest, including the mechanism of the arrest (eg, traumatic or asphyxial), location (eg, out-of-hospital versus in-hospital or ward versus PICU), response (eg, monitored versus unmonitored or witnessed versus unwitnessed), and underlying pathophysiology (eg, cardiomyopathy, congenital defect, single ventricle physiology, drug toxicity, or metabolic derangement). Additionally, discontinuation of resuscitation in the prehospital setting is further complicated as the environment may not be conducive to making such an important decision. A study of emergency medical system providers found most to be uncomfortable discontinuing CPR of pediatric patients in cardiac arrest before transport to the hospital [60]. These factors all should be considered before deciding to terminate resuscitative efforts. Continuation of CPR has been considered futile beyond 15 to 20 minutes of CPR or when more than two doses of epinephrine are needed [61–63]. Monitoring children in the prearrest phase, prompt CPR to minimize the no-flow phase, excellent CPR quality during the low-flow phase, and excellent critical care management in the postresuscitation phase seem to have improved outcomes from cardiac arrests. Survival with good outcomes is increasingly common despite greater than 15 minutes of CPR and more than two doses of epinephrine [9–11,56–59,64–68]. The age-old question, “When should we stop?” does not seem to have an easy answer.

**Summary**

In many ways this is the Golden Age of CPR research. Through the work of several CPR pioneers, most of whom still are actively involved in CPR
research, a greater understanding has been gained in many areas. In the past, the concept of evidence-based pediatric cardiac arrest recommendations seemed fanciful. Recommendations were based on extrapolated animal and adult data. These approaches no longer are acceptable. The past 2 decades have brought advances in understanding the pathophysiology of cardiac arrest and VF, better treatment strategies (eg, emphasis on chest compressions before defibrillation for prolonged out-of-hospital cardiac arrests), and a more robust standard for CPR research reporting with the development and acceptance of the Utstein criteria. Moreover, rigorous methodology is improving outcomes at the intersection of CPR science and practical application with the advent of public access defibrillation and a greater emphasis on teaching techniques and CPR performance. Evolving understanding of the pathophysiology of events and titration of the interventions to the timing, etiology, duration, and intensity of the cardiac arrest event can improve resuscitation outcomes. Interventions increasingly are tailored to the phase of cardiac arrest and the etiopathophysiology (ie, arrhythmogenic versus asphyxial versus ischemic arrests). Outcomes from pediatric cardiac arrest and CPR seem to be improving from less than 10% survival in the 1970s to 27% survival in the NRCPR data in the twenty-first century.

Exciting discoveries in basic and applied science are on the immediate horizon for study in specific populations of cardiac arrest victims. By strategically focusing therapies to specific phases of cardiac arrest and resuscitation and to evolving pathophysiology, there is great promise that critical care interventions will lead the way to more successful CPR and cerebral resuscitation in children. Treatment of sudden death in children in the future requires more evidence-based and less anecdotal interventions. Emerging technology interfaced with evolving teams and systems of postresuscitative care likely will facilitate high quality interventions and ensure optimal odds for survival.

New epidemiologic initiatives, such as the NRCPR for in-hospital cardiac arrests and the large-scale, multicenter National Heart, Lung, and Blood Institute Resuscitation Outcome Consortium for out-of-hospital arrests, are providing new data to guide resuscitation practices and generate hypotheses for new approaches to improve outcomes. Excellent basic life support often is not provided. Innovative technical advances, such as directive and corrective real-time feedback, can increase the likelihood of effective basic life support. In addition, team dynamic training and debriefing can improve self-efficacy and operational performance substantially. Mechanical interventions, such as ECMO or other cardiopulmonary bypass systems, already are used in many centers during prolonged in-hospital pediatric cardiac arrests.

Clinical trials are necessary for appropriate evidence-based recommendations for the treatment of pediatric cardiac arrest. The first successful randomized, controlled, blinded trial of any pediatric cardiac arrest intervention was
completed and published in the twenty-first century [39]. Evidence-based pediatric cardiac arrest therapeutics will be based on randomized controlled trails of important interventions. It is likely that the evolution of systems, such as cardiac arrest centers, similar to trauma, stroke, and myocardial infarction centers, will help direct children who require specialized postresuscitation care to centers best able to provide this.

References


