PEDIATRIC CARDIOPULMONARY RESUSCITATION

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Summary

Outcomes following pediatric out-of-hospital arrests are much worse than in-hospital arrests. Survival to hospital discharge typically occurs in less than 10 % of these children, and many have severe neurological sequelae. These poor outcomes are in part because of prolonged periods of 'no flow' and in part because of specific diseases (e.g., traumatic cardiac arrest and sudden infant death syndrome [SIDS]). The major role of CPR is to supply vital organs and tissues with blood flow, oxygen and nutrients. Coronary perfusion pressure below 15 mmHg during CPR is a poor prognostic factor for a return of spontaneous circulation. The most critical elements are to "push hard" and "push fast". Because there is no flow without chest compressions, it is important to minimize interruptions in chest compressions. The best ratio depends upon many factors including the compression rate, the tidal volume, the blood flow generated by compressions, and the time that compressions are interrupted to perform ventilations. A chest compression to ventilation ratio of 15:2 delivered the same minute ventilation as CPR with a chest compression to ventilation ratio of 5:1 in a manikin model of pediatric CPR, but the number of chest compressions delivered was 48 % higher with the 15:2 ratio. Intraosseous vascular access has largely replaced the need for endotracheal drug administration. Absorption of drugs into the circulation after endotracheal administration depends on dispersion over the respiratory mucosa, pulmonary blood flow, and the matching of the ventilation (drug dispersal) to perfusion. Myocardial dysfunction and vascular instability are common folowing resuscitation from cardiac arrest. Therefore in infants and children with cardiovascular dysfunction after cardiac arrest it is reasonable to administer vasoactive drugs (epinephrine, dopamine, dobutamine and norepinephrine) titrated to improve myocardial function and organ perfusion. VF is an uncommon, but not rare, EKG rhythm during out-of-hospital pediatric cardiac arrests. The incidence of VF varies by setting and age. In special circumstances, such as tricyclic antidepressant overdose, cardiomyopathy, post-cardiac surgery, and prolonged QT syndromes, VF is a more likely rhythm during cardiac arrest. Commotio cordis, or mechanically-initiated VF due to relatively low-energy chest wall impact during a narrow window of repolarization (10-30 msec before the T wave peak in swine models) is reported predominantly in children 4-16 years old. 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 pulse. Provision of high quality CPR can improve outcome and save lives. Because pediatric cardiac arrests are commonly due to progressive asphyxia and/or shock, the initial treatment of choice is prompt CPR. The earlier that VF can be diagnosed, the more successfully we can treat it. ECMO is commonly used for circulatory support in pediatric cardiac surgical patients with refractory low cardiac output, persistent hypoxemia, arrhythmias, cardiac arrest, or failure to wean from CPB. CPR guidelines recommend target values for selected CPR parameters related to rate and depth of chest compressions and ventilations, avoidance of CPR-free intervals, and complete release of sternal pressure between compressions. An approach to "Push Hard, Push Fast, Minimize Interruptions, Allow Full Chest Recoil and Don't Over-ventilate" can markedly improve myocardial, cerebral, and systemic perfusion, and will likely improve outcomes. Outcomes from pediatric cardiac arrest and CPR appear to be improving. The evolution of practice to understand the pathophysiology and timing, intensity, duration, and variability of the hypoxic-ischemic insult leads to goal directed therapy gated to the phase of cardiac arrest encountered.

Key words: pediatric cardiopulmonary resuscitation, defibrillation

Rezumat. Resuscitarea cardiopulmonară pediatrică

Rezultatele resuscitării în prespital la copii sunt mult mai modeste în comparaţie cu cele intraspitaliceşti. Supravieţuirea la externarea din spital a acestor copii constituie mai puţin de 10% şi mulţi dintre ei rămân cu sechele neurologice severe. Aceste rezultate nesatisfăcătoare sunt cauzate de alungirea perioadei de „no flow" şi parţial, inclusiv şi de patologia de bază (stopul cardiac traumatic, moartea subită cardiacă a sugarului). Rolul major al resuscitării cardiopulmonare este de a asigura ţesuturile şi organele vitale cu oxigen şi substanţe nutritive prin menţinerea perfuziei sanguine adecvate. Presiunea de perfuzie coronariană sub 15 mm Hg în timpul resuscitării cardiorespiratorii este un indicator de prognostic nefavorabil de restabilire a circulaţiei spontane. Accentul se pune pe compresiile toracice de calitate care trebuie să fie frecvente şi puternice. Din cauza lipsei debitului sanguin în lipsa compresiunilor este important de minimalizat pauzele în compresiunile toracice. Succesul resuscitării este în dependenţă de mulţi factori, inclusiv frecvenţa compresiunilor, volumul tidial, presiunea de perfuzie generată de compresiuni şi de pauzele de întrerupere a compresiunilor şi fără circulaţie. Raportul compresiuni-ventilări de 15:2 asigură aceeaşi ventilare într-un minut ca şi în raportul 5:1 de resuscitare cardiorespiratorie, dar în modele experimentale pe animale s-a demonstrat că numărul de compresiuni toracice este mai înalt cu 48% în raportul de 15:2. Accesul intraosos este prioritar şi a înlocuit calea de administrare intratraheală. Absorbţia medicamentelor în circulaţie după administrare endotraheală este în dependenţă de gradientul de dispersie în mucoasă, de debitul sanguin pulmonar şi de gradul de ventilaţie-perfuzie. Disfuncţia miocardică este frecventă după resuscitarea cardiorespiratorie. Medicaţia vasoactivă (adrenalina, dobutamina, dopamina şi noradrenalina) poate ameliora statusul hemodinamic al copilului, postresuscitare cardiorespiratorie, dar dozele vor fi titrate în funcţie de starea clinică.

Fibrilaţia ventriculară este un ritm mai puţin frecvent întâlnit, dar nu rar în stopul cardiac la copii. Incidenţa fibrilaţiei ventriculare variază în dependenţă de circumstanţe şi vârstă. În supradozările cu antidepresante triciclice, chirurgie cardiacă, cardiomiopatie şi sindromul QT lung fibrilaţia ventriculară ca ritm de instalare a morţii subite la copii este frecvent întâlnit. Comoţia cordului sau iniţierea mecanică a fibrilaţiei ventriculare este raportată mai frecvent în categoriile de vârste 14-16 ani. Defibrilarea (definită ca terminare a fibrilaţiei ventriculare) este vital necesară în stopul cardiac prin fibrilaţie ventriculară şi tahicardie ventriculară fără puls. Asigurarea resuscitării cardiorespiratorii înalt calitative ameliorează rezultatele resuscitării. Stopul cardiac pediatric, de obicei, este cauzat de progresia şocului şi/sau asfixiei, tratamentul iniţial al cauzei este primordial. Din aceste considerente recunoaşterea ritmului şi tratamentul cauzal sunt importante în succesul resuscitării. Suportul respirator extracorporeal: oxigenarea extracorporeală (ECMO) este utilizată în suportul circulator la copii în chirurgia cardiacă cu debit cardiac refractar scăzut, hipoxemie persistentă, aritmii, stop cardiac sau insuficienţă cardiacă secundară bypassului cardiopulmonar. Ghidul resuscitării cardiopulmonare recomandă selectarea parametrilor resuscitării şi asigurarea lor vizând frecvenţa şi adâncimea compresiunilor toracice, frecvenţa şi eficacitatea ventilaţiei. Compresiile puternice şi frecvente, minimalizarea pauzelor, asigurarea revenirii peretelui toracic şi excluderea supraventilării asigură îmbunătăţirea perfuziei miocardice, cerebrale şi sistemice şi ameliorează prognosticul supravieţuirii. Rezultatele resuscitării cardiorespiratorii la copii în stopul cardiac necesită îmbunătăţire. Evoluţia practicilor bazate pe înţelegerea mecanismelor patofiziologice a cauzelor, duratei şi gravităţii, hipoxiei şi/sau ischemiei, mecanismelor stopului cardiac sunt orientate în elaborarea terapiilor ţintite, etapizate şi argumentate pe dovezi.

Cuvinte-cheie: resuscitarea cardiopulmonară pediatrică, defibrilarea

Резюме. Педиатрическая сердечно-легочная реанимация

Результаты догоспитальном реанимации у детей намного более скромны по сравнению с госпитальным уровнем. Выживаемость этих детей до выписки из больницы составляют порой менее 10%, а многие из них остаются с тяжелыми неврологическими осложнениями. Эти плохие результаты обусловлены удлинение периода „no flow" и, в том числе, основной патологией (травматические остановки сердца, внезапная сердечная смерть младенца). Основной ролью сердечно-легочной реанимации является обеспечение тканей и жизненно важных органов кислородом и питательными веществами путем поддержанием адекватного кровоснабжения. Ишемическая перфузионное давление ниже 15 мм рт.ст. в течение кардиореспираторной реанимации указывает на плохой прогноз для восстановления спонтанного кровообращения. Акцент делается на качество компрессий грудной клетки, которые должны быть частыми и сильными. Из-за отсутствия кровотока важно чтобы паузы в компрессии грудной клетки были сведены к минимуму. Успех реанимации зависит от многих факторов, в том числе частоты компрессии, перфузии и давления, создаваемого за счет сжатий грудной клетки, пауз и перерывов в компрессионных движениях. Соотношение сжатия-вентиляции 15:02 обеспечивает тот же объем вентиляции за одну минуту при сердечно-легочной реанимации, как и соотношении 5:01, но экспериментальные модели на животных показали, что эффективность компрессии грудной клетки выше на 48% при соотношении 15: 02. Внутрикостное доступа становится одним из приоритетных, заменяя эндотрахеальные пути введения. Поглощение препарата после эндотрахеальнго введения находится в зависимости от градиента дисперсии слизи, кровотока и степени вентиляции легких. Дисфункция миокарда является общим явлением после проведенной сердечно-легочной реанимации. Вазоактивный лекарства (адреналин, добутамин, допамин и норадреналин) могут улучшить гемодинамический статус ребенка, после сердечной реанимации, но дозы необходимо рассчитывать исходя из клинического состояния. Фибрилляция желудочков встречается реже, но нередко приводит к остановке сердца у детей. Частота случаев фибрилляции желудочков колеблется в зависимости от условий и возраста. При передозировке с трициклическими антидепрессантами в кардиохирургии, при кардиомиопатии и долгосрочной QT синдром желудочковой фибрилляции встречается как причина внезапной смерти у детей достаточно часто. Сотрясение сердца или механическое инициирование фибрилляции желудочков отмечается чаще в возрастных группах 14-16 лет. Дефибрилляции (определяется как прекращение фибрилляции желудочков) имеет жизненно важное значение при остановке сердца при фибрилляции желудочков и желудочковой тахикардии без пульса. Обеспечение высокого качества кардиореспираторной реанимации улучшает исходы реанимации. Остановка сердца у детей обычно происходит при прогрессировании шока и / или асфиксии, поэтому первоначальное устранение и лечение причины имеет первостепенное значение. В связи с этим, распознавание ритма и лечение причины имеет важное значение в успехе реанимации. Экстракорпоральная респираторная поддержка: экстракорпоральная оксигенация используется для вспомогательного кровообращения в сердечной хирургии у детей с низким сердечным выбросом, постоянной гипоксемией, аритмией, остановками сердца или сердечной недостаточностью. Руководства по сердечно-легочной реанимации рекомендует выбор параметров и их обеспечение касательно частоты и глубины компрессии грудной клетки, частоты и эффективности вентиляции. Сильные и частые сжатия, минимизация перерывов и исключение гипервентиляции грудной стенки обеспечивают улучшение перфузии миокарда, центральной нервной системы, улучшая прогноз выживаемости. Актуальные результаты кардио-респираторной реанимации при остановке сердца у детей требуют мероприятий по их совершенствованию. Эволюция практического опыта основана на понимании патофизиологических причин продолжительности и тяжести гипоксии и / или ишемии, механизмов остановки сердца, разработке методов лечения и их научного обоснования.

Ключевые слова: педиатрическая сердечно-легочная реанимация, дефибрилляция

Introduction

Paediatric cardiorespiratory arest is often caused by hypoxia as the body has limited compensatory mechanisms to deal with severe illness or injury. Ventricular fibrillation or pulseless ventricular tachycardia is uncommon in children compared to adults as primary heart disease occurs infrequently. Pronounced hypoxia arising from progressive illness (or the effects of injury) causes myocardial dysfunction, leading to profound bradycardia, which can degenerate to asystole or pulseless electrical activity (PEA). Other vital organs also suffer from severe hypoxia. Both asystole and PEA have poor outcomes.

In this review we will briefly review the current state-of-the-art in pediatric cardiac arrest: Epidemiology, the four phases of cardiac arrest, mechanisms of blood flow during cardiopulmonary resuscitation (CPR), interventions, post-arrest supportive care, special resuscitation circumstances (pediatric ventricular fibrillation [VF], post-congenital heart surgery, extracorporeal membrane oxygenation [ECMO]-CPR), and innovative implementation of training programs.

Epidemiology of Pediatric Cardiac Arrest

Bystander CPR is only provided to approximately 30% of pre-hospital pediatric cardiac arrest victims [1]. Dependent on the setting of pediatric cardiac arrest, initial return of spontaneous circulation occurs in 5-64% of cases, with approximately half of those surviving their arrest event living to hospital discharge and approximately 75% of survivors having a favorable neurologic outcome [2, 3]. Critical factors that influence survival outcomes include the environment in which arrest occurs, the pre-existing condition of the child, the duration of no flow prior to resuscitation, the initial electrocardiograph (EKG) rhythm detected, and the quality of the basic and advanced life support interventions provided (**table 1**).

Long-term survival from pediatric out-of-hospital cardiac arrest is generally reported as < 5%, while survival from arrest in a pediatric ICU is 15-27%. Morbidity and mortality remain high if cardiorespiratory arrest occurs as the profound hypoxia leads to multi-organ failure in many cases. For cardiorespiratory arrests that occur out of hospital, survival is between 6 and 12%, with fewer than 5% having no neurological consequences. In hospital, 27% of cardiac arrest patients survive to discharge and of those having a respiratory arrest where cardiac output is still maintained, more than 70% have good long-term outcomes (**table 2**).

Table 1

Summary of representative studies of outcome following out-of-hospital pediatric cardiac arrest

Author, year	Setting	Number of patients	Return of spontaneous circulation	Survival to discharge	Favorable neurological survival
Osmond, 2006 [6]	Out-of-hospital cardiac arrest, Canada	503	Not reported	10 (2%)	Not reported
Donoghue, 2005 [75]	Out-of-hospitai cardiac arrest systematic review	5693	Not reported	689 (12%)	228 (4%)
Lopez-Herce, 2005 [8]	Mixed in-hospital and out-of-hospital cardiac arrest	213	110 (52%)	45 (21%)	34 (16%)

Table 2

Summary of representative studies of outcome following in-hospital pediatric cardiac arrest

Author, year	Setting	Number of patients	Return of spontaneous circulation	Survival to discharge	Good neurological survival
Meaney, 2006 [16]	All ICU patients < 21	464	50%	(22%)	(14%)
Samson, 2006 [19]	In-hospital cardiac arrest, (initial VF/VT rhythm)	272 (104)	(70 %)	(35%)	(33%)
Nadkarni, 2006 [3]	In-hospital cardiac arrest	880	459 (52 %)	236 (27 %)	154 (18%)
Extracorporeal Life Support Organization, 2005 [15]	In-hospital cardiac arrest resuscitation by ECMO	232	N/A All needed ECMO	88 (38 %)	Not reported
Lopez-Herce, 2005 [8]	Mixed in-hospital and out-of-hospital cardiac arrest	213	110(52%)	45 (21 %)	34(16%)

Pediatric Out-of-hospital Arrests

Outcomes following pediatric out-of-hospital arrests are much worse than in-hospital arrests [1, 2, 4-13]. Survival to hospital discharge typically occurs in less than 10% of these children, and many have severe neurological sequelae. These poor outcomes are in part because of prolonged periods of 'no flow' and in part because of specific diseases (e.g., traumatic cardiac arrest and sudden infant death syndrome [SIDS]). Many pediatric out-of-hospital cardiac arrests are not witnessed, and only 30 % of children are provided with bystander CPR. Therefore, the 'no flow' period is typically quite prolonged before emergency medical service (EMS) personnel provide CPR **(figure 1).**

Mechanism of Blood Flow to Vital Organs during Cardiac Arrest

The major role of CPR is to supply vital organs and tissues with blood flow, oxygen and nutrients. Minimally interrupted flow of oxygen and nutrients is necessary to sustain viability and promote restoration of normal function. A combination of direct cardiac compression and thoracic pump mechanisms appears to be important in blood flow generation during CPR.

Coronary Blood Flow during CPR

During cardiac arrest (asystole or VF) coronary flow ceases. During chest compression, aortic pressure rises at the same time as right atrial pressure. During the decompression phase of chest compression, the right atrial pressure falls faster and lower than the aortic pressure generating a pressure gradient that perfuses the heart with oxygenated blood during 'diastole'. Coronary perfusion pressure below 15 mmHg during CPR is a poor prognostic factor for a return of spontaneous circulation [24].

The importance of negative intrathoracic pressure on coronary perfusion pressure and myocardial blood flow during CPR has been recently discovered. During the decompression phase, negative intrathoracic pressure can be enhanced by briefly impeding air flow to the lungs (e.g., with an inspiratory impedance threshold device), which promotes venous return, cardiac output and mean aortic pressure. The application of this concept has been shown in animal and adult human trials of CPR to improve vital organ perfusion pressures, myocardial blood flow, and survival rates, but has not yet been explored in children [25-27].

Phases of Resuscitation

Interventions to improve outcome from pediatric cardiac arrest should be targeted to optimize therapies according to the etiology, timing, duration, intensity, and 'phase' of resuscitation as suggested in **Table 3**. There are at least four phases of cardiac arrest: 1) pre-arrest; 2) no flow (untreated cardiac arrest); 3) low flow (CPR); and 4) post-resuscitation. The pre-arrest phase represents the greatest opportunity

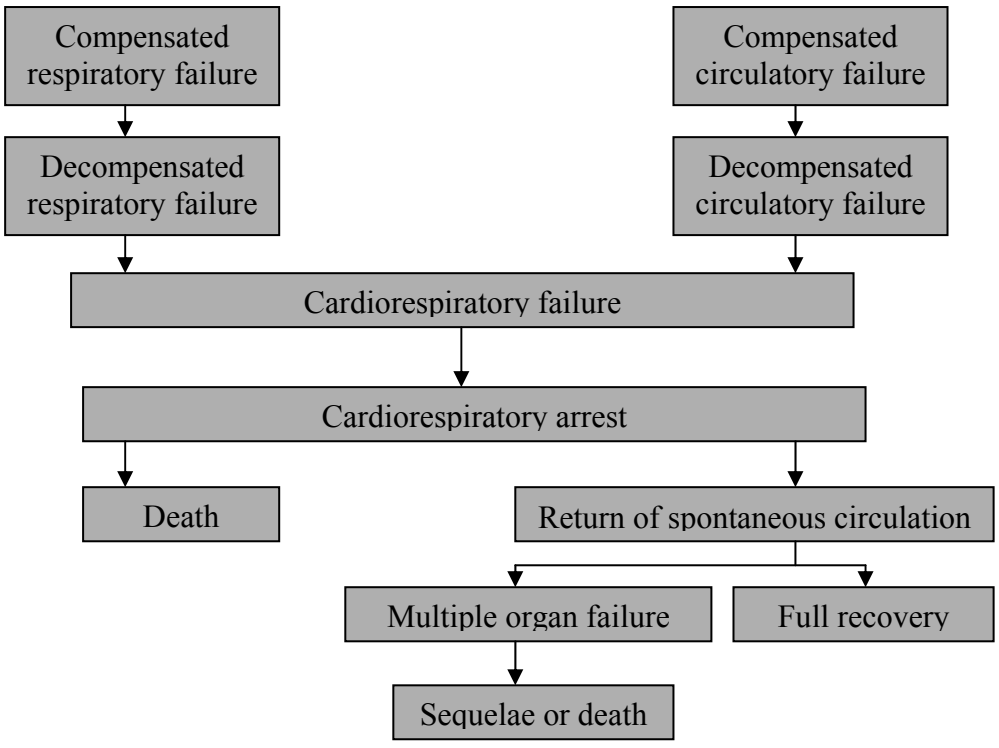

Figure 1. *The sequence of events in the seriously ill/injured child who deteriorates over time*

to impact patient survival by preventing pulseless cardiopulmonary arrest. This phase includes recognition and treatment of pre-existing conditions (e.g., neurological, cardiac, respiratory, infectious, or metabolic problems), developmental status (e.g., premature neonate, mature neonate, infant, child, or adolescent), and precipitating events (e.g., respiratory failure or shock).

Interventions during the pre-arrest phase focus on prevention. Since early recognition, prevention, and anticipation of cardiac arrest is better than treatment, Medical Emergency Teams (Rapid Response Teams) are being trained to recognize and intervene when cardiac arrest is impending [28, 29].

Interventions during the no flow phase of pulseless cardiac arrest focus on early recognition of cardiac arrest and prompt initiation of basic life support. The goal of effective CPR is to optimize coronary and cerebral perfusion and blood flow to critical organs during the low flow phase. Basic life support with near continuous effective chest compressions (e.g., push hard, push fast, allow full chest recoil, minimize interruptions, and don't over ventilate) is the emphasis in this phase. For VF and pulseless ventricular tachycardia (VT), rapid determination of EKG rhythm and prompt defibrillation when appropriate are important. For cardiac arrests due to asphyxia and/or ischemia, provision of adequate myocardial perfusion and myocardial oxygen delivery with ventilation titrated to blood flow is important.

The post-resuscitation phase is a high-risk period for brain injury, ventricular arrhythmias, and extension of reperfusion injuries. Injured cells can hibernate, die, or partially or fully recover function. Myocardial dysfunction and severe hypotension are common during the post-resuscitation phase [30], Interventions, such as systemic hypothermia, during the immediate post-resuscitation phase strive to minimize reperfusion injury and support cellular recovery. The post-arrest phase may have the most potential for innovative advances in the understanding of cell injury and death, inflammation, apoptosis and hibernation, ultimately leading to novel interventions. Thoughtful attention to management of temperature (avoid hyperthermia), glucose (normoglycemia), blood pressure (normotension), coagulation, and optimal ventilation (avoid hyperventilation) may be particularly important in this phase.

The specific phase of cardiac arrest and resuscitation should dictate the timing, intensity, duration and focus of interventions. Emerging data suggest that interventions that can improve short-term outcome during one phase may be deleterious during another. For instance, intense vasoconstriction during the low flow phase of cardiac arrest may improve coronary perfusion pressure and probability

Table 3

Phases of cardiac arrest and resuscitation

Phase	Interventions
Pre-arrest (Protect)	• Optimize community education regarding child safety • Optimize patient monitoring and rapid emergency response • Recognize and treat respiratory failure and /or shock to prevent cardiac arrest
Arrest (no-flow) (Preserve)	• Minimize interval to BLS and ALS (organized response) • Minimize interval to defibrillation, when indicated
Low-flow (CPR) (Resuscitate)	• 'Push Hard', 'Push Fast' • Allow full chest recoil • Minimize interruptions in compressions • Avoid overventilation • Titrate CPR to optimize myocardial blood flow (coronary perfusion pressures and exhaled CO_2) • Consider adjuncts to improve vital organ perfusion during CPR • Consider ECMO if standard CPR/ALS not promptly successful
Post-resuscitation Short-term	• Optimize cardiac output and cerebral perfusion • Treat arrhythmias, if indicated • Avoid hyperglycemia, hyperthermia, hyperventilation • Consider mild post-resuscitation systemic hypothermia • Debrief to improve future responses to emergencies
Post-resuscitation Longer-term rehabilitation (Regenerate)	• Early intervention with occupational and physical therapy • Bioengineering and technology interface • Possible future role for stem cell transplantation

Note: **CPR: cardiopulmonary resuscitation; BLS: basic life support; ALS: advanced life support; ECMO: extracorporeal membrane oxygenation.**

of return of spontaneous circulation. The same intense vasoconstriction during the post-resuscitation phase may increase left ventricular afterload and worsen myocardial strain and dysfunction. Current understanding of the physiology of cardiac arrest and recovery only enables the titration of blood pressure, global oxygen delivery and consumption, body temperature, inflammation, coagulation, and other physiologic parameters to attempt to optimize outcome. Future strategies will likely take advantage of emerging discoveries and knowledge of cellular inflammation, thrombosis, reperfusion, mediator cascades, cellular markers of injury and recovery, and transplantation technology.

Interventions during Cardiac Arrest (No-Flow) and CPR (Low Flow) Phases

Airway and Breathing

During CPR, cardiac output and pulmonary blood flow are approximately 10-25% of that during normal sinus rhythm. Consequently, much less ventilation is necessary for adequate gas exchange from the blood traversing the pulmonary circulation during CPR. Animal and adult data indicate that over-ventilation during CPR is common and can substantially compromise venous return and cardiac output. Most concerning, these adverse hemodynamic effects during CPR combined with the interruptions in chest compressions typically contribute to worse survival outcomes. In animal models of sudden VF cardiac arrest, acceptable PaO_2 and $PaCO_2$, persist for four to eight minutes during chest compressions without rescue breathing. Adequate oxygenation and ventilation can continue without rescue breathing because the lungs serve as a reservoir for oxygen during the low flow state of CPR, and chest compressions alone with an open airway can provide about 33% of normal minute ventilation. Several retrospective studies of witnessed VF cardiac arrest in adults also suggest that outcomes are similar or better after bystander-initiated CPR with either chest compressions alone or chest compressions plus rescue breathing [31]. Animal studies of asphyxia-precipitated cardiac arrests have established that rescue breathing is a critical component of successful CPR [32]. Asphyxia results in significant arterial hypoxemia and acidemia prior to resuscitation in contrast to VF. In this circumstance, rescue breathing can be life-saving (**figure 2**).

Circulation

Basic life support with minimally interrupted effective chest compressions is generally not provided. The most critical elements are to "push hard" and "push fast". Because there is no flow without chest compressions, it is important to minimize interruptions in chest compressions. To allow good venous return in the decompression phase of external cardiac massage, it is important to allow full chest recoil, and to avoid over ventilation. The latter can prevent venous return because of increased intrathoracic pressure.

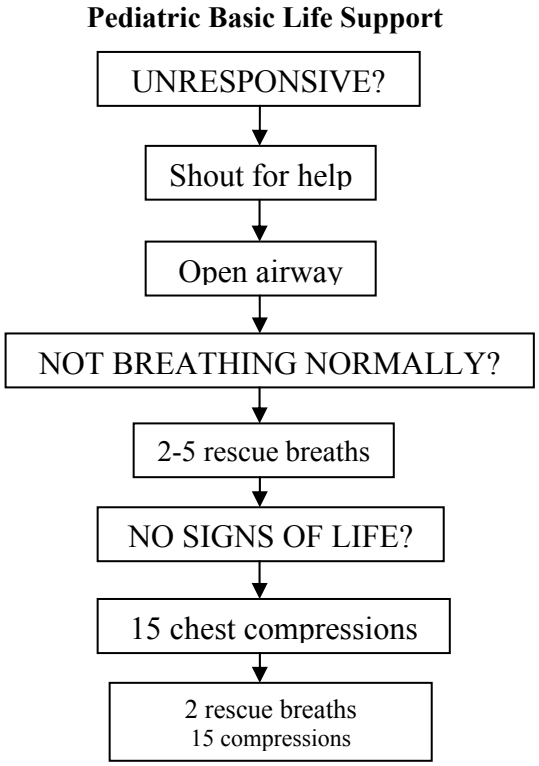

Figure 2. *Pediatric basic life support algorithm*

Open chest CPR

Excellent standard closed chest CPR generates approximately 10-25% of baseline myocardial blood flow and a cerebral blood flow that is approximately 50% of normal. By contrast, open chest CPR can generate a cerebral blood flow that approaches normal. Although open chest massage improves coronary perfusion pressure and increases the chance of successful defibrillation in animals and humans, surgical thoracotomy is impractical in many situations. Open chest CPR is often provided to children after open-heart cardiac surgery and sternotomy. Earlier institution of open chest CPR may warrant reconsideration in selected special resuscitation circumstances such as penetrating trauma and tamponade.

Ratio of Compressions to Ventilation

Ideal compression-ventilation ratios for pediatric patients are unknown. Recent physiologic estimates [33] suggest the amount of ventilation needed during CPR is much less than the amount needed during a normal perfusing rhythm because the cardiac output during CPR is only 10-25% of that during normal sinus

rhythm. The best ratio depends upon many factors including the compression rate, the tidal volume, the blood flow generated by compressions, and the time that compressions are interrupted to perform ventilations. A chest compression to ventilation ratio of 15:2 delivered the same minute ventilation as CPR with a chest compression to ventilation ratio of 5:1 in a manikin model of pediatric CPR, but the number of chest compressions delivered was 48 % higher with the 15:2 ratio [34, 35]. The benefits of positive pressure ventilation (increased arterial content of oxygen and carbon dioxide elimination) must be balanced against the adverse consequence of impeding circulation.

Drug Administration

Intraosseous vascular access has largely replaced the need for endotracheal drug administration. Absorption of drugs into the circulation after endotracheal administration depends on dispersion over the respiratory mucosa, pulmonary blood flow, and the matching of the ventilation (drug dispersal) to perfusion. Although animal studies indicate that epinephrine can improve initial resuscitation success after both asphyxial and VF cardiac arrests, no single medication has been shown to improve survival to hospital discharge outcome from pediatric cardiac arrest. Medications commonly used for CPR in children are vasopressors (epinephrine or vasopressin), calcium chloride, sodium bicarbonate, and anti-arrhythmics (amiodarone or lidocaine). During CPR, epinephrine's ct-adrenergic effect increases systemic vascular resistance, increasing diastolic blood pressure which in turn increases coronary perfusion pressure and blood flow and increases the likelihood of the return of spontaneous circulation. Epinephrine also increases cerebral blood flow during CPR because peripheral vasoconstriction directs a greater proportion of flow to the cerebral circulation (**table 4**).

Table 4

Medications for Pediatric Resuscitation

Medication	Dose	Remarks
Adenosine	0,1 mg/kg (maximum 6 md) Second dose: 0,2 mg/kg (maximum 12 mg)	Monitor ECG Rapid IV/IO bolus with flush
Amiodarone	5 mg/kg IV/IO; may repeat twice up to 15 mg/kg Maximul single dose 300 mg	Monitor ECG and blood pressure; adjust administration rate to urgency (IV push during cardiac arrest, more slowly-over 20-60 minutes with perfusing rhythm). Expert consultation strongly recommended prior to use when patient has a perfusing rhythm. Use caution when administering with other drugs that prolong QT (obtain expert consultation)
Atropine	0,02 mg/kg IV/IO 0,04-0,06 mg/kg ET Repetat once if needed Minimum dose: 0,1 mg Maximul single dose 0,5 mg	Higher doses may be used with organophosphate poisoning
Calcium Chloride (10%)	20 mg/kg IV/IO (0,2 ml/kg) Maximul single dose 2 g	Administer slowly
Epinephrine	0,01 mg/kg (0,1 ml/kg 1:10000) IV/IO 0,1 mg/kg (0,1 ml/kg 1:1000)ET Maximul dose 1 mg IV/IO; 2,5 mg ET	May repeat every 3-5 minutes
Glucose	0,5-1 g/kg IV/IO	Newborn: 5-10 ml/kg $D_{10}W$ Infants and Children: 2-4 ml/kg $D_{25}W$ Adolescents: 1-2 ml/kg $D_{10}W$
Lidocaine	Bolus: 1 mg/kg IV/IO Infusion: 20-50 mcg/kg/minute	
Magnesium Sulfate	25-50 mg/kg IV/IO over 10-20 minutes, faster in torsades de pointes Maximul dose 2 g	
Noloxone	Full Reversal: < 5 y or ≤ 20 kg: 0,1 mg/kg IV/IO/ET ≥5y or > 20 kg: 2 mg IV/IO/ET	Uselower doses to reverse respiratory depression associated with therapeutic opioid use (1-5 mcg/kg titrate to effect)
Procainamide	15 mg/kg IV/IO Adult Dose: 20 mg/min IV infusion to total maximum dose of 17 mg/kg	Monitor ECG and blood pressure Give slowly-over 30-60 minutes. Use caution when administering with other drugs that prolong QT (obtain expert consultation)
Sodium bicarbonate	1 mEq/kg per dose IV/IO slowly	After adequate ventilation

The 3-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 increases the vigor and intensity of VF, 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 return of spontaneous circulation [36, 37]. However, prospective and retrospective studies indicate that use of high-dose epinephrine in adults or children does not improve survival and may be associated with a worse neurological outcome [38, 39]. A randomized, blinded controlled trial of rescue high-dose epinephrine versus standard-dose epinephrine following failed initial standard-dose epinephrine for pediatric in-hospital cardiac arrest demonstrated a worse 24-hour survival in the high-dose epinephrine group (1/27 vs 6/23, $p < 0.05$) [40].

Data suggest that hyperoxemia (i.e. a high PaO_2) enhances the oxidative injury observed following ischemia-reperfusion. Therefore, one goal of the postresuscitation phase is to reduce the risk of oxidative injury while maintaining adequate oxygen delivery.

Specifically, use the lowest inspired oxygen concentration that will maintain the arterial exyhemoglobin saturation ≥ 94%.

Post-resuscitation Interventions

Temperature management

Mild induced hypothermia is the most celebrated goal-directed post-resuscitation therapy for adults. Two seminal articles [41, 42] established that induced hypothermia (32-34°C) could improve outcome for comatose adults after resuscitation from VF cardiac arrest. In both randomized, controlled trials, the inclusion criteria were patients older than 18 years who were persistently comatose after successful resuscitation from non-traumatic VF. Interpretation and extrapolation of these studies to children is difficult. Fever following cardiac arrest, brain trauma, stroke, and other ischemic conditions is associated with poor neurological outcome. Hyperthermia following cardiac arrest is common in children [43]. It is reasonable to believe that mild induced systemic hypothermia may benefit children resuscitated from cardiac arrest. However, benefit from this treatment has not been rigorously studied and reported in children or in any patients with non-VF arrests. Emerging neonatal trials of selective brain cooling and systemic cooling show promise in neonatal hypoxic-ischemic encephalopathy, suggesting that induced hypothermia may improve outcomes [44, 45].

Post-resuscitation Myocardial Support

Post-arrest myocardial stunning occurs commonly after successful resuscitation in animals, adults, and children. Post-arrest myocardial stunning is pathophysiologically similar to sepsis-related myocardial dysfunction and post-cardiopulmonary bypass (CPB) myocardial dysfunction, including increases in inflammatory mediator and nitric oxide (NO) production. Optimal treatment of post-arrest myocardial dysfunction has not been established. The hemodynamic benefits seen in animal studies of post-arrest myocardial dysfunction, in pediatric studies of post-CPB myocardial dysfunction, and in pediatric sepsis-related myocardial dysfunction support the use of inotropic/vasoactive agents in this setting [46-51], although there are no data demonstrating improvements in outcome.

Blood Pressure Management

Laurent and colleagues [30] demonstrated that 55% of adults surviving out-of-hospital cardiac arrests required in-hospital vasoactive infusions for hypotension unresponsive to volume boluses. It is rational to presume that blood pressure variability should be minimized as much as possible following resuscitation from cardiac arrest. A brief period of hypertension following resuscitation from cardiac arrest may diminish the no-reflow phenomenon. In animal models, brief induced hypertension following resuscitation results in improved neurological outcome compared to normotension. In a retrospective human study, post-resuscitative hypertension was associated with a better neurological outcome after controlling for age, gender, duration of cardiac arrest, duration of CPR, and preexisting diseases [39].

Glucose Control

Hyperglycemia following adult cardiac arrest is associated [52] with worse neurological outcome after controlling for duration of arrest and presence of cardiogenic shock. In animal models of asphyxial and ischemic cardiac arrest, administration of insulin and glucose, but not administration of glucose alone, improved neurological outcome compared to administration of normal saline [53]. Data for evidence-based titration of specific endpoints is not available.

Post-resuscitation Outcomes and Quality of Life

Neuropsychological Issues

Information about neurological outcomes and predictors of neurological outcome after both adult and pediatric cardiac arrests is limited. Barriers to assessment of neurological outcomes of children after cardiac arrests include the constantly changing developmental context that occurs with brain

maturation. Prediction or prognosis for future neuropsychological status is a complex task, particularly after an acute neurological insult. There is little information about the predictive value of clinical neurological examinations, neurophysiological diagnostic studies (e.g., electroencephalogram [EEG], or somatosensory evoked potentials), biomarkers, or imaging (computed tomography [CT], magnetic resonance imaging [MRI], or positron emission tomography [PET]) on eventual outcome following cardiac arrest or other global hypoxic-ischemic insults in children. CT scans are not sensitive in detecting early neurological injury. The value of MRI studies following pediatric cardiac arrest is not yet clear. However, MRI with diffusion weighting should provide valuable information about hypoxic/ischemic injury in the subacute and recovery phases. Emerging data suggest that a burst-suppression pattern on post-arrest EEG is sensitive and specific for poor neurological outcome [54]. One study showed that somatosensory evoked potential (SSEP) was highly sensitive and specific in pediatric patients after cardiac arrest [55]. However, SSEP is not standardized in the pediatric population and is difficult to interpret. Many children who suffer a cardiac arrest have substantial pre-existing neurological problems. For example, 17% of the children with in-hospital cardiac arrests from the NRCPR were neurologically abnormal before the arrest [3]. Thus, comparison to pre-arrest neurological function of a child is difficult and adds another dimension/barrier to the assessment and prediction of post-arrest neurological status.

Biomarkers are emerging tools to predict neurological outcome. In an adult study, serum levels of neuron-specific enlace (NSE) and Sloob protein showed prognostic value. NSE > 33 (μg/1 and Sloob > 0.7 μg/1 were highly sensitive and specific for poor neurological outcome (death or persisting unconsciousness) [56]. The validation of these biomarkers in pediatric post-arrest patients requires further study.

Special Resuscitation Circumstances

Pediatric Ventricular Fibrillation

VF is an uncommon, but not rare, EKG rhythm during out-of-hospital pediatric cardiac arrests. Two studies reported VF as the initial rhythm in 19-24% of out-of-hospital pediatric cardiac arrests, but these studies excluded SIDS deaths. In studies that include SIDS victims, the frequency drops to the range of 6-10% [57]. The incidence of VF varies by setting and age. In special circumstances, such as tricyclic antidepressant overdose, cardiomyopathy, post-cardiac surgery, and prolonged QT syndromes, VF is a more likely rhythm during cardiac arrest (**table 5**).

Commotio cordis, or mechanically-initiated VF due to relatively low-energy chest wall impact during a narrow window of repolarization (10-30 msec. before the T wave peak in swine models) is reported predominantly in children 4-16 years old. Out-of-hospital VF cardiac arrest is uncommon in infants, but occurs more frequently in children and adolescents. The variance of VF by age was highlighted in a study documenting VF/VT in only 3 % of children 0-8 years old in cardiac arrest versus 17 % of children 8-19 years old [58]. Although VF is often associated with underlying heart disease and generally considered the 'immediate cause' of cardiac arrest, 'subsequent' VF can also occur during resuscitation efforts. Asphyxia-associated VF is also well documented among pediatric near-drowning patients [59]. Traditionally, VF and VT have been considered 'good' cardiac arrest rhythms, resulting in better outcomes than asystole and pulseless electrical activity. Among the first 1,005 pediatric in-hospital cardiac arrests in the NRCPR [19], 10 % had an initial rhythm of VF/VT; an additional 15 % had subsequent VF/VT (i.e., some time later during the resuscitation efforts). Of note, survival to discharge was much more common among children with an initial shockable rhythm than among children with shockable rhythms occurring later during the resuscitation. These data suggest that outcomes after initial VF/VT are 'good', but outcomes after subsequent VF/VT are substantially worse, even compared to asystole/pulseless electrical activity rhythms.

Termination of ventricular fibrillation: Defibrillation

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 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 mins of adult witnessed VF, long-term survival can occur in more than 70% [60, 61]. In general, mortality increases by 7%-10% per minute of delay to defibrillation. Early and effective, near-continuous chest compressions can attenuate 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 due to progressive asphyxia and/or shock, the initial treatment of choice is prompt CPR. Therefore, rhythm recognition is relatively less emphasized compared with adult cardiac arrests. The earlier that VF can be diagnosed, the more successfully we can treat it.

Table 5

Pediatric emergency treatment chart

		Adrenaline	**Fluid bolus**	**Glucose**	**Sodium bicarbonate**		**Tracheal tube uncuffed**	**Tracheal tube cuffed**	**Defibrilllation**
	Strength dose	1:10000 10 microgram kg^{-1}	0,9% saline 20 ml kg^{-1}	10% 2 ml kg^{-1}	4,2% 8,4%¤ 1 mmol kg^{-1}				4 Joules kg^{-1}
	Route	i.v., i.o.	i.v., i.o.	i.v., i.o.	i.v., i.o., UVC, i.v., i.o.				Transthoracic
	Notes		Consider warmed fluid	For known hypoglycaemia				Monitor cuff pressure	Monophasic or biphasic
Age	Weight (kg)	ml	ml	Recheck glucose after dose (ml)	ml	ml	ID mm	ID mm	Manual
< 1 month	3,5	0,35	70	7	7	-	3,0	-	20
1 month	4	0,4	80	8	8	-	3,0-3,5	3,0	20
3 months	5	0,5	100	10	10	-	3,5	3,0	20
6 months	7	0,7	140	14	-	7	3,5	3,0	30
1 year	10	1,0	200	20	-	10	4,0	3,5	40
2 years	12	1,2	240	24	-	12	4,5	4,0	50
3 years	14	1,4	280	28	-	14	4,5-5,0	4,0-4,5	60
4 years	16	1,6	320	32	-	16	5,0	4,5	60
5 years	18	1,8	360	36	-	18	5,0-5,5	4,5-5,0	70
6 years	20	2,0	400	40	-	20	5,5	5,0	80
7 years	23	2,3	460	46	-	23	5,5-6,0	5,0-5,5	90
8 years	26	2,6	520	52	-	26	-	6,0-6,5	100
10 years	30	3,0	600	60	-	30	-	7,0	120
12 years	38	3,8	760	76	-	38	-	7-7,5	150
Adolescent	> 40 kg	10	1000	80	-	50	-	7-8	As for adults
Cardioversion	Syncronised Shock – 0,5-1,0 Joules/kg^{-1} escalating to 2,0 Joules/kg^{-1} if unsuccessful								
Amiodarone	5 mg/kg^{-1} i.v. or i.o bolus in arrest (0,1 ml/kg^{-1} of 150 mg in 3 ml) after 3rd and 5th shocks. Flush line with 0,9% saline or 5% glucose								
Atropine	20 microgram/kg^{-1}, minimum dose 100 microgram, maximum dose 600 microgram								
Calcium chloride 10%	0,2 ml/kg^{-1} for hypocalcaemia/hyperkalaemia								
Lorazepam	100 microgram/kg^{-1} i.v. or i.o for treatment of seizures. Can be repeated after 10 minutes. Maximum single dose 4 mg								
Nolaxone	Resuscitation dose for full reversal 100 microgram/kg^{-1}. For partial reversal of opiate analgesia 10 microgram/kg^{-1} boluses, titrated to effect								
Anaphylaxis	Adrenaline 1:1000 intramuscularly (< 6 years 150 microgram (0,15 ml), 6-12 years 300 microgram (0,3 ml), > 12 years 500 microgram (0,5 ml)) can be repeated after five minutes. OR titrate boluses of 1 $microgram^{-1}$ i.v. ONLY if familiar with giving i.v. adrenaline								

Wights averaged on lean body mass from 50th centile weights for males and females. Recommendations for tracheak tubes are based on full term neonates

For newborns glucose at 2,5 ml/kg^{-1} is recommended

Post-operative Congenital Heart Disease Considerations

The post-operative cardiac patient may require resuscitation due to a persistent low cardiac output state, but is also likely to experience an acute decompensation such as a respiratory event, an arrhythmia, a feed-associated vagal episode, aortopulmo-nary shunt occlusion, pulmonary hypertensive crisis, or a coronary event. Although the incidence of cardiac arrest is higher in children admitted to a cardiac intensive care unit (ICU) compared to those admitted to a pediatric ICU, the outcome of these patients is better - 44% survival in the cardiac ICU versus 15-27% in the pediatric ICU. Likely explanations for the disparity in survival rates are that the populations' arrest etiologies, arrest interventions, and post-arrest management are inherently different.

Mechanical Circulatory Support

ECMO is commonly used for circulatory support in pediatric cardiac surgical patients with refractory low cardiac output, persistent hypoxemia, arrhythmias, cardiac arrest, or failure to wean from CPB. Some centers report using ECMO to support neonates early after surgical palliation for hypoplastic left heart syndrome to avoid hypoxemia and potential cardiac

arrest during the low cardiac output syndrome [15]. According to the 2004 Extracorporeal Life Support Organization registry, survival to hospital discharge was 41%. Following a stage 1 palliation for hypoplastic left heart syndrome (2002 to 2006, n = 269), 33 patients (13 %) required CPR and of these patients 21 (63%) were stabilized with ECMO. In particular, patients with shunted single-ventricle circulation supported with ECMO show survival to hospital discharge ranging from 39 to 64% [62]. Perhaps the ultimate technology to control post-resuscitation temperature and hemodynamic parameters is ECMO. In addition, the concomitant administration of heparin may optimize microcirculatory flow.

Reports of the use of veno-arterial ECMO to reestablish circulation and provide controlled reperfusion following cardiac arrest have 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 post-operative myocardial dysfunction or arrhythmias [17, 63-67]. In one study [64], 11 children who suffered cardiac arrest in the pediatric ICU after cardiac surgery were placed on ECMO during CPR after 20-110 minutes of CPR. Prolonged CPR was continued until ECMO cannulae, circuits, and personnel were available. Six of these 11 children were long-term survivors without apparent neurological sequelae. Most remarkably, Morris et al. [67] reported 66 children who were placed on ECMO during CPR over 7 years. The median duration of CPR prior to establishment of ECMO was 50 minutes, and 35% (23/66) of these children survived to hospital discharge. It is important to emphasize that these children had brief periods of 'no flow', excellent CPR during the 'low flow' period, and a well-controlled post-resuscitation phase. CPR and ECMO are not curative treatments; they are simply cardiopulmonary supportive measures that may allow tissue perfusion and viability until recovery from the precipitating disease process (**table 6**).

Quality of CPR and Resuscitation Interventions

Despite evidence-based guidelines, extensive provider training, and provider credentialing in resuscitation medicine, the quality of CPR is typically poor. CPR guidelines recommend target values for selected CPR parameters related to rate and depth of chest compressions and ventilations, avoidance of CPR-free intervals, and complete release of sternal pressure between compressions [68]. Slow compression rates, inadequate depth of compression, and substantial pauses are the norm. An approach to "Push Hard, Push Fast, Minimize Interruptions, Allow Full Chest Recoil and Don't Over-ventilate" can markedly improve myocardial, cerebral, and systemic perfusion, and will likely improve outcomes [69]. Recent technology has been developed that monitors quality of CPR and implements a force sensor and accelerometer into a defibrillator monitor to quantitatively provide verbal feedback to the CPR administrator on the frequency and volume of ventilations. Recent studies show that rescuers can use feedback obtained from the defibrillator electrode pads placed on the chest to improve compliance with these guidelines. Quality of post-resuscitative management has also been demonstrated to be critically important to improve resuscitation survival outcomes [70].

Special Issues in Simulation, Advanced Education, and Implementation of Programs

Just-in-time' and 'just-in-place' training concepts were developed based on studies and recent review by experts for resuscitation training. Psychomotor skills and team function are the primary skills necessary during resuscitation; however, it is well recognized that these skills are subject to decay within six weeks after resuscitation training [71]. Just-in-time' and

Table 6

Medications to Maintain Cardiac Output and for Postresuscitation Stabilization

Inamrinone	0,75-1 mg/kg IV/IO over 5 minutes; may repeat x 2 then: 5-10 mcg/kg per minute	Inodilator
Dobutamine	2-20 mcg/kg per minute IV/IO	Inotrope; vasodilator
Dopamine	2-20 mcg/kg per minute IV/IO	Inotrope; chronotrope; renal and splanchnic vasodilator in low doses; pressor in high doses
Epinephrine	0,1-1 mcg/kg per minute IV/IO	Inotrope; chronotrope; vasodilator in low doses; pressor in high doses
Milrinone	Loading dose: 50 mcg/kg IV/IO over 10-60 min then 0,25-0,75 mcg/kg per minute	Inodilator
Norepinephrine	0,1-2 mcg/kg per minute	Vasopressor
Sodium nitroprusside	Initial: 0,5-1 mcg/kg per minute; titrate to effect up to 8 mcg/kg per minute	Vasodilator Prepare only in D_5W

'Just-in-place' refresher training seems reasonable to enhance operational performance and improve patient safety based on the facts that psychomotor skills decay over time. This can incorporate some of the advantages of simulation such as abilities to plan and shape training opportunities, safe environment for both patients and students, unexpected exposure to rare but complicated and important clinical events, and opportunity to repeat performance [72]. DeVita et al. evaluated the efficiency of the code team (crisis management team) training with adult high fidelity simulation manikins [73]. These authors measured the survival of the manikin in a simulated scenario and the task completion rate as outcomes in three simulated training sessions. The team performance showed improvement in overall simulation survival rate and task completion rate from 0% to 90%, and 31% to 89%, respectively. Hunt et al. successfully used simulated trauma stabilization "mock codes" to identify deficiencies in stabilization of children with trauma presenting to the hospital emergency departments [74]. Evaluation-tool inter-rater reliability was excellent, and 57% of the stabilization tasks needed improvement (estimating a child's weight, preparing for intraosseous needle placement, ordering fluid boluses, applying warm measures, and ordering dextrose for hypoglycemia). Such simulations are likely to drive resuscitation implementation in the future.

Future Directions and Potential Obstacles

Exciting new epidemiological studies, such as the NRCPR for in-hospita cardiac arrests and the large-scale, multicenter Resuscitation Outcome Consortium funded by the National Heart, Lung and Blood Institute (NHLBI), are providing new data to guide our resuscitation practices and generate hypotheses for new approaches to improve outcomes. It is increasingly clear that excellent basic life support is often 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 substantially improve self-efficacy and operational performance. Induced hypothermia is a promising neuro-protective and cardio-protective post-arrest intervention. Mechanical interventions, such as ECMO or other CPB systems, are already commonplace interventions during prolonged in-hospital cardiac arrests. Technical advances are likely to further improve our ability to provide such mechanical support.

Clinical trials are necessary for appropriate evidence-based recommendations for treatment of pediatric cardiac arrests. It is likely that the evolution of systems such as cardiac arrest centers, similar to trauma, stroke, and myocardial infarction centers, is likely to facilitate the administration of appropriate intensive care to patients who require specialized post-resuscitation care.

Human factors

Human factors (HF) are the environmental and behavioral elements that influence the way that people or teams of people, interact with one another and the equipment and devices they use.

The content is focused on three specific areas that are directly relevant to those attending and managing collapsed patients on an infrequent basis: how accidents occur, communication issues and the concept of situation awareness.

It is vital to recognize that we all make errors. Sometimes, even when multiple stops and checks have been put into place, things can still go wrong. On occasions, accidents may even occur as a consequence of the procedures put in the place to prevent them.

The key to recognizing, understanding and preventing accidents is to understand that people do not seek to cause them. Bad or catastrophic events usually occur because a series of small errors or adverse circumstances that come together at a particular time and place. This creates a situation where the final error that triggers the accident is either inevitable or occurs through an action which seems entirely reasonable at the time. This concept, known as an error chain, was originally described by James Reason. It is also often referred to as "the Swiss cheese model" and is shown in **figure 3**.

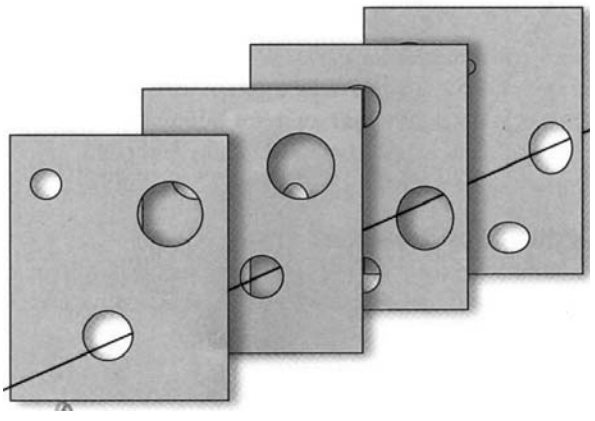

Figure 3. *James Reason's Swiss cheese model of an error chain. (Reproduced from Reason J. Human error: models and management. BMJ. 2000 March 18;320:pp. 768-770, with permission from BMJ Publishing Group Ltd.)*

Each layer of cheese represents a layer of defense such as a protocol, procedure or an environmental condition that serves to prevent an accident occurring. The holes represent the imperfections. When all the

holes line up an accident occurs. Accidents can be prevented by identifying and plugging these holes.

In practice, everyone needs to maintain vigilance for the presence of factors that might be seen as a hole in the cheese.

Situation awareness refers to the mental models that we have in our head of the world around us. We use it to plan our actions and we base our communications with others upon it. It is not difficult to see that if this model is wrong then disaster may ensue.

Obtaining complete and accurate information can be very difficult. At the simplest level our eyes often deceive us. Is the illustration in **figure 4** a cowboy looking away or an old lady?

Figure 4. *What do you see?*

Good teams talk to one another; they don't just assume they are all following the same principles. Key issues must always be shared, ideally in a formal briefing.

In a resuscitation attempt, vocalizing the algorithms as they are actioned is an excellent way of ensuring both concordance of thought and allowing for others to highlight oversights.

Conclusion

Outcomes from pediatric cardiac arrest and CPR appear to be improving. The evolution of practice to understand the pathophysiology and timing, intensity, duration, and variability of the hypoxic-ischemic insult leads to goal directed therapy gated to the phase of cardiac arrest encountered. Exciting discoveries in basic and applied science laboratories are on the immediate horizon for study in specific sub-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 cardio-pulmonary and cerebral resuscitation in children.

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