Chapter 39: Cardiopulmonary Resuscitation

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INTRODUCTION

FOCUS POINTS

1. Highest risk patients for cardiopulmonary resuscitation (CPR) include infants under one year of age, ASA Physical Status Classification ≥3, and children having cardiac surgery.
2. The American Heart Association (AHA) adopted a major change to the sequence of chest compressions and ventilation from Airway-Breathing-Circulation (ABC) to Circulation-Airway-Breathing (CAB).
3. Medication-related intraoperative cardiac arrest is commonly associated with local anesthetic toxicity and anaphylaxis secondary to antibiotic or muscle relaxant administration.
4. The alpha agonist action of epinephrine is probably the most important in increasing coronary blood flow to maintain myocardial blood flow and in providing cerebral blood flow with peripheral vasoconstriction.
5. If available, capnography should be used during CPR with target end-tidal CO_2 ($ETCO_2$) of ≥15 mm Hg.
6. During the postresuscitative phase the patient is at the highest risk for brain injury, ventricular arrhythmias, and reperfusion injury.
7. Avoidance of hyperthermia should be a periresuscitation goal.
8. Extracorporeal Life Support (ECLS) should be considered in reversible causes that include hyperkalemia, local anesthetic toxicity, general anesthetic overdose, and airway emergency.

HISTORY OF CPR AND EPIDEMIOLOGY

Perioperative cardiac arrest is rare and the incidence in children can be difficult to define as the time frame for anesthesia-related cardiac arrest can range from intraoperative up to 30 days postoperatively.[1] There is a discrepancy in whether studies include events during cardiac surgery or noncardiac cases. It is estimated that in-hospital pediatric cardiac arrest involves 5000 to 10,000 children per year in the United States.[2,3] Pediatric perioperative cardiac arrests excluding cardiac surgery have been found to occur in 2.9 to 7.4 per 10,000 procedures.[4,5] The incidence of specific anesthesia-related cardiac arrests when cardiac surgery is included ranges from 0.8 to 4.6 per 10,000 procedures with the highest incidence of 79 to 127 per 10,000 procedures in cardiac surgery.[4,6]

Beyond those having cardiac surgery, the highest risk patients include infants under 1 year of age (greatest in neonate) and those with an ASA physical status of 3 or higher.[4,5,7] The etiology of cardiac arrest can be divided into four broad categories to include medication-related (anesthesia overdose), cardiovascular (hypovolemia), respiratory, and equipment-related causes.[8] Within these categories the most common causes associated with perioperative pediatric arrest include hypovolemia, hyperkalemia, laryngospasm, inhaled induction, central-line complications, venous air embolism, and hypoxia.[9] Cardiovascular (CV) etiologies make up 40% of anesthesia-related cardiac arrests with decreased intravascular volume being the most common culprit. Hypovolemia due to unrecognized ongoing hemorrhage is the most common cause of intraoperative hypovolemia and is worsened

by the lack of vital sign variations (e.g., lack of heart rate increase) and inadequate IV access for appropriate resuscitation.[4,7,9] Following CV causes, respiratory etiologies make up about 31% of cardiac arrest related to anesthesia in children and involve inadequate ventilation, inadequate oxygenation, and the "loss of airway" in the form of laryngospasm, bronchospasm, and airway difficulty.[4,7]

Determining the outcome for pediatric patients after a cardiac arrest varies depending on whether events are defined as "anesthesia-related" versus "perioperative" in nature. "Anesthesia-related" mortality is estimated to be 0.1 to 1.6 per 10,000 procedures, whereas perioperative cardiac arrest (including anesthesia, surgical, and patient disease) mortality is higher at 3.8 to 13.4 per 10,000 procedures.[1,4,8,10] Overall the quality of survival is more favorable after cardiac arrest from an anesthesia-related cause compared to a nonanesthesia cause in that patients have a 62% chance of surviving and survivors have a 92% chance of having a positive neurological outcome after an anesthesia-related cause. Survival rates are only estimated to be 36% with 22% of survivors returning to their neurological status when all perioperative cardiac arrest causes are assessed.[8] It is difficult to pinpoint factors that may lead to improved outcomes after anesthesia-related causes but may include reversible causes of operating room cardiac arrest, continuous hemodynamic and respiratory monitoring during anesthesia, and the advanced preparation paired with the resuscitation skills of the anesthesiologist.[8]

RECOGNIZING ARREST

In hospital pediatric cardiac arrest most commonly occurs in the pediatric ICU due to progression of respiratory failure and/or circulatory shock.[11] One study using a multicenter registry of adverse events in pediatric anesthesia demonstrated the incidence of cardiac arrest in the perioperative period to be 5.3 per 10,000 anesthetic cases.[12] There are four recognized phases of cardiac arrest: prearrest, no flow (untreated cardiac arrest), low flow (CPR), and postresuscitation. The goals of care during each phase are vastly different; however, early recognition of the early phase gives the greatest chance of changing patient outcomes by potentially preventing the subsequent phases. Delay in starting CPR and prolonged periods of no flow are all associated with worse outcomes.[11] Thus, recognizing that a patient has progressive respiratory or circulatory failure is important for preventing progression or time spent in the no-flow phase of cardiac arrest and for prompt management with rapid initiation of basic life support (BLS) or pediatric advanced life support (PALS) once in the no-flow phase. The postresuscitative phase will be discussed later in this chapter.

MECHANICS OF CPR

The goal of CPR is to maintain coronary perfusion pressure, which is the primary factor of myocardial blood flow. Maintaining appropriate diastolic blood pressure (DBP) and thus coronary perfusion pressure is associated with increased likelihood of survival to discharge and favorable neurological outcomes after in-hospital cardiac arrest (IHCA).[13] It is important to recognize that CPR has limitations, providing only about 10% to 30% of normal blood flow to the heart and 30% to 40% of normal blood flow to the brain.[14] Being well-versed in the mechanics of BLS during pediatric CPR and resuscitation is vital to pediatric providers in order to provide optimal support during cardiac arrest.

To minimize the time to first compressions and to better align with the adult BLS recommendations that emphasize chest compressions to ventilations,[15] in 2015, the American Heart Association (AHA) adopted a major change to the sequence of chest compressions and ventilation from Airway-Breathing-Circulation (ABC) to Circulation-Airway-Breathing (CAB).[16,17] Recognizing that asphyxia and other respiratory etiologies are more common in pediatric cardiac arrest, the AHA acknowledges that ventilation may be more important in pediatric resuscitation; however, simulation studies suggest a greater delay in first compressions in the ABC approach than delay in initiation of ventilation in the CAB approach.[17–19] For in-hospital cardiac arrest, the first rescuer should begin immediate chest compressions and call for further assistance, while the second rescuer to arrive should focus on the airway and breathing components.[20]

Depth and Recoil

The AHA guidelines for pediatric cardiac arrest recognize that there is limited evidence supporting the targeted depth for compression in pediatric patients. The consensus recommendations are that pediatric patients require compression of the chest by at least one-third of the anterior-posterior diameter. In infants, this is approximately 1.5 in., while in children this is 2 in. or greater. Of note, overly deep compressions have been associated with potentially worse patient outcomes.[15]

Allowing for complete chest recoil between compressions is a key element often missed in high-quality CPR. Incomplete chest recoil can lead to higher intrathoracic pressures, decreased venous return, decreased coronary perfusion and blood flow, and decreased cerebral perfusion.[17,21]

Focal Versus Circumferential Compressions in Infants

Due to children's size and chest wall compliance, the use of focal versus circumferential compressions is debated. Focal compressions utilize two fingers placed over the infant's sternum to administer compressions, while circumferential compressions are administered by encircling the chest with two hands and placing the rescuer's thumbs to depress the sternum.[17,20] The AHA endorses the use of two fingers or focal technique in pediatric resuscitation; however, several animal and simulation studies have supported the use of the two hands or circumferential technique.[17] The latter method in these studies resulted in higher mean arterial, systolic, and diastolic blood pressures.[22,23] The AHA recommendations suggest either technique can be utilized, as long as adequate depth and recoil can be achieved.[17]

Ratios and Rate

During cardiac arrest, the ratio of compressions to ventilations depends on the presence of an advanced airway and the number of rescuers. This ratio is based on achieving a balance that optimizes oxygen delivery to the vital organs, which requires compressions and minute ventilation. Rescuers must also recognize the positive pressure ventilation (PPV) benefits, but know that PPV can be detrimental if it is used too aggressively due to the impedance to forward flow PPV may cause.[20] At the writing of this text, the most updated AHA recommendations for compression to ventilation ratios come from their 2010 consensus.[24]

Single Rescuer

In a study on out-of-hospital cardiac arrests (OHCA) comparing favorable neurological outcomes for children receiving standard bystander CPR (compressions with rescue breaths) versus compression only, CPR showed that children with noncardiac etiology of their cardiac arrest had more favorable neurological outcomes with standard bystander CPR.[25] Several animal studies also support this finding as well, which is why the AHA continues to recommend a compression to ventilation of 30 compressions to 2 rescue breaths for infants and children (same recommendation for adults).[24]

Two Rescuers; No Advanced Airway in Place

For most IHCAs, there will likely and fortunately be more than one provider available. If there is no advanced airway present, the compression-to-ventilation ratio should be 15 compressions for every 2 breaths, provided by bag-mask ventilation.[24] Efforts should be made to obtain a secure, advanced airway as soon as possible, while minimizing interruptions to chest compressions when in a hospital setting.

Two Rescuers; Advanced Airway in Place

Once an advanced airway has been established, compressions should be performed continuously with no pauses.[20,24] A rapid ventilation rate should be avoided during CPR. A CPR study on a pig model revealed that excessive ventilation led to significantly decreased coronary perfusion pressure, increased intrathoracic pressure, and decreased survival rates.[26,27] The AHA does not provide a suggested rate for ventilation during pediatric cardiac arrest, but merely recommends avoidance of excessive ventilation.[24] A generally accepted rate is 8 to 12 breaths per minute during cardiac arrest. This should be titrated by the provider based on end-tidal CO_2 or signs of increased intrathoracic pressure.

SPECIAL CONSIDERATIONS FOR INTRAOPERATIVE CPR

Surgical procedures require an array of positions including the prone and sitting positions that may expose pediatric patients to a higher risk of both cardiac arrest and delay in CPR. Patients are also exposed to multiple drugs that are necessary for the anesthetic, which might predispose the child to toxicity that may lead to cardiac arrest. Open-chest CPR is a viable option in postoperative cardiac patients or abdominal surgeries during which the chest or abdomen are left open. Studies show that open-chest CPR can achieve near-normal blood flow and increase cardiac output two to three times above conventional closed-chest CPR.[28,29]

Spine surgeries and posterior cranial surgeries may delay conventional supine CPR due to the stage of the surgery requiring surgical stabilization and preparation for repositioning. To prevent delay of life-saving therapies, CPR should be initiated in the prone position. One method to deliver prone compressions involves one hand over each scapula while counterpressure is provided by a second rescuer's hand/fist or sandbag on the sternum. The second method is identical except the compressions are done with the heel of one hand on the spine with the second hand on top, similar to that of conventional sternum compressions and can be done when surgical exposure does not include the midline spine.[30,31] Prone compressions have been shown to achieve an effective depth 75% of the time when compared to supine compressions using a high-fidelity simulation model.[32] The efficacy of prone compressions was noted in a clinical study that showed six adults in an ICU had better hemodynamics when CPR was done in the prone position versus the supine position.[33]

Not only does alternative positioning lead to limitations to CPR, but procedures in which large open blood vessels are above the level of the heart can place the patient at higher risk for a venous air embolus (VAE). Craniofacial, other neurosurgical procedures, and spine surgeries are common culprits for this complication as they also may be associated with hypovolemia that can lower the CVP and worsen entrainment of air.[9] A VAE is typically recognized as an abrupt decrease in blood pressure and $ETCO_2$, which can be further verified by end-tidal nitrogen, transesophageal echocardiography, or audible doppler bubbles.[8] Initial steps should involve lowering the operative site below the heart, flooding the site with fluid, and if possible positioning the patient in the left lateral decubitus position to prevent air entry into the pulmonary artery which could cause further obstruction to left heart flow.

Inhalational induction exposes patients to rapid physiological changes such as vasodilation and a hyperstimulating state that can lead to significant hemodynamic changes and respiratory compromise in an unsecured airway. It is estimated that 25% of perioperative cardiac arrests occur during induction due to hypotension and laryngospasm.[7] Hypotension may be significant when the vasodilation from volatile anesthetics is paired with the relative hypovolemic patient from causes such as NPO requirements, bowel preparations, and shock. Treatment should involve decreasing the inhalational agent and administering fluid or blood with possible temporization with vasoactive medications. If laryngospasm occurs, positive pressure with deepening of the anesthetic should be initiated. Depending on vascular access, a muscle relaxant (such as succinylcholine) should be administered via the IV or IM route. Careful induction and vigilant awareness with appropriate response to cardiovascular and respiratory changes can improve the safety profile during an inhalational induction.

The risk for intraoperative cardiac arrest from hyperkalemia in children is high when rapid infusion of blood occurs and increases proportionately with the age of the blood transfused.[7] Hemodynamic signs of hyperkalemia include peaked T waves, arrhythmias (VT or VF), and widening QRS complexes which should be immediately treated with discontinuation of the blood transfusion, alkalosis (hyperventilation and bicarbonate), and stabilization of the cardiac membrane with calcium. Potassium can be further driven intracellularly with insulin/glucose and beta-agonists.

Medication-related intraoperative cardiac arrest is commonly associated with local anesthetic toxicity and anaphylaxis. Regional anesthesia is typically performed when children are already under general anesthesia, so early signs of toxicity (neurological changes) are not present in prevention of the later effects of conduction abnormalities and cardiovascular collapse. Cardiac arrest caused by local anesthetic toxicity is unique in that hemodynamic support is accomplished with lower doses of epinephrine (less than 1 mcg/kg per bolus) and drugs that could further compromise cardiac function (vasopressin, calcium channel blockers, beta blockers, and lidocaine) should be avoided. The mainstay treatment is lipid emulsion 20% 1.5 mL/kg (maximum dose 12 mL/kg) followed by an infusion 0.25 mL/kg/min. If hemodynamic stability cannot be accomplished medically, extracorporeal life support should be an early consideration.[34] Another medication-associated etiology for sudden cardiovascular collapse is anaphylaxis which is most commonly related to the administration of antibiotics or neuromuscular blockers. Anaphylaxis may be exhibited by tachycardia, bronchospasm, angioedema, and rash. Treatment includes 100% oxygen, epinephrine IV or IM, corticosteroids, antihistamines, and supportive care with albuterol and H1 blockers. CPR should commence as indicated by the AHA guidelines for PALS if anaphylaxis progresses to shock.[8]

VENTRICULAR FIBRILLATION/TACHYCARDIA

Ventricular fibrillation is an uncommon yet fatal arrhythmia. Children with a sudden, witnessed collapse in an out-of-hospital situation should arouse a strong suspicion for ventricular fibrillation (VF) or ventricular tachycardia (VT).[17] In a large, multicenter study, it was found that 27% of pediatric patients who had an IHCA had VF or VT at some point during their arrest. In 10% of the patients, it was the initial pulseless rhythm. Notably, this study found that in patients whose initial rhythm was not VT/VF but subsequently deteriorated into VT/VF during their resuscitation had worse outcomes

than those patients who were in VF/VT as their initial rhythm.[35]

Causes

Underlying cardiac disease is one of the most common causes of IHCA from VF/VT, with up to 40% of in-hospital cases occurring in pediatric cardiac patients.[36] Other common causes include tricyclic antidepressant overdose, hyperkalemia, cardiomyopathy, prolonged QT syndromes, and asphyxiation.[20]

Treatment

A study of witnessed OHCA in adults showed that rapid defibrillation of VF led to a long-term survival of >70% when defibrillation was administered in less than 3 minutes.[37] Although mortality increases by as much as 7% to 10% with each minute of delay of defibrillation, earlier electrical treatment increases the success rate of returning to an organized rhythm.[20] Thus, it is imperative to both recognize and aggressively treat VT/VF when present during a cardiac arrest.

For IHCA, manual defibrillation should be administered by trained healthcare providers. The evidence supporting the dosing of defibrillation is limited in pediatrics and extrapolated from adult data. The initial recommended dose for pediatric VT/VF is 2 J/kg. Subsequent doses should be increased by 2 J/kg to a maximum of 10 J/kg or maximum adult dose.[20] In between delivered shocks, efforts should be made to minimize interruptions of chest compressions, with resumption immediately upon delivery of the shock and analyzed rhythm after 2 minutes of effective CPR.[17]

CARDIOPULMONARY RESUSCITATION MEDICATIONS (TABLE 39-1)

Table 39-1

Resuscitation Drug Guide

Drug	Dose/Route	Indication	Action
Epinephrine (1:10,000 IV/IO) (1:1,000 ETT)	IV/IO (1:10K): 0.01 mg/kg (0.1 mL/kg) (max 1 mg) ETT (1:1K): 0.1 mg/kg IM/SQ (1:1K): 0.01 mg/kg (anaphylaxis)	• Bradycardia • Pulseless arrest • Anaphylaxis	• Alpha-adrenergic • Beta-adrenergic
Vasopressin	IV/IO: 0.4 U/kg (max 40 U)	• Refractory VF	• V1 (vasoconstriction) • V2 (renal water reabsorption)
Atropine	IV/IO/IM: 0.02 mg/kg (max 1 mg) ETT: 0.03–0.05 mg/kg	• Bradycardia • AV node block	• Blocks cholinergic stimulation
Adenosine	IV/IO: 0.1 mg/kg (first dose; max 6 mg) 0.2 mg/kg (second dose; max 12 mg)	• SVT	• Prolongs AV-node refractory period • Slows conduction
Amiodarone	IV/IO: 5 mg/kg (pulseless) over 20–60 minutes (if perfusing rhythm) (max 20 mg/kg/day)	• VF, VT and SVT	• Alpha-adrenergic blockade • Beta-adrenergic blockade • Ca channel blockade • Action potential prolongation
Lidocaine	IV/IO: 1 mg/kg (max 100 mg) ETT: 1.5–2.5 mg/kg	• Ventricular • arrhythmias	• Deceases automaticity of ventricular ectopic foci
Magnesium	IV/IO: 25–50 mg/kg (max 2 g)	• Torsades de pointes • Hypomagnesemia	• In enzymatic reactions as intracellular cation
Calcium	IV/IO: 20 mg/kg ($CaCl_2$) 60 mg/kg (CaGlc) (max 2 g)	• Hypocalcemia • Hyperkalemia • Hypermagnesemia • Calcium channel blocker overdose	• Myocardial excitation-contraction coupling • Myocardial contractility
Sodium Bicarbonate	IV/IO: 1 meq/kg or calculate base deficit	• Metabolic acidosis • Hyperkalemia • Long CPR time	• Buffers excess H^+ thus increasing the pH

Epinephrine

Epinephrine has long been proven to increase diastolic pressure and systemic vascular resistance, thereby becoming the mainstay medication for CPR as it directly improves coronary perfusion which increases the chances for a successful resuscitation.[38] The alpha-agonist action of epinephrine is probably the most important in increasing coronary blood flow to maintain myocardial blood flow and in providing cerebral blood flow with peripheral vasoconstriction.[39] Although the beta-adrenergic stimulation may increase the "vigor" of VF to help in defibrillation, it increases myocardial oxygen demand which can increase the risk of ischemic injury.[40] There is varying evidence about the use of high-dose epinephrine (0.1 mg/kg) versus standard dose epinephrine (0.01 mg/kg). Some studies have found that cerebral and coronary blood flow may be increased, but other studies have noted that this may result in detrimental increase in myocardial oxygen consumption which may lead to postresuscitation adverse effects with no benefits in return of spontaneous circulation (ROSC), neurological outcome, or survival to hospital discharge.[41] A randomized, prospective study by Perondi et al revealed that there was no difference in survival in children given high-dose versus standard-dose epinephrine in witnessed in-hospital cardiac arrest and patients with asphyxia-related cardiac arrest did worse.[42] The situations in which high-dose epinephrine may be helpful are beta-blocker or calcium channel–blocker overdose, severe anaphylaxis, or septic shock.[24] In summary, the 2015 AHA guidelines recommend standard dose epinephrine for serial doses in pulseless cardiac arrest.[18]

Vasopressin

Vasopressin tends to be compared in efficacy to epinephrine in that it provides vasoconstriction (V1 receptors) and causes water reabsorption by binding to renal tubule receptors (V2 receptors) without the adrenergic effects that cause increased myocardial demand. The use of vasopressin in pediatric cardiac arrest is not well studied and in one multivariate analysis from October 1999 to November 2004 it was noted that vasopressin was associated with worse ROSC, but without any difference in discharge survival.[43] The AHA guidelines do not provide recommendations for vasopressin in pediatric patients, but in adults with refractory VF it is recommended to use 40 units of vasopressin. In one retrospective case series of four children undergoing prolonged CPR (> 60 minutes) there was ROSC in three of the four patients following vasopressin (0.4 U/kg) administration, so this may warrant further study about the use of vasopressin in prolonged pediatric cardiac arrest.[44]

Atropine

Atropine is a parasympatholytic drug that increases heart rate by its effects on the sinus node, shortening of AV conduction, and activating latent ectopic pacemakers.[45] It is especially useful for bradycardia as a result of increased parasympathetic tone. Atropine should be considered in second- and third-degree AV blocks, and bradycardia with associated hypotension.[46] In pediatric patients, atropine is occasionally given before intubation to prevent the parasympathetic response that might result from airway manipulation in a neonate.

Adenosine

Adenosine is a purine nucleoside that binds to adenosine receptors in the myocardium and peripheral vasculature to cause slower conduction through the AV node by prolonging the AV-node refractory period. Adenosine is used to treat supraventricular tachycardia by terminating the reentrant circuit.[47] It must be given very rapidly (typically in a stopcock followed by a 10-mL flush) due to its very rapid metabolism by adenosine deaminase on red blood cells, which clears it within 30 seconds.[48]

Amiodarone

Amiodarone is an antiarrhythmic drug that is classified as a class III antiarrhythmic (increases action potential and refractory period), but spans over all the classes in its effect as potassium channel blocker, sodium influx blocker, noncompetitive beta-blocker, and calcium channel blocker.[49] The effect of amiodarone is directly associated with the route of administration. Oral administration leads to a mostly class III effect, but the IV loading dose most commonly used in an emergency results in a class II effect with increased AV node refraction and AV node conduction.[50] The alpha-adrenergic blockade effect of amiodarone leads to coronary and systemic vasodilation, so while enhancing heart perfusion it may lead to overall hypotension.[51] With its wide pharmacological profile, amiodarone has become one of the recommended drugs (along with lidocaine) for shock refractory VF and pulseless VT (pVT).[18]

Lidocaine

Lidocaine is a class 1B antiarrhythmic that acts on the sodium channels to increase the refractory period and decrease the action potential to stop reentrant ventricular arrhythmias. As there is no effect on AV conduction time, lidocaine does not have an effect on atrial or junctional arrhythmias, but is useful in decreasing the ventricular accelerated ectopic foci.[52] Lidocaine is used for ventricular tachyarrhythmias and can be used interchangeably with amiodarone for shock-refractory VF or pVT.[18]

Magnesium

Hypomagnesemia may occur in critically ill children and with coming off cardiopulmonary bypass, which may increase the risk of arrhythmias. Administering magnesium emergently (during CPR) is only indicated for hypomagnesemia and torsades de pointes VT. If magnesium is given rapidly it can lead to hypotension due to a decrease in systemic vascular resistance, so the infusion rate may be limited by this adverse effect.[8]

Calcium

Calcium directly affects contractility and ventricular automaticity, but despite its essential role in cardiac contractility it has been found to be associated with poor survival and neurological outcomes in pediatric patients when used during CPR.[53] This negative effect may be due to the role of calcium in cell death of many organs. The only indications for calcium during cardiopulmonary resuscitation are suspected hypocalcemia, hyperkalemia, hypermagnesemia, and calcium channel blocker overdose.[52] Giving calcium during an intraoperative cardiac arrest may be warranted as there is a higher potential for hypocalcemia and hyperkalemia with rapid blood product administration.[54] As electrolyte imbalance was noted to be associated with 5% of pediatric perioperative arrests in the Pediatric Perioperative Cardiac Arrest Registry, intraoperative CPR is a special situation during which calcium administration should likely occur.[7] Calcium administration should be carried out with care as it can lead to bradycardia, heart block, and ventricular standstill.[7]

Sodium Bicarbonate

Sodium bicarbonate administration during cardiopulmonary arrest remains very controversial. The use of sodium bicarbonate is beneficial in correcting the metabolic acidosis that can lead to depressed myocardial function and decreased response to catecholamines. It may also treat the acidosis that leads to systemic vasodilation and increased pulmonary vascular resistance.[8,55] The worrisome effects of bicarbonate administration include metabolic alkalosis that can impair oxygen delivery to tissues (shift the oxyhemoglobin dissociation curve to the left), hypernatremia, hypercapnia, and hyperosmolarity which all may be associated with increased mortality.[56,57] This leaves the use of sodium bicarbonate limited to hyperkalemic arrest, hypermagnesemia, long CPR time, tricyclic antidepressant overdose, and overdose from sodium-blocking drugs (cocaine, beta-blockers, and diphenhydramine).[8,51]

QUALITY CPR

While survival outcomes have continued to improve over the years, the degree of mortality and morbidity remains quite high even among IHCA patients.[2,58] With this knowledge, there has been a growing focus on identifying methods to monitor the quality of CPR efforts being provided by healthcare providers, particularly in the inpatient setting (ICU or OR) where patients often already have advanced airways and invasive monitoring. The AHA recommends the use of arterial line waveforms and in-line capnography as guidance for the quality of compressions being provided.

Capnography

Capnography should be used to verify tracheal tube placement during perfusing rhythms and assess cardiac output by chest compressions during cardiac arrest. If $ETCO_2$ is not detected during cardiac arrest, the position of the tracheal tube should be confirmed by direct laryngoscopy, taking care to minimize interruptions to compressions.

The AHA previously recommended a target $ETCO_2$ of 15 mm Hg or higher, as animal studies supported a direct association between $ETCO_2$ and ROSC. Adjustments to the quality of compressions should occur if this target is not being met by optimizing the depth, rate, and recoil. An effort to ensure the

patient is not being ventilated excessively should also be made. A rapid rise in $ETCO_2$ can also be a visual indicator of ROSC, which is another significant utility of capnography monitoring during cardiac arrest.

In cases of low pulmonary blood flow, such as pulmonary embolism or obstructed caval shunts, $ETCO_2$ may not be detectable regardless of adequacy of compressions. In cases of severe airway obstruction, such as status asthmaticus or pulmonary edema, the $ETCO_2$ may be undetectably high.

Invasive Hemodynamic Monitoring

If a patient has an invasive arterial catheter during cardiac arrest, the waveform can be used to optimize the delivery of chest compressions. Animal studies showed an increased likelihood of ROSC when invasive monitoring is used to guide CPR. At this time, there are no evidence-based guidelines for goal systolic or diastolic blood pressures.[18]

POSTRESUSCITATION CONSIDERATIONS

In the postresuscitative phase, after achieving ROSC, the goals of management focus on preserving neurological function and preventing further secondary organ damage. During this phase, the patient is at the highest risk for brain injury, ventricular arrhythmias, and reperfusion injury. After stabilization, the goal should be to diagnose the underlying etiology of the arrest and provide appropriate interventions, otherwise the patient remains at risk for additional events.

Myocardial Function

Myocardial stunning is common after cardiac arrest and usually involves global, biventricular systolic and diastolic dysfunction. The severity of myocardial dysfunction increases with prolonged untreated cardiac arrest time, prolonged CPR, and after administration of multiple shocks of higher energy for defibrillation. Myocardial stunning will often lead to hypotensive shock, further exacerbating secondary organ damage.[11] Several pediatric studies demonstrated that post-ROSC hypotension was associated with worse survival to discharge and less likelihood of discharge with favorable neurological outcomes.[59] Specific treatment for this phenomenon has not been well-described in pediatric patients; however, the use of fluid resuscitation followed by continuous infusions of vasoactive agents with inotropic properties is recommended to maintain a systolic blood pressure greater than the 5th percentile for age.[18] Care should be taken not to increase the afterload to the left ventricle through aggressive use of vasoactive agents leading to hypertension in the postarrest phase of care, which may cause further myocardial dysfunction.[20]

Targeted Temperature Management

Hyperthermia is known to lead to worse outcomes and is common in postarrest children.[20,60] Fevers should be treated aggressively, with the goal to achieve normothermia. Trials in adults have shown induced hypothermia to be neuroprotective postcardiac arrest; however, this was not replicated in a pediatric study.[61] At this time, there is inadequate evidence to support cooling after IHCA for pediatric patients, thus the mainstay of treatment should be avoidance of hyperthermia.[18]

Oxygenation

Hyperoxemia after cardiac arrest was shown to increase oxidative stress in animal studies. There is limited evidence in the pediatric population about the effect of hyperoxemia on outcomes, though one large observational study showed improved survival to ICU discharge in patients with normoxemia when compared to those with hypoxemia ($PaO_2 < 60$ mm Hg) or hyperoxemia ($PaO_2 > 300$ mm Hg).[62] Thus, it is suggested to target normoxemia in pediatric patients in the postresuscitative phase.

Extracorporeal Life Support (ECLS)

Initiating CPR is the gold standard for the low-flow and no-flow states during a cardiopulmonary arrest, but early activation of ECLS should be considered in cardiac arrest with reversible causes that are refractory to standard resuscitation measures.[18] ECLS is now part of the 2015 AHA PALS guidelines and is recommended for in-house cardiac arrest and has been especially useful in patients with congenital heart disease, as several studies

have found that ECLS in this patient cohort has higher survival rates.[36,63,64] The use of ECLS in the operating room is important as typically this is an environment in which cardiac arrest is recognized very quickly by personnel highly trained in resuscitation and many times the surgeon is readily available to initiate ECMO. ECLS should be considered in reversible causes that include hyperkalemia, local anesthetic toxicity, general anesthetic overdose, and airway emergency. Patients with bleeding issues may be a challenge on ECMO, but the only contraindications to ECMO are patients with irreversible pathology or disability that would prevent an acceptable quality of life after conclusion of ECMO.[8]

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