Chapter

45 CARDIOPULMONARY RESUSCITATION

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Cardiopulmonary resuscitation (CPR) was initially defined nearly 50 years ago, as the administration of mouth-to-mouth ventilation and closed chest cardiac compressions in a pulseless patient. Since that time, significant advances in CPR and cardiovascular life support have been made. Today, the early descriptions of CPR are termed *basic life support* (BLS), whereas adult advanced cardiovascular life support (ACLS) and pediatric advanced cardiovascular life support (PALS) include additional invasive techniques by experienced practitioners.

Out-of-hospital resuscitation is well described, whereas in-hospital resuscitation and life support are less commonly studied. A retrospective analysis of in-hospital CPR found that between 2000 and 2009, 1 in 393 hospitalized patients received CPR, and 23% survived to discharge.[1] Cardiac arrest in the perioperative period is unique in that it can frequently be anticipated, and health care providers and resources are immediately available.

The American Heart Association (AHA), in conjunction with the International Liaison Committee on Resuscitation (ILCOR), published updated guidelines for the administration of CPR and emergency cardiovascular care (ECC) in 2015. These guidelines, revised from the 2010 version, include added emphasis on systems of care in the prehospital, in-hospital, and postresuscitation settings, and on the continued education of CPR techniques to providers. Furthermore, instead of periodic overall updates, new evidence will now be continually evaluated and revised guidelines will be available online.[2,3]

BASIC LIFE SUPPORT

BLS includes a number of key measures, including recognition of unresponsiveness and cardiac arrest, activation

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of an emergency response system, early administration of CPR, and early defibrillation if indicated. In the hospital setting, a health care provider will perform the following sequence of steps, as described by the AHA algorithm: (1) ensure safety; (2) check for response; (3) activate resuscitation team; (4) simultaneously check for adequate breathing and pulse; (5) retrieve automated external defibrillator (AED) and emergency equipment; (6) begin CPR and defibrillate when defibrillator becomes available; and (7) provide two-person CPR as help arrives[4] (Fig. 45.1).

Recognition

The recognition and management of cardiac arrest in an unresponsive patient differ between laypersons and health care providers. The AHA guidelines recognize this

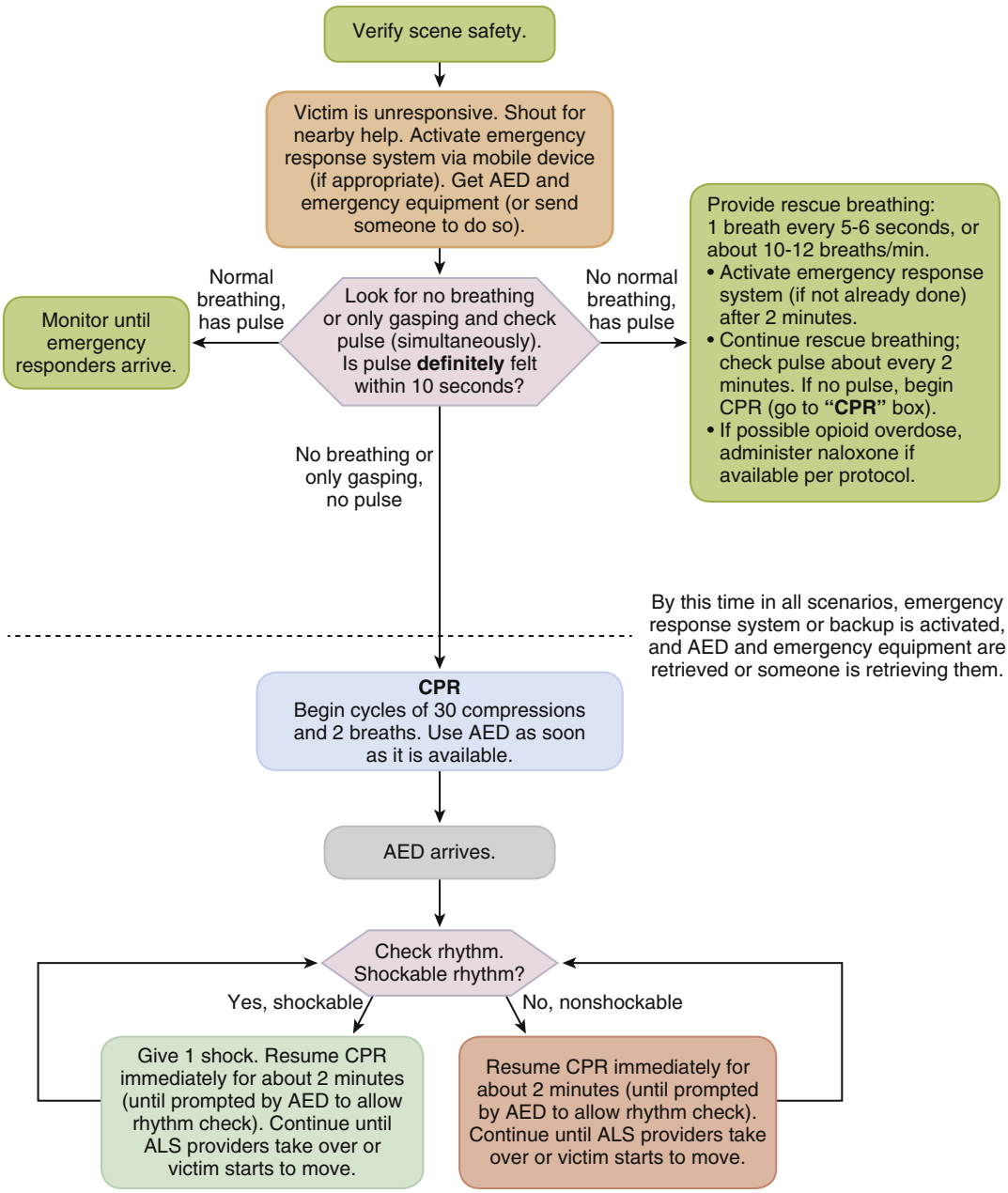


Fig. 45.1 BLS Healthcare Provider Adult Cardiac Arrest Algorithm—2015 Update. *AED,* Automated external defibrillator; *ALS,* advanced life support; *BLS,* basic life support; *CPR,* cardiopulmonary resuscitation. (©2015 American Heart Association.)

distinction and include increased flexibility in emergency response activation either before or after breathing and pulse assessment for health care providers. The 2015 guidelines also include a larger role for dispatcher-guided CPR for laypersons in treating out-of-hospital cardiac arrest.

Health care providers should check for a pulse while simultaneously evaluating for adequate ventilation. The pulse should be assessed at either the carotid or femoral artery. The elapsed time for the pulse check should not exceed 10 seconds, in order to minimize time to start chest compressions. When monitoring respiration, occasional gasps should not be mistaken for normal breathing.

Early Cardiopulmonary Resuscitation

When initiating chest compressions, the heel of the hand is placed longitudinally on the lower half of the sternum, between the nipples. The sternum is depressed at least 5 cm (2 inches) at a rate of at least 100 compressions per minute but no faster than 120 compressions per minute. Rates more rapid than 120 compressions per minute lead to a decrease in the depth of compressions.[5] A depth of no more than 6 cm is also recommended, as excessive compression depth has been associated with an increased rate of thoracic injury. Complete chest recoil is necessary to allow for venous return and is important for effective CPR. The pattern is 30 compressions to 2 breaths (30:2 equals 1 cycle of CPR), regardless of whether one or two rescuers are present.

Since 2010, the importance of definitive airway management has taken a secondary role to chest compressions. The old mnemonic ABCD (airway, breathing, circulation, and defibrillation) has given way to CAB (compression, airway, breathing). This is because the early initiation of high-quality chest compressions improves the likelihood of a return of spontaneous circulation (ROSC). Airway maneuvers are still attempted, but they should occur quickly, efficiently, and minimize interruptions in chest compressions. Opening the airway can be achieved by a simple head tilt–chin lift technique (Fig. 45.2). A jaw thrust maneuver can be used in patients with suspected cervical spine injury. Simple airway devices, such as nasal or oral airways, can be inserted to displace the tongue from the posterior oropharynx.

Although several large out-of-hospital studies have demonstrated that chest compression-alone CPR is not inferior to traditional compression-ventilation CPR, health care providers are still expected to provide assisted ventilation.[6,7] Care should be taken to avoid rapid or forceful breaths. Concern exists for reducing preload and cardiac output with excessive positive-pressure ventilation.[8] Establishing an advanced airway during in-hospital cardiac arrest (IHCA) results in fewer interruptions to chest compressions during CPR.[9] Complications may also occur from gastric insufflation and subsequent aspiration of gastric contents. Maximum oxygen concentration

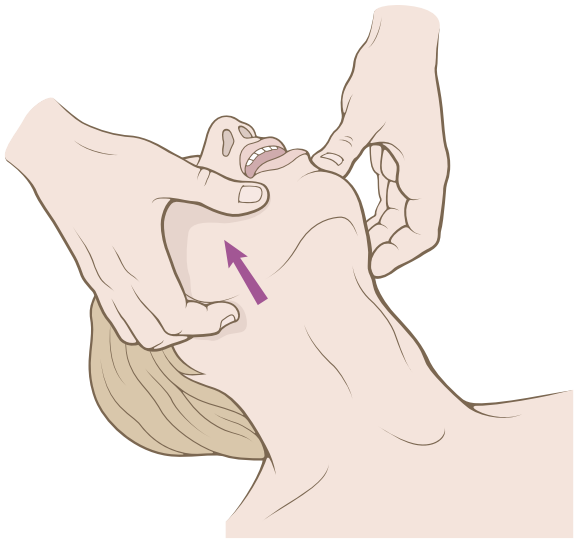


Fig. 45.2 The head tilt–jaw thrust maneuver provides a patent upper airway by tensing the muscles attached to the tongue, thus pulling the tongue away from the posterior pharynx. Forward displacement of the mandible is accomplished by grasping the angles of the mandible and lifting with both hands, which serves to displace the mandible forward while tilting the head backward.

is administered in order to provide optimally saturated arterial hemoglobin concentrations. Delivered tidal volumes of approximately 400 to 600 mL are given over 1 second and should produce visible chest rise. Once an advanced airway has been established, a respiratory rate of 10 breaths/min is the goal because hyperventilation is detrimental for neurologic recovery. The decreased minute ventilation is also appropriate because cardiac output is much smaller than normal during resuscitation.

Early Defibrillation

A defibrillator is attached to the patient as soon as possible. Proper electrode pad placement on the chest wall is to the right of the upper sternal border below the clavicle and to the left of the nipple with the center in the midaxillary line (Fig. 45.3). Most electrode pads now come with diagrams showing their correct positioning. Alternative locations include anterior-posterior, anterior–left infrascapular, and anterior–right infrascapular. Right anterior axillary to left anterior axillary is not recommended.

The amount of energy (joules [J]) delivered is dependent on the type of defibrillator used. Two major defibrillator types (monophasic and biphasic) are available. Monophasic waveform defibrillators deliver a unidirectional energy charge, whereas biphasic waveform defibrillators deliver an in-series bidirectional energy charge. Based on evidence from implantable defibrillators, bidirectional energy delivery is probably more successful in

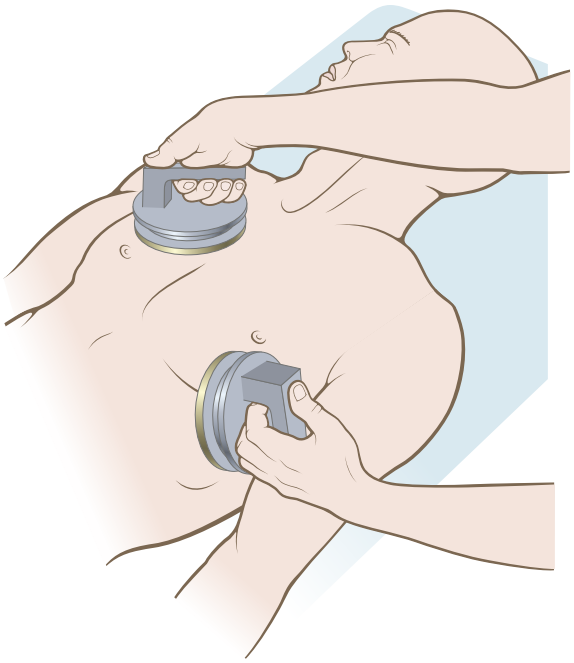


Fig. 45.3 Schematic depiction of the proper placement of paddle electrodes in an adult.

terminating ventricular tachycardia (VT) and ventricular fibrillation (VF). In addition, biphasic waveform shocks require less energy than traditional monophasic waveform shocks (120 to 200 J vs. 360 J, respectively) and may therefore cause less myocardial damage.

The time until defibrillation is critical to survival, especially because the most frequent initial cardiac rhythm in adult patients is VT/VF. Defibrillation should occur as soon as possible when recognizing a VT/VF arrest. CPR should be initiated while emergency equipment is being retrieved. In one study of IHCAs, 30% of patients received delayed defibrillation. Patients receiving delayed defibrillation have slower rates of ROSC and survival to hospital discharge. Furthermore, each additional minute of delay was associated with worse outcomes.[10] Chest compressions should be resumed immediately following defibrillation.

Ancillary Devices and Alternative Techniques

The 2015 AHA guidelines reviewed the evidence for ancillary devices used during CPR and found insufficient support to recommend any of the following: impedance threshold device, active compression-decompression CPR with impedance threshold device, mechanical piston device for chest compressions, and load distributing band devices.

There was also insufficient evidence to recommend the routine use of extracorporeal CPR (venoarterial extracorporeal membrane oxygenation [ECMO]) for patients in cardiac arrest. However, there may be some benefit in carefully selected patients who suffer from witnessed in-hospital arrest secondary to reversible causes.

ADULT ADVANCED CARDIAC LIFE SUPPORT

Adult ACLS includes several interventions besides BLS in order to manage cardiac arrest. These interventions can include airway manipulation, medication administration, arrhythmia management, and transition to postresuscitation care. However, the key element of ACLS remains high-quality CPR, which includes correctly performed chest compressions, minimal compression interruption, and early cardiac defibrillation. The additional components of ACLS and specific arrhythmia management will be discussed later. As there were no updates to the bradycardia and tachycardia algorithms from 2010, they will not be reviewed in detail. Figs. 45.4 and 45.5 summarize the management of the patient with bradycardia or tachycardia with a pulse. All algorithms are readily available online.[3]

Monitoring Cardiopulmonary Resuscitation

A number of physiologic variables can be used to monitor CPR. Continuous monitoring of end-tidal carbon dioxide (P_{ETCO_2}) with waveform capnography can be beneficial during resuscitation. In addition to confirmation of advanced airway placement, P_{ETCO_2} can guide the rescuers in adequacy of chest compressions.[11] Alternative physiologic measures during CPR include arterial relaxation diastolic pressure, arterial pressure monitoring, and central venous oxygen saturation. Specific target values during resuscitation are still being evaluated.[12] Furthermore, a prolonged reduction in P_{ETCO_2} should not be used in isolation for prognostication, and it should certainly not be used in patients without an endotracheal tube. Bedside cardiac ultrasound can also be considered when managing cardiac arrest, but its use is not routinely recommended. If it is utilized, an experienced sonographer should perform the ultrasound and interruptions in chest compressions should be minimized.

Airway Management

The 2015 AHA guidelines, consistent with the ILCOR review, recommend either a bag-mask or advanced airway device (endotracheal tube or supraglottic airway) for providing oxygenation and ventilation during CPR.[13] The choice of technique depends on the skill of the provider. Because chest compressions are often not performed during endotracheal intubation, the rescuer should compare the need for compressions against the need for definitive airway management Chest compressions are not interrupted for longer than 10 seconds during airway management and are resumed immediately following

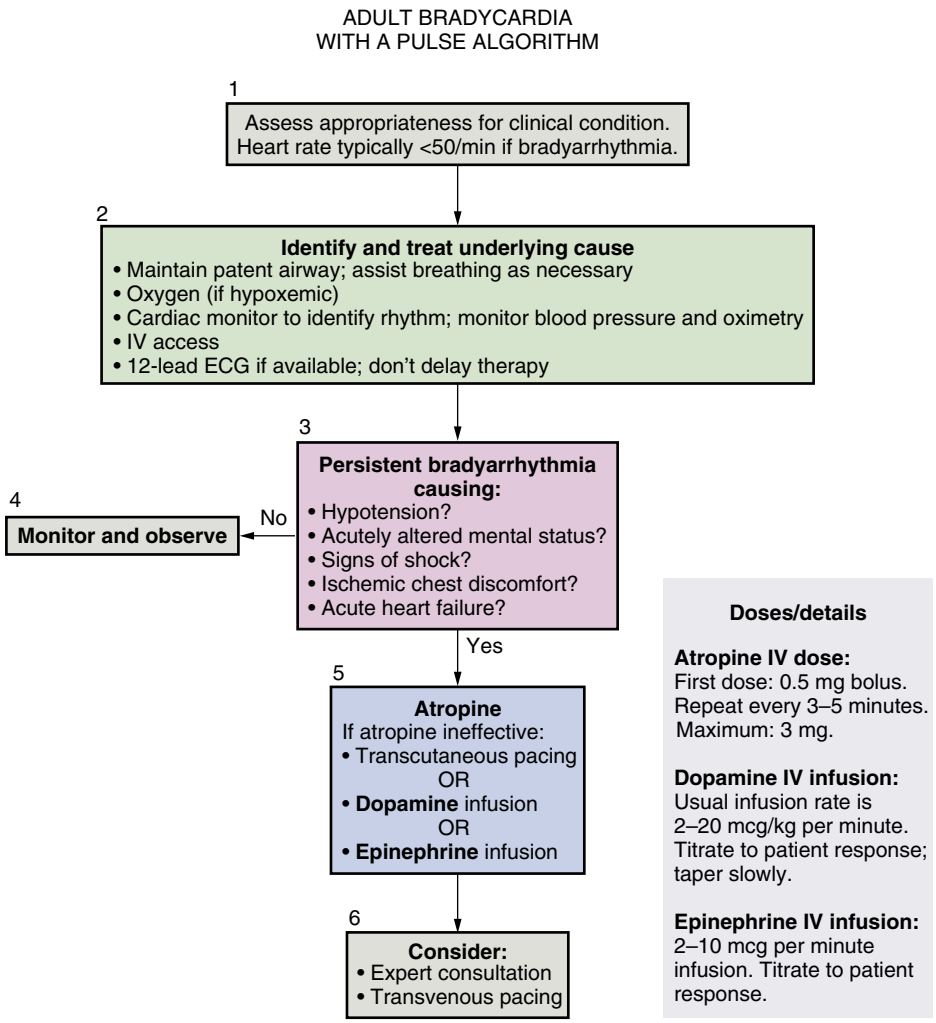


Fig. 45.4 Resuscitation algorithm for bradycardia with a pulse. *ECG*, Electrocardiogram; *IV*, intravenous. (From American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care – Part 7: Adult Advanced Cardiovascular Life Support. ECCguidelines.heart.org © Copyright 2015 American Heart Association, Inc.)

endotracheal intubation. If the intubation attempt is unsuccessful, placement of a laryngeal mask airway can be considered (also see Chapter 16). Insertion of an advanced airway can be deferred until after the patient fails to respond to several cycles of CPR and defibrillation. However, the clinical course of the arrest should be considered. For example, a patient with severe pulmonary edema may benefit from endotracheal intubation sooner rather than later. There are no formal recommendations for the timing of advanced airway placement.

Continuous waveform capnography is recommended as the measurement of choice for the assessment of advanced airway placement. Clinical evaluation should also occur, which includes auscultation of bilateral breath sounds and visualization of bilateral chest rise. If capnography is not available, alternative methods include esophageal detector device, nonwaveform capnogram, and ultrasound. Once the endotracheal tube is confirmed to be in the trachea, it is secured in place. One breath is delivered every 6 seconds (10 breaths/min) without synchronization with compressions.

Algorithms

Pulseless Arrest

Cardiac dysrhythmias that produce pulseless cardiac arrest are (1) VF, (2) VT, (3) pulseless electrical activity (PEA), and (4) asystole (Fig. 45.6). During pulseless cardiac arrest, the primary goals are to provide effective chest compressions and early defibrillation if the rhythm is VF or VT. Drug administration is of secondary importance because the efficacy of pharmacologic interventions has been difficult to measure or prove. After initiating CPR and defibrillation, rescuers can then establish intravenous

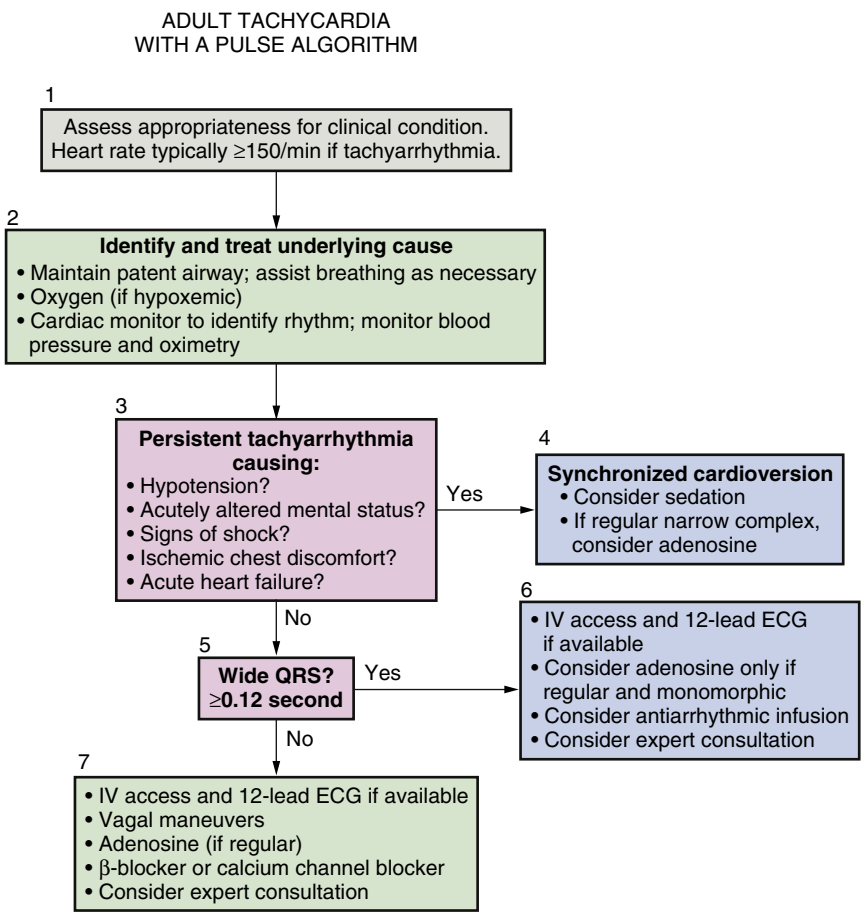


Fig. 45.5 Resuscitation algorithm for tachycardia with a pulse. *CHF*, Congestive heart failure; *ECG*, electrocardiogram; *IV*, intravenous; J, joule; *NS*, normal saline; *VT*, ventricular tachycardia. (From American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care – Part 7: Adult Advanced Cardiovascular Life Support. ECCguidelines.heart.org

access, obtain a more definitive airway, and consider drug therapy, all while providing continued chest compressions and ventilation.

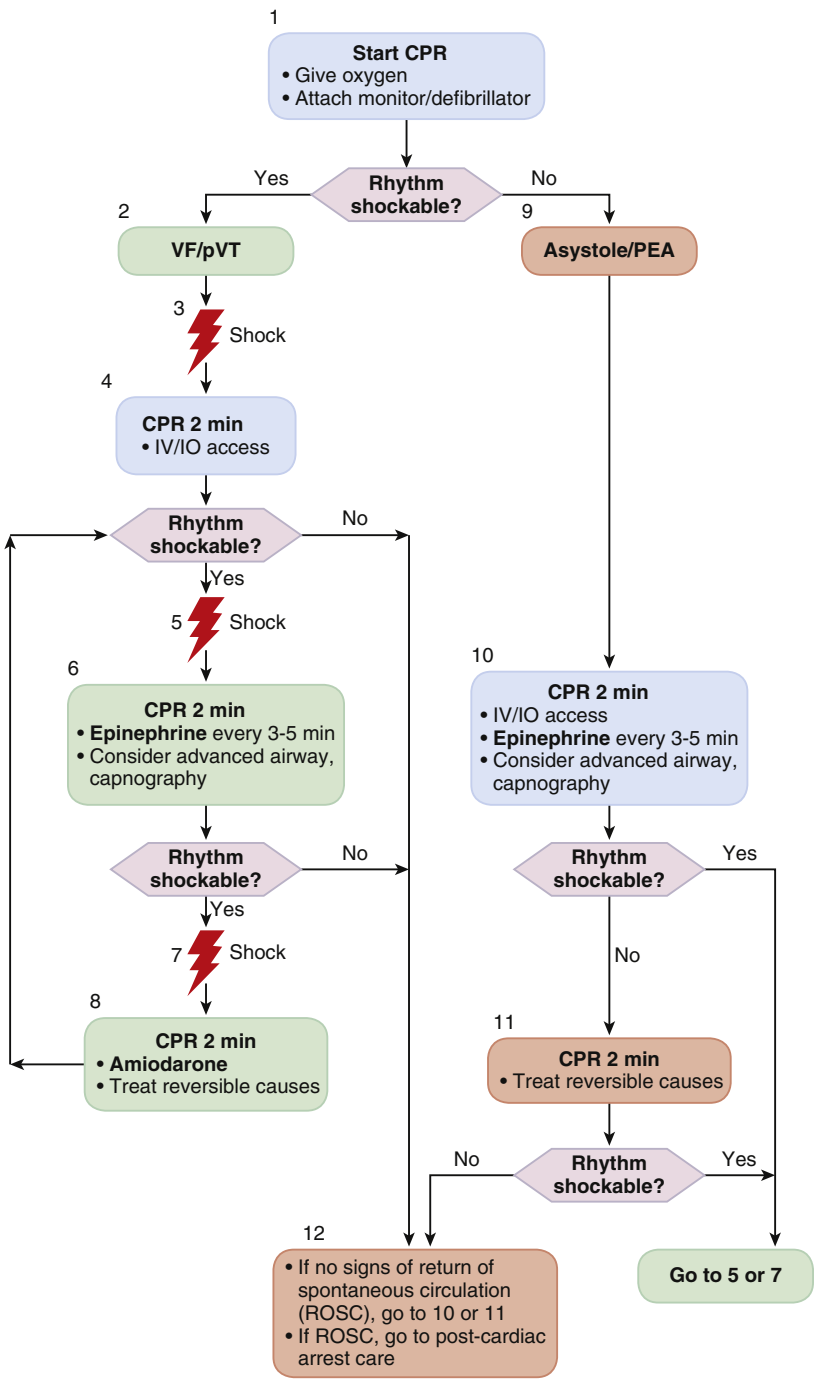
Ventricular Fibrillation/Ventricular Tachycardia

If the cardiac arrest is witnessed, the health care provider immediately places the defibrillator pads on the patient's chest, determines the rhythm, and delivers a shock if VF or VT is present (see Fig. 45.6). CPR is resumed immediately after delivery of the shock and continued for five cycles or about 2 minutes, followed by reevaluation of the cardiac rhythm. If the patient remains in VF/VT, the defibrillator is charged to the appropriate energy level while CPR is still being performed, as determined by the manufacturer's instructions. A biphasic defibrillator is preferred over monophasic, and a single shock is preferred over sequential (also called *stacked*) shocks.

If VF or VT persists after one to two sets of CPR-defibrillation cycles, a vasopressor is given (Table 45.1). Epinephrine, 1 mg intravenously (IV), may be administered every 3 to 5 minutes. Drug administration is timed to minimize interruptions in chest compressions. If the patient remains in VT/VF, amiodarone, an antiarrhythmic, can improve the likelihood of restoring and maintaining ROSC. The role of antiarrhythmics in improving survival following VF/VT arrest is not clear. A currently ongoing trial, ROC-ALPS seeks to provide information regarding the use of lidocaine, amiodarone, and placebo in managing arrhythmia during cardiac arrest.[14] Magnesium sulfate can be considered if torsades de pointes is suspected.

Asystole/Pulseless Electrical Activity

Asystole is the absence of any ventricular electrical activity and is usually a moribund rhythm, whereas PEA is



CPR quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Rotate compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
  - If P_{ETCO_2} <10 mm Hg, attempt to improve CPR quality.
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality.

Shock energy for defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Drug therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.

Advanced airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in P_{ETCO_2} (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia
- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

Fig. 45.6 Adult Cardiac Arrest Algorithm – 2015 Update. *CPR*, Cardiopulmonary resuscitation; *ET*, endotracheal; *IO*, intraosseus; *IV*, intravenous; *PEA*, pulseless electrical activity; P_{ETCO_2}, partial pressure of end-tidal carbon dioxide; *pVT*, pulseless ventricular tachycardia; *VF*, ventricular fibrillation. (From Link MS, Berkow LC, Kudenchuk PJ, Halperin HR, Hess EP, Moitra VK, Neumar RW, O'Neil BJ, Paxton JH, Silvers SM, White RD, Yannopoulos D, Donnino MW. Part 7: adult advanced cardiovascular life support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(suppl 2):S444–S464.

Table 45.1 Drugs Used During Adult Cardiopulmonary Resuscitation

Drug Name	Dose	Indication
Adenosine	6 mg IV/IO May repeat 12 mg IV/IO (cut dose in half if using central line)	For stable narrow QRS tachycardia or monomorphic VT (contraindicated with preexcitation syndrome)
Amiodarone	300 mg IV/IO May repeat 150 mg IV/IO 150 mg IV/IO over a 10-min period Maintenance infusion of 1 mg/min for 6 h, then 0.5 mg/min Maximum total dose of 2.2 g/24 h	For pulseless VT/VF For stable VT or uncertain wide QRS tachycardia and narrow QRS tachycardias
Atropine[a]	0.5 mg IV/IO May repeat to total dose of 3 mg	For bradycardia
Diltiazem	15 to 20 mg (0.25 mg/kg) IV/IO over a 2-min period May repeat in 15 min at 20-25 mg/kg (0.35 mg/kg) Maintenance infusion of 5-15 mg/h, titrate to heart rate	For stable narrow QRS tachycardia (contraindicated with preexcitation syndrome)
Dopamine	2 to 10μg/kg/min by infusion	For bradycardia instead of a pacer, while awaiting a pacer, or if a pacer is ineffective or not tolerated
Epinephrine[a]	1 mg IV/IO Repeat every 3 to 5 min 2 to 10μg/min by infusion	For pulseless cardiac arrest For bradycardia instead of a pacer, while awaiting a pacer, or if a pacer is ineffective or not tolerated
Esmolol	0.5 mg/kg IV/IO load, followed by an infusion at 0.05 mg/kg/min May repeat the 0.5-mg/kg bolus and increase the infusion to 0.1 mg/kg/min Maximum infusion of 0.3 mg/kg/min	For stable narrow QRS tachycardias (contraindicated with preexcitation syndrome)
Lidocaine[a]	1 to 1.5 mg/kg IV/IO May repeat 0.5 mg to 0.75 mg/kg Maximum total of three doses or 3 mg/kg	For pulseless VT/VF when amiodarone is NOT available
Magnesium	1 to 2 g IV/IO	For torsades de pointes
Metoprolol	5 mg IV/IO May repeat every 5 min Maximum total dose of 15 mg	For stable narrow QRS tachycardias (contraindicated with preexcitation syndrome)
Procainamide	20 to 50 mg/min IV/IO (max 17 mg/kg) until arrhythmia suppressed Maintenance infusion of 1 to 4 mg/min	For stable wide QRS tachycardia
Sotalol	100 mg (1.5 mg/kg) IV/IO over 5 min	For stable wide QRS tachycardia
Verapamil	2.5 to 5 mg IV/IO over a 2-min period May repeat 5 to 10 mg over a 15- to 30-min period Maximum total dose of 20 mg	For stable narrow QRS tachycardia (contraindicated with preexcitation syndrome)

[a]Also effectively delivered by tracheal mucosal absorption when administered through an endotracheal tube.
IO, Intraosseus; *IV*, intravenous; *VF*, ventricular fibrillation; *VT*, ventricular tachycardia.

often caused by a reversible condition and can be treated if the inciting cause is identified (Table 45.2). These two cardiac rhythms have been combined as the second part of the pulseless arrest algorithm because of similarities in their management (see Fig. 45.6). Neither will benefit from defibrillation; effective CPR with minimal interruptions, identifying and treating reversible causes, and establishing an advanced airway are the primary interventions. A bedside cardiac ultrasound may provide valuable information regarding the cause of arrest. Additionally, the absence of ventricular wall motion on ultrasound predicts an unlikely ROSC.[15] A vasopressor may be administered after initiation of CPR. Epinephrine, 1 mg IV, is given every 3 to 5 minutes. Cardiac rhythm checks

Table 45.2 Major Causes of Cardiovascular Collapse in the Perioperative Period

8 Hs	8 Ts
Hypovolemia	**T**oxins (anaphylaxis/anesthesia)
Hypoxia	**T**amponade
Hydrogen ion (acidosis)	**T**ension pneumothorax
Hyperkalemia/hypokalemia	**T**hrombosis in coronary artery
Hypoglycemia	**T**hrombus in pulmonary artery
Hypothermia	**T**rauma
Malignant **h**yperthermia	Q**T** interval prolongation
Hypervagal response	Pulmonary hyper**t**ension

Modified from the 5 Hs and 5 Ts proposed by the American Heart Association (AHA).

are performed after every five cycles or 2 minutes of CPR. The administration of high-quality chest compressions can be monitored by P_{ETCO_2}, coronary perfusion pressure, or central venous saturation (S_{CVO_2}), if available. If an organized cardiac rhythm is present, the rescuer checks for a pulse. If there is no pulse, CPR should be continued. If a pulse is present, the rescuer identifies the rhythm and treats accordingly.

Medications

Establishing intravenous access is important, but it should not interfere with CPR and defibrillation. A single, large peripheral intravenous or intraosseus catheter is sufficient for resuscitating most pulseless patients. Drugs are administered rapidly and followed with a 20-mL fluid bolus if given peripherally. If intravenous/intraosseus access cannot be obtained, or is lost, certain drugs (epinephrine, lidocaine, atropine, naloxone) can be given via the endotracheal tube. The endotracheal tube dose is 2 to 10 times the recommended intravenous dose, and the drug is diluted in 5 to 10 mL of sterile water before instillation down the endotracheal tube.

Epinephrine and amiodarone are among the most commonly used drugs in the ACLS algorithms (see Table 45.1) and deserve special attention. Epinephrine is a combined direct α- and β-adrenergic receptor agonist. In multiple animal studies, administration of epinephrine was beneficial in establishing ROSC. Epinephrine can increase diastolic blood pressure and thereby restore coronary perfusion pressure and blood flow back to the myocardium. However, epinephrine also increases myocardial oxygen consumption by increasing heart rate and afterload.

Amiodarone was initially developed as an antianginal drug in the 1950s but was abandoned because of its side effects. Because it has effects on cardiac sodium and potassium channels, as well as α- and β-receptors, amiodarone has been reinvestigated for its antiarrhythmic effects. In this regard, amiodarone prolongs repolarization and refractoriness in the sinoatrial node, the atrial and ventricular myocardium, the atrioventricular (AV) node, and the His-Purkinje cardiac conduction system. Amiodarone can exacerbate or induce arrhythmias especially torsades de pointes. This drug may interact with volatile anesthetics to produce heart block, profound vasodilation, myocardial depression, and severe hypotension. Amidarone has many drug interactions and can prolong the effects of oral anticoagulants, phenytoin, digoxin, and diltiazem. Despite its multiple disadvantages, administration of amiodarone improves survival to hospital admission in adults with out-of-hospital VF/VT arrest when compared with placebo and lidocaine.[8,9] The recommended dose of amiodarone for VF/VT is 300 mg IV. An additional bolus dose of 150 mg IV may be given for persistent VF/VT.

Vasopressin, a nonadrenergic vasopressor, was removed from the 2015 ACLS guidelines because of a lack of demonstrated benefit when compared to epinephrine.[16] Drugs used for ACLS are associated with ROSC but not with improved survival to hospital discharge or neurologic recovery. There are no specific recommendations for timing of ACLS drug delivery, though for a nonshockable rhythm, epinephrine may be given as soon as possible. Administration of steroids along with vasoactive drugs may improve the likelihood of survival and favorable neurologic outcome for IHCAs; however, there is no recommendation for their routine use.[17] Finally, in patients with the potential for cardiac arrest secondary to opioid overdose, administration of naloxone should be considered (see Chapter 9).

PEDIATRIC ADVANCED CARDIOVASCULAR LIFE SUPPORT

Cardiorespiratory resuscitation of infants and children follows the same basic principles as those for adults (also see Chapter 34). Most pediatric cardiac events are a result of arterial hypoxemia and respiratory compromise, and, thus, airway management and breathing are critical to successful pediatric resuscitation. In contrast, adults tend to experience cardiac arrest as a result of VT or VF secondary to myocardial ischemia. Regardless, pediatric BLS follows the same algorithm as for adults: CAB. Naturally, there are several specific differences between adult and pediatric patients. Infants are younger than 1 year in age, whereas children are between the age of 1 year and adolescence. Adult BLS resuscitation guidelines can be used for adolescent children (Table 45.3). The elements of high-quality CPR in pediatrics remain unchanged from adults and include: (1) adequate chest compression

Table 45.3 Comparative Resuscitation Techniques Between Adults, Children, and Infants (Summary of Key BLS Components for Adults, Children, and Infants[a])

Component	Recommendations		
	Adults	Children	Infants
Recognition	Unresponsive (for all ages)		
	No breathing or no normal breathing (i.e., only gasping)	No breathing or only gasping	
CPR sequence	C-A-B		
Compression rate	At least 100-120/min		
Compression depth	At least 2 inches (5 cm) but not more than 2.4 inches (6 cm)	At least one third AP diameter of chest (about 2 inches [5 cm])	At least one third AP diameter of chest (about 1½ inches [4 cm])
Chest wall recoil	Allow complete recoil between compressions		
Compression interruptions	Minimize interruptions and limit interruptions to <10 s		
Airway	Head tilt–chin lift Jaw thrust, if suspected trauma		
Compression-to-ventilation ratio (until advanced airway is placed)	30:2 (1 or 2 rescuers)	30:2 (single rescuer) 15:2 (2 rescuers)	
Ventilations: when the rescuer is not proficient	Compressions only		
Ventilations with advanced airway	1 breath every 6 seconds (10 breaths/min) Asynchronous with chest compressions (about 1 second/breath) Visible chest rise	1 breath every 6 seconds (10 breaths/min) Asynchronous with chest compressions (about 1 second/breath) Visible chest rise	1 breath every 6 seconds (8-10 breaths/min) Asynchronous with chest compressions (about 1 second/breath) Visible chest rise
Defibrillation	Attach and use AED as soon as available. Minimize interruptions in chest compressions before and after shock. Resume CPR beginning with compressions immediately after each shock.		

[a]Excluding the newly born, in whom the cause of an arrest is nearly always asphyxial.
AED, Automated external defibrillator; *AP*, anteroposterior; *C-A-B*, compression, airway, breathing; *CPR*, cardiopulmonary resuscitation.
(From 2015 AHA Summary of Key Basic Life Support Components [Adults, Children, Infants].)

rate; (2) adequate chest compression depth; (3) adequate recoil between chest compressions; (4) minimal interruptions to chest compressions; and (5) avoidance of excess ventilation.[18]

Circulation

In a child, the heel of one or both hands should be placed on the lower half of the sternum, between the nipples, while keeping the fingers off the rib cage and staying above the xiphoid process. In an infant, chest compressions are delivered via the two-finger technique. Two fingers of one hand are placed over the lower half of the sternum approximately one fingerwidth below the intermammary line while keeping above the xiphoid process. For both infants and children, the sternum should be depressed at least one third to one half the anteroposterior diameter of the chest (4 cm infants, 5 cm children) at a rate of 100 to 120 compressions per minute.

Pulse checks and closed chest compressions are performed slightly differently, depending on whether the patient is a child or an infant. In children, the pulse is palpated at the carotid or femoral artery, similar to adults. In infants, the pulse is checked at the brachial or femoral artery. As with adults, P_{ETCO_2} can be used to evaluate the quality of CPR. If invasive monitoring, such as an arterial catheter, is already in place, it may also be used to evaluate and guide CPR.

ECMO can be considered in all pediatric cardiac arrest patients who are refractory to standard conventional therapies.

Airway

The airway of pediatric patients is slightly different from that of an adult, but head tilt–chin lift is still the technique of choice to open the airway. Children tend to have a larger tongue and epiglottis in relation to the mouth and larynx. In addition, they have a larger head in relation to the body. Overextension or excessive flexion of the head can lead to difficulty visualizing the glottic opening during direct laryngoscopy. Straight laryngoscope blades may be preferred over curved blades to lift the epiglottis anteriorly and away from the glottic opening in young children (also see Chapter 34).

Breathing

Given the likely causes of pediatric cardiopulmonary arrest, conventional CPR (compressions and ventilation) is recommended over compression-only resuscitation. The pattern should be 30 compressions to 2 breaths (30:2) if there is a single rescuer and 15 compressions to 2 breaths (15:2) if there are two rescuers.

Defibrillation

In children, defibrillation should be performed when a pulseless shockable rhythm (VT, VF) is present. An initial energy of 2 to 4 J/kg should be attempted, regardless of the waveform type. Subsequent defibrillations should be at least 4 J/kg but should not exceed 10 J/kg. Biphasic AEDs can be used in children older than 1 year old outside the hospital setting. AHA guidelines recommend the use of a pediatric dose attenuator system that will decrease the amount of delivered energy. If one is not available, a standard external defibrillator can be substituted.

Drugs

Most drug dosages are calculated by using current known weight or ideal body weight based on height. Most pediatric units have resuscitation carts divided by weight to facilitate drug administration in an emergency so that calculations do not need to be performed and valuable time is not wasted. As with adults, epinephrine has been associated with increased rate of ROSC and can be used in cardiac arrest. For refractory VF or pulseless VT in pediatric patients, either lidocaine or amiodarone can be administered.

POSTRESUSCITATION CARE

After successful resuscitation with ROSC, patients are admitted to the intensive care unit for further definitive and supportive treatment (Fig. 45.7). Post–cardiac arrest care includes improving cardiopulmonary function optimally to ensure adequate organ perfusion. It should be consistent, integrated, and multidisciplinary. If arrest occurs at a center that is not equipped to manage elements of postresuscitation care, transfer to a larger regional center should be considered.[19]

When possible, therapies are administered concurrently. Specifically, percutaneous coronary interventions (PCIs) should not be delayed to institute hypothermia, and the institution of hypothermia should not delay PCI. Often, vasopressors and inotropes need to be administered during the immediate postresuscitation period because of the presence of myocardial stunning and hemodynamic instability. Central venous access for drug administration may be necessary, along with an arterial catheter to facilitate hemodynamic monitoring.

Acute Coronary Syndrome

An electrocardiogram is obtained as soon as possible after ROSC in order to evaluate for ST-segment elevation myocardial infarction. If acute ST-segment elevation is noted, the patient should be taken for emergent angiography. Some patients with non-ST-segment elevation may also benefit from emergent angiography.[20] These evaluations are made regardless of neurologic status.

Hemodynamic Goals

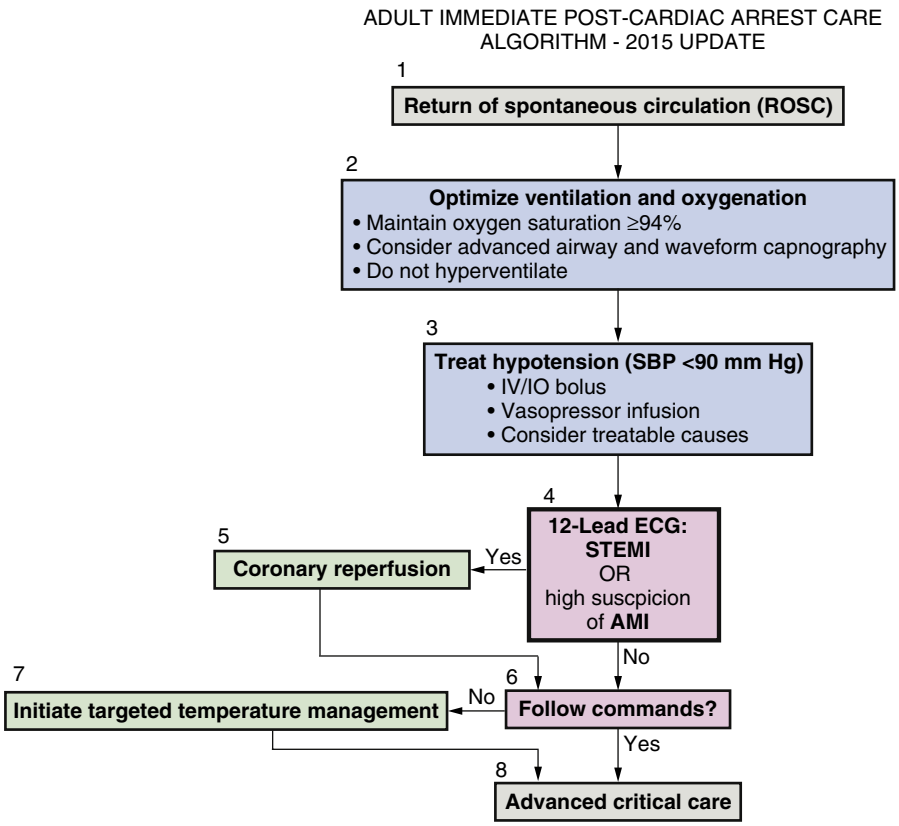
Following an ROSC, oxygenation and ventilation should be evaluated and optimized. Advanced airway placement may be considered, and hyperventilation is avoided. The goal for oxygenation is a saturation more than 94% and for ventilation the goal is a $Paco_2$ of 35 to 45 mm Hg. A chest radiograph is obtained. Hypotension is treated, avoiding systolic arterial blood pressure less than 90 mm Hg or mean arterial blood pressure less than 65 mm Hg. This can be achieved with a combined administration of intravenous fluids and vasoactive drugs. No specific hemodynamic variables, including arterial blood pressure, cardiac output, venous oxygen saturation, or urine output have been recommended, as these likely vary widely between individual patients. Reversible causes for cardiac arrest are assessed. An electrocardiogram, echocardiogram, and serial cardiac enzymes are obtained. Serum lactate is monitored to assess for adequate tissue perfusion.

Neurologic Monitoring

In addition to cardiac recovery, neurologic recovery is of vital importance. This is especially true during the immediate postresuscitation phase. An electroencephalogram can be obtained to evaluate for seizure, and anticonvulsants can be administered for status epilepticus.

Targeted Temperature Management

Temperature should be monitored closely, and hyperthermia is avoided at all times as this can worsen ischemic brain injury (also see Chapter 30). The 2010 ACLS guidelines



Doses/details

Ventilation/oxygenation:
Avoid excessive ventilation. Start at 10 breaths/min and titrate to target P_{ETCO_2} of 35–40 mm Hg.
When feasible, titrate F_{IO_2} to minimum necessary to achieve Spo_2 ≥94%.

IV bolus
Approximately 1–2 L normal saline or lactated Ringer's

Epinephrine IV infusion:
0.1–0.5 mcg/kg per minute (in 70-kg adult: 7–35 mcg per minute)

Dopamine IV infusion:
5–10 mcg/kg per minute

Norepinephrine IV infusion:
0.1–0.5 mcg/kg per minute (in 70-kg adult: 7–35 mcg per minute)

Reversible causes
– **H**ypovolemia
– **H**ypoxia
– **H**ydrogen ion (acidosis)
– **H**ypo-/hyperkalemia
– **H**ypothermia
– **T**ension pneumothorax
– **T**amponade, cardiac
– **T**oxins
– **T**hrombosis, pulmonary
– **T**hrombosis, coronary

Fig. 45.7 Algorithm for postcardiac arrest care. *AMI*, Acute myocardial infarction; *ECG*, electrocardiogram; *F_{IO_2}*, fraction of inspired oxygen; *IO*, intraosseous; *IV*, intravenous; *P_{ETCO_2}*, partial pressure of end-tidal carbon dioxide; *SBP*, systolic blood pressure; *Spo_2*, pulse oximeter oxygen saturation; *STEMI*, ST-segment elevation myocardial infarction. (From American Heart Association. Web-based Integrated Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care – Part 8: Post-Cardiac Arrest Care. ECCguidelines.heart.org. © Copyright 2015 American Heart Association, Inc.)

recommended therapeutic hypothermia of 32° C to 34° C for comatose patients after out-of-hospital VF/VT arrest. Milder therapeutic hypothermia, to 36° C, may confer similar benefit.[21] Temperature management in patients who suffer from IHCA or nonshockable cardiac arrest has not clearly been defined. Given the relative ease and safety of controlling body temperature and the risk of poor neurologic outcome because of hyperthermia, the 2015 guidelines recommend that all comatose patients after cardiac arrest and ROSC be treated with targeted temperature management between 32° C and 36° C. Complications of therapeutic hypothermia include impaired coagulation and increased risk of infection at lower targeted levels, but are quite minimal at 36° C. Thus, patient factors can be taken into consideration when selecting the targeted temperature. Pre-hospital-induced hypothermia is not recommended.

Blood Glucose Control

Increased blood glucose concentrations after resuscitation from cardiac arrest are associated with poor neurologic outcome. Yet, tight control of serum glucose has not been verified to improve neurologic outcome. Regardless,

glucose levels after resuscitation should be monitored closely to avoid hypoglycemia and hyperglycemia.

Prognosis

Patients should not be evaluated for prognosis sooner than 72 hours following ROSC and targeted temperature management in most circumstances.

SPECIAL PERIOPERATIVE CONSIDERATIONS

Though inadequately studied, intraoperative cardiac arrest occurs in up to 43 out of 100,000 procedures. One study identified hemorrhage and anaphylaxis as the two most common causes of intraoperative cardiac arrest.[22] Cardiac arrest during anesthesia is distinct from cardiac arrest in other settings in that our patients have a different pathophysiology. Cardiac arrests during anesthesia are usually witnessed, and frequently anticipated. Furthermore, the cause is often surgical or otherwise more easily reversible. Because of this, perioperative cardiac arrests are associated with a higher survival rate and better neurologic outcomes than other in-hospital arrests.[23] The traditional guidelines do not often translate well into the perioperative setting. Because of that, the American Society of Anesthesiologists (ASA) Committee on Critical Care Medicine published a monograph specific to advanced life support for anesthesia. They expanded common causes of cardiac arrest events to the following categories and factors.[24]

1. Medications: anesthetic overdose, high neuraxial blockade, local anesthetic toxicity, drug administration errors
2. Respiratory: hypoxemia, autopositive end-expiratory pressure (PEEP), acute bronchospasm
3. Cardiovascular: vasovagal, hypovolemic/hemorrhagic shock, distributive shock, obstructive shock, right ventricular failure, left ventricular failure, arrhythmia, acute coronary syndrome

The ASA monograph also suggests the following interventions when performing resuscitation in the operating room: (1) call for help, (2) initiate chest compressions, (3) discontinue anesthetic, (4) discontinue surgery, (5) retrieve emergency equipment, (6) increase fraction of inspired oxygen (F_{IO_2}) to 100%, (7) manually ventilate the lungs, (8) open all intravenous lines, and (9) use capnography to assess CPR.

Four unique circumstances to anesthesia providers are detailed in the next paragraphs.

Anaphylaxis

Minor drug reactions such as a rash are not an uncommon occurrence in the operating room. Major reactions, such as anaphylactic shock, occur much less often. Common drugs associated with anaphylaxis are latex, β-lactam antibiotics, succinylcholine, all muscle relaxants, and intravenous contrast material. The treatment of anaphylaxis involves the administration of epinephrine to interrupt the cascade of profound vasodilation and significant vascular leak. If possible, the offending drug should be removed or stopped. Epinephrine and vasopressin can be used to support arterial blood pressure, while steroids and antihistamines are administered to further attenuate the response. Intravenous fluid administration is essential secondary to the vascular leak. CPR and ACLS should be immediately instituted if there is no pulse palpated. In the event of complete cardiovascular collapse, larger doses of epinephrine may be required (Box 45.1).

Box 45.1 Treatment of Anaphylaxis

Stop or remove inciting agent or drug
Oxygen at F_{IO_2} 1.0
Intravenous fluids
CPR/ACLS if pulseless
Epinephrine, intravenous
- *Bolus:* 10 to 100 μg—if not pulseless
- *Bolus:* 1-3 mg—if pulseless
- *Infusion:* 4-10 μg/min

Vasopressin, intravenous
- *Bolus:* 0.5 to 2 units—if not pulseless
- *Bolus:* 40 units—if pulseless

H_1 blocker, intravenous: diphenhydramine 50 mg
H_2 blocker, intravenous: famotidine 20 mg
Steroid, intravenous: hydrocortisone 50 to 150 mg

ACLS, Advanced cardiovascular life support; *CPR*, cardiopulmonary resuscitation; *H_1, H_2,* histamine receptor types 1 and 2.

Gas Embolism

Although a very rare event, the incidence of gas embolism has the potential to increase in parallel with the worldwide increase in laparoscopic surgical procedures, posterior spine surgery, and endobronchial laser procedures (also see Chapter 30). The initial management includes cessation of the offending cause (i.e., halt insufflation), occlusion of open veins, and flooding the surgical field with saline. Patients should be placed in Trendelenburg position with left side down to keep the gas in the apex of the ventricle and allow for filling. Complete circulatory collapse should be treated with CPR and ACLS.

Local Anesthetic Systemic Toxicity

Local anesthetics affect sodium channels throughout the body, including the brain and the heart. In general, toxicity occurs in a dose-dependent fashion with cardiovascular collapse occurring at the end of the spectrum (also see Chapter 10). In nonanesthetized patients, central nervous system symptoms are vital to recognize as they tend

Box 45.2 Treatment of Local Anesthetic Toxicity

Stop local anesthetic
CPR/ACLS if pulseless
20% Intralipid IV:
- *Load:* 1.5 mL/kg
- *Infusion:* 0.25 mL/kg/h

Sodium bicarbonate to maintain pH >7.25 in a prolonged resuscitation
Consider transcutaneous or transvenous pacing for bradycardic rhythms
Continue CPR for at least 60 min

ACLS, Advanced cardiovascular life support; *CPR*, cardiopulmonary resuscitation; *IV*, intravenous.

to precede cardiac manifestations. Cardiac rhythms can range from premature ventricular contractions to asystole. If possible, the administration of the local anesthetic should be stopped. Intralipid should be given for cardiovascular toxicity.[25] Good neurologic recovery in these patients can occur despite the prolonged resuscitation (Box 45.2). Vasopressin should be avoided, and epinephrine doses should be decreased (<1 μg/kg).[26]

Cardiovascular Collapse From Neuraxial Anesthesia

Cardiovascular collapse from neuraxial anesthesia has been described but is inadequately understood.[27] It seems to occur in younger, otherwise healthy patients undergoing routine surgical procedures with neuraxial anesthesia (also see Chapter 17). Proposed mechanisms causing the cardiac arrest include a shift in autonomic balance toward the parasympathetic system, a decrease in venous return from pooling in the splanchnic circulation, and activation of baroreceptors that stimulate a paradoxic Bezold-Jarisch response. A high level of spinal anesthesia seems to be the most frequent culprit. Regardless, treatment follows standard CPR and ACLS recommendations.

SYSTEMS OF CARE

Health care delivery systems differ significantly between out-of-hospital cardiac arrest and IHCAs. The AHA discusses both of these distinct systems; here we will address IHCA and the myriad roles the anesthesia provider may play. A patient at risk of cardiac arrest while in the hospital depends on appropriate surveillance and prevention, prompt recognition and response by a multidisciplinary team, high-quality CPR, early defibrillation, and additional ACLS as needed.[28] Though recovery from IHCA has improved over the last few decades, there remains considerable variability and room for improvement.

The AHA guidelines recommend the establishment of rapid response teams or medical emergency teams in order to reduce the incidence of cardiac arrest in patients who are at increased risk. These patients should be transferred to a higher acuity setting, such as an intensive care unit. Discussions with the patient or family members regarding preference for aggressive resuscitation should ideally be conducted prior to an actual cardiac arrest event. Crisis resource management techniques should be utilized in order to optimize resuscitation team dynamics. These include a designated resuscitation team, with predetermined roles and communication strategies, and a plan for debriefing after an event.

In 2015 the National Academy of Medicine (formerly Institute of Medicine) released a report titled "Strategies to Improve Cardiac Arrest Survival: A Time to Act." This report noted the significant morbidity associated with cardiac arrest, and the need to improve outcomes.[29,30] The report introduced eight recommendations to improve resuscitation practices: (1) robust data collection and dissemination, (2) improved public response, (3) enhanced emergency medical service (EMS) capability, (4) updated national accreditation standards, (5) continuous quality improvement, (6) increased research funding in resuscitative science, (7) increased speed in adopting existing strategies, and (8) establishment of a new nationwide cardiac arrest collaborative. The AHA has responded to this call to action by announcing commitments to improve systems of care, resuscitation research, and creation of a national cardiac arrest collaborative.[31]

QUESTIONS OF THE DAY

1. What are the components of effective chest compressions in an infant, child, and adult?
2. According to advanced cardiovascular life support (ACLS) guidelines, what is the role of epinephrine, amiodarone, and vasopressin in the management of cardiac arrest?
3. Which patients are most likely to benefit from targeted temperature management after a cardiac arrest?
4. During the perioperative period, what factors are potential contributors to the risk of cardiac arrest?
5. What interventions should be part of cardiac arrest management in the operating room?

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