Contents

Video Contents

Chapter 8

Standard Normal Imaging Planes

Miscellaneous Disease

Imaging Enhancement

Myocardial Disease/Complications of Myocardial Infarction

Pulmonary Embolus

Valvular Disease

on the tricuspid valve *(TV, arrow)*. Note the failure of the leaflets to coapt. *RA,* Right atrium; *RV,* right ventricle.

Pericardial Effusion

Video 8.49. PLAX Posterior Eff. PLAX view of large predominantly posterior postoperative pericardial effusion *(Eff)*. The *arrow* points to small anterior collection of fluid. *Ao,* Aorta; *Dao,* descending aorta; *RV,* right ventricle.

Video 8.50. PSAX Posterior Eff. PSAX view demonstrates very large posterior effusion *(Eff)* in the same patient as Video 8.49. *LV,* Left ventricle.

Video 8.51. Subcostal Peri Eff. Subcostal view demonstrating large inferoposterior accumulation of fluid *(Eff)* in the same patient as Video 8.49.

Video 8.52. PLAX Circ Eff. PLAX demonstrating large circumferential pericardial effusion with right ventricular *(RV)* collapse *(arrow)* suggesting tamponade. *Ao,* Aorta; *DA,* descending aorta; *LV,* left ventricle.

Video 8.53. PSAX Circ Eff. PSAX of the same patient as Video 8.52 demonstrating large pericardial effusion *(Eff)* and right ventricular *(RV)* collapse *(arrow)*. *LV,* Left ventricle.

Video 8.54. Subcostal Circ Eff. Subcostal view of same patient as Video 8.52 demonstrating large circumferential effusion *(Eff)*.

Video 8.55. Dilated IVC/Eff. The inferior vena cava *(IVC)* in the same patient as Video 8.52 is dilated and nonreactive. *Ple,* Pleural effusion.

Video 8.56. Massive Eff. Massive pericardial effusion *(Eff)* secondary to bacterial pericarditis. The image on the left is subcostal; the image on the right is apical four-chamber view, which demonstrates frank collapse of right heart chambers *(arrows)*. *LV,* Left ventricle.

Video 8.57. Massive Eff/IVC. Same patient as Video 8.56 demonstrating dilated nonreactive inferior vena cava *(IVC)*. *Eff,* Pericardial effusion; *LV,* left ventricle.

Video 8.58. PLAX p/op Eff. Massive pericardial effusion *(Eff)* in this postoperative patient, demonstrating almost no filling of either ventricle secondary to fluid compression. The *arrow* shows right ventricle *(RV)* collapse. *LA,* Left atrium; *LV,* left ventricle.

Video 8.59. PLAX p/op Eff 2. Same patient as Video 8.58 demonstrating near cardiac standstill while the operator is struggling to prepare for emergent pericardiocentesis. The *arrow* points to the aortic valve.

Video 8.60. PLAX p/op Eff Tap. Same patient as Video 8.58 demonstrating ventricles beginning to fill as fluid is tapped. *Eff,* Pericardial effusion; *LV,* left ventricle.

Video 8.61. PLAX p/op Eff Tap 2. Same patient as Video 8.58 after more than 1400 mL of bloody pericardial fluid has been drained and ventricles appear at near-normal volume. The patient eventually succumbed of prolonged hypoxia. *Ao,* Aorta; *LV,* left ventricle; *RV,* right ventricle.

Video 8.62. Subcostal Myxedema Eff. Subcostal views of large circumferential effusion *(Eff)* in a patient with myxedema. The image on the right demonstrates collapse of the inferior vena cava *(IVC)*, suggesting a chronic effusion. The heart rate and blood pressure were normal.

Video 8.63. Eff/Tap. Off-axis apical views of patient sitting upright in the catheter laboratory during pericardiocentesis of malignant effusion. The *arrow* points to the needle in pericardial space. *LV,* Left ventricle; *MV,* mitral valve.

Video 8.64. Eff/Tap Saline Solution. Injection of agitated saline solution *(blue arrow)* in the same patient as Video 8.63 during pericardiocentesis confirms correct location of the needle *(white arrow)* in the pericardial space.

Video 8.65. SAX Base Hematoma. PSAX view at the level of the aortic valve *(Ao)* demonstrates large mediastinal (not pericardial) hematoma *(H)* after blunt injury to the chest. In this view it can be difficult to distinguish a pericardial from a mediastinal process.

Video 8.66. AP4CH Hematoma. Apical four-chamber view of same patient as Video 8.65 demonstrates hematoma *(H)* in pleural space and trivial pericardial effusion (indicated by the *asterisk*). *LV,* Left ventricle.

Video 8.67. Subcostal Hematoma. Subcostal view of same patient as Video 8.65 demonstrating no pericardial effusion. This was a very useful view in this patient. *LV,* Left ventricle; *RV,* right ventricle.

Video 8.68. AP4CH RA Coll. Apical four-chamber view demonstrating right atrial *(RA)* collapse but no significant pericardial fluid. *Dao,* Descending aorta; *RV,* right ventricle.

Video 8.69. AP4CH RA/IVC Coll. Subcostal view in same patient as Video 8.68 *(right image)* demonstrating complete collapse of the inferior vena cava *(IVC)*, consistent with hypovolemia.

Video 8.70. PLAX Fat Pad. PLAX of the left ventricle *(LV)*. The arrow points to an epicardial fat pad, demonstrating "linear streaking," suggesting tissue planes c/w fat, in the typical anterior plane over the right ventricle *(RV)*. *RA,* Right atrium.

Video 8.71. Subcostal Fat Pad. Subcostal view of the same patient as Video 8.70, demonstrating epicardial fat *(arrow),* which moves with the epicardial surface and demonstrates tissue planes. *LV,* Left ventricle; *RA,* right atrium; *RV,* right ventricle.

Diseases of the Aorta

Video 8.72. PLAX Diss. PLAX of markedly dilated proximal aorta demonstrating dissection flap *(arrow).* It is less common to identify dissections with transthoracic imaging. *AoV,* Aortic valve; *LA,* left atrium; *LV,* left ventricle.

Video 8.73. AP4CH Diss. Apical four-chamber view in same patient as Video 8.72 demonstrates marked dilatation of proximal ascending aorta and dissection flap *(arrow). AoV,* Aortic valve.

Video 8.74. AP4CH Diss/Color. Apical four-chamber view in same patient as Video 8.73 demonstrates proximal dissection flap *(arrow)* and severe aortic regurgitation.

Video 8.75. AP4CH/Myxoma. Typical left atrial myxoma *(M)* seen from apical four-chamber view, prolapsing through the mitral valve in diastole. *LA,* Left atrium; *LV,* left ventricle.

Video 8.76. TEE Ao Debris. Gr5 mixed atheromatous debris *(arrow)* in aorta as seen on TEE.

Video 8.77. TEE Ao Debris 2. TEE image of proximal descending aorta (Prox Desc Ao) demonstrating Gr5 atheromatous debris *(arrow).*

Chapter 14

Video 14.1. Cook Medical Ciaglia Blue Rhino (Bloomington, IN). Percutaneous tracheostomy introducer. Technique of percutaneous dilational tracheostomy. (Courtesy Cook Medical.)

Chapter 18

Video 18.1. Inferior vena cava longitudinal view.

Video 18.2. Lung sliding.

Video 18.3. A-lines.

Video 18.4. B-lines.

Video 18.5. Pleural effusion.

Video 18.6. Atelectasis.

Video 18.7. Deep vein thrombosus in the internal jugular vein.

Video 18.8. Examination for a lower extremity deep vein thrombosus.

Chapter 38

Video 38.1. Auto–positive end-expiratory pressure (auto-PEEP).

1

Cardiac Arrest and Cardiopulmonary Resuscitation

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CHAPTER OUTLINE

Epidemiology and General Principles

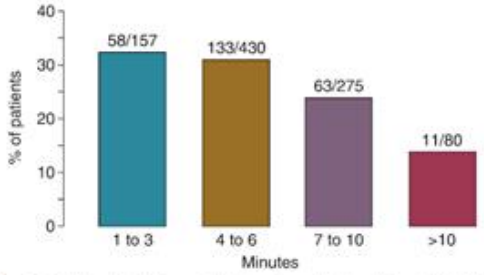
Sudden cardiac arrest is defined as the cessation of effective cardiac mechanical activity as confirmed by the absence of signs of circulation. Sudden cardiac arrest is the most common fatal manifestation of cardiovascular disease and a leading cause of death worldwide. The exact incidence of sudden cardiac arrest is unclear, but in the United States alone, it has been estimated to be as high as 450,000 persons annually.[1] Approximately 21% to 25% of sudden cardiac arrest events are due to pulseless ventricular arrhythmias (i.e., ventricular fibrillation [VF] or pulseless ventricular tachycardia [VT]), whereas the rest can be attributed to other cardiac rhythms (i.e., asystole or pulseless electrical activity [PEA]).[2,3] Patients who suffer cardiac arrest due to VF or VT have a much higher chance of surviving the event compared with patients who present with PEA/asystole.[4] The prognosis is better in patients with ventricular arrhythmias because (1) ventricular arrhythmias are potentially treatable with defibrillation (i.e., "shockable" initial rhythm) to restore circulation, whereas the other initial rhythms are not, and (2) ventricular arrhythmias are typically a manifestation of a cardiac etiology of cardiac arrest (e.g., acute myocardial infarction), whereas the other initial rhythms are more likely to be related to a noncardiac etiology and perhaps an underlying condition that is less treatable. Clinical outcomes for cardiac arrest are poor. Approximately 11% of out-of-hospital cardiac arrest (OHCA)[5] and 20% of in-hospital cardiac arrest (IHCA) patients survive to hospital discharge.[6]

The basic principles of resuscitation are an integral part of training for many health care providers (HCPs). Because timely interventions for cardiac arrest victims have the potential to be truly lifesaving, it is especially important for critical care practitioners to have a sound understanding of the evaluation and management of cardiac arrest. A number of critical actions (*chain of survival*) must occur in response to a cardiac arrest event. The chain of survival paradigm (Fig. 1.1) for the treatment of cardiac arrest remained unchanged in the 2015 American Heart Association (AHA) Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care from 2010 and is similar to other cardiac arrest guidelines. The chain of survival paradigm consists of five separate and distinct elements: immediate recognition that cardiac arrest has occurred and activation of the emergency response system; application of effective cardiopulmonary resuscitation (CPR); early defibrillation (if applicable); advanced cardiac life support; and initiation of postresuscitation care (e.g., targeted temperature management).[7–9] When the chain of survival is implemented effectively survival for VF, OHCA can exceed 45%.[3]

Cardiopulmonary Resuscitation

For CPR to be effective in restoring spontaneous circulation, it must be applied immediately at the time of cardiac arrest. Therefore immediate recognition that a cardiac arrest has occurred and activation of the emergency response system are essential. Patients become unresponsive at the time of cardiac arrest. Agonal gasps may be observed in the early moments after a cardiac arrest event, although normal breathing ceases. Pulse checks (i.e., palpation of femoral or carotid arteries for detection of a pulse) are often

• **Fig. 1.1** The American Heart Association chain of survival paradigm.[89] This figure represents the critical actions needed to optimize the chances of survival from cardiac arrest. The links (from left to right) include (1) immediate recognition of cardiac arrest and activation of the emergency response system; (2) early and effective cardiopulmonary resuscitation; (3) defibrillation (if applicable); (4) advanced cardiac life support; and (5) post–cardiac arrest care (including target temperature management if appropriate). (From Travers AH, Rea TD, Bobrow BJ, et al. Part 4: CPR overview: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2010;122(18 suppl 3):S676–S684.)



• **Fig. 1.2** Relationship between the time interval before attempted defibrillation and the proportion of patients discharged from the hospital alive after out-of-hospital cardiac arrest. (Modified from Weaver WD, Cobb LA, Hallstrom AP, et al. Factors influencing survival after out-of-hospital cardiac arrest. *J Am Coll Cardiol*. 1986;7:752–757.)

unreliable, even when performed by experienced HCPs.[10] Because delays in initiating CPR are associated with worse outcome, and prolonged attempts to detect a pulse may result in a delay in initiating CPR, prolonged pulse checks are to be avoided. CPR should be started immediately if the patient is unresponsive and either has agonal gasps or is not breathing.[7]

Chest Compressions

In CPR, chest compressions are used to circulate blood to the heart and brain until a pulse can be restored. High-quality chest compressions are vital for successful resuscitation. The mechanism by which chest compressions generate cardiac output is through an increase in intrathoracic pressure plus direct compression of the heart. As in 2010, the 2015 AHA guidelines recommend HCPs initiate the CPR sequence with chest compressions instead of rescue breaths (i.e., compressions-airway-breathing [C-A-B] vs. airway-breathing-compressions [A-B-C]) to minimize the time to chest compressions.[7] With the patient lying in the supine position, the rescuer applies compressions to the lower half of the patient's sternum. The heel of one hand is placed over the lower half of the sternum and the heel of the other hand on top in an overlapping and parallel fashion. In addition to manual CPR, there has been an increase in the use of mechanical CPR devices. Studies have not demonstrated a difference in patient outcomes between manual and mechanical CPR.[8,11]

The recommended compression depth in adults is at least 2 inches (5 cm) while avoiding chest compression depths greater than 2.4 inches (6 cm).[7–9] The recommended rate of compression per the 2010 AHA guidelines was *at least* 100 compressions per minute.[12] The 2015 AHA guidelines now recommend a rate of 100 to 120 compressions per minute.[7] These new AHA guidelines are in line with the 2010 and 2015 European Resuscitation Council guidelines for resuscitation, as well as the 2015 International Liaison Committee on Resuscitation recommendations.[8,9,13] In addition, incomplete recoil of the chest impairs the cardiac output that is generated. Incomplete recoil occurs when HCPs lean on the chest wall between compressions; thus HCPs should avoid leaning to allow the chest wall to recoil completely between compressions. Owing to rescuer fatigue, the quality of chest compressions predictably decreases as the time providing chest compressions increases, and the persons providing chest compressions (even experienced HCPs) may not perceive fatigue or a decrease in the quality of their compressions.[14] Therefore it is recommended that rescuers performing chest compressions rotate every 2 minutes.[7,9]

The quality of CPR is a critical determinant of surviving a cardiac arrest event.[15] Minimization of interruptions in chest compressions is imperative. Interruptions in chest compressions during CPR have been quite common historically, and the "hands off" time has been shown to comprise a substantial amount of the total resuscitation time.[15] The proportion of time chest compressions are performed during a cardiac arrest has been termed chest compression fraction. A higher chest compression fraction has been associated with increased survival in VF cardiac arrest,[16] and increased return of spontaneous circulation (ROSC) in non-VF cardiac arrest.[17] Guidelines recommend a chest compression fraction as high as possible, with a goal of at least 60%.[7,9] A chest compression fraction of 80% is likely ideal and achievable in many settings.[18] Potential reasons for decreased chest compression fraction include pulse checks, rhythm analysis, switching compressors, procedures (e.g., airway placement), and pauses before defibrillation ("preshock pause"). All of these potential reasons for interruptions should be minimized. Pauses related to rotating compressors, pulse checks, and delivering rescue breaths should take no longer than 10 seconds.[7,9] Eliminating (or minimizing) preshock pauses has been associated with a higher likelihood of ROSC and improved clinical outcome.[19,20]

Defibrillation

The next critically important action in the resuscitation of patients with cardiac arrest caused by pulseless ventricular arrhythmias (i.e., VF or pulseless VT) is rapid defibrillation. Delays in defibrillation are clearly deleterious, with a sharp decrease in survival as the time to defibrillation increases.[21] With the advent of automatic external defibrillators and their dissemination into public places, both elements of effective CPR (effective chest compressions and rapid defibrillation) can be performed by lay rescuers in the field for patients with OHCA. Fig. 1.2 shows the importance of rapid defibrillation, with decreasing success of resuscitation with increasing time to defibrillation. Thus defibrillation should be performed as soon as possible. The amount of energy for defibrillation should be based on the specific manufacturer's guidance. To increase the chest compression fraction, chest compressions should resume immediately after defibrillation for adult cardiac arrest in any setting.[7,9]

Rescue Breathing

Recommendations regarding ventilation during CPR depend on the rescuer (i.e., trained HCPs vs. layperson).[7,9] For trained HCPs, the recommended ventilation strategy is a cycle of 30 chest compressions to 2 breaths until an endotracheal tube is placed, and then continuous chest compressions with 1 breath every 6 seconds after the endotracheal tube is placed. Excessive ventilations can be deleterious from a hemodynamic perspective owing to increased intrathoracic pressure and reduction in the cardiac output generated by CPR and thus should be avoided during resuscitation.[22] Excessive ventilation could also potentially result in alkalemia. For OHCA is it reasonable for emergency medicine service providers to delay positive-pressure ventilation while providing up to 3 cycles of 200 continuous chest compressions with passive oxygenation.[7]

For laypersons who are attempting CPR in the field for a victim of OHCA, rescue breathing is no longer recommended. Rather, the recommended strategy is compression-only (or hands-only) CPR.[7,9] The rationale is that compression-only CPR can increase the number of effective chest compressions that are delivered to the patient (i.e., minimizes interruptions for rescue breaths), and does not require mouth-to-mouth contact. Mouth-to-mouth contact is one of the perceived barriers to CPR in the field. By removing this element, the hope is that an increase in attempts at bystander CPR will result. Hands-only CPR has been found to be noninferior to conventional CPR including rescue breaths for victims of OHCA[23–25]; thus hands-only CPR has become the preferred technique to teach lay rescuers. Fig. 1.3 displays the AHA algorithm for adult basic life support.

Advanced Cardiac Life Support

There are several additional elements of resuscitation that are intended specifically for trained HCPs (e.g., advanced cardiac life support [ACLS]), and specifically this includes pharmacologic therapy. Fig. 1.4 displays the AHA algorithm for ACLS.[26]

It is notable that the impact of recommended ACLS therapies on outcome from cardiac arrest remains a matter of debate. Some studies have shown that ACLS interventions did not improve clinical outcomes when compared to basic life support alone.[27]

Vasopressors

The primary goal of pharmacologic interventions is to assist the achievement and maintenance of spontaneous circulation. The mainstay of pharmacologic interventions is vasopressor drugs. It is reasonable to administer epinephrine (1 mg) by intravenous (IV) or intraosseous (IO) route every 3 to 5 minutes during CPR until ROSC is achieved.[8,26,28] If IV/IO access cannot be established, epinephrine could be administered via an endotracheal tube, but at a higher dose (2–2.5 mg). The optimal timing of epinephrine administration is currently unclear; however, observational studies have demonstrated that early administration of epinephrine is associated with improved outcomes in nonshockable rhythms.[29,30]

Vasopressin as a substitute for epinephrine was removed from the 2015 AHA guidelines. One study found the use of intracardiac arrest vasopressin (20 IU/CPR cycle), epinephrine (1 mg/CPR cycle), and methylprednisolone (40 mg during first CPR cycle) followed by postcardiac arrest hydrocortisone (300 mg daily for 7 days maximum and gradual taper) increased the probability of successful ROSC and discharge with good neurologic outcome[31]; however, the evidence for this practice is not currently strong enough to recommend its routine use.[26]

Antiarrhythmic Drugs

Amiodarone is the preferred antiarrhythmic agent for refractory VF/VT cardiac arrest.[8] Refractory VF/VT refers to VF or pulseless VT that persists or recurs after one or more defibrillation attempts. It is unlikely that antiarrhythmic drug therapy itself will convert refractory VT/VF; rather, the goal of antiarrhythmic drug therapy is to aid in reestablishing and maintaining an organized perfusing rhythm in conjunction with subsequent defibrillation. The recommended dose of amiodarone is 300 mg IV/IO for the first dose, 150 mg IV/IO for the second dose.[26,28] Of note, antiarrhythmic drug therapy has not been demonstrated to improve survival or neurologic outcomes after VF/VT cardiac arrest.

Other Drug Therapies

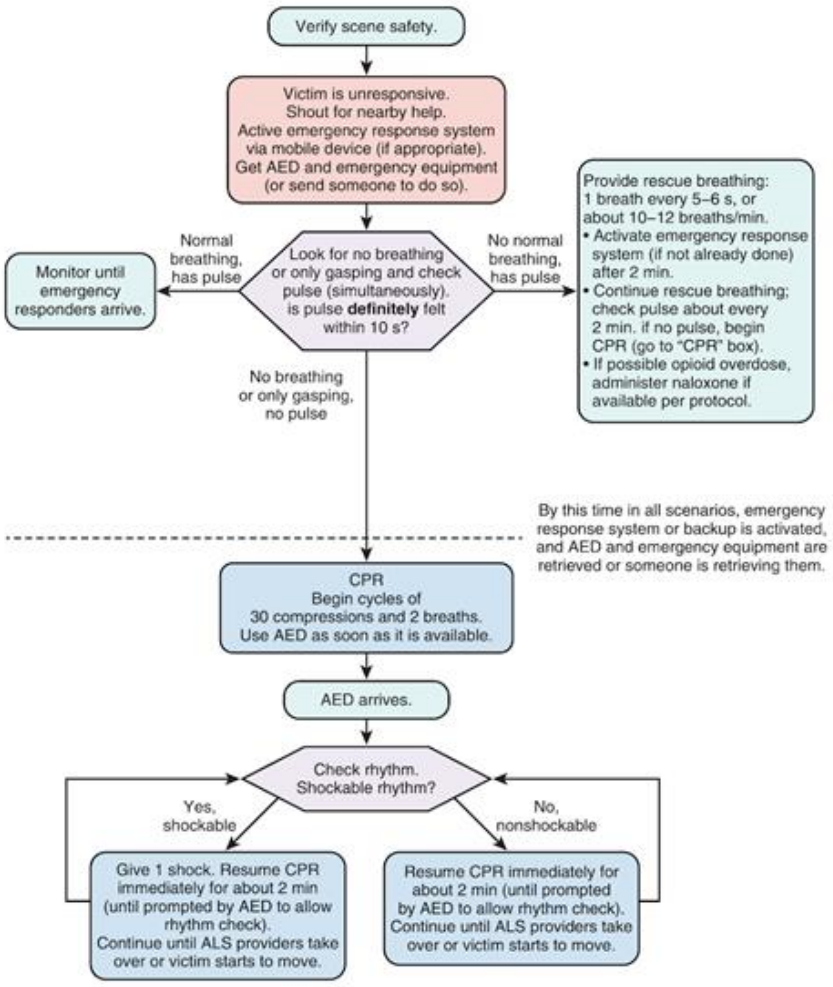
The use of atropine for PEA/asystole was removed from the ACLS guidelines in 2010. Along these lines, there is also insufficient evidence to recommend routine administration of sodium bicarbonate or calcium during CPR.[28,32] In the setting of confirmed or suspected pulmonary embolism, limited evidence suggests that systemic thrombolysis administration during ongoing CPR is associated with ROSC and short-term survival,[33] with earlier administration associated with greater benefit.[34] However, in the setting of presumed cardiac etiology of cardiac arrest there is currently no evidence to support clinical benefit.[35] The decision to administer systemic thrombolytics during cardiac arrest may be considered in cases with a strong suspicion for pulmonary embolism, or cardiac etiology without immediate access to percutaneous coronary intervention.

End-Tidal Carbon Dioxide

End-tidal carbon dioxide ($ETCO_2$) may reflect cardiac output and pulmonary blood flow. $ETCO_2$ can be measured during CPR to monitor the quality of chest compressions in intubated patients. An $ETCO_2$ less than 10 mm Hg after 20 minutes of high-quality CPR has been demonstrated to be predictive of mortality,[36,37] whereas an $ETCO_2$ greater than 20 mm Hg was associated with survival to hospital discharge.[36] Although predictive of outcome, a low $ETCO_2$ should not be used alone to discontinue CPR efforts. It is also important to realize a low $ETCO_2$ could be a result of bronchospasm, plugging or kinking of the endotracheal tube, hyperventilation, or an air leak in the airway.[26] A sudden, sustained increase in $ETCO_2$ (i.e., >40 mm Hg) during chest compressions can be an indication of ROSC.[26,28,38] $ETCO_2$ monitoring in nonintubated patients may not accurately reflect the true $ETCO_2$ level and should not be used as an adjunct prognostication tool.

Extracorporeal CPR

Extracorporeal CPR (ECPR) refers to venoarterial extracorporeal membrane oxygenation and cardiopulmonary bypass during cardiac arrest. In theory ECPR could be used as a bridge to allow physicians time to treat reversible causes of cardiac arrest. To date there has not been a randomized control trial comparing ECPR to conventional CPR for prolonged resuscitations. The limited case reports and observational studies do not provide sufficient evidence to recommend the routine use of ECPR; however, it may be reasonable

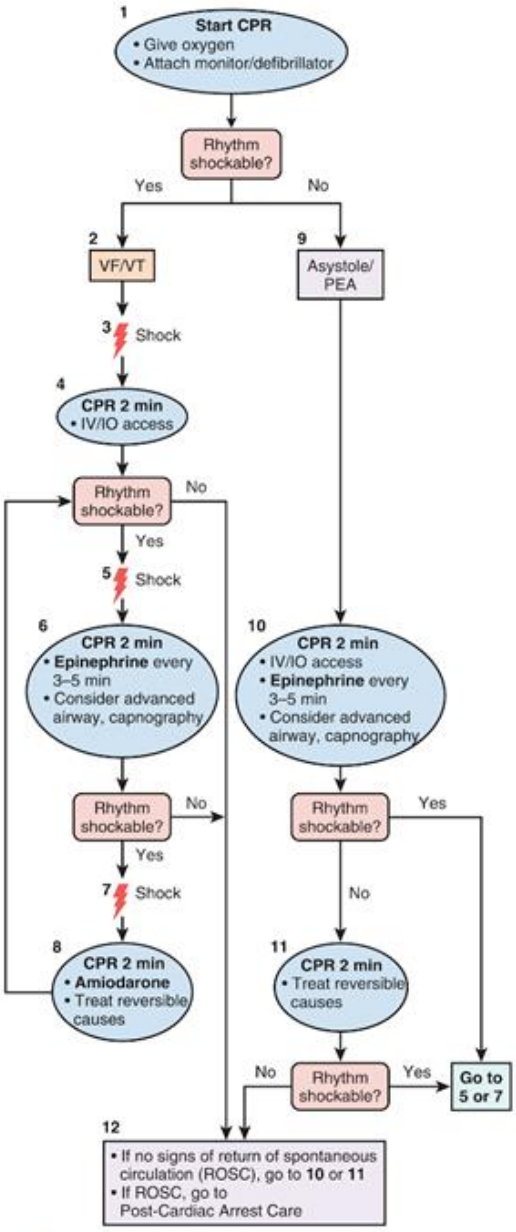


• **Fig. 1.3** American Heart Association adult Basic Life Support *(BLS)* algorithm. *AED*, Automated external defibrillator; *ALS*, advanced life support; *CPR*, cardiopulmonary resuscitation. (From Kleinman ME, Brennan EE, Goldberger ZD, et al. Part 5: adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 suppl 2):S414–S435.)

to consider ECPR for specific patients with a potentially reversible etiology of cardiac arrest.[26,28]

Reversible Causes

When managing a cardiac arrest it is important to consider the potential etiologies of the arrest. In many situations it is not possible to maintain an organized perfusing rhythm until the underlying cause of the cardiac arrest is reversed. A frequently used pneumonic for common potential reversible causes involves the "Hs and Ts."[28] The "Hs" include hypoxia, hypovolemia (including hemorrhage), hyperkalemia, hypokalemia, hypoglycemia, hydrogen ions (i.e., metabolic acidosis), and hypothermia. The "Ts" include thrombosis (i.e., coronary occlusion and pulmonary embolism), tension pneumothorax, cardiac tamponade, and ingestion of therapeutic or toxic substances. Once the etiology of the cardiac arrest is identified (or strongly presumed), treatment should be aimed at reversing the specific cause.



CPR Quality
- Push hard (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 min 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 (e.g., 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 min
- **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 s (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. 1.4** American Heart Association Advanced Cardiac Life Support (ACLS) algorithm. *CPR*, Cardiopulmonary resuscitation; *ET*, endotracheal; *IO*, intraosseous; *IV*, intravenous; *PEA*, pulseless electrical activity; P_{ETCO_2}, partial pressure of end tidal carbon dioxide; *pVT*, pulseless ventricular tachycardia; *ROSC*, return of spontaneous circulation; *VF*, ventricular fibrillation; *VT*, ventricular tachycardia. (From Link MS, Berkow LC, Kudenchuk PJ, et al. Part 7: adult advanced cardiovascular life support: 2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation*. 2015;132(18 suppl 2):S444–S464.)

Postresuscitation Care

Even if ROSC is achieved with CPR and defibrillation, cardiac arrest victims are at extremely high risk of dying in the hospital, and many who survive sustain permanent crippling neurological sequelae. Approximately 50% to 60% of patients successfully resuscitated from OHCA do not survive. After ROSC global ischemia/reperfusion (I/R) injury results in potentially devastating neurologic disability. The primary cause of death among postresuscitation patients is brain injury. However, clinical trials have shown that targeted temperature management (TTM) after ROSC can improve outcomes. These landmark clinical trials have dramatically transformed the classical thinking about anoxic brain injury after cardiac arrest; this condition is, in fact, *treatable*. Early therapeutic interventions such as TTM initiated in the post-ROSC period can improve the trajectory of the long-term disease course. Accordingly, the postresuscitation care is now considered to be a crucial fifth link in the chain of survival paradigm (see Fig. 1.1).[7–9]

General Approach

Post–cardiac arrest syndrome is a heterogeneous disease process with varying precipitating factors and underlying etiologies. As such, post–cardiac arrest management must be tailored on a case-by-case basis for each specific patient. For patients resuscitated from cardiac arrest, admission to a critical care unit with the following capabilities should be considered[39]:

- Critical care support to optimize cardiovascular indices and vital organ perfusion, and prevent repeat cardiac arrest (or provide rapid treatment of rearrest if it occurs)
- Interventional cardiac catheterization for possible percutaneous coronary intervention (PCI) if needed
- TTM for at least 24 hours in attempts to prevent permanent neurologic injury
- Systematic application of an evidence-based approach to neurologic prognostication to refrain from inappropriately early final determinations of poor neurologic prognosis (i.e., to prevent inappropriately early withdrawal of life support before the neurologic outcome can be known with certainty).

Hemodynamic Support

I/R triggers profound systemic inflammation. In clinical studies, ROSC has been associated with sharp increases in circulating cytokines and other markers of the inflammatory response. Accordingly, some investigators have referred to the post–cardiac arrest syndrome as a "sepsis-like" state. The clinical manifestations of the systemic inflammatory response may include marked hemodynamic derangements such as sustained arterial hypotension similar to septic shock. Hemodynamic instability occurs in approximately 50% of patients who survive to intensive care unit admission after ROSC, and thus the need for aggressive hemodynamic support (e.g., continuous infusion of vasoactive agents and perhaps advanced hemodynamic monitoring) should be anticipated.[40]

In addition to a systemic inflammatory response, an equally important contributor to post-ROSC hemodynamic instability is myocardial stunning. Severe, but potentially reversible, global myocardial dysfunction is common after ROSC. The etiology is thought to be I/R injury, but treatment with defibrillation (if applied) could also contribute. Although the myocardial dysfunction occurs in the absence of an acute coronary event, myocardial ischemia may be an ongoing component of myocardial depression if an acute coronary syndrome caused the cardiac arrest. Severe myocardial stunning may last for hours, but it often improves by the 24-hour mark after ROSC. An echocardiogram may be helpful in hemodynamic assessment after ROSC to determine if global myocardial depression is present, as this may affect decisions on vasoactive drug support (e.g., dobutamine) or mechanical augmentation (e.g., intraaortic balloon counterpulsation) until the myocardial function recovers. However, when needed, clinicians should be aware that β-adrenergic agents may increase the likelihood of dysrhythmia.

Observational studies have demonstrated postresuscitation arterial hypotension to be associated with sharply lower survival,[40,41] and a post-ROSC mean arterial blood pressure greater than 70 mm Hg to be associated with improved neurologic outcome at hospital discharge.[42] However, it is currently unclear whether a specific blood pressure target or other hemodynamic goals are beneficial.[43] Expert opinion (and clinical intuition) suggests that hemodynamics and organ perfusion should be optimized, and rapidly reversing and preventing hypotensive after ROSC is recommended.[44,45] Whether or not postresuscitation hypotension has a cause-and-effect relationship with worse neurologic injury or is simply a marker of the severity of the I/R injury that has occurred remains unclear.

Oxygenation and Ventilation

Given the ongoing I/R injury during the early period after ROSC, hypoxia should be avoided in all post–cardiac arrest patients. Exposure to hyperoxia (excessively high partial pressure of arterial oxygen [PaO_2]) has also been associated with poor clinical outcome among adult patients resuscitated from cardiac arrest and admitted to an intensive care unit.[46] These data corroborate the findings of numerous laboratory studies in animal models in which hyperoxia exposure after ROSC worsens brain histopathologic changes and neurologic function.[47–51] A paradox may exist regarding oxygen delivery to the injured brain, where inadequate oxygen delivery can exacerbate cerebral I/R injury, but excessive oxygen delivery can accelerate formation of oxygen free radicals and subsequent reperfusion injury. Although the results of observational studies of hyperoxia are mixed[52,53] and no interventional studies have been performed, expert opinion advocates initially using the highest available oxygen concentration after ROSC to prevent hypoxia, and once the arterial oxygen saturation or PaO_2 can be measured, the fraction of inspired oxygen can be titrated down as much as possible while maintaining an arterial oxygen saturation of at least 94% to limit unnecessary exposure to an excessively high postresuscitation PaO_2.[45,54]

Post-ROSC partial pressure of arterial carbon dioxide ($PaCO_2$) levels have been demonstrated to be associated with clinical outcomes. Specifically, hypocapnia is associated with worse neurologic outcome.[55–57] It is possible that hypocapnia induces cerebral vasoconstriction and decreased cerebral blood flow, resulting in worsening cerebral ischemia. The effects of hypercapnia on clinical outcomes are currently unclear.[58] Current recommendations advocate maintaining normocapnia (i.e., $PaCO_2$ 35–45 mm Hg) after cardiac arrest.[54]

Seizures

Seizures are not uncommon after anoxic brain injury. Routine seizure prophylaxis is not currently recommended in post–cardiac arrest syndrome. However, it is important to be vigilant in clinical assessment for any motor responses that could represent seizure