Chapter 24: Cardiac Resuscitation

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Content Update

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Cardiac resuscitation is a rapidly changing clinical science. Recommendations of the November 2015 AHA-ACLS updates[1] are provided in this chapter and are augmented by the most current resuscitation research literature. To summarize:

CHEST COMPRESSIONS

The recommended chest compression rate is 100-120 per minute, updated from 100 per minute.

Chest compression depth should be 5-6 cm, not more than 6 cm. Chest compression ratio should be > 60 and as close to 90 as possible.

There does not appear to be an advantage to asynchronous or interpolated breaths as long as the highest possible compression ratio is maintained.

The compression to breath ratio is 30:2. If adjunctive or definitive airway is in place, provide 10 breaths per minute.

DEFIBRILLATION

Early defibrillation is the most effective modality for return of spontaneous circulation (ROSC).

DRUGS

Amiodarone or lignocaine (lidocaine) may not provide added benefit to defibrillation.

Vasopressin has been removed from the ACLS algorithm.

Routine use of beta blockers after cardiac arrest is not recommended, with benefits for ROSC only demonstrated through animal studies and case reports.

Steroids may provide some benefit when bundled with vasopressin and epinephrine in in-hospital cardiac arrest, though routine use is not recommended.

OXYGENATION AND CAPNOGRAPHY

Provide 100% FiO_2 during cardiac arrest, with oxygen saturation titrated to greater than 94% post ROSC.

End tidal CO_2 can be used to monitor for ROSC. Low end tidal CO_2 (less than 10 mm Hg) after 20 minutes is associated with low likelihood of survival.

ECMO

ECMO or ECPR can be considered in patients with refractory cardiac arrest who have not responded to conventional CPR, where it can be rapidly implemented with suspicion of reversible cause of cardiac arrest.

POST ROSC CARE

Emergency percutaneous coronary intervention is recommended for patients with ST elevation on ECG and for hemodynamically or electrically unstable patients with no ST elevation but suspected cardiovascular cause.

Targeted temperature management between 32-36^{o}C for comatose patients with ROSC for at least 24 hours is recommended.

SOCIAL MEDIA AND EDUCATION

Use of social media technologies that summon rescuers in close proximity to a victim of out-of-hospital cardiac arrest may be reasonable. Audiovisual devices can be used to educate providers and improve CPR quality.

INTRODUCTION AND EPIDEMIOLOGY

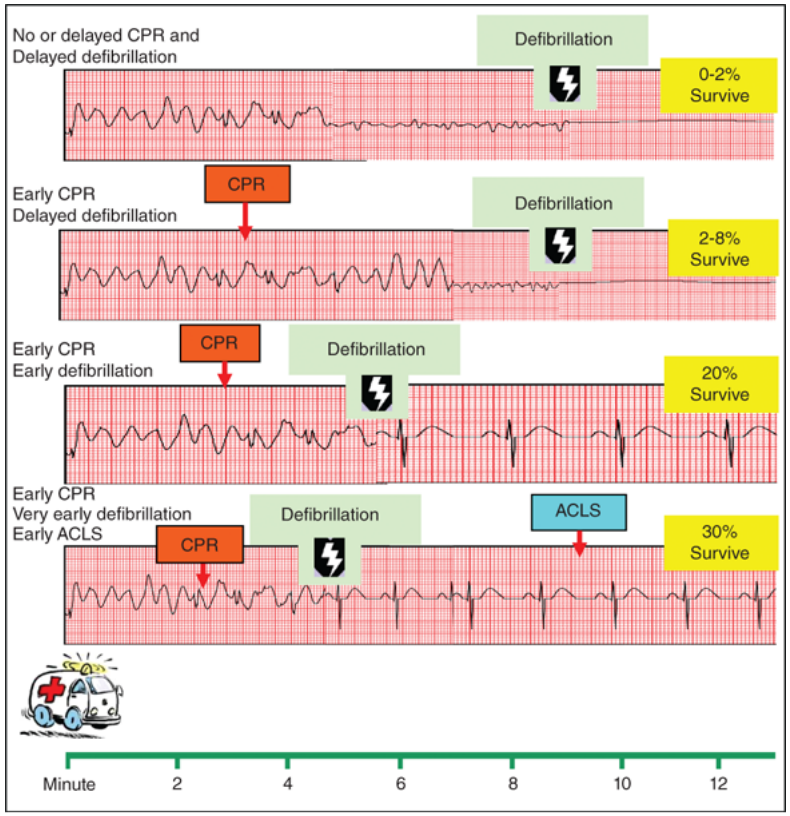
Every year, approximately 6.8 to 8.5 million persons throughout the world[2] sustain cardiac arrest. About 70% of cardiac arrests occur out of hospital. The proportion of cardiac arrest patients who are treated varies from about 54.6% (United States) to about 28.3% (Asia). The proportions with ventricular fibrillation (VF) and survival vary from 11% and 2%, respectively, in Asia, to 28% and 6% in North America, 35% and 9% in Europe, and 40% and 11% in Australia.[3] About half of cardiac arrest victims are <65 years old.

Ventricular tachyarrhythmias are the initiating event in about 80% of patients with out-of-hospital primary cardiac arrest. During ambulatory electrocardiogram (ECG) monitoring of 157 witnessed cardiac arrests, Bayés de Luna et al[4] documented 70% with ventricular tachycardia (VT) and VF, 13% with torsades de pointes, and 17% with bradyarrhythmias. Untreated VF deteriorates to asystole in about 15 minutes.[5] For patients with sudden cardiac arrest, the rate of survival declines rapidly by about 7% to 10% for each minute without defibrillation.[6] If delay to defibrillation exceeds 12 minutes,[7] survival is of the order of 0% to 5%.

THE CHAIN OF SURVIVAL

The structured emergency care system concept for treatment of cardiac arrest is called the **Chain of Survival and includes five components: recognition and activation of the emergency response system, immediate high-quality CPR, rapid defibrillation, basic and advanced emergency medical services, and advanced life support and post arrest care for out-of-hospital cardiac arrest (OHCA).** If a community's prehospital EMS can be activated promptly, reach the patient within 5 minutes of collapse, and deliver the first shock shortly thereafter, survival in excess of 15% to 20% can be expected, with recent reports of >30% survival.[8,9] With delayed initiation of CPR, defibrillation, and access to the patient by the emergency services, the impact of advanced life support measures is small (Figure 24–1). Improved survival can only occur if structured emergency care systems allow trained providers to access the patient rapidly and deliver the appropriate treatment in a timely fashion. Delays in initiating the various links weaken the chain and adversely affect the next link, resulting in a decreased chance of a good outcome for the patient.

FIGURE 24–1.
Incremental survival benefits by the links in the Chain of Survival. ACLS, advanced cardiac life support. [Reproduced, with permission, from the National Resuscitation Council, Singapore.]

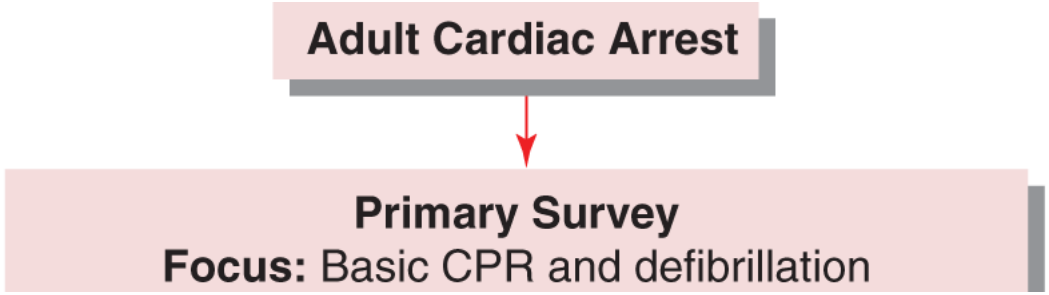


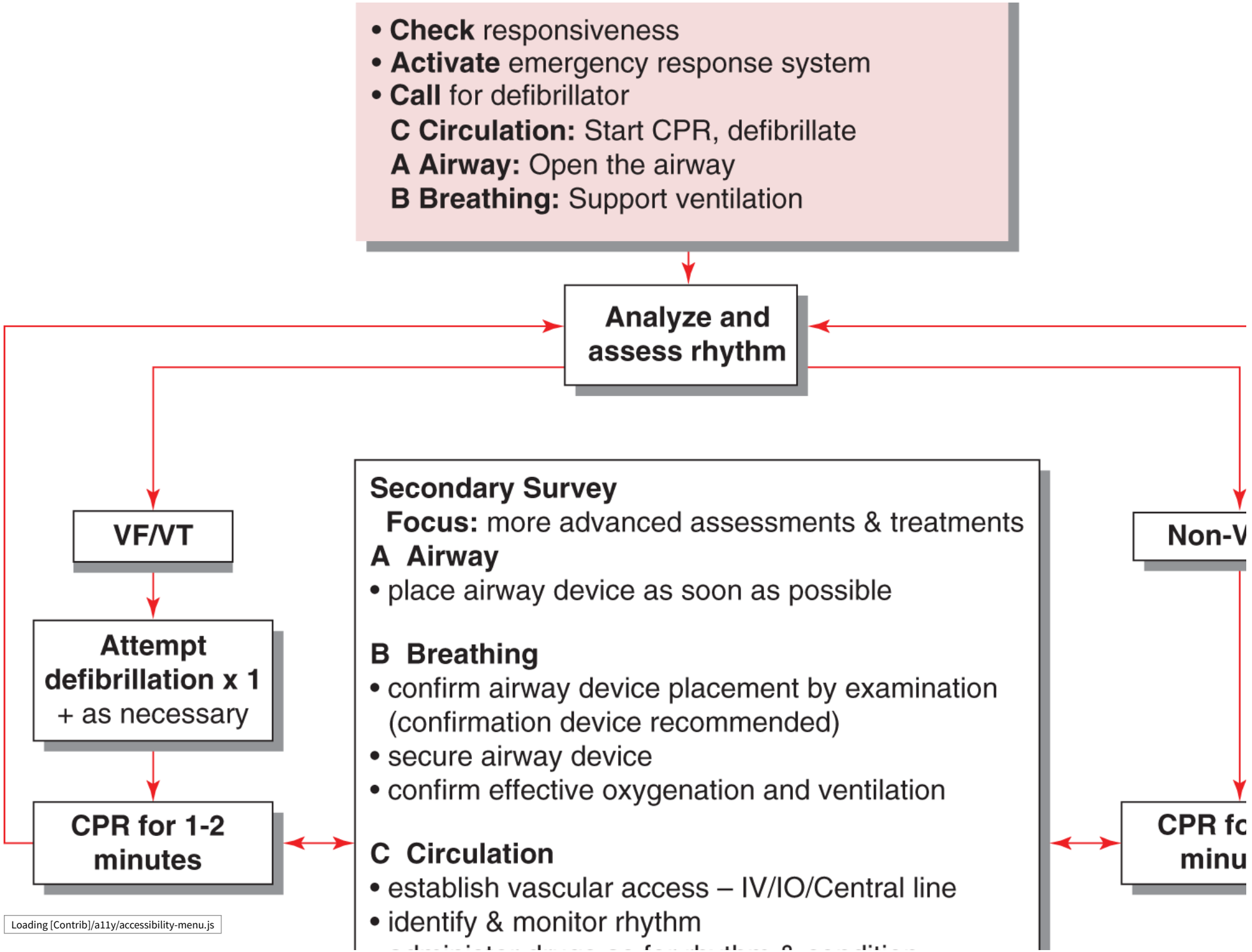
ADVANCED CARDIAC LIFE SUPPORT (ACLS)

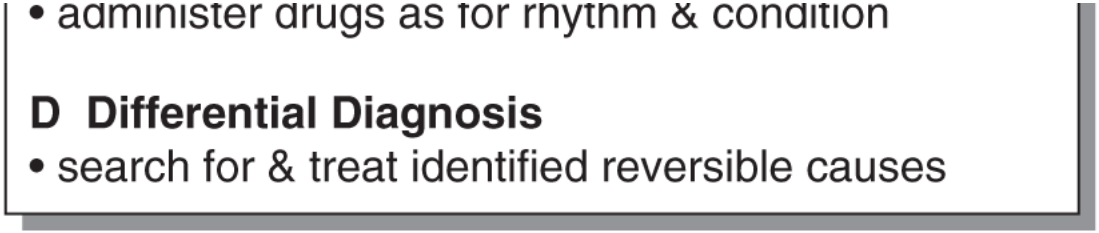
The basic life support assessments and interventions are often called the **Primary Survey**, and the advanced life support assessments and interventions are often called the **Secondary Survey**.

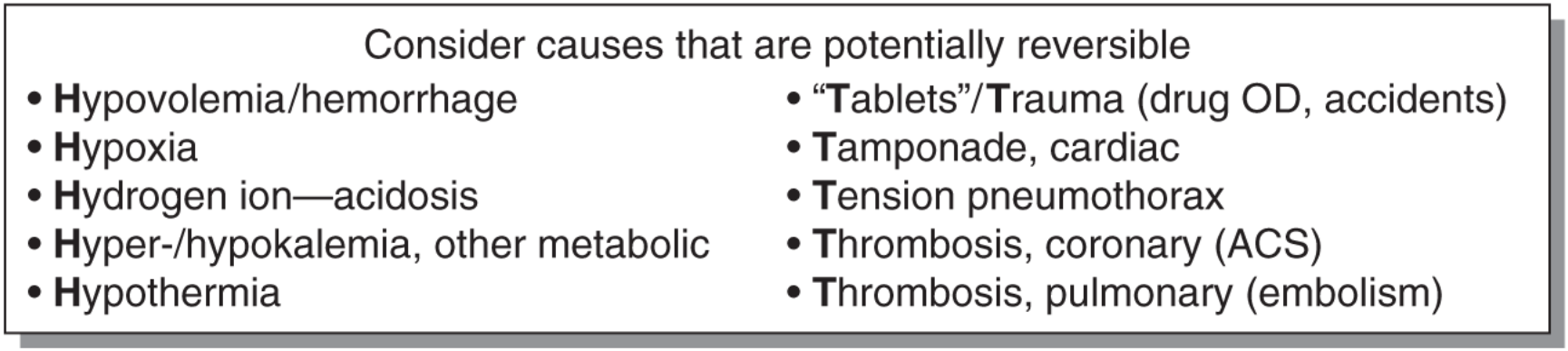
An organized approach to resuscitation begins with the Primary Survey and blends smoothly with the Secondary Survey. These are summarized in the **Universal ACLS Algorithm** (Figure 24–2). This method helps any ACLS provider remember the sequence of resuscitation actions and, therefore, be less likely to miss any of the vital steps in the care of the patient.

Figure 24–2.
Universal ACLS Algorithm. ACS, acute coronary syndrome; OD, overdose.









PRIMARY SURVEY

The **Primary Survey** addresses the identification of cardiac arrest and performance of good-quality CPR (including ventilation) and defibrillation. The procedures for the primary survey are described in chapters 22 and 23. The 2015 ACLS guidelines emphasize chest compressions, starting with Circulation-Airway-Breathing (CAB) as opposed to Airway-Breathing-Circulation (ABC). Maximize chest compression fraction to >60% with minimal pauses in compressions.

Defibrillate as soon as possible for ventricular fibrillation or pulseless ventricular tachycardia. Start at 200 joules biphasic, or at maximum energy. Make sure CPR interruptions are as brief as possible when defibrillating. Maintain continuous compressions during rhythm analysis and defibrillator recharging. Resume one cycle of CPR after defibrillation even with ROSC.

For ventilation, bag-valve masks deliver 21% oxygen if using room air, 60% oxygen when connected to an oxygen source with flow at 12 L/min, and 90% to 95% with a reservoir bag. Chest compressions need not be stopped to initiate or continue ventilation.

During cardiac arrest, 100% FiO_2 during compressions is recommended due to unreliable measurement of O_2 saturation. The time required to perform endotracheal intubation may decrease compression fraction.[10] Any airway device may be used during CPR.

The 2015 updates recommend 30:2 compression to breath ratio, maximizing compression time, with no pauses longer than 10 seconds.[11,1] The target compression rate is now 100-120, rather than at least 100. The maximum compression rate is 120, as quality decreases with rates greater than this. The maximum depth of compression is 6 cm, with target of 5-6 cm, rather than at least 5 cm in the 2010 guidelines. Audiovisual devices may be used to optimize CPR quality.

For one and two rescuer CPR, the 2015 ACLS guidelines recommend thirty compressions followed by two breaths (30:2 ratio) in patients without an advanced airway. During breaths, compressions may be withheld. If an advanced airway is in place, the 2015 updates recommend a breath rate of 10 per minute, or one breath every six seconds, asynchronous from compressions. However, a 2015 study by the Resuscitation Outcomes Consortium (ROC) compared continuous chest compressions with asynchronous bag-valve-mask ventilation against CPR with compression pauses for breaths in patients without an advanced airway in place. This study found no significant difference between the two ventilation techniques for survival to hospital discharge, as long as the chest compression fraction was > than 75%. Therefore, the key point is to provide meticulous CPR, maximizing compressions, with the briefest of pauses (< 10 seconds).[12]

Mechanical CPR devices may also be used to provide cardiac compression. The two most common types of mechanical CPR devices employed in clinical practice are the LUCAS-2, with a piston attached to an active compression-decompression cup (Figure 24–3) and the load-distributing band Autopulse (Figure 24–4). These devices allow provision of either 30:2 CPR or continuous chest compressions with interposed manual ventilations. Apply defibrillator pads to the bare chest before placing a mechanical CPR device.

The evidence to date does not suggest benefit from mechanical versus manual chest compression.[13,14] However, mechanical compressions are reasonable if sustained high quality compressions compromise provider safety or are impractical. There is little evidence to support the use of impedance threshold devices.

FIGURE 24–3.
The LUCAS-2 chest compression gadget with its attached active compression-decompression device.

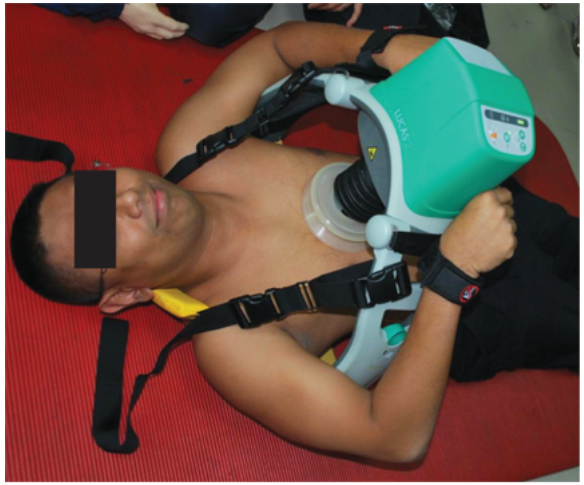
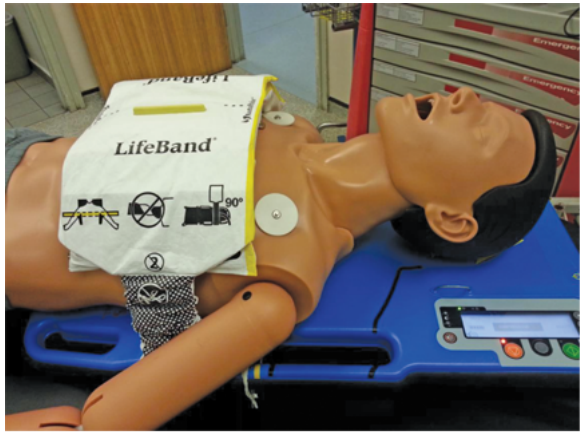


FIGURE 24–4.
The Autopulse mechanical CPR device with its load-distributing band.



The critical rhythms associated with cardiac arrest are **VF** (Figure 24–5), pulseless **VT** (Figure 24–6), asystole (Figure 24–7), and pulseless electrical activity (**PEA**) (Figure 24–8).

FIGURE 24–5.

Ventricular fibrillation.

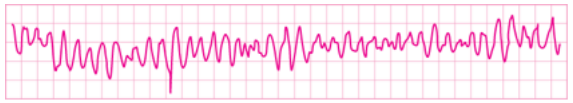


FIGURE 24–6.

Pulseless ventricular tachycardia.

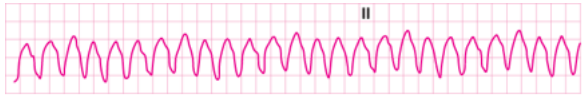


FIGURE 24–7.

Asystole.

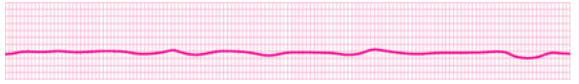
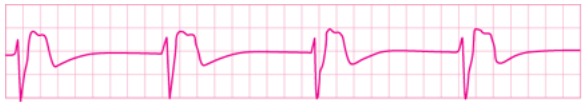


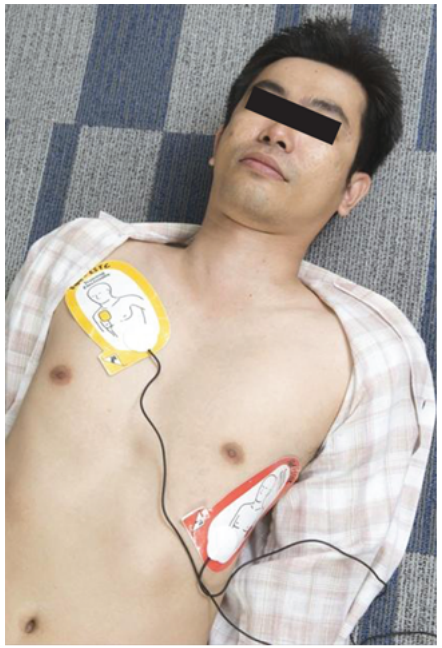
FIGURE 24–8.

Pulseless electrical activity.



Defibrillation may be done with either an automated external defibrillator (**AED**) or a manual defibrillator (see chapter 23). **Deliver shocks for VF or pulseless VT with minimal interruption of chest compressions only during actual shock delivery**. Make sure that the position of the defibrillator pads (**Figure 24–9**) does not interfere with monitoring leads.

FIGURE 24–9.
Placement of defibrillator pads.



After delivery of the first shock, resume CPR (**Figure 24–10**) immediately for up to about 2 minutes before reviewing the ECG monitor for a rhythm diagnosis. If a viable rhythm has returned, check for pulse and breathing. If breathing and pulse have returned, begin care for post–return of spontaneous circulation (**ROSC**) management. If breathing is absent, continue rescue breathing with a bag-valve mask at the rate of about 10 breaths per minute; if pulse is not present, continue CPR and move to the **Secondary Survey**.

FIGURE 24–10.
Continuing CPR after shock using an AED.

SECONDARY SURVEY

Components of the Secondary Survey are listed in Table 24–1 .

Table 24–1

Components of the Secondary Survey

Component and Function	Detailed Components
A: Securing the Airway	Endotracheal intubation or other supraglottic airway, eg, Laryngeal Mask Airway
B: Breathing (Ventilation)	1. Ventilation with Bag-Valve Mask 2. Oxygen 3. Mechanical ventilation
C: Maintaining the Circulation	1. Continue CPR 2. Provide defibrillation and cardioversion to correct life threatening 'shockable' rhythms 3. Continuous ECG monitoring to monitor the circulation's rhythm and identify arrhythmias that need to be corrected 4. Gain vascular access through IV/IO line 5. Provide drugs to help maintain circulation 6. Consider use of ECMO
D: Differential diagnosis	1. Determine why the patient is not responding to the resuscitative efforts 2. Look for correctible causes of collapse and provide necessary interventions
Specialized Care after ROSC*	Therapeutic hypothermia, glycemic control, early goal directed therapy, percutaneous coronary interventions

*ROSC, return of spontaneous circulation

The **Secondary Survey** includes endotracheal intubation or the placement of another airway adjunct, assessment of ventilatory status, gaining intravenous access, identifying ECG rhythms, delivering drugs to enhance circulation, and addressing the reasons for the occurrence or persistence of cardiac arrest.

ECG and ultrasound may assist in determining etiology of arrest, particularly in PEA.[15] PEA with narrow QRS is generally due to mechanical problems caused by right ventricular inflow or outflow obstruction, while PEA with wide-complex QRS is associated with metabolic pathology or myocardial ischemia with left ventricular failure. The use of ultrasound to assess cardiac contractility and etiology of arrest may provide benefit, but make sure US assessment does not compromise compressions. Ultrasound may also be used to confirm ROSC and ETT placement.

While the Secondary Survey steps are in progress, continue the basic resuscitative actions of the primary survey. Usually, defibrillation pads placed carefully left of the apex and to the right of the sternum just below the right clavicle (Figure 24–9) can also act as monitoring leads. Otherwise, true monitoring leads may be placed at the front of the right and left shoulders and over the left iliac crest. Such placement does not interfere with defibrillation or cardiac pacing procedures. Further management is based on the cardiac rhythm.

VASCULAR ACCESS

Vascular access techniques are discussed in Chapter 31, "Vascular Access." A large peripheral vein allows a rapid rate of fluid administration, if needed. If unable to cannulate a peripheral vein, establish intraosseous access. Central venous lines take time to establish and, because of the length of the cannula, cannot deliver fluids as rapidly as a peripheral line, and every bolus dose of drugs requires flushing with at least 20 mL of normal saline. Central lines are useful for central venous pressure monitoring to guide fluid resuscitation and circulatory management.

In cardiac arrest, circulation of blood grinds to a halt. With good-quality CPR, one can expect to generate up to about 30% of normal cardiac output, which is just about what is required to supply vital organs such as the h circulation is associated with a slower circulation time. Therefore, drugs given during cardiac resuscitation are best administered via a proximal peripheral vein. Once drugs are

given, flush the line with normal saline, and continue CPR for at least 30 to 60 seconds before repeat external defibrillation, so that the heart is optimally primed to respond.

The optimal infusion fluid is normal saline and not dextrose, Ringer's lactate, or sodium bicarbonate. However, unless hypovolemia is a contributing factor to cardiac arrest, fluid infusion should be slow, if at all necessary. Drug dosages are the same whether given by the intraosseous, IV, or central line routes. The endotracheal tube route is no longer recommended for drug administration because drug absorption is limited if the airway is filled with pulmonary edema fluid.

DRUGS USED FOR ROSC FROM CARDIAC ARREST

Drugs available for cardiac resuscitation and cardiac dysrhythmia management are also described in chapters 19 and 20. This section will cover those drugs specifically used for resuscitation from cardiac arrest to ROSC. Drugs are an adjunct in the management of cardiac arrest patients. Good CPR, ventilation, and early defibrillation are the cornerstones of management of cardiac arrest. The effectiveness of standard resuscitative drugs on ROSC and survival to hospital discharge has not been well demonstrated.[16–18] However, consensus documents recommend the standard resuscitative drugs described below with the presumptive rationale that drugs help "restart" the heart and preserve coronary and cerebral circulation.

EPINEPHRINE

Epinephrine (adrenaline) is an endogenous catecholamine. It has an important role in cardiac arrest, although the evidence base for improved outcomes in humans is weak.[19] Epinephrine seems to improve ROSC and short-term survival[20], but does not appear to improve survival to hospital discharge, or neurologic outcome.[21,22] The primary beneficial effect appears to be peripheral vasoconstriction, which improves cerebral and coronary blood flow. Potential adverse effects include an increase in myocardial oxygen consumption and an increase in pulmonary shunting. The most common adverse reaction is tachycardia. Epinephrine may worsen myocardial ischemia and induce ventricular ectopy and VT. **Epinephrine is used mainly to treat cardiac arrest from VF or pulseless VT unresponsive to the initial shock, asystole, PEA, and profoundly symptomatic bradycardia**. The 2015 updates provide a class IIb recommendation, stating standard dose epinephrine may be reasonable for patients with cardiac arrest. The standard dose in cardiac arrest is 1.0 milligram diluted to 10 mL (10 mL of 1:10,000) given IV. Repeat if needed at 3- to 5-minute intervals. There is no maximum dose. Escalating doses at 2 to 5 milligrams IV every 3 to 5 minutes (high-dose epinephrine) have not resulted in increased long-term survival.[19] For IV infusion in patients with cardiogenic shock or symptomatic bradycardia, the dose is 1 milligram in 500 mL normal saline beginning at 2 to 10 micrograms/min, escalating as needed at 3- to 5-minute intervals. Do not add epinephrine to infusions that contain alkaline solutions because epinephrine has ineffective clinical activity in alkaline solutions.

AMIODARONE

Amiodarone is generally considered a Class III antiarrhythmic drug, but it possesses electrophysiologic characteristics of all four Vaughn-Williams classes. It causes coronary and peripheral artery vasodilation. **Its main use in cardiac arrest is for persistent VT or VF after defibrillation and epinephrine**. While the 2015 ACLS guidelines recommend amiodarone for refractory VF, a 2016 large multi-center trial failed to demonstrate a difference in survival to hospital discharge for shock-refractory VF or pulseless VT with amiodarone or lidocaine compared to placebo.[23]

Amiodarone can also be used for hemodynamically stable VT, hemodynamically stable polymorphic VT, and hemodynamically stable wide-complex tachycardia of uncertain origin. It is also used for the pharmacologic conversion of atrial fibrillation, control of rapid ventricular rate in preexcitation supraventricular dysrhythmias, and as an adjunct to electrical cardioversion of refractory paroxysmal supraventricular tachycardia/atrial tachycardia. For pulseless VT or VF, the dose is a bolus of 300 milligrams IV followed by a 20-mL flush with 5% dextrose in water or saline. Give another 150-milligram bolus if there is no response to the first dose. For stable ventricular and supraventricular dysrhythmias, administer amiodarone IV 150 milligrams over 10 to 15 minutes (not to exceed 30 milligrams/min), followed by a maintenance infusion of 1 milligram/min for 6 hours and then 0.5 milligrams/min for the next 18 hours. Infusions exceeding 2 hours should be administered in glass or polyolefin bottles because the drug precipitates in plastic tubing. Hypotension and bradycardia are the most common unwanted effects. These may be addressed by slowing the infusion rate, giving an IV fluid challenge, or using pressors or positive chronotropic agents. Occasionally, temporary pacing for refractory bradycardia from amiodarone may be required, especially if other measures are ineffective.

Recent literature suggests no improvement in survival or favorable neurologic outcome in patients with OHCA due to initial shock-refractory VF or pulseless VT with antiarrhythmic medications such as amiodarone or lidocaine.[23] Patients with in-hospital cardiac arrest may receive some benefit when given steroids, vasopressin, and epinephrine as a bundle, though routine use is not recommended.[24]

LIDOCAINE (LIGNOCAINE)

Lidocaine (lignocaine) (see also chapter 12, Approach to Shock) is a Class I antiarrhythmic drug. It reduces automaticity, suppresses ventricular ectopy, and may be used for hemodynamically stable VT and refractory VF/pulseless VT. In the 2010 recommendations, lidocaine was the second-choice drug after amiodarone, or was used if amiodarone was not available. However, a 2016 multicenter study suggests no improvement in survival or neurologic outcome with use of lidocaine (or amiodarone or placebo) for OHCA from VF or pulseless VT.[23] In the 2010 guidelines for cardiac arrest, the dose was an IV bolus of 1 to 1.5 milligrams/kg body weight.

A second bolus of 0.5 to 0.75 milligrams/kg was provided if the rhythm persisted. The 2015 algorithm no longer contains lidocaine, and guidelines state the routine use of lidocaine after ROSC is not recommended, though it may be considered in patients with ROSC after VF or pulseless VT. Upon restoration of spontaneous circulation, lidocaine may be given as an infusion at a rate of 1 to 4 milligrams/min.

If dysrhythmia reappears during the infusion of lidocaine, give a bolus of 0.5 milligrams/kg and increase the infusion rate to 4 milligrams/min. Toxicity may occur with doses exceeding 3 milligrams/kg body weight bolus or in patients with liver disease, since the drug is hepatically metabolized. Symptoms include neurologic changes such as drowsiness, disorientation, reduced hearing ability, perioral paresthesia, muscle tremors, and seizures. Myocardial depression and circulatory depression are also features of toxicity, and these may be illustrated by widening QRS complexes and falling blood pressures. In patients with known impaired liver function or patients >70 years old, give the same recommended bolus doses, but decrease the normal infusion rate by 50%.

β-Blockers

In animal studies, β-blockers can reduce myocardial oxygen requirements, decrease the number of shocks needed for defibrillation, and can prolong survival from VF/pulseless VT. Successful use in humans is based primarily upon case reports. However, one retrospective series of patients with refractory VF or VT who received at least 3 defibrillation attempts, 300 mg of amiodarone, and 3 mg of adrenalin, without success, reported that esmolol given during resuscitation improved rate of ROSC, ICU admission, survival to hospital discharge, and favorable neurologic outcome.[25] Further study is required.

MAGNESIUM

Magnesium is a cofactor in numerous enzymatic reactions. It is essential for the function of the Na-K-ATPase pump. Magnesium deficiency may be associated with cardiac arrhythmias, sudden death, and precipitation of VF. Magnesium is used to treat hypomagnesemia, with or without dysrhythmias.

Magnesium is initial treatment for torsades de pointes and dysrhythmias secondary to hypomagnesemia, cardiac arrest from QT_c prolongation, or cardiac glycoside toxicity.[16,26] For patients in VF or pulseless VT due to the above conditions, give 1 to 2 grams in 10mL 5% dextrose in water IV over 1 minute. Magnesium provides no benefit for routine use in cardiac arrest. For patients with a pulse, the dose is 1 to 4 grams in 50 mL 5% dextrose in water over 60 minutes. Adverse reactions include flushing, sweating, mild bradycardia, hypotension, asystole with circulatory collapse (with too rapid administration), and respiratory depression. Hypermagnesemia may produce depressed reflexes, flaccid paralysis, diarrhea, respiratory depression, and circulatory collapse.

OTHER DRUGS IN CARDIAC ARREST

Atropine

Atropine is a parasympatholytic agent that enhances sinus node automaticity and atrioventricular conduction by direct vagolytic action. **It is not recommended for PEA or for treatment of cardiac arrest**. It is indicated for symptomatic bradycardia. The dose is 0.5 or 0.6 milligrams IV (dose depends on formulation in country of use) and may be repeated at 5- to 10-minute intervals up to a maximum of 0.04 milligrams/kg body weight. Atropine may induce tachycardia or premature ventricular contractions and cause worsening of myocardial ischemia. Symptoms of overdosage include tachycardia, delirium, coma, flushed and hot skin, ataxia, and blurred vision. Administration of low doses less than 0.5 milligrams IV may produce paradoxical bradycardia and precipitate VF.

Calcium

Calcium is given during resuscitation only for cardiac arrest from hyperkalemia, hypocalcemia, or calcium channel blocker overdose. Calcium is not recommended for routine administration for VF/pulseless VT or PEA.[16,27] The dose of calcium chloride is 0.2 mL/kg of 10% calcium chloride, given as a slow IV bolus.

Sodium Bicarbonate

The use of sodium bicarbonate during cardiac arrest was advocated in the past to treat presumptive acidosis, because severe acidosis decreases myocardial contractility.[28] However, **routine use during cardiac arrest is no longer recommended** due to a number of potential adverse effects. Sodium bicarbonate causes hypernatremia, hyperosmolality, and alkalosis (which in turn induces a left shift of the oxyhemoglobin dissociation curve), and IV sodium bicarbonate produces carbon dioxide, resulting in hypercarbia unless ventilation is increased. It does not appear to improve defibrillation success. **It may be given in cardiac arrest from hyperkalemia or cyclic antidepressant overdose**. It is also acceptable and possibly helpful for intubated patients with a long arrest interval until ROSC and with persistent severe metabolic acidosis. The dose is 1 to 1.5 mEq/kg IV bolus, followed by 0.75 mEq/kg every 10 to 15 minutes as needed. If continuous infusion of sodium bicarbonate is used, check pH to guide therapy.

Vasopressin

Vasopressin, also called antidiuretic hormone, is a naturally occurring neurohypophysial peptide hormone synthesized in the hypothalamus and stored in the pituitary gland. It increases water absorption in the nephron and increases peripheral vascular resistance. Vasopressin levels elevate during cardiac arrest, and this observation led to investigation of its role in resuscitation. Vasopressin has a longer duration of action and, in laboratory studies, maintains coronary perfusion pressure, myocardial blood flow, and cerebral blood flow better than epinephrine. However, most studies do not show superiority of vasopressin over epinephrine.[19,27] Vasopressin as a first-line agent in cardiac arrest (40-unit dose) does not improve long-term survival when compared to epinephrine,[29] although vasopressin might improve short-term survival in patients with prolonged cardiac arrest.[29] Combining vasopressin (40 units) with epinephrine does not appear to improve outcomes.[19] The 2015 updates remove vasopressin from all algorithms. A combination of vasopressin, epinephrine, and steroids is not recommended at this time for routine use, due to low quality of evidence.

DOUBLE SEQUENTIAL EXTERNAL DEFIBRILLATION

Double sequential external defibrillation may be appropriate for patients with shock-refractory VF. This is initially described in the 1980s, with several current descriptions of success in patients with refractory VF.[30–32] It is theorized double-sequential defibrillation reduces the VF threshold and overrides the relative refractory period of cardiac tissue. To complete this, a second set of defibrillator pads are placed in the anterior-posterior positions (if the first set is placed in the standard apex-sternum orientation), for a total of two sets (4 separate pads). Both sets are charged to maximal settings, with simultaneous shock delivery, which requires coordination. This may be appropriate for patients in refractory VF after quality CPR and several attempts at defibrillation.

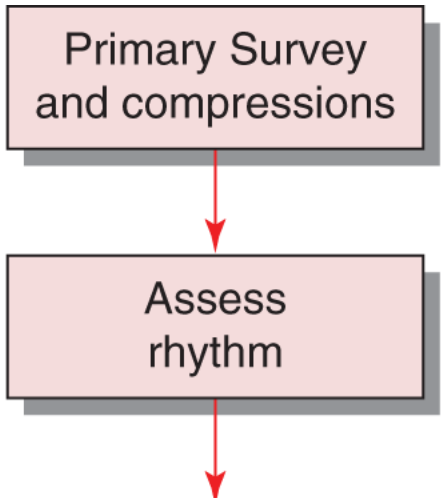
COMMON CARDIAC ARREST ALGORITHMS

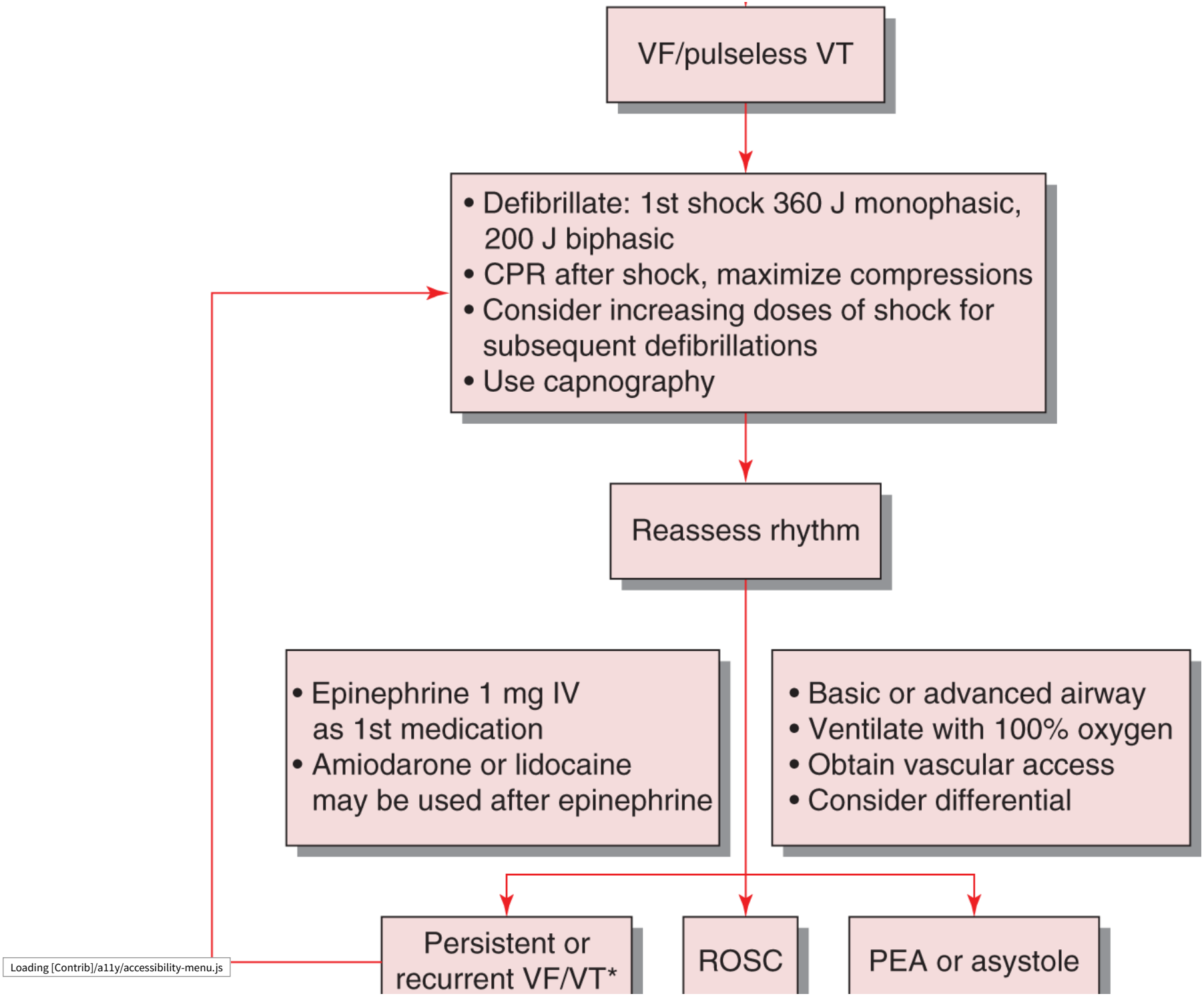
The algorithmic approach to cardiac arrest management allows a structured decision-making process when managing cardiac arrest victims. The cardiac arrest rhythms discussed here are pulseless VT/VF, asystole, and PEA. The use of ultrasound to assess contractility and ECG for length of the QRS interval (narrow versus wide) has been evaluated and may provide benefit in determining etiology of arrest. Peri-arrest ultrasound should not compromise compressions, but it may assist in identifying reversible causes of arrest. It is currently unclear whether the use of US affects clinical outcomes.

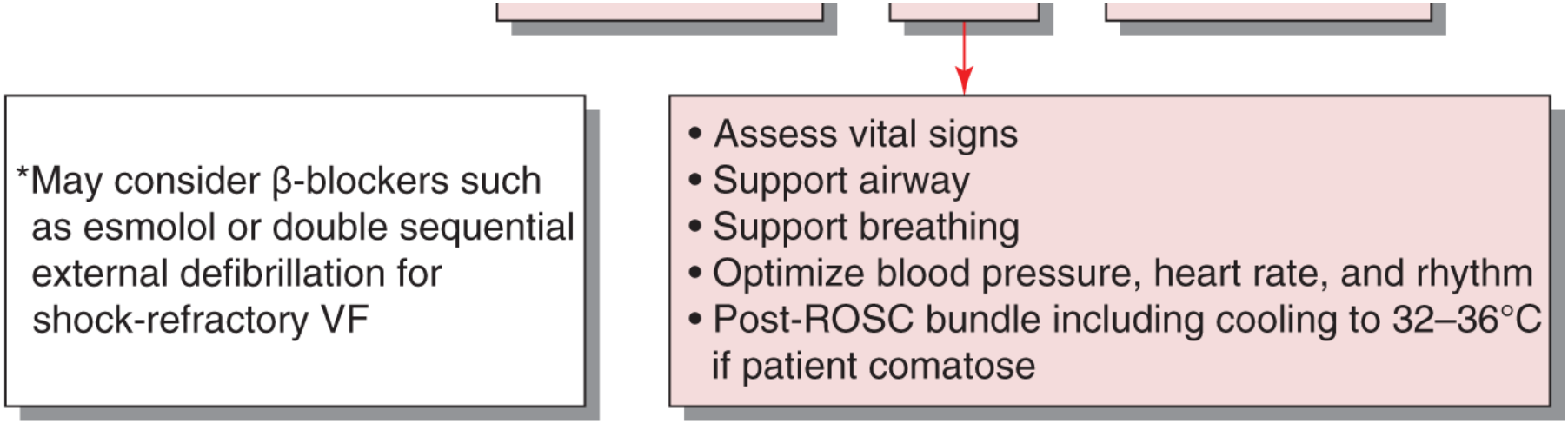
VENTRICULAR FIBRILLATION (VF)/PULSELESS VENTRICULAR TACHYCARDIA (VT)

Once VF or pulseless VT is diagnosed, prepare for defibrillation while CPR is in progress. If ROSC does not occur after the first shock, move to the mega-VF approach (**Figure 24–11**). Continue CPR for 1 to 2 minutes followed by rhythm analysis, and if VF persists or recurs, deliver electrical shocks, usually biphasic, beginning usually at 150 J and escalating, if needed, to 360 J. For monophasic defibrillators, begin at 360 J. After delivery of shock, follow with CPR again for 1 to 2 minutes before rhythm analysis. Maintain good-quality CPR for optimal coronary and vital organ perfusion. Parallel to the CPR-analysis-shock-CPR-analysis-shock-CPR cycle, begin secondary ABCDs and give drugs that may help to lower defibrillation thresholds, such as epinephrine (adrenaline), amiodarone, and/or lidocaine (lignocaine). Any or all of these drugs may be administered in any combination and repeated at roughly 3- to 5-minute intervals. After administration of any of these drugs, provide at least 30 to 60 seconds of effective CPR to allow the injected drug to reach the central circulation before the next shock. Continue the resuscitation cycle for as long as the rhythm remains as VF or pulseless VT.

Figure 24–11.
Management of VF/pulseless VT.



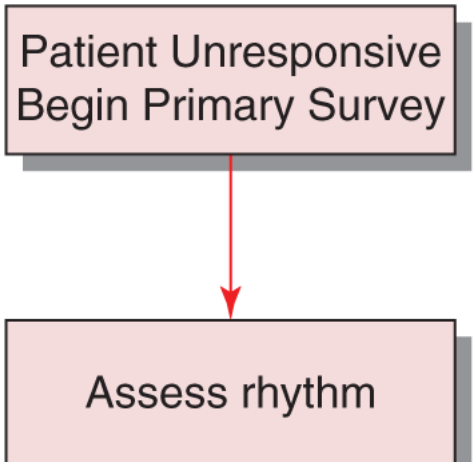


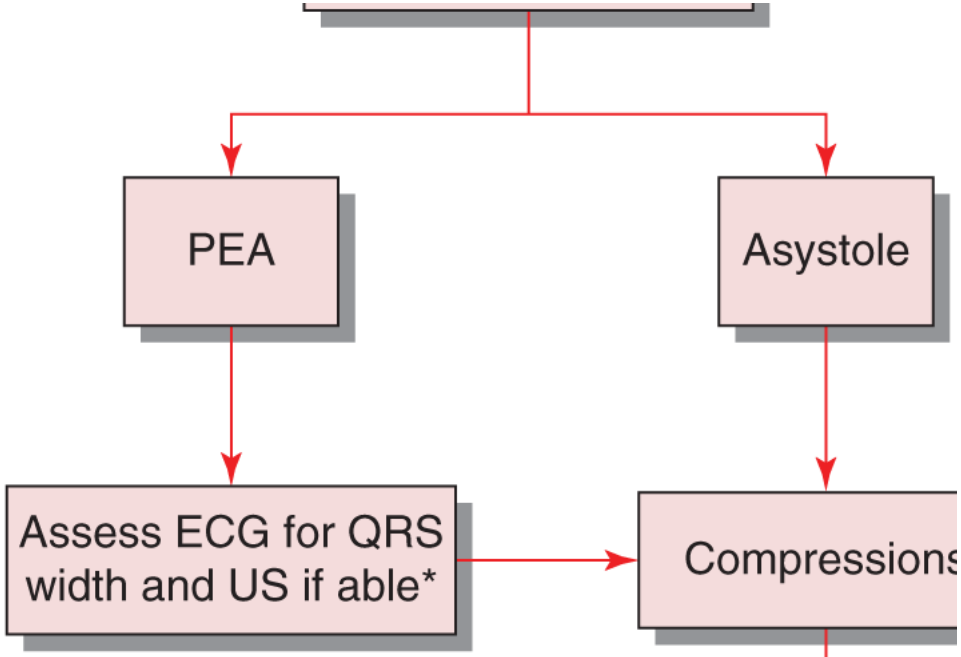


ASYSTOLE/PULSELESS ELECTRICAL ACTIVITY (PEA)

For asystole or PEA, (a condition in which cardiac contractions are absent in the presence of coordinated electrical activity), continue CPR and institute the secondary ABCDs. Give drugs to enhance the chances of ROSC (Figure 24–12). Epinephrine (adrenaline) 1.0 milligram as a bolus dose in 10 mL is the drug of choice. Vasopressin alone or in combination with epinephrine is no more effective than epinephrine alone. Repeat epinephrine as needed in 3- to 5-minute intervals. Consider other possible causes of asystole or PEA, and identify and correct them. It is currently assumed that narrow-complex PEA[33] could be the result of mechanical problems such as cardiac tamponade, pneumothorax, mechanical hyperinflation, pulmonary embolism, or myocardial rupture. Point-of-care US and assessment of the clinical scenario can direct specific treatment. Wide-complex PEA, on the other hand, can result from a metabolic problem (ie, hyperkalemia), drug toxicity (ie, sodium channel blocker toxicity), or cardiac ischemia, and left ventricular failure and should be treated as appropriate. Survival is low if patients in asystole do not achieve ROSC in the field or convert to a shockable rhythm.[34–36]

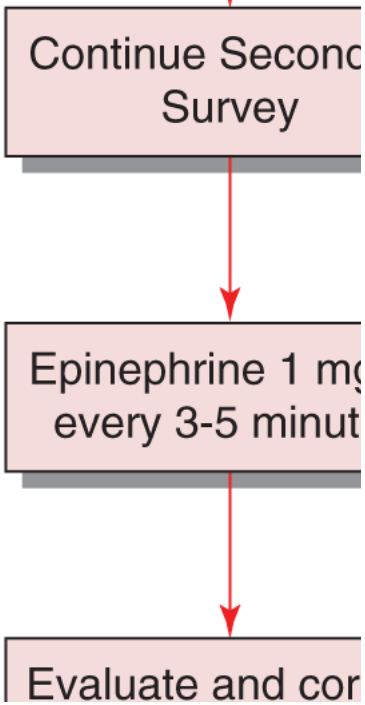
Figure 24–12.
Asystole/PEA management algorithm..





*PEA Evaluation

NARROW COMPLEX PEA	WIDE COMPLEX PEA
Consider mechanical cause: • Tamponade • PE • Hypovolemia/bleeding (IVC) • Tension PTX • Acute MI with cardiac rupture	Consider metabolic cause: • Hyperkalemia • Sodium channel blocker overdose • Severe acidosis • Agonal rhythm • Acute MI with pump failure
Bedside US: Hyperdynamic LV	**Bedside US:** Hypokinetic or akinetic LV
Management: IV fluid bolus and treat underlying condition • Tamponade → Pericardiocentesis • PE → Thrombolysis	**Management:** Treat underlying cause • Hyperkalemia → IV calcium, sodium bicarbonate bolus, insulin/glucose • Sodium channel blocker overdose → IV



• Hypovolemia/bleeding → Resuscitation • Tension PTX → Decompression	sodium bicarbonate boluses

reversible caus

DIFFERENTIAL DIAGNOSES OF CARDIAC ARREST

In every resuscitation, it is useful to identify important causes of cardiac arrest and reasons for lack of response to standard resuscitation algorithms. Causes can be grouped as the **5 H's—hypovolemia or hemorrhage, hypoxia, hydrogen ion (acidosis), hypo- or hyperkalemia, and hypothermia**—and the **5 T's—trauma + tablets (overdose), cardiac tamponade, coronary thrombosis, tension pneumothorax, and thrombosis (pulmonary embolism)**. The use of ultrasound to assess contractility and ECG for length of the QRS interval (narrow versus wide) has been evaluated and may provide benefit in determining etiology of arrest. Peri-arrest ultrasound should not compromise compressions, but it may assist in identifying reversible causes of arrest. It is currently unclear whether the use of US affects clinical outcomes. However, use of US in PEA may affect clinical decision-making through identification of pathologies requiring emergent intervention.[37]

H: HYPOVOLEMIA OR HEMORRHAGE

History of fluid or blood loss may be available. Rectal examination can identify massive lower GI bleeding; nasogastric intubation can identify massive upper GI bleeding; and bedside FAST can diagnose massive intraperitoneal bleeding. Treat with fluids and blood products.

H: HYPOXIA

Hypoxia occurs with lack of oxygen and alveolar ventilation. Make sure that the airway adjunct is placed correctly. Check breath sounds at intervals to ensure that the endotracheal tube has not slipped out of the trachea or to identify pneumothorax. Verify the source of oxygen—an oxygen cylinder or the piped oxygen supply.

H: HYDROGEN ION (ACIDOSIS)

The acidosis of cardiac arrest is a combination of respiratory and metabolic acidosis. Respiratory acidosis is addressed by early endotracheal intubation and alveolar ventilation. Metabolic acidosis can be somewhat addressed by good-quality CPR. Sodium bicarbonate is administered for severe metabolic acidosis from prolonged or poor initial resuscitation, at a dose of 1 to 1.5 mEq/kg. Half the initial dose can be readministered after 10 to 15 minutes, depending on pH.

H: HYPER- OR HYPOKALEMIA, OTHER METABOLIC DISORDERS

Suspect hyperkalemia in patients on hemodialysis or peritoneal dialysis (look for presence of arteriovenous fistula or dialysis catheter). Other metabolic disorders are extremely difficult to confidently identify in cardiac arrest. If hyperkalemia is suspected, administer calcium chloride, sodium bicarbonate, insulin, and glucose. Treat hyperkalemia from suspected digitalis toxicity with IV magnesium sulfate or digoxin-specific Fab fragments (Digibind®).

H: HYPOTHERMIA

Treat hypothermia with gradual rewarming with blankets and warm IV fluids. If there is no recovery of consciousness in the hypothermic patient following ROSC, maintain a body core temperature of 33°C until further assessment and decisions can be made.

T: "TABLETS" (DRUG OVERDOSE)

Drug overdose is rarely identified as a cause of cardiac arrest during the resuscitation process. In the event of antidepressant overdose, administer IV sodium bicarbonate. Lipid emulsion infusion may be useful in cardiac arrest associated with cyclic antidepressants or local anesthetics.

T: CARDIAC TAMPONADE

Cardiac tamponade is best identified during resuscitation by bedside transthoracic US. This requires brief interruption of chest compressions. Treatment of tamponade causing cardiac arrest is bedside pericardiocentesis.

T: TENSION PNEUMOTHORAX

Suspect tension pneumothorax during cardiac resuscitation if breath sounds are unequal on chest auscultation after verifying correct endotracheal tube placement. Treatment is immediate needle decompression.

T: CORONARY THROMBOSIS

Acute coronary thrombosis or acute myocardial infarction is one of the most common causes of cardiac arrest. Risk factors are a history of coronary artery disease and initial rhythm of VF/VT.[38–42] Cardiac catheterization after resuscitation is an underused procedure.[42] A 12-lead ECG in the immediate post–cardiac arrest state can identify an ST-elevation acute myocardial infarction and allow arrangements for immediate coronary angiography. Myocardial and neurologic function can improve after percutaneous coronary intervention following cardiac arrest.[43] Thus, after ROSC, especially in the face of post-ROSC ECG evidence of acute myocardial infarction, advocate for cardiac catheterization and percutaneous coronary revascularization if available and appropriate. A few case reports describe fibrinolysis during CPR with resultant ROSC and good neurologic outcome.[44] Coronary angiography is reasonable in select patients with suspected cardiac origin of arrest but without ST elevation, or in those electrically unstable patients without ST elevation with suspected cardiovascular lesion. Reports are too few to determine whether thrombolysis together with CPR results in more severe bleeding than thrombolysis without CPR.[45]

T: THROMBOSIS (PULMONARY EMBOLISM)

Pulmonary embolism causing cardiac arrest requires fibrinolysis or embolectomy. However, the diagnosis is rarely made at time of collapse, and even then, most systems are not geared to make such prompt diagnosis and initiate the necessary procedures for embolectomy. Fibrinolytic agents could be considered during cardiac arrest from suspected pulmonary embolism on a case-by-case basis.[46] Factors suggestive of pulmonary embolism causing cardiac arrest include two of three signs/symptoms (prearrest respiratory distress, altered mental status, or shock); arrest witnessed by a physician or emergency medical technician; and PEA as the first or primary arrest rhythm.

POST-ROSC COMPLICATIONS

After ROSC, many factors affect survival: anoxic brain injury, post–cardiac arrest myocardial dysfunction, the systemic reperfusion response, and the cause of cardiac arrest. Ischemic-reperfusion injury is discussed in chapter 26, Post-Cardiac Arrest Syndrome. Anoxic brain injury results in disturbance of cerebral microvascular hemostasis and manifests as coma, seizures, myoclonus, and varying degrees of neurocognitive dysfunction, including brain death. Post–cardiac arrest myocardial dysfunction results from myocardial stunning with cardiac hypokinesis and a low left ventricular ejection fraction. Clinical manifestations include tachycardia and elevated left ventricular end-diastolic pressures progressing to hypotension and reduced cardiac output. The systemic ischemia-reperfusion response consists of inflammation, endothelial activation, and disturbed vasoregulation with generalized activation of immunologic and coagulation pathways, causing increased risk of multiple organ failure and infection. Clinical manifestations of the systemic ischemia-reperfusion response include impaired oxygen delivery and utilization and increased susceptibility to infection.

OXYGENATION AND VENTILATION

Hyperoxia during the early phase of reperfusion after ROSC harms postischemic neurons and increases brain lipid peroxidation.[47–49] After ROSC, adjust the rate of ventilation and tidal volume to maintain arterial oxyhemoglobin saturation at 94% to 98%.[50] Hyperventilation is not recommended because it can increase intrathoracic pressures and decrease venous return and cardiac output. In addition, hypocarbia resulting from hyperventilation decreases cerebral blood flow and aggravates anoxic brain damage. The suggested ventilator parameters during the post-ROSC phase[48–50] are as follows: Pa_{CO2} between 35 and 45 mm Hg (5 to 6 kPa); Sa_{O2} between 94% and 98%; tidal volume between 6 and 8 mL/kg ideal body weight; P_{ETCO2} between 35 and 40 mm Hg; and 10 to 12 ventilations per minute.

HEMODYNAMIC MANAGEMENT

Obtain 12-lead ECG after ROSC and repeat at 8 hours or as needed. Administer IV fluids and drugs to optimize blood pressure, cardiac output, and urine output. The target for blood pressure is a mean arterial pressure of 65 to 100 mm Hg, and the target for blood oxygenation is an S_{CVO2} of ≥70%.[50] Pharmaceutical agents to support the circulation include epinephrine, norepinephrine, dopamine, dobutamine, nitroglycerine, and esmolol. Routine use of lidocaine after ROSC is not recommended.Obtain an echocardiogram at 24 hours after ROSC to detect regional wall motion abnormalities and determine ejection fractions.

TARGETED TEMPERATURE MANAGEMENT (THERAPEUTIC HYPOTHERMIA)

Brain cooling decreases cerebral oxygen demand, reduces cellular effects of reperfusion, and decreases the production of reactive oxide radicals. Targeted temperature management (cooling to 32 to 36°C; temperature goal is controversial) during the first 24 hours after ROSC improves survival and neurologic recovery[51–53] in patients who remain comatose soon after ROSC. Hyperpyrexia during the first 48 hours is usually associated with a lowered chance of optimal neurologic recovery. The optimal temperature is not known, and avoiding fever is paramount. See chapter 26, Post-Cardiac Arrest Syndrome, for detailed discussion.

ECPR (ECMO) in Cardiac Arrest

Extracorporeal Membrane Oxygenation (ECMO), also known as Extracorporeal Life Support (ECLS) is a recent introduction in the management of cardiac arrest. Its use is well-documented in the neonatal and pediatric population, and in adults, for refractory respiratory failure, and cardiogenic shock. Use in refractory cardiac arrest is also known as ECPR. ECPR is a bridging therapy to definitive treatments, such as percutaneous coronary interventions, cardiac bypass surgery, or heart transplant.

The ECMO equipment consists of a blood pump, a venous reservoir, an oxygenator for exchanging both oxygen and carbon dioxide, and a heat exchanger to warm the blood used. The whole system is monitored through pressure, oxygen saturation, and temperature monitors. Three types of ECMO circuits are available:

1. A veno-arterial ECMO (VA-ECMO) pumps blood from the venous side to the arterial side to facilitate gas exchange and provide hemodynamic support. The blood is pumped from the venous circulation through a cannula inserted in either the inferior vena cava or right atrium, through the oxygenator where gas exchange occurs, then warmed and returned to the patient through a cannula placed in either the aortic arch or femoral artery into the arterial circulation. This is the modality that is used to support cardiac arrest patients.
2. A veno-venous (VV) ECMO removes blood from the right atrium, passes it through the gas exchanger, and returns it across the tricuspid valve into the right ventricle. It does not provide hemodynamic support. This modality is used mainly for refractory respiratory failure.
3. An arterio-venous ECMO (AV-ECMO) makes use of the patient's own arterial pressure to pump the blood from the arterial to the venous side and facilitates gas exchange in the process. This does not require the use of a separate blood pump.

The ECMO circuit is initially primed with fresh blood, which is then pumped through the circuit. During maintenance of ECMO, haemodynamic parameters, urinary output, hematological indices, fluids, and electrolytes are monitored.

ECPR complications can be mechanical or medical, or both. Mechanical complications consist of clots in the circuit, mediastinal bleeding from tears to the great vessels, oxygenator failure and malfunction of the blood pump, oxygenator, heat exchanger and sensors. Medical complications include intracranial and systemic hemorrhage, initial cardiac stunning that may occur soon after initiation of ECMO, pneumothorax, acute kidney injury, gastrointestinal bleeding, sepsis, and metabolic derangement.

ECPR must also be accompanied by good post-cardiac arrest management, to include targeted temperature and hemodynamic management, and early coronary angiography for definitive treatment. At the present time, survival rates are low, but can be improved with EMS and ED training, appropriate patient selection, and more widespread application of the technique.

Figure 1.
ECPR in the emergency department, Singapore General Hospital.

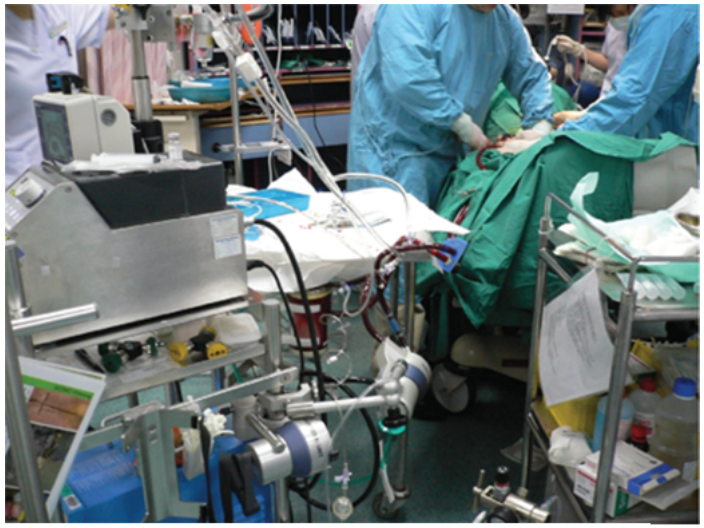


TABLE 1

General Indications and Contraindications for ECPR (ECMO in Cardiac Arrest)

Indications	Contraindications
Good pre-morbid status before cardiac arrest	Advanced age; advanced malignancy; poor baseline neurologic function; baseline inability to perform activities of daily living; pre-exisitng 'do not resuscitate' order
Intervention to be curative, not palliative	Suspect aortic dissection or severe aortic regurgitation; traumatic cardiac arrest
Reversible trigger event for cardiac arrest (dysrhythmia, STEMI, etc.)	Unwitnessed cardiac arrest and no bystander CPR Long prehospital transport time Prolonged cardiac arrest unless good perfusion and metabolic support is documented

GLYCEMIC CONTROL

Post-ROSC hyperglycemia is associated with increased mortality and worse neurologic outcomes.[54] Hypoglycemia, similarly, is also associated with poor outcomes in critically ill patients. Maintain blood sugar levels between 100 and 180 milligrams/dL (6 and 10 mmol/L).

NEUROLOGIC ASSESSMENT

Features of brain injury after ROSC include coma, seizures, myoclonus, and various degrees of neurocognitive dysfunction ranging from memory deficits to a persistent vegetative state and finally brain death. Treat seizures promptly. The neurologic prognosis in the majority of comatose cardiac arrest survivors undergoing therapeutic hypothermia cannot be reliably predicted in the ED.

OTHER TECHNOLOGY

CAPNOGRAPHY

Waveform capnography is the most reliable means of confirming and continuously monitoring tracheal tube placement, as well as monitoring resuscitation. An end-tidal reading of less than 10 mm Hg CO_2 after 20 minutes of resuscitation is associated with low chance of survival. However, this should not be used alone when deciding to cease resuscitation efforts. Capnography may also be used to monitor ventilation rate and CPR quality, as high quality compressions should produce an end-tidal CO_2 of at least 12-15 mm Hg, if not greater. A sudden rise in end tidal CO_2 is an early indication of ROSC.

SOCIAL MEDIA

Social media currently possesses a role in cardiac arrest, as it is reasonable for communities to incorporate social media to notify and summon potential rescuers in close proximity to a patient of suspected out of hospital cardiac arrest. These rescuers should be willing and able to provide CPR.

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