Chapter 7: Cardiac Arrest

INTRODUCTION

- *Cardiac arrest* is defined as cessation of cardiac mechanical activity as confirmed by absence of signs of circulation (eg, detectable pulse, unresponsiveness, apnea).

PATHOPHYSIOLOGY

- Coronary artery disease is the most common finding in adults with cardiac arrest and causes ~75% of sudden cardiac deaths. In pediatric patients, cardiac arrest typically results from respiratory failure, asphyxiation, or progressive shock.
- Noncardiac causes include drowning, choking, asphyxia, electrocution, trauma, poisoning, severe asthma, pneumonia, drug overdose, and sudden infant death syndrome.
- Two different pathophysiologic conditions are associated with cardiac arrest:
  - ✔ Primary: arterial blood is fully oxygenated at the time of arrest.
  - ✔ Secondary: respiratory failure with lack of ventilation leads to severe hypoxemia, hypotension, and cardiac arrest.
- Cardiac arrest in adults usually results from arrhythmias. Historically, ventricular fibrillation (VF) and pulseless ventricular tachycardia (PVT) were most common. Recent data indicate that nonshockable rhythms (ie, asystole, pulseless electrical activity [PEA]) are now more prevalent. This change is of concern because survival rates to hospital discharge are higher after shockable rhythms like VF and PVT than with nonshockable rhythms like asystole and PEA.

CLINICAL PRESENTATION

- Cardiac arrest may be preceded by anxiety, shortness of breath, crushing chest pain, nausea, vomiting, and diaphoresis.
- Signs include apnea; hypotension; no detectable pulse; cyanosis; cold, clammy extremities; and unresponsiveness.

DIAGNOSIS

- Rapid diagnosis is vital to success and is made by observing clinical manifestations consistent with cardiac arrest.
- Electrocardiography (ECG) identifies the cardiac rhythm, which in turn determines drug therapy.
  - ✔ VF is electrical anarchy of the ventricle resulting in no cardiac output and cardiovascular collapse.
  - ✔ PEA is absence of a detectable pulse and presence of some type of electrical activity other than VF or PVT.
  - ✔ Asystole occurs when there is no electrical activity in the heart, indicated by a flat line on the ECG.

TREATMENT

- Goals of Treatment: Resuscitation goals are to preserve life; restore health; relieve suffering; limit disability; and respect the individual's decisions, rights, and privacy. This can be accomplished via cardiopulmonary resuscitation (CPR) by return of spontaneous circulation (ROSC) with effective ventilation and perfusion quickly to minimize hypoxic damage to vital organs. After successful resuscitation, the primary outcome is survival to hospital discharge with good neurologic function.

General Approach

- The American Heart Association (AHA) guidelines for CPR and emergency cardiovascular care (ECC) are now updated annually (published online) and emphasize timely implementation of the "chain of survival":

    ✔ For out-of-hospital arrests: (1) immediate recognition of cardiac arrest and activation of the emergency response system, (2) immediate high-quality CPR, (3) rapid defibrillation, (4) effective advanced cardiac life support (ACLS), and (5) integrated post-arrest care.

    ✔ For in-hospital arrests: (1) surveillance and prevention of cardiac arrest, (2) prompt notification and response by a multidisciplinary team of healthcare providers, (3) high-quality CPR, (4) prompt defibrillation and ACLS when appropriate, and (5) integrated post-arrest care.

Basic Life Support Given by Healthcare Providers

- The mnemonic for the CPR sequence is "CAB" (circulation, airway, breathing). Basic life support *given by healthcare providers trained in CPR* includes the following actions performed in this order:

    ✔ First, determine patient responsiveness. If unresponsive with no breathing or no normal breathing (ie, only gasping), activate the emergency medical response team and obtain an automated external defibrillator (AED).

    ✔ Check for a pulse, but if not definitely felt within 10 seconds, begin CPR with chest compressions and use the AED when available.

    ✔ Begin CPR with 30 chest compressions at a rate of 100–120/min and a compression depth of at least 2 in (5 cm) in adults and at least one third of the anteroposterior chest diameter in infants and children (~1.5 in [4 cm] in infants and 2 in [5 cm] in children).

    ✔ Open the airway and deliver 2 rescue breaths, then repeat chest compressions. Follow each cycle of 30 chest compressions by 2 rescue breaths.

    ✔ Continue CPR until an AED is ready for use or ACLS providers take over care.

    ✔ If/when an AED is available, check rhythm to determine if defibrillation is advised. If so, deliver one shock with immediate resumption of chest compressions/rescue breaths for 2 minutes or until prompted by the AED to allow for another rhythm check.

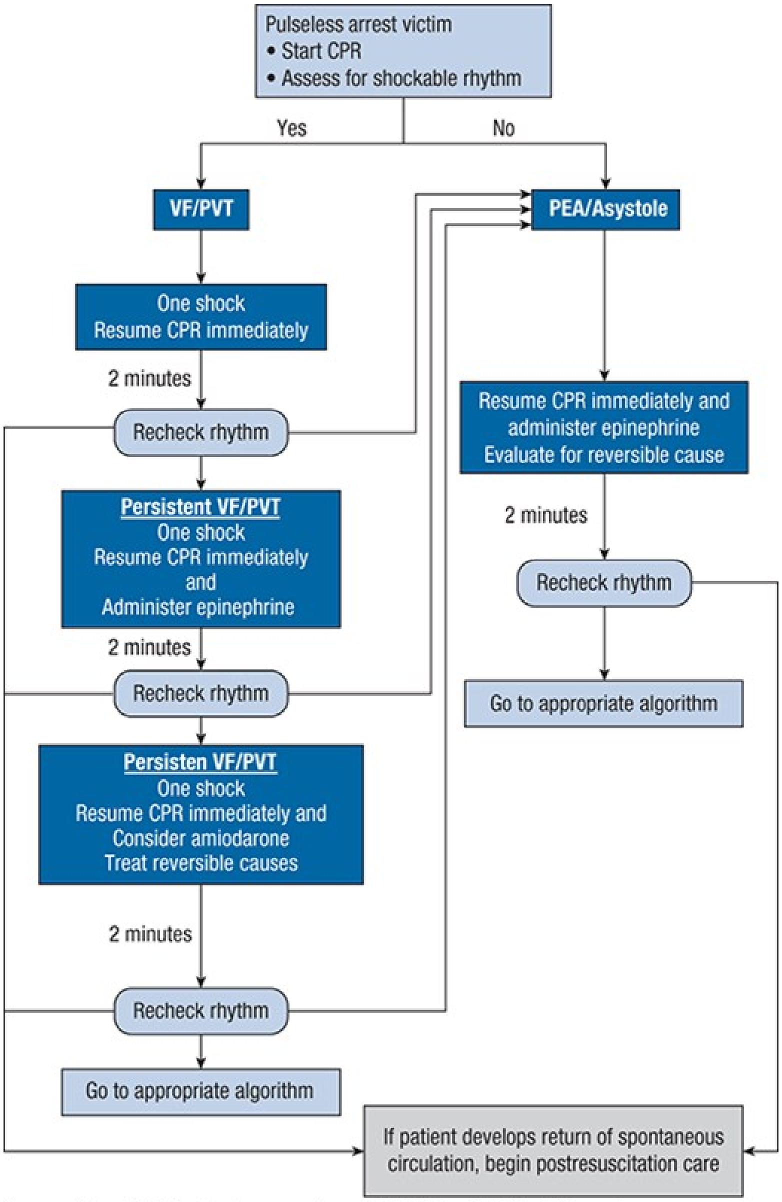
    ✔ If the rhythm is not shockable, resume CPR immediately for 2 minutes or until prompted by the AED. Continue this cycle until ACLS providers take over or the victim starts to move.

Advanced Cardiac Life Support

- Once ACLS providers arrive, further definitive therapy is given following the ACLS algorithm shown in **Figure 7-1**.
- A bag-mask device or an advanced airway (eg, endotracheal tube, supraglottic device) may be used to provide ventilation. One provider can deliver 1 breath every 6 seconds while a second provider performs continuous chest compressions.
- Central venous catheter access results in faster and higher peak drug concentrations than peripheral venous administration, but central line access is not needed in most resuscitation attempts. However, if a central line is already present, it is the access site of choice. If IV access (either central or peripheral) has not been established, insert a large peripheral venous catheter. If this is unsuccessful, insert an intraosseous (IO) device.
- If neither IV nor IO access can be established, **lidocaine**, **epinephrine**, **naloxone**, and **vasopressin** may be administered endotracheally. The endotracheal dose should generally be 2–2.5 times larger than the IV/IO dose. Dilute the medications in 5–10 mL of either sterile water (preferred) or normal saline.

FIGURE 7-1

Treatment algorithm for adult cardiac arrest: Advanced cardiac life support (ACLS).



Source: Terry L. Schwinghammer, Joseph T. DiPiro, Vicki L. Ellingrod, Cecily V. DiPiro: *Pharmacotherapy Handbook, 11e*

Treatment of Ventricular Fibrillation and Pulseless Ventricular Tachycardia

Nonpharmacologic Therapy

- Electrical defibrillation is the only effective method of restoring a cardiac rhythm for VF and PVT. For a witnessed adult cardiac arrest, use an AED immediately when available. When the arrest is not witnessed or when an AED is not immediately available, initiate CPR while the AED is being retrieved, and attempt defibrillation as soon as the device is available. For in-hospital cardiac arrest with an AED available, begin CPR while the AED is being placed.

- After 2 minutes of chest compressions, recheck the rhythm; if there is still evidence of VF or PVT, give pharmacologic therapy with repeat attempts at single-discharge defibrillation.

- Obtain IV access and an advanced airway when feasible, but not at the expense of stopping chest compressions. Once an airway is obtained, use caution to avoid providing excessive ventilations.

Pharmacologic Therapy

Epinephrine

- **Epinephrine** is a drug of first choice for treating VF, PVT, asystole, and PEA. It is an α- and β-receptor agonist, causing vasoconstriction and increased rate and forcefulness of heart contractions, thereby augmenting low coronary and cerebral perfusion pressures.

- The recommended adult dose of epinephrine is 1 mg administered by IV or IO injection every 3–5 minutes. Although higher doses have been studied, they are not recommended for routine use in cardiac arrest.

Vasopressin

- **Vasopressin** is a potent nonadrenergic vasoconstrictor that increases blood pressure (BP) and systemic vascular resistance. Despite some theoretical advantages, clinical outcomes are not superior to use of standard-dose epinephrine alone or to the combination of vasopressin and epinephrine. For in-hospital cardiac arrest, the combination of vasopressin, epinephrine, and methylprednisolone with post-arrest hydrocortisone may be considered, but further studies are needed before this can be routinely recommended.

Antiarrhythmics

- The purpose of antiarrhythmic drug therapy after unsuccessful defibrillation and vasopressor administration is to prevent development or recurrence of VF and PVT. However, clinical evidence demonstrating improved survival to hospital discharge is lacking.

- **Amiodarone** may be considered in patients with VF/VT unresponsive to CPR, defibrillation, and vasopressors. The dose is 300 mg IV/IO followed by a second dose of 150 mg.

- **Lidocaine** may be considered as an alternative to amiodarone for VF or PVT unresponsive to CPR, defibrillation, and a vasopressor, but it has not been shown to improve rates of ROSC, admission to the hospital, or survival to discharge. The initial dose is 1–1.5 mg/kg IV. Additional doses of 0.5–0.75 mg/kg can be administered at 5- to 10-minute intervals to a maximum total dose of 3 mg/kg if VF/PVT persists.

Magnesium

- Severe hypomagnesemia has been associated with VF/PVT, but routine administration of magnesium during cardiac arrest has not improved clinical outcomes. Two trials showed improved ROSC in cardiac arrests associated with torsades de pointes; therefore, limit magnesium administration to these patients. The dose is 1–2 g diluted in 10 mL of 5% dextrose in water administered IV/IO push over 15 minutes.

Thrombolytics

- Thrombolytic use during CPR has been investigated because most adult cardiac arrests are related to either myocardial infarction (MI) or pulmonary embolism (PE). Although early small studies demonstrated some benefit, larger randomized trials showed no improvement in ROSC or survival to hospital discharge, and the incidence of intracranial hemorrhage was greater than with placebo. Therefore, fibrinolytic therapy should not be used routinely in cardiac arrest but may be considered when PE is suspected.

Treatment of Pulseless Electrical Activity and Asystole

Nonpharmacologic Therapy

- Successful treatment of PEA and asystole depends on diagnosis of the underlying cause. Potentially reversible causes include: (1) hypovolemia, (2) hypoxia, (3) acidosis, (4) hyper- or hypokalemia, (5) hypothermia, (6) hypoglycemia, (7) drug overdose, (8) cardiac tamponade, (9) tension pneumothorax, (10) coronary thrombosis, (11) pulmonary thrombosis, and (12) trauma.

- PEA and asystole are treated the same way. Both conditions require CPR, airway control, and IV access. Avoid defibrillation in asystole because the resulting parasympathetic discharge can reduce the chance of ROSC and decrease the likelihood of survival. If available, transcutaneous pacing can be attempted.

Pharmacologic Therapy

- **Epinephrine** (1 mg by IV or IO injection every 3–5 minutes) is the primary pharmacologic agent used; vasopressin is no longer recommended.
- Atropine is no longer recommended for treatment of asystole or PEA because there are no prospective controlled trials showing benefit and the evidence from observational reports is inconsistent.

Acid–Base Management

- Acidosis occurs during cardiac arrest because of decreased blood flow or inadequate ventilation. Chest compressions generate only about 25% of normal cardiac output, leading to inadequate organ perfusion, tissue hypoxia, and metabolic acidosis. Lack of ventilation causes CO_2 retention, leading to respiratory acidosis. The combined acidosis reduces myocardial contractility and lowers the fibrillation threshold.
- Routine use of sodium bicarbonate in cardiac arrest is not recommended because there are insufficient data to support its use, and it may have detrimental effects. It can be used in special circumstances (eg, hyperkalemia, tricyclic antidepressant overdose, salicylate toxicity).

Postresuscitative Care

- ROSC from a cardiac arrest may be followed by a post-cardiac arrest syndrome characterized by hypoxic brain injury, myocardial dysfunction, systemic ischemia/reperfusion response, and the underlying persistent precipitating pathology.
- It is imperative to ensure adequate airway and oxygenation. After use of 100% oxygen during the resuscitation effort, titrate the oxygen fraction down as tolerated to maintain an oxyhemoglobin saturation of at least 94% (0.94). Overventilation can be avoided by using end-tidal (ET) CO_2 measurements.
- Evaluate for ECG changes consistent with acute MI immediately and perform revascularization if present.
- Because cerebral hypoperfusion may persist for several hours after resuscitation, augmenting BP to achieve a goal of mean arterial pressure (MAP) >80 mm Hg has been recommended.
- Therapeutic hypothermia or targeted temperature management is an integral component of postresuscitative care. Hypothermia can protect against cerebral injury by suppressing chemical reactions that occur after restoration of blood flow. All comatose adult patients should have targeted temperature management after ROSC, with a constant temperature maintained between 32° and 36°C for at least 24 hours. A rewarming phase should consist of slow and controlled warming at a rate of 0.2–0.5°C per hour. It is also reasonable to actively prevent fever after targeted temperature management. Potential complications of hypothermia include coagulopathy, dysrhythmias, bradycardia, diuresis, electrolyte disorders, hyperglycemia, infection risks, and effects on drug distribution and clearance.

EVALUATION OF THERAPEUTIC OUTCOMES

- The optimal outcome following CPR is an awake, responsive, spontaneously breathing patient. Patients must remain neurologically intact with minimal morbidity after resuscitation if it is to be considered successful.
- In many cases, rhythm assessment via ECG and pulse checks are the only physiologic parameters available to guide therapy. However, palpating a pulse to determine efficacy of blood flow during CPR has not been shown to be useful.
- Invasive hemodynamic monitoring (eg, coronary perfusion pressure, central venous oxygenation) can provide useful information during CPR but is seldom available. Arterial diastolic pressure may be a reasonable surrogate for coronary perfusion pressure with a suggested goal of >25 mm Hg. An arterial central venous oxygen saturation <30% (0.30) indicates poor CPR quality.

- $ETCO_2$ monitoring is a useful method to assess cardiac output during CPR and has been associated with ROSC. Persistently low $ETCO_2$ values (<10 mm Hg [1.3 kPa]) during CPR in intubated patients suggest that ROSC is unlikely.
- In the postresuscitative phase, monitoring should be directed toward the components of the post-cardiac arrest syndrome. Identify and treat the precipitating cause of the arrest (eg, coronary angiography with prompt recanalization if indicated). Optimize hemodynamics with avoidance of hypotension (MAP <65 mm Hg or SBP <90 mm Hg). Monitor oxygenation closely and maintain arterial blood oxygen saturation >94% (0.94). Because seizures can occur after cardiac arrest, EEG monitoring is indicated. Avoid hyperthermia and maintain normoglycemia. A complete review of systems is recommended because the post-cardiac arrest syndrome can affect most organ systems.

See Chapter 40, Cardiac Arrest, authored by Jeffrey F. Barletta, for a more detailed discussion of this topic.