Standard Shock and Resuscitation

Competency Applies fundamental knowledge to provide basic and selected advanced emergency care and transportation based on assessment findings for a patient in shock, respiratory failure or arrest, cardiac failure or arrest, and postresuscitation management.

TOPIC 23

CARDIAC ARREST AND RESUSCITATION

INTRODUCTION

Today, the wealth of information on the treatment of cardiac arrest far exceeds that in any other period in the history of prehospital medicine. Research is booming, and new strategies and tactics are evolving rapidly. It is certainly an exciting area of paramedic care, but with this evolution comes distinct responsibilities for providers to stay current and learn how new trends can affect the care they deliver.

Although the data set is still immature, evidence-based trends are driving the modern care of the out-of-hospital cardiac arrest patient. Debates about specific therapies may be ongoing, but immutable truths have begun to emerge. The first and foremost of these truths is that successful resuscitation is centered around quality, uninterrupted chest compressions and that for all the paramedic drugs and interventions, this basic skill is most meaningful.

The second evidence-based truth is that early defibrillation is essential. Research has demonstrated that these two links in the American Heart Association (AHA) chain of survival are most important when considering outcomes. Coupling early defibrillation with ongoing chest compressions maximizes the chance of successful resuscitation.

Many other therapies have evolved around these two truths. A changing paradigm of on-scene care is evolving. Furthermore, we are now realizing just how important postarrest care is as well.

As a paramedic, you must integrate these dynamic changes and recognize that treatment modalities will likely change as research continues on. Although you will most likely be driven by local protocol, it is essential that you understand the best strategies for caring for cardiac arrest.

TRANSITION highlights

- *Frequency of annual cardiac arrest rates and similar trends over the past several years.*
- *Pathophysiologic changes that occur in cardiac arrest; how quickly cells may become injured and die.*
- *Electrical changes to the heart that accompany cardiac arrest.*
- *Relationship between cardiac arrest and the metabolic changes that lead to cellular damage and death.*
- *Assessment findings indicative of cardiac arrest, and findings suggestive of a patient who may go into cardiac arrest.*
- *Importance of prehospital interventions and why they are successful in reversing cardiac arrest.*
- *The best way to integrate care interventions for a successful reversal of cardiac arrest:*
  - Difference in management of witnessed and unwitnessed cardiac arrests.
  - Provision of high-quality CPR.
  - Automated external defibrillator operation.
  - Proper airway and ventilation.
  - Importance of not interrupting compressions.

EPIDEMIOLOGY

Cardiovascular disease is the most prevalent chronic condition in the United States, as well as the leading contributor to death. It has been said that cardiovascular disease is actually a "young person's" disease process that results in "old age" complications. In other words, the way a younger person treats his body in the present time will have a direct impact on disease presence and progression by the time he reaches late adulthood. In fact, with many cardiovascular disease processes, the first clinical indication of its presence may well be the patient's first heart attack, stroke, or even sudden cardiac arrest (SCA).

The statistics representing cardiovascular disease are as alarming as the disease process itself. It has been estimated that more than 62 million Americans have some form of cardiovascular disease (this number does not include those yet to be diagnosed). As the result of cardiovascular disease, 1.5 million people will suffer a heart attack each year, which will result in cardiac arrest and the death of 500,000 of them.

Studies from 2009 indicate that up to 350,000 patients each year suffer cardiac arrest within one hour following the onset of the signs and symptoms, which means that the majority of cardiac arrests will occur in the prehospital environment—in fact, about 60 percent of all cardiac arrests are treated by EMS. In a more sobering light, about once every minute someone will collapse from SCA.

About once every minute someone will collapse from sudden cardiac arrest.

SCA is not always caused by cardiovascular disease. Statistics described previously have shown that cardiovascular disease is the leading cause of arrest; however, approximately 30 percent to 35 percent of cardiac arrests occur because of other etiologies. These include traumatic injuries (head and chest), nontraumatic hemorrhage (gastrointestinal [GI] bleeds, aortic rupture, intracranial bleeds), drug overdoses (accidental or purposeful), and pulmonary embolism.

During cardiac arrest, the normal electrical impulses are usually absent or disrupted, or the mechanical response to the electrical impulse does not occur.

Regardless of etiology, all cardiac arrest patients require the same assessment and treatment goals: rapid identification and assessment, airway maintenance, support of absent breathing, artificial circulatory support, provision of electrical therapy as warranted, and attempts at reversing or remediating the offending cause of the arrest situation.

PATHOPHYSIOLOGY

To better understand the signs and symptoms of cardiac arrest, the paramedic must first understand the basic pathophysiologic underpinnings related to the condition. *Cardiac arrest* (also known as *cardiopulmonary arrest* or *circulatory arrest*) is the cessation of normal circulation of the blood; if this is unexpected (as it often is), it can be termed a *sudden cardiac arrest*.

With cardiac arrest, ventricular contraction is absent or ineffective, resulting immediately in the cessation of blood flow and systemic circulatory failure—in fact, it is the final common pathway to human death. Although it is often difficult to determine the cause of cardiopulmonary arrest at the time of presentation, a working differential diagnosis of the causes can be formulated based on the patient's history, physical examination, and automated external defibrillator (AED) rhythm analysis.

With stoppage of the heart, blood flow ceases and no oxygenated blood is being delivered to the capillary beds of the body. Lack of blood flow initially causes pulselessness and unresponsiveness in the patient, but the lack of oxygen supply to the body's cells results in irreversible tissue damage and death.

All organs have different susceptibilities to ischemic injury from cardiac arrest, with the brain being the most vulnerable. Literature suggests that brain damage occurs after 4 to 6 minutes of normothermic cardiac arrest, and the damage is irreversible. The heart is the second most susceptible organ to ischemic injury. The renal, GI, musculoskeletal, and integumentary systems are much more resistant to ischemia than the heart and brain; these organs rarely sustain irreversible damage in patients who are successfully resuscitated. As such, for the best chance of survival and neurologic recovery, immediate and decisive treatment is imperative.

Cardiac arrest can also be caused by multiple etiologies, either medical or traumatic. As discussed, the primary risk factor for an acute coronary syndrome is coronary artery disease. Although cardiac arrest is commonly caused by an acute coronary syndrome, it can also be caused by myriad other conditions. For example, a traumatized patient may be in arrest following severe head trauma or significant blood loss.

A person who accidentally or intentionally overdoses on his medication may be in arrest owing to the effects of the medication or asphyxia. Even stroke or seizure patients may go into cardiac arrest secondary to permanent brain damage from inadequate oxygenation of cerebral tissue.

It is also important to note the common changes that occur to the electrical conduction system of the heart when a person experiences cardiac arrest, as specific treatment modalities may be necessary based on the heart rhythm. During cardiac arrest, the normal electrical impulses are usually absent or disrupted, or the mechanical response to the electrical impulse does not occur. In most situations of cardiac arrest caused by cardiovascular disease, instead of smooth, coordinated contractions the heart often shows a different type of electrical activity, most commonly the uncoordinated twitching known as *ventricular fibrillation*, which cannot produce any ventricular contraction.

Conversely, in some situations in which the cause of arrest was hemorrhagic or trauma related, the heart might show organized electrical activity without evidence of actual mechanical contraction or blood flow because of the loss of volume or direct damage to the heart muscle itself. The final common pathway in these and other arrest scenarios is that after cardiac arrest has existed for several minutes, the heart will eventually cease all electrical activity and the patient will "flatline" (an electrocardiogram [ECG] rhythm known as *asystole*). Asystole is the least viable rhythm, which most often leads to an unsuccessful resuscitation attempt.

Tissue metabolic concerns also must be taken into account when managing any patient in cardiac arrest. Research has indicated that, during cardiac arrest, the metabolic demands of the cells are no longer being met secondary to the lack of perfusion. Despite this, the cells still attempt to maintain functioning as long as possible with the residual oxygen and metabolic substrates remaining in the adjacent bloodstream. Eventually, the normal cellular activity and ongoing creation of energy (adenosine triphosphate [ATP]) becomes so deranged from a lack of perfusion that the cells cease aerobic metabolism in favor of anaerobic metabolism in an attempt to maintain ATP production.

The consequence of this metabolic shift is the creation of overwhelming acidosis that actually hastens ongoing cellular damage and death. This has a clinical consideration regarding the management of cardiac arrest (the provision of cardiopulmonary resuscitation, artificial ventilation, and defibrillation), based on the estimated downtime of the patient and whether or not bystander cardiopulmonary resuscitation (CPR) was started prior to EMS arrival. These considerations will be discussed more thoroughly in the "Emergency Medical Care" section of this topic.

The discussion thus far has centered on cardiac arrest in the adult patient. However, cardiac arrest can also occur in the pediatric patient—the most common etiologies are acute airway/breathing compromise or body system trauma. In children, the presenting cardiovascular change is typically caused by airway/breathing compromise with resulting bradycardia, which, left untreated, will eventually deteriorate into asystole. However, in about 15 percent to 20 percent of pediatric cardiac arrests, the patient may present with ventricular tachycardia or fibrillation, which is managed with the AED. Thus, the need for rapid defibrillation should be considered in children who meet AED utilization criteria with SCA not preceded by respiratory symptoms.

ASSESSMENT FINDINGS

Dispatch may provide information that will lead you to suspect cardiac arrest. Reports that a patient has no pulse or that emergency medical responders are performing CPR clearly indicate cardiac arrest. But also be alert to the possibility of cardiac arrest in calls to patients with other complaints, including chest pain or discomfort, difficulty in breathing, seizures, unresponsiveness, or serious motor vehicle crashes or other significant trauma.

Often the patient in cardiac arrest is not hard to identify. During the primary assessment, the paramedic will note the patient in cardiac arrest to be unresponsive, pulseless and apneic, and frequently cyanotic. The determination of pulselessness is commonly determined in a large core body artery, at either the carotid or femoral locations. Determining pulselessness can be difficult; therefore, a pulse check should be limited to 10 seconds or less. According to the 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care, unresponsiveness and absence of breathing or absence of normal breathing should be the key assessment indicators of cardiac arrest. Health care providers perform pulse checks simultaneously with assessment for unresponsiveness and apnea or agonal respirations.

Usually following cessation of blood flow, the patient will lose consciousness within 15 seconds; this may be followed by brief seizure activity. A near-immediate loss of bowel/bladder control may also occur with the onset of arrest. Agonal or gasping respirations may last up to 60 seconds. Pupils also typically dilate within 60 seconds of arrest.

Dependent lividity and rigor mortis, which are also associated with cardiac arrest patients, will not develop for hours after cardiac arrest occurs. If those signs are present, they are indicative of a prolonged downtime; in these cases, the patient should not be resuscitated. The following are the most important clinical indications of cardiac arrest:

- Unresponsiveness (usually occurs about 10 to 15 seconds after the heart stops)
- Absence of breathing or normal breathing (breathing may last up to 60 seconds following arrest)
- No detectable pulse (central)

Historical information gathered from on-scene family or bystanders may provide key information regarding etiology and potential outcome. If the cardiopulmonary arrest is witnessed, there exists a potential for bystander/health care provider CPR, which increases the likelihood of a successful resuscitation. If the cardiac arrest occurs prior to arrival of the EMS, then information obtained from family, bystanders, or other emergency personnel may provide key information that will assist in resuscitation of the patient. Important historical information includes the following questions:

- Was the arrest witnessed?
- How quickly was CPR started?
- Was an AED used?
- How much time passed since the arrest was first recognized (remembering that such estimates are often inaccurate)?
- What was the patient doing at the time of arrest and during the several hours just prior to the arrest?
- What are the patient's past medical history and current medications?

Not all patients with chest pain will experience cardiac arrest, nor will all unresponsive or traumatized patients. What is important to note is that these patients can rapidly deteriorate to cardiac arrest. In these situations, in which the patient is suspected to be in critical condition, it may be best to bring the monitor/defibrillator from the ambulance to the patient on initial approach.

EMERGENCY MEDICAL CARE

Although medical direction and protocol will ultimately determine what interventional steps are recommended or taken, the paramedic should follow the current guidelines for cardiopulmonary resuscitation as written by the AHA. Remember also the importance of the "chain of survival," which underscores the importance of a systematic approach toward cardiac arrest management.

The 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care emphasize the need to begin chest compressions in the cardiac arrest patient immediately and minimize any interruptions once the chest compressions are begun. Quality chest compressions provide around 25 percent of normal circulation and help cells stay alive as the heart arrhythmia is corrected. Appropriately performed chest compressions sustain adequate cerebral perfusion pressure (which perfuses brain tissue) and adequate coronary perfusion pressure (which perfuses heart muscle). Not only are these essential to sustain life, but they have also proven to be at least theoretical predictors of successful resuscitation.

Because of the importance of chest compressions, the standard ABC sequence of initial assessment is altered to CAB in suspected cardiac arrest patients. In cardiac arrest, chest compressions (C) are initiated immediately; after 30 compressions, the airway is opened (A), and the first two ventilations (B) are delivered. This sequence reduces the delay to first compressions and restores coronary and cerebral perfusion pressure as soon as possible. The following are key treatment considerations during the management of a patient in cardiac arrest:

1. Witnessed versus unwitnessed cardiac arrest. If the EMS personnel did not witness the cardiac arrest, immediately initiate CPR beginning with

Figure 23-1 Direct ventilation with high-concentration oxygen.

chest compressions. Attach the monitor/defibrillator but continue compressions until you are ready to analyze the rhythm. Cease compressions to analyze and defibrillate if necessary, and then return immediately to chest compressions following the defibrillation (or following the analyzation if a nonshockable rhythm is identified). CPR is then continued in 2-minute cycles (5 cycles of 30 compressions and 2 ventilations). At the conclusion of the 2-minute cycle, the rhythm will be analyzed again and a pulse check performed if an organized rhythm is identified.

If the paramedic witnessed the cardiac arrest, the goal is to defibrillate within 3 minutes from the onset of cardiac arrest (or as soon as possible). As in an unwitnessed arrest, chest compressions should be initiated while the monitor defibrillator is prepared, but they should not delay immediate defibrillation when indicated.

2. Open the airway, insert an oropharyngeal airway, and ventilate the patient with a bag-valve mask and 100 percent oxygen (Figure 23-1). Ventilate the patient carefully, being sure not to exceed the recommended ventilation rate of one ventilation after every 30 compressions (8 to 10/minute). Research has indicated that overventilation (ventilations that are either too fast or deliver too large a tidal volume) is detrimental to cardiac output in arrested patients. Remember, in the cardiac arrest patient, chest compressions precede opening the airway and ventilation (CAB). Adequate resources are often available in the prehospital setting to perform airway management simultaneously with chest compressions. The key is to not interrupt or compromise the effectiveness of chest compressions at any time.

In nonarrest states, blood is returned to the heart via muscular contraction of the muscles that "milk" the veins in conjunction with the one-way valves in the veins' lumina. Blood also returns to the heart via gravity from the head and upper torso and because of the negative intrathoracic pressure created during spontaneous breathing (known as the *cardiothoracic pump*). In a cardiac arrest, obviously the patient will be motionless, which eliminates blood return facilitated by muscular contraction. Furthermore, the arrested patient is typically in a supine position; therefore, gravity cannot facilitate blood return to the heart. Finally, if the patient is apneic and positive pressure ventilation (PPV) is being provided, there is minimal generation of negative intrathoracic pressure during the recoil of chest compressions to facilitate blood flow to the lungs.

Although the first two influences (supine position and lack of motion) cannot realistically be mediated by the paramedic, the third influence (provision of PPV) and its effect on the cardiothoracic pump can be at least minimized by providing ventilations at a rate of 8 to 10 per minute, with a tidal volume just sufficient to achieve visible chest rise. Only after an advanced airway is in place should ventilations be delivered asynchronously to chest compressions at a rate of 8 to 10 ventilations per minute. In support of this, it has been shown through recent research that, historically, the patient in cardiac arrest has been overventilated by care providers, which in turn diminishes cardiac output states during compressions by altering cardiac preload. Ultimately, it was shown that overventilation is negatively correlated with cardiac arrest survival rates.

As a paramedic, you may also have to make a decision about the most appropriate method to manage the airway. Although the airway in cardiac arrest patients is by default unsecure, definitive airway management in the form of endotracheal intubation (ETI) may not be the most appropriate intervention. Even though the net result of ETI is a secure airway, the process of placing the tube likely interrupts chest compressions for an unacceptable length of time. Because chest compressions are so vital to a successful outcome, the paramedic must weigh the benefit of a secured airway against the cost of interrupted compressions. Remember also that blind insertion airway devices such as the King airway and the laryngeal mask airway do not interrupt compressions and may provide a viable alternative to ETI in the cardiac arrest setting.

3. Assess the pulse and provide cardiac compressions. As noted previously, assess a core pulse location for determining whether cardiac compressions are warranted. Traditionally, the carotid pulse is used for this determination, but some research has shown that lay providers and health care providers alike may have trouble determining whether the pulse is present or absent in a hemodynamically unstable or critical patient. Therefore, quickly (maximum 10-second count) assess the pulse (Figure 23-2). This is done correctly by being sure to assess the pulse on the same side as the paramedic, assessing with the fingertips of the first and second digit (do not use the thumb), and never assessing for carotid

Figure 23-2 Checking the patient's carotid pulse (maximum 10 seconds).

pulses simultaneously on each side of the neck. If the patient is unresponsive, with no breathing or agonal breathing, and a central pulse cannot be quickly located, immediately begin chest compressions. Do not waste precious compression time trying to ensure that the patient is pulseless. Chest compressions delivered to patients who are not pulseless rarely lead to significant injury.

The second concern with assessing the pulse and providing compressions is the fashion in which the paramedic delivers external compressions. Since 2005, the AHA has been advocating "push hard, push fast" to underscore the importance of high-quality compressions in increasing survival rates of arrested patients. It has been well documented in previous research that even perfectly performed compressions can achieve only a small portion of normal cardiac output. It has been more recently documented in the literature that shallow compressions, slow compressions, or frequent interruptions of compressions result in reduced cardiac output, which ultimately translates into poor survival rates. Research has shown that compressions that are delivered at a rapid rate (100/min) and depress the sternum to a depth of at least 2 inches with complete recoil of the chest wall will optimize the effectiveness of compression.

The paramedic must also realize that any interruption of compressions, even for brief periods, causes the cardiac output and coronary perfusion to drop to nothing. On resumption of compressions it can take up to 45 seconds of constant compressions to return cardiac output to what it was prior to ceasing compressions. As such, always minimize the number of times you have to stop compressions and minimize the length of time compressions have to be stopped (this is also why the compression/ventilation ratio is now 30:2 and why the initial focus in cardiac arrest is on chest compressions).

Advocating and performing the "push hard, push fast" approach to compressions at a 30:2 ratio is correlated with increased cardiac output and is correlated with higher survival rates in arrested patients.

4. Examine other cardiac arrest considerations. Additional CPR adjuncts and interventions may become more commonplace during prehospital management of arrested patients. One device is a mechanical CPR adjunct that will alleviate one person from performing CPR. Because it is mechanical in nature, it may provide more consistent compressions; however, no adjunct to date has been proven to be superior to standard manual CPR (▶ Figure 23-3).

5. Consider postarrest care. Cardiac arrest is but a symptom of a larger problem; although it requires immediate resuscitative efforts, paramedics should never lose sight that there is some underlying cause that made the arrest occur. Once resuscitation is successful, paramedics should rapidly shift their focus toward identifying and treating this cause. The most likely concern (and most likely cause of cardiac arrest) is acute coronary syndrome. Paramedics should consider a 12-lead ECG following return of a perfusing rhythm and also consider transport to an appropriate facility capable of restoring coronary blood flow or treating other underlying causes. Another intervention is the use of therapeutic hypothermia during the postarrest resuscitation phase. It has been learned through controlled clinical trials that by carefully lowering the body core temperature, metabolic demands decrease, edema diminishes, and increased survival rates have been realized. This intervention is implemented into some EMS protocols and, as a paramedic, you may initiate this therapy. Always follow local protocols.

OTHER ADVANCED CARDIAC LIFE SUPPORT CHANGES

The 2010 AHA Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care contain several other changes that are significant to paramedic-level care. The highlights include the following:

- **Removal of atropine from the asystole and pulseless electrical activity treatment algorithm.** The 2010 Guidelines state, "Available evidence

Figure 23-3 The AutoPulse™ Model 100: (A) applied to a patient; (B) close-up view.

suggests that the routine use of atropine during PEA or asystole is unlikely to have a therapeutic benefit. For this reason atropine has been removed from the cardiac arrest algorithm."

- **Waveform capnography in cardiac arrest.** Recent research has shown that using waveform capnography in cardiac arrest care may be valuable. During cardiac arrest, CO_2 is not returned to the lungs; therefore, $PETCO_2$ (exhaled CO_2) will be relatively nonexistent. When chest compressions are initiated, blood flow to the lungs is at least partially restored and $PETCO_2$ should become detectable. $PETCO_2$ therefore correlates well with cardiac output during chest compressions; trending can be used to assess the adequacy of CPR and, according to the 2010 Guidelines, "optimize compression depth and rate and detect fatigue in the provider performing compressions." Abrupt sustained increase in $PETCO_2$ during CPR can also indicate the return of spontaneous circulation.
- **Vasopressin.** Topic 15, "Medication Administration," discussed the role of vasopressin in cardiac arrest. However, in summary, it can be said that vasopressin is a nonadrenergic peripheral vasoconstrictor that also causes coronary and renal vasoconstriction. It is used in cardiac arrest care to provide effects similar to epinephrine (although through a slightly different mechanism). The 2010 Guidelines note:

> Because the effects of vasopressin have not been shown to differ from those of epinephrine in cardiac arrest, 1 dose of vasopressin 40 units IV/IO may replace either the first or second dose of epinephrine in the treatment of cardiac arrest. Although there is little evidence to suggest vasopressin performs better than epinephrine, some systems may implement this drug into their prehospital formularies.

Although specific chronological interventions for the management of a cardiac arrest patient in the prehospital environment were not discussed in their entirety, the previous discussion was intended to address the current research and physiology regarding cardiac arrest management and how to maximize the effects of the interventions to improve resuscitation rates. Treatment for the patient in cardiac arrest must be delivered in a timely manner, without error. Failure to do so will invariably lead to lower success rates in patient resuscitation.

When caring for a patient in cardiac arrest, always do your best in each of the interventions you are providing. Remember to "push hard, push fast" with compressions so that the brain and heart can still receive blood flow during arrest management. Ensure adequate ventilations, and provide oxygen during the arrest as well. Minimize the amount of time that CPR is interrupted.

The AED should be used specifically as addressed earlier in this topic so that patients in ventricular fibrillation or pulseless ventricular tachycardia will have the greatest opportunity for survival. Patients in nonventricular fibrillation cardiac arrest should also receive this high level of care so that they, too, may have the greatest chance for survival.

The person with impending cardiac arrest, in cardiac arrest, or just coming out of cardiac arrest is probably one of the most challenging and dynamic patients the paramedic will ever encounter. The situation requires a thorough understanding of the body, application of multiple skills simultaneously, and coordination of multiple EMS providers—all done in the shortest time possible so the patient can be transported to the hospital for definitive care.

TRANSITIONING

REVIEW ITEMS

1. Of the following clinical findings, which is most reliable for determining whether the patient is in cardiac arrest or not?
   a. unresponsiveness
   b. brief seizure activity
   c. agonal breathing pattern
   d. absence of a core pulse
2. What is the most significant contributor to cardiac arrest in the prehospital environment?
   a. trauma
   b. cardiovascular disease
   c. gastrointestinal hemorrhage
   d. occlusion of a cerebral artery
3. A patient is found in cardiac arrest; no bystanders or family were present. All that is known is that it took EMS 8 minutes to arrive. Given this, the first intervention should be to ______.
   a. start CPR
   b. attach the defibrillator
   c. contact medical direction
   d. initiate ventilations at 12 per minute
4. Which of the following would best describe why a blind insertion airway device (BIAD) might be preferred to endotracheal intubation in the treatment of a cardiac arrest patient?
   a. A BIAD secures the airway more definitively.
   b. Insertion of a BIAD does not interrupt chest compressions.
   c. Endotracheal intubation improves blood return to the heart.
   d. Hyperventilation is more likely when an endotracheal tube is in place.
5. Overventilation of the patient in cardiac arrest can have what detrimental effect?
   a. inability to properly compress the sternum
   b. failures of the AED to read the cardiac rhythm correctly
   c. decrease in cardiac output achieved through compressions
   d. hyperventilation causing hyperoxemia, which damages the central nervous system

APPLIED PATHOPHYSIOLOGY

While nearing the end of your 12-hour night shift, you are paged for a possible cardiac arrest in a 67-year-old man. On arrival at the scene nine minutes later, the family member who found the patient states that he was "fine last night." Currently nothing is being done for the patient, who is in fact pulseless and apneic. The patient is still warm to the touch, there is cyanosis to the fingertips and hands, and no rigor or dependent lividity is present.

1. Should the paramedic initiate CPR or apply the defibrillator first, assuming that both could be done simultaneously?

2. Explain your rationale in support of your answer to the preceding question. What clinical condition or change in the circumstances would have to be present for you to change your answer?

3. Describe the anticipated physiologic benefit(s) for each of the interventions that may be administered to a patient suffering from a cardiac arrest:
   a. PPV delivered at a rate of 10–12/min
   b. Providing compressions at a rate >100/min
   c. Use of defibrillation after CPR has been initiated in an unwitnessed cardiac arrest

4. What three clinical findings are most reliable for determining that an adult patient is in cardiac arrest?

5. What are four or five specific questions regarding the patient's history that the paramedic should try to ascertain as rapidly as possible when confronted with a patient in cardiac arrest?

6. What is the most common underlying etiology to pediatric cardiac arrest, and what is typically the presenting cardiac rhythm in these patients?

CLINICAL DECISION MAKING

You are transporting to the hospital a 59-year-old obese patient with a history of hypertension and diabetes secondary to chest pain. Thus far, you have placed the patient in a position of comfort, administered oxygen via nonrebreather mask, and assisted in the administration of two doses of nitroglycerin. While reassessing the patient's vital signs, he moans loudly and then suddenly loses consciousness. You then witness him experiencing a mild full-body seizure that lasts about 10 seconds. You can visually see the patient is now apneic, and his skin is rapidly becoming ashen in color.

1. Based on the information provided, what assessment parameter should the paramedic determine first, before initiating any other patient care interventions?

2. What cardiac rhythm disturbance does this patient most likely have?

After confirming that the patient is unresponsive to external stimuli and is apneic and pulseless, as you reach for the defibrillator, the ambulance stops and paramedic from a backup unit from the same EMS company climbs into the back of the ambulance with you, and you are again en route to the hospital with lights and sirens.

3. What are your immediate interventions? Support your answer with an understanding of the metabolic changes seen in cardiac arrest.

After another 5 minutes of transport time toward the hospital, you now have an airway in place, compressions are ongoing, and the patient is receiving positive pressure ventilation via bag-valve mask attached to high-flow oxygen. You notice that your partner is ventilating the patient at a rate of about 18 per minute.

4. Is this an appropriate rate for an adult patient in cardiac arrest? If not, what is the appropriate rate?

5. What detrimental effects, if any, may occur secondary to the overventilation of a patient in cardiac arrest?