For reasons that are still not completely understood, a brittle plaque will sometimes develop a crack, exposing the inside of the atherosclerotic wall. Acting like a torn blood vessel, the ragged edge of the crack activates the blood-clotting system, just as when an injury has caused bleeding. In this situation, however, the resulting blood clot will partially or completely block the lumen of the artery. If it does not occlude the artery at that location, the blood clot may break loose and begin floating in the blood, becoming what is known as a thromboembolism. A **thromboembolism** is a blood clot that is floating through blood vessels until it reaches an area too narrow for it to pass, causing it to stop and block the blood flow at that point. Tissues downstream from the blood clot will experience a lack of oxygen (hypoxia). If blood flow is restored in a short time, the hypoxic tissues will recover. However, if too much time goes by before blood flow returns, the hypoxic tissues will die. If a blockage occurs in a coronary artery, the condition results in an **acute myocardial infarction (AMI)**, a heart attack (**FIGURE 17-9**). **Infarction** means the death of tissue. The same sequence may also cause the death of cells in other organs, such as the brain. The death of heart muscle decreases the heart's ability to pump and can also cause it to stop pumping completely (**cardiac arrest**).

In the United States, coronary artery disease is the number one cause of death for men and women. The peak incidence of heart disease is between the ages of 45 and 64 years, but it can also strike teens and people in their 90s. You must be alert to the possibility that, although less likely, a 26-year-old with chest pain could be having an AMI, especially if he or she has a higher than usual risk.

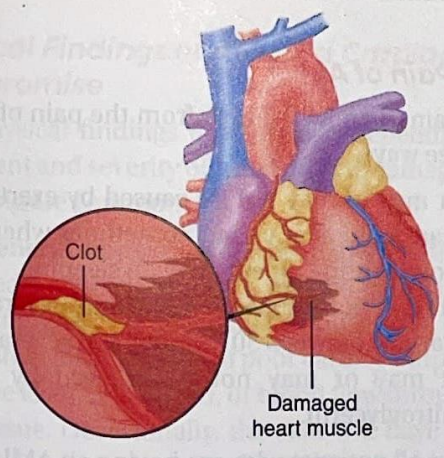

FIGURE 17-9 An acute myocardial infarction (heart attack) occurs when a blood clot prevents blood flow to an area of the heart muscle. If left untreated, death of myocardium can result.

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Factors that place a person at higher risk for an AMI are called risk factors. The major controllable factors are cigarette smoking, high blood pressure, elevated cholesterol level, elevated blood glucose level (diabetes), lack of exercise, and obesity. The major risk factors that cannot be controlled are older age, family history of atherosclerotic coronary artery disease, race, ethnicity, and male sex. Other factors that play a role in heart disease are stress, excessive alcohol, and poor diet.

Acute Coronary Syndrome

Many patients who call for EMS assistance because of chest pain have acute coronary syndrome. **Acute coronary syndrome (ACS)** is a term used to describe a group of symptoms caused by myocardial ischemia. As discussed earlier, myocardial ischemia is a decrease in blood flow to the heart, which leads to chest pain through reduced supply of oxygen and nutrients to the tissues of the heart. This can be a temporary situation known as angina pectoris, or a more serious condition, an AMI. Because the signs and symptoms of these two conditions are very similar, they are treated the same under the designation of ACS. To understand them better, we will examine each one separately.

Angina Pectoris

Chest pain does not always mean that a person is having an AMI. When, for a brief time, heart tissues are not getting enough oxygen, the pain is called **angina pectoris**, or angina. Although angina can result from a spasm of an artery, it is most often a symptom of atherosclerotic coronary artery disease. Angina occurs when the heart's need for oxygen exceeds its supply, usually during periods of physical or emotional stress when the heart is working hard. A large meal or sudden fear may also trigger an attack. When the increased oxygen demand goes away (eg, the person stops exercising), the pain typically goes away.

Anginal pain is commonly described as crushing, squeezing, or "like somebody standing on my chest." It is usually felt in the midportion of the

chest, under the sternum. However, it can radiate to the jaw, the arms (frequently the left arm), the midportion of the back, or the epigastrium (the upper-middle region of the abdomen). The pain usually lasts from 3 to 8 minutes, rarely longer than 15 minutes. It may be associated with shortness of breath, nausea, or sweating. It usually disappears promptly with rest, supplemental oxygen, or nitroglycerin (NTG), all of which decrease the need for or increase the supply of oxygen to the heart. Although angina pectoris is frightening, it does not mean that heart cells are dying, nor does it usually lead to death or permanent heart damage. It is, however, a warning that you and the patient should take seriously. With angina, the electrical system can be compromised because the oxygen supply to the heart is diminished, and the person is at risk for problems with cardiac rhythm.

Angina can be further differentiated into stable and unstable angina. Unstable angina is characterized by pain or discomfort in the chest of coronary origin that occurs in the absence of a significant increase in myocardial oxygen demand. If untreated, it is associated with a very high risk of spontaneous AMI. Stable angina is characterized by pain in the chest of coronary origin that occurs in response to exercise or some activity that increases the demand on the heart muscle beyond the heart's capacity to increase its own blood flow. EMS often becomes involved when stable angina becomes unstable, such as when a patient whose pain is normally relieved by sitting down and taking one nitroglycerin tablet has taken three tablets with no relief. Keep in mind that it can be difficult, even for physicians in hospitals, to distinguish between the pain of angina and the pain of an AMI. Patients experiencing chest pain or discomfort, therefore, should always be treated initially as if they are having an AMI.

Acute Myocardial Infarction

The pain of an AMI signals the actual death of cells in the area of the heart muscle where blood flow is obstructed. Once dead, the cells cannot be revived. Instead, they will eventually turn to scar tissue and become a burden to the beating heart. Therefore, fast action is critical in treating a heart attack. The sooner the arterial blockage can be cleared, the fewer the cells that may die. About 30 minutes after blood flow is cut off, some heart muscle cells begin to die. After about 2 hours, as many as one-half of the cells in the area can be dead; in most cases, after 4 to 6 hours, more than 90% will be dead. In many cases, however, opening the coronary artery with clot-busting (thrombolytic) medications or angioplasty (mechanical clearing of the artery) can prevent permanent damage to the heart muscle if done within the first few hours after the onset of symptoms. Therefore, immediate prehospital treatment and transport to the emergency department (ED) are essential.

An AMI is more likely to occur in the larger, thick-walled left ventricle, which needs more blood and oxygen than the right ventricle.

Signs and Symptoms of AMI

A patient with an AMI may show any of the following signs and symptoms:

- Sudden onset of weakness, nausea, and sweating without an obvious cause
- Chest pain, discomfort, or pressure that is often crushing or squeezing and that does not change with each breath
- Pain, discomfort, or pressure in the lower jaw, arms, back, abdomen, or neck
- Irregular heartbeat and **syncope** (fainting)
- Shortness of breath, or dyspnea
- Nausea/vomiting
- Pink, frothy sputum (indicating possible pulmonary edema)
- Sudden death

The Pain of AMI

The pain of an AMI differs from the pain of angina in three ways:

- It may or may not be caused by exertion but can occur at any time, sometimes when a person is sitting quietly or even sleeping.
- It does not resolve in a few minutes; rather, it can last between 30 minutes and several hours.
- It may or may not be relieved by rest or nitroglycerin.

Not all patients who are having an AMI experience pain or recognize it when it occurs. Approximately one-third of patients never seek medical attention. This can be attributed, in part, to the fact that people are afraid of dying and do not want to

face the possibility that their symptoms may be serious (cardiac denial). Middle-aged men, in particular, are likely to minimize their symptoms. However, some patients, particularly older people, women, and people with diabetes, may not experience any pain during an AMI but may have other common complaints associated with ischemia discussed earlier. Others may feel only mild discomfort and call it indigestion. It is not uncommon for the only complaint, especially in older patients and women, to be fatigue. AMI without the classic chest pain is often referred to as a silent myocardial infarction. Heart disease is the number one killer of women in the United States, and EMTs should consider AMI even when the classic symptom of chest pain is not present. This is also true for older people and people with diabetes.

Therefore, when you are called to a scene where the chief complaint is chest pain, complete a thorough assessment, no matter what the patient says. Patients with cardiac risk factors should also be carefully assessed if they have any of the associated symptoms, even if no chest pain is present. Any complaint of chest discomfort is a serious matter. The best thing you can do is to assume the worst.

Street Smarts

If it appears a patient is having a heart attack, be sure the AED is close by. That way, if it is needed, the delay to the first shock will be minimized.

Physical Findings of AMI and Cardiac Compromise

The physical findings of AMI vary, depending on the extent and severity of heart muscle damage. The following are common:

- **General appearance.** The patient often appears frightened. There may be nausea, vomiting, and a cold sweat. The skin is often pale or ashen gray because of poor cardiac output and the loss of perfusion, or blood flow through the tissue. Occasionally, the skin will have a blue tint, called cyanosis; this is the result of poor oxygenation of the circulating blood.
- **Pulse.** Generally, the pulse rate increases as a normal response to pain, stress, fear, or actual injury to the myocardium. Because dysrhythmias are common in an AMI, you may feel an irregularity or even a slowing of the pulse. The pulse may also be dependent on the area of the heart that has been affected by the AMI. Damage to the inferior area of the heart often presents with bradycardia.
- **Blood pressure.** Blood pressure may fall as a result of diminished cardiac output and diminished capability of the left ventricle to pump. However, most patients with an AMI will have a normal or possibly even elevated blood pressure.
- **Respiration.** The respiratory rate is usually normal unless the patient has CHF. In that case, respirations may become rapid and labored with a higher likelihood of cyanosis and possibly frothy sputum. A complaint of difficulty breathing is common with cardiac compromise, so even if the rate seems normal, look at the work of breathing, and treat the patient as if respiratory compromise were present.
- **Mental status.** Patients with AMIs often experience confusion or agitation and sometimes experience an almost overwhelming feeling of impending doom. If a patient tells you, "I feel like I'm going to die," pay attention.

Words of Wisdom

Documenting exactly how a patient describes chest discomfort, in the patient's own words, is a valuable source of information for hospital staff. Remember OPQRST (Onset, Provocation/Palliation, Quality, Radiation, Severity, Time of onset).

Consequences of AMI

An AMI can have three serious consequences:

- Sudden death
- Cardiogenic shock
- Congestive heart failure

Sudden Death

Sudden death is usually the result of cardiac arrest, in which the heart fails to generate effective blood flow. Although you cannot feel a pulse in someone experiencing cardiac arrest, the heart may still be twitching, though erratically. The heart is using up energy without pumping any blood. Such an

abnormality of heart rhythm is a ventricular **dysrhythmia**, known as ventricular fibrillation (VF).

A variety of other lethal and nonlethal dysrhythmias may follow an AMI, usually within the first hour. In most cases, premature ventricular contractions, or extra beats in the damaged ventricle, occur. Premature ventricular contractions by themselves may be harmless and are common among healthy people, as well as sick people. Other dysrhythmias include the following:

- **Tachycardia**. Rapid beating of the heart, 100 beats/min or more.
- **Bradycardia**. Unusually slow beating of the heart, 60 beats/min or less.
- **Ventricular tachycardia**. Rapid heart rhythm, usually at a rate of 150 to 200 beats/min. The electrical activity starts in the ventricle instead of the atrium. This rhythm usually does not allow adequate time between beats for the left ventricle to fill with blood. Therefore, the heart pumps less volume and the patient's blood pressure may fall, or the pulse may be lost altogether. The patient may also feel weak or lightheaded or may even become unresponsive. In some cases, existing chest pain may worsen or chest pain that was not there before onset of the dysrhythmia may develop. Most cases of ventricular tachycardia (VT) will be sustained but may deteriorate into VF.
- **Ventricular fibrillation**. Disorganized, ineffective quivering of the ventricles. No blood is pumped through the body, and the patient becomes unconscious within seconds. The only way to convert this dysrhythmia is to defibrillate the heart. To **defibrillate** means to shock the heart with a specialized electric current in an attempt to stop the chaotic, disorganized contraction of the myocardial cells and allow them to start again in a synchronized manner to restore a normal rhythmic beat. Defibrillation is most likely to be successful in terms of saving a life if delivered within the first few minutes of sudden death. If a defibrillator is not immediately available, CPR must be initiated until the defibrillator arrives. Even if CPR is begun at the time of collapse, chances of survival diminish approximately 7% to 10% each minute until defibrillation is accomplished.

If uncorrected, unstable VT or VF will eventually lead to **asystole**, the absence of all heart electrical activity. Without CPR, asystole may occur within minutes. Asystole usually reflects a long period of ischemia, and nearly all patients you find in asystole will die.

Cardiogenic Shock

Shock is a simple concept but one that few people without medical training really understand. For that reason, Chapter 13 is devoted to a discussion of shock. The discussion of shock in this chapter is limited to that associated with cardiac problems; however, many other medical problems may cause shock as well (**TABLE 17-1**).

Shock is present when body tissues do not get enough oxygen, causing body organs to malfunction. In **cardiogenic shock**, often caused by a heart attack, the problem is that the heart lacks enough power to force the proper volume of blood through the circulatory system. Cardiogenic shock is more commonly found after an AMI that affects the inferior and posterior regions of the left ventricle of the heart because this ventricle provides circulation to the majority of the body. Cardiogenic shock can develop immediately or sometime after an AMI. The various signs and symptoms of cardiogenic shock are produced by the improper functioning of the body's organs. The challenge for you is to recognize shock in its early stages, when treatment is much more likely to be successful.

Congestive Heart Failure

Failure of the heart occurs when the ventricular myocardium is so profoundly damaged that it can no longer keep up with the return flow of blood from the atria. **Congestive heart failure (CHF)** can occur at any time after a myocardial infarction, in the setting of heart valve damage, or as a consequence of long-standing high blood pressure. Any condition that weakens the pumping strength of the heart may cause CHF, and this often happens after a heart attack.

Just as the pumping function of the left ventricle can be damaged by coronary artery disease, it can also be damaged by diseased heart valves or chronic hypertension. In any of these cases, when the muscle can no longer contract effectively, the

TABLE 17-1 Cardiogenic Shock and Congestive Heart Failure Facts

Cardiogenic Shock

Signs and Symptoms

- One of the first signs of shock is anxiety or restlessness as the brain becomes relatively starved for oxygen. The patient may report "air hunger." Think of the possibility of shock when the patient is saying that he or she cannot breathe. Obviously, the patient can breathe, because he or she can talk. However, the patient's brain is sensing that it is not getting enough oxygen.
- As the shock continues, the body tries to send blood to the most important organs, such as the brain and heart, and away from less important organs, such as the skin. Therefore, you may see pale, cool, clammy skin in patients with shock.
- As the shock gets worse, the body will attempt to compensate by increasing the amount of blood pumped through the heart. Therefore, the pulse rate will be higher than normal. In severe shock, the heart rate usually, but not always, is greater than 120 beats/min. As the shock progresses, the pulses may become irregular and weak.
- Shock can also present with rapid and shallow breathing, nausea and vomiting, and a decrease in body temperature.
- Finally, as the heart and other organs begin to malfunction, the blood pressure will fall below normal. A systolic blood pressure less than 90 mm Hg is easy to recognize, but it is a late finding that indicates decompensated shock. It is important not to assume that shock is not present just because the blood pressure is normal (compensated shock).

Treatment of Cardiogenic Shock

Take the following steps when treating patients with signs and symptoms of cardiogenic shock:

1. Position the patient comfortably. Some patients will be more comfortable in a semi-Fowler position (head and knees slightly elevated); however, patients with low blood pressure may not tolerate a semi-upright position and may be more comfortable and more alert in a supine position.
2. Administer oxygen at a rate to keep the oxygen saturation between 95% and 99%.
3. Assist ventilations as necessary.
4. Cover the patient with sheets or blankets as necessary to preserve body heat. Be sure to cover the top of the patient's head in very cold weather, as this is where much heat is lost.
5. Provide prompt transport to the ED.

Congestive Heart Failure

Signs and Symptoms

- This patient usually finds it easier to breathe when sitting up. When the patient is lying down, more blood is returned to the right ventricle and lungs, causing further pulmonary congestion.
- The patient is often agitated.
- Chest pain may or may not be present.
- The patient often has distended neck veins that do not collapse even when the patient is sitting.
- The patient may have swollen ankles from dependent edema (backup of fluid).
- The patient generally will have high blood pressure, a rapid heart rate, and rapid respirations.
- The patient will usually be using accessory breathing muscles of the neck and ribs, reflecting the additional hard work of breathing.
- Skin is usually pale or cyanotic and sweaty.
- The fluid surrounding small airways may produce crackles (formerly known as rales), best heard by listening to either side of the patient's chest, about midway down the back. In severe CHF, these soft sounds can be heard even at the top of the lung.

Once CHF develops, it can be treated but not cured. Regular use of medications may alleviate the symptoms. However, patients with CHF often become ill again and are frequently hospitalized. Approximately one-half will die within 5 years of the onset of symptoms.

Treatment of CHF

Treat a patient with CHF and pulmonary edema urgently. Call for ALS back-up.

1. Take the vital signs, and give oxygen by continuous positive airway pressure (CPAP) to move some of the fluid out of the lungs and improve oxygenation. If CPAP is not available or not tolerated by the patient, you may give oxygen by mask or cannula to maintain the oxygen saturation between 95% and 99%.
2. Allow the patient to remain sitting in an upright position with the legs down.
3. Be reassuring; many patients with CHF are quite anxious because they feel as if they cannot breathe.
4. Patients who have had problems with CHF before will usually have specific medications for its treatment. Gather these medications and take them along with you to the hospital.
5. Nitroglycerin may be of value in reducing pulmonary edema if the patient's systolic blood pressure is more than 100 mm Hg. If medical control or standing orders advise you to do so, administer nitroglycerin sublingually.
6. Prompt transport to the ED is essential.

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heart tries other ways to maintain an adequate cardiac output. Two specific changes in heart function occur: The heart rate increases, and the left ventricle enlarges to increase the amount of blood pumped each minute.

When these adaptations can no longer make up for the decreased heart function, CHF eventually develops. With left-sided heart failure, the lungs become congested with fluid because the left side of the heart fails to pump the blood effectively. Blood tends to back up in the pulmonary veins, increasing the pressure in the capillaries of the lungs. When the pressure in the capillaries exceeds a certain level, fluid (mostly water) passes through the walls of the capillary vessels and into the alveoli. This condition is called pulmonary edema. It may occur suddenly, as in an AMI, or slowly over months, as in chronic CHF. Sometimes, in patients with an acute onset of CHF, severe pulmonary edema will develop, in which the patient has pink, frothy sputum and severe dyspnea. With right-sided heart failure, blood backs up in the venae cavae, resulting in edema in the lower extremities or distention of the veins in the neck.

If the right side of the heart is damaged, fluid collects in the body, often showing up as swelling in the feet and legs. The collection of fluid in the part of the body that is closest to the ground is called **dependent edema**. The swelling causes relatively few symptoms other than discomfort. However, chronic dependent edema may indicate underlying heart disease even in the absence of pain or other symptoms. Because the right side of the heart supplies the preload for the left side of the heart, right heart failure can result in an inadequate supply of blood to the left ventricle, resulting in a drop in the systemic blood pressure. It is important to realize that some patients may present with signs of both left- and right-side heart failure because left-side failure often leads to right-side failure.

Hypertensive Emergencies

Hypertension is defined as any systolic blood pressure greater than 130 mm Hg or a diastolic blood pressure greater than 80 mm Hg. Another cardiac-related condition is a hypertensive emergency. A **hypertensive emergency** is defined as a

YOU are the Provider

You arrive at the scene and are escorted by the patient's son to her bedroom. She is sitting up in bed with her fist clutched against her chest. She is conscious and alert, but is notably anxious. Her skin is pale and diaphoretic. Your partner opens the jump kit as you assess the patient.

Recording Time: 0 Minutes	
Appearance	Anxious, pale, and diaphoretic
Level of consciousness	Conscious and alert
Airway	Open; clear of secretions and foreign bodies
Breathing	Increased respiratory rate; adequate depth
Circulation	Radial pulse rapid and irregular; skin pale and diaphoretic

After confirming that she has not taken any medication and that she is not allergic to any medications, you give the patient four 81-mg aspirin tablets to chew and swallow according to your protocols. As you continue your assessment and further inquire about her medical history, your partner applies the pulse oximeter, which shows that the patient's oxygen saturation is 91%. Based on this he applies oxygen via nasal cannula at 4 L/min and prepares to take her vital signs. She tells you that she had a heart attack 3 years ago; has high blood pressure; and takes enalapril (Vasotec), nitroglycerin, and one aspirin per day.

4. Why is aspirin given to patients with an acute cardiac event?
5. What type of medication is nitroglycerin? How may it help relieve chest pain, pressure, or discomfort?
6. When is nitroglycerin indicated for a patient? What is the typical dose?

systolic pressure greater than 180 mm Hg in the presence of impending or progressive organ damage. Because patients do not feel their blood pressure, the signs and symptoms of hypertensive emergency are related to the effects of the hypertension. Some patients with chronic hypertension may not experience signs or symptoms until their systolic pressure is significantly higher than this value. One of the most common signs is a sudden severe headache. If described as "the worst headache I have ever felt," this may also be a sign of cerebral hemorrhage. Other signs and symptoms include strong bounding pulse, ringing in the ears, nausea and vomiting, dizziness, warm skin (dry or moist), nosebleed, altered mental status, and even the sudden development of pulmonary edema. Untreated hypertensive emergencies can lead to a stroke or a dissecting aortic aneurysm.

If you suspect your patient is experiencing a hypertensive emergency, attempt to make him or her comfortable and monitor the blood pressure regularly. Position the patient with the head elevated, and transport rapidly to the ED. Depending on the distance and time involved in transport, you should consider ALS assistance for the patient. Paramedics may be able to administer medications to lower the blood pressure to a safer level. If ALS personnel can be on the scene quickly, contact them early and allow them to transport the patient from the scene. If the transport distance is long, consider asking for an ALS unit to meet you along the way and take over patient care and transportation from that point. Remember that getting the patient with a hypertensive emergency to the hospital as quickly and safely as possible is the best prehospital treatment you can provide.

An **aortic aneurysm** is a weakness in the wall of the aorta. The aorta dilates at the weakened area, which makes it susceptible to rupture. A **dissecting aneurysm** occurs when the inner layers of the aorta become separated, allowing blood (at high pressures) to flow between the layers. Uncontrolled hypertension is the primary cause of dissecting aortic aneurysms. This separation of layers weakens the wall of the aorta significantly, making it more likely to be ruptured under conditions of continued high blood pressure. If the aorta ruptures, the amount of internal blood loss will be so large that the patient will die almost immediately. The signs and symptoms of a dissecting aortic aneurysm include very sudden chest pain located in the anterior part of the chest or in the back between the shoulder blades. It may be difficult to differentiate the chest pain of a dissecting aortic aneurysm from that of an AMI, but several distinctive features may help. The pain from an AMI is often preceded by other symptoms—nausea, indigestion, weakness, and sweating—and tends to come on gradually, getting more severe with time and often described as "pressure" rather than "stabbing." By contrast, the pain of a dissecting aortic aneurysm usually comes on full force from one minute to the next (**TABLE 17-2**). A patient with a dissecting aortic aneurysm also may exhibit a difference in blood pressure between arms or diminished pulses in the lower extremities. Aortic aneurysms are often difficult to diagnose in the

TABLE 17-2 AMI Versus Dissecting Aortic Aneurysm

	AMI	Dissecting Aneurysm
Onset of pain	Gradual, with additional symptoms	Abrupt, without additional symptoms
Quality of pain	Tightness or pressure	Sharp or tearing
Severity of pain	Increases with time	Maximal from onset
Timing of pain	May wax and wane	Does not abate once it has started
Region/radiation	Substernal; back is rarely involved	Back possibly involved, between the shoulder blades
Clinical signs	Peripheral pulses equal	Blood pressure discrepancy between arms or decrease in a femoral or carotid pulse

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prehospital setting, but you must consider them a possibility in any patient with chest and abdominal pain and significant hypertension. Transport the patient without delay.

Patient Assessment

While en route to the scene, consider the standard precautions that will be needed. The precautions can be as simple as gloves for a patient with chest pain or full precautions for a patient in cardiac arrest. Remember, the patient's condition can change rapidly between the time you are dispatched and your arrival.

Scene Size-up

Do not let your guard down on medical calls. Always ensure that the scene is safe for all. As you approach the scene, look for and address any hazards. Determine the necessary standard precautions and whether you will need additional resources.

Identification of the nature of illness is important to start your patient assessment in the right direction. Use the information you get from the dispatcher, clues at the scene, and comments of bystanders or family members to begin to develop an idea about the type of problem your patient might be experiencing. For patients with cardiac problems, the clues often include a report of chest pain, difficulty breathing, or sudden loss of consciousness. Once you establish a preliminary nature of illness, you will be able to guide your assessment to find the important information much more effectively. Just remember not to become fixated on a specific condition at this early point in the assessment; sometimes the situation turns out to be very different from how it initially appeared.

Primary Assessment

As you approach the patient, form a general impression of his or her condition to recognize and address life threats. You will likely begin by determining whether the patient is responsive. Perform a primary assessment of the patient. If the patient is unresponsive and pulseless, begin CPR, starting with chest compressions, and call for an AED. Use of the AED is discussed in the section on cardiac arrest later in this chapter. Generally, an AED should be applied if the patient is pulseless, not breathing (apneic), and unresponsive. Consider calling for ALS backup if possible.

Once you have formed a general impression, the next step in the primary assessment of a conscious patient is to assess airway and breathing. Unless the patient is unresponsive, the airway will most likely be patent. Responsive patients should be able to maintain their own airway. Some episodes of cardiac compromise may produce dizziness or even fainting spells (syncope). If dizziness or fainting has occurred, consider the possibility of a spinal injury from a fall. Assess and treat the patient as appropriate.

Assess the patient's breathing to determine if it is adequate to provide enough oxygen to an ailing heart. If the rate is too fast or too slow, the depth of respiration seems to be too shallow, or if the patient is struggling to breathe, respirations are inadequate. Listen for abnormal breath sounds at this time because these can also be important indicators of respiratory distress. Some patients feel shortness of breath even though there are no obvious signs of respiratory distress. Pulse oximetry is a valuable tool in treatment of cardiac disease and should be applied at this time. If the patient is having any difficulty breathing or if his or her oxygen saturation is less than 94%, administer oxygen at 4 L/min via a nasal cannula. If the saturation does not improve quickly, increase the oxygen concentration. If you cannot get a pulse oximetry reading, apply a nonrebreathing mask at 15 L/min. In general, the goal is to maintain the oxygen saturation level between 95% and 99%. If the patient is not breathing or has inadequate breathing, ensure adequate ventilations with a bag-mask device and 100% oxygen.

Patients experiencing pulmonary edema may require positive-pressure ventilation with a bag-mask device or CPAP. CPAP is the most effective way to assist a person with CHF to breathe effectively and prevent the need to use an invasive airway management technique. Be aware of the indications and contraindications of CPAP and be competent in utilizing this equipment.

After assessing airway and breathing, assess the patient's circulation. Determine the rate and quality of the patient's pulse. Is the pulse rhythm regular or irregular? Is the pulse too fast or too slow? If you find abnormalities in the pulse, you should be more suspicious. Assess the patient's skin condition, color, moisture, and temperature, as well