Standard Medicine

Competency Integrates assessment findings with principles of epidemiology and pathophysiology to formulate a field impression and implement a comprehensive treatment/disposition plan for a patient with a medical complaint.

TOPIC 28

CARDIOVASCULAR EMERGENCIES: HEART FAILURE

INTRODUCTION

Diagnosing and treating a heart failure patient in acute pulmonary edema is one of the most challenging situations a paramedic can face. Although there are literally thousands of heart failure patients living in America each day, the vast majority will not need the services of EMS. However, a small percentage of those will present with life-threatening side effects, such as pulmonary edema, and will require prehospital intervention in the most desperate terms.

The acute presentation of heart failure not only can be life threatening, but also can be difficult to discern from other chronic respiratory illnesses. As a result, paramedics are faced every day with the dilemma of making important treatment decisions with only limited information at their disposal. To make matters worse, recent research has demonstrated that some commonly used therapies may be harmful if the diagnosis is wrong.

Although diagnostic technology has improved and will continue to advance, paramedics must rely on a knowledge of pathophysiology and quality assessment skills to rapidly identify this category of patient. Treatments should be goal oriented and aggressive, but must address the reality of acute pulmonary edema, not the outdated myths. Furthermore, EMS systems, in the development of protocols, should use treatment options that are forgiving and broader in scope so as to minimize the risk of misdiagnosis.

This topic provides a brief overview of the pathophysiology of acute heart failure and includes a review of both right- and left-sided failure. It also reviews key diagnostic findings and treatment, especially with regard to acute pulmonary edema.

TRANSITION *highlights*

- ***Frequency, ethnic and gender predisposition, and morbidity rates for patients with heart failure.***
- ***Pathophysiologic changes that accompany this disease process:***
  - Heart failure.
  - Left heart failure.
  - Right heart failure.
- ***Common signs and symptoms of heart failure with specific findings that will best delineate between these emergencies.***
- ***Assessment phases for a patient suffering from heart failure and the specific etiologies that accompany this disease process.***
- ***Treatment interventions:***
  - Oxygen.
  - Pulse oximetry.
  - Body positioning.
  - Continuous positive airway pressure (CPAP).
  - Medications.

EPIDEMIOLOGY

In the United States, heart failure is a disease state of significant concern. It has been estimated that the prevalence rate for heart failure is about 1 percent to 2 percent in the adult population. In more specific numbers, more than 400,000 patients are diagnosed yearly with heart failure, and currently about 3 million Americans have this disease state. In fact, this pathology is a common reason for hospital admittance, as up to 40 percent of heart failure patients are hospitalized every year. Heart failure has been found to be the cause of death in blacks more often than in whites (blacks are 1.5 times more likely to die from heart failure) and has a higher prevalence in men than in women (although in the population over 75 years of age, heart failure shows no gender preferences).

As to be expected, the prevalence of heart failure increases with age, affecting about 10 percent of the population older than 75 years. Finally, as a comorbid factor, patients with insulin-dependent diabetes have a significantly higher mortality rate.

PATHOPHYSIOLOGY

Heart failure (sometimes referred to as *congestive heart failure*) is a medical diagnosis, but, more important, it is a pathophysiologic state in which the heart muscle is unable to pump the blood needed to meet the venous return of the body. The resultant drop in cardiac output most typically results in a condition in which a buildup of fluid (congestion) in the body occurs as a result of pump failure. In essence, it represents the condition in which the left, right, or both ventricles fail to meet the body's needs. It may be a chronic condition that develops over a period of time (perhaps years), or it may be more acute in nature should it be associated with a large myocardial infarction (MI) or other serious disruption in the heart's ability to pump blood mechanically.

The many causes of heart failure include coronary artery disease, valvular dysfunction, and myocardial disease. Other factors that may contribute to heart failure include excessive salt or water intake, hypertension, thyrotoxicosis, pulmonary embolism, alcohol/drug abuse, and anemia.

Heart failure, as discussed, results in the reduction of cardiac output and may be caused by a decrease in stroke volume or a change in heart rate. By definition, cardiac output is the amount of blood pumped by the heart for 60 seconds. The relationship of cardiac output (CO), stroke volume (SV), and heart rate (HR) is found in the following formula:

$$CO = SV \times HR$$

A reduction in cardiac output leads to compensatory mechanisms that act to restore cardiac output. For instance, when a patient sustains an MI, dead heart muscle can prevent the heart from pumping normally, thus leading to decreased cardiac output. The body senses the decrease in cardiac output by way of baroreceptors in the aortic arch and carotid bodies and tries to compensate by increasing sympathetic tone.

Because the myocardium cannot increase stroke volume because of the damaged pump, it must compensate by increasing the heart rate. If, however, a patient has a dysrhythmia that affects only the heart rate (i.e., bradycardia), the decreased heart rate leads to a decreased cardiac output. In that case, the body tries to compensate by increasing the stroke volume and systemic vascular resistance.

The body has several other mechanisms it can use to compensate for decreased cardiac output. These include vasoconstriction of peripheral vessels and activation of the hormonal systems of the body designed to increase intravascular volume. An example of the hormonal response is brain-type natriuretic peptide (BNP). This substance is released in response to distention of the ventricles seen in heart failure. The substance, tested for as an indicator of heart failure, promotes natriuresis and tends to lower blood volume. Unfortunately, these compensatory mechanisms actually increase myocardial oxygen demand and thus are potentially detrimental to myocardial function.

Heart failure is generally divided into left ventricular (LV) failure and/or right ventricular (RV) failure, although this is somewhat arbitrary in nature, owing to the fact that the right and left ventricles perfuse different portions of the circulation. It can also be described as "backward failure" (leading to congestion) and "forward failure" (leading to diminished end-organ perfusion).

Left-Sided Failure

Left ventricular (LV) failure occurs when the left ventricle is unable to pump adequately; the heart pumps inadequately for multiple reasons. Dysfunction of the heart muscle itself, as is seen with MI, is one of the main causes of LV pump failure. Dysrhythmias also inhibit the heart's ability to pump normally.

With *backward* failure of the left ventricle, pulmonary congestion (pulmonary edema) results, leading to signs and symptoms that are primarily respiratory in nature. With *forward* failure of the left ventricle, diminished peripheral perfusion and systemic circulation result. Conditions that may be responsible for this include obstruction of outflow from the heart, as is seen in valvular disease or chronic systemic hypertension.

Right-Sided Failure

In this type of heart failure, the right side of the heart fails to function as an adequate pump to the lungs, which commonly leads to back pressure of blood into the venous and systemic circulation with *backward* failure of the right ventricle. Backward failure of the right ventricle results in excess fluid that accumulates in the body, often in dependent extremities (▶ Figure 28-1), and may cause jugular venous distention (JVD) (▶ Figure 28-2), enlargement of the liver, and possible abdominal distention in severe cases.

Right ventricular (RV) failure is most commonly caused by backward failure of the left heart muscle, which then causes an eventual backlogging of blood into the right heart circuit. Similar to the causes of LV failure, other disorders that can cause the right side to fail include dysfunction of

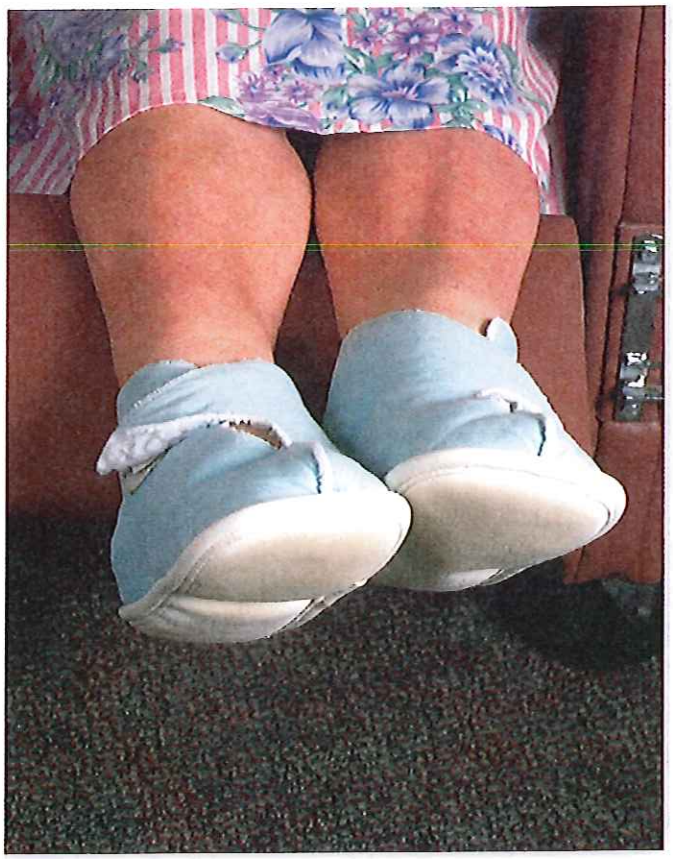

Figure 28-1 Edema to the lower extremities is a classic sign of heart failure.

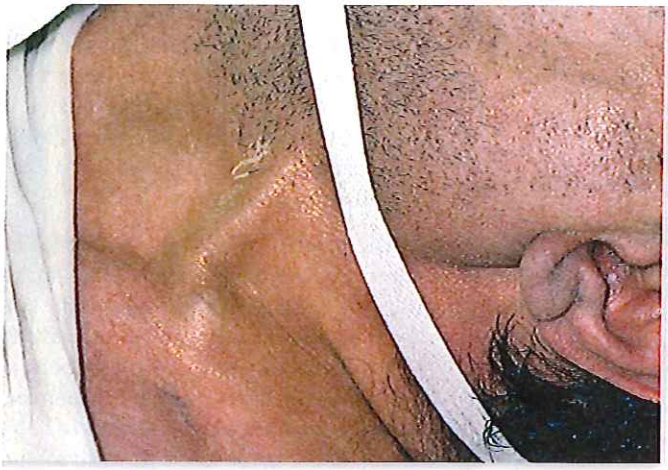

Figure 28-2 Jugular vein distention is a late sign of heart failure. (© *David Effron, M.D.*)

the heart muscle itself from chronic pulmonary hypertension or, in acute cases, right ventricular MI. Right ventricular MI is less common than LV infarctions, but it is seen. Pulmonary hypertension and stenotic pulmonary valvular disease can also result in *forward* failure of the right heart and may result in lungs being underperfused, leading to subjective respiratory distress and diminished preload to the left heart circuit.

Table 28-1 summarizes these pathophysiologic changes in right and left heart failure leading to the diagnosis of congestive heart failure. Remember, though, heart failure is the underlying problem; when the medical condition exacerbates, the presentation is often centered around dyspnea, weakness, changes in breath sounds, possible chest pain, weakness, and fluid retention.

ASSESSMENT FINDINGS

The signs and symptoms of heart failure will depend on the severity of the condition and whether it is an acute-onset or a long-term problem. During the assessment, recall that several exceptions apply to a simple left-versus-right division of heart failure symptoms. Left-sided *forward* failure overlaps with right-sided *backward* failure. Because the most common cause of right heart failure is left heart failure, patients may present with symptoms of both types (a condition known as *biventricular failure*). Regardless, the signs and symptoms of congestive heart failure include the following (▷ Figure 28-3):

- Marked or severe dyspnea (shortness of breath)
- Tachycardia (rapid heart rate, greater than 100 bpm)
- Difficulty breathing when supine (orthopnea)
- Suddenly waking at night with dyspnea (paroxysmal nocturnal dyspnea)
- Fatigue on any type of exertion
- Anxiety
- Tachypnea (rapid respiratory rate)
- Diaphoresis (sweating)
- Upright position with legs, feet, arms, and hands dangling
- Cool, clammy, pale skin
- Chest discomfort
- Cyanosis
- Agitation and restlessness from the hypoxia
- Edema (swelling) to the hands, ankles, and feet
- Crackles and possibly wheezes on auscultation
- Decreased SpO_2 reading
- Blood pressure normal, elevated, or low
- Distended neck veins—jugular venous distention (JVD)
- Distended and soft, spongy abdomen

Table 28-2 summarizes common findings of heart failure and discusses the pathophysiologic change that underlies them.

Medications can often help diagnose a history of heart failure.

Medications can often help diagnose a history of heart failure. Early and mild cases of heart failure are commonly treated with beta blockers to help control rate and myocardial oxygen demand. As heart failure progresses, patients are frequently prescribed diuretics to manage fluid volume levels. Patient also frequently take angiotensin-converting enzyme (ACE) inhibitors to control afterload.

Diagnosing Acute Pulmonary Edema

Acute pulmonary edema is the common byproduct of left-sided failure. As pressure builds in the circuit behind the left ventricle, fluid begins to migrate out of the system. The specific challenge occurs in the acute setting when that fluid leaves the vascular space, crosses the membranes of the pulmonary capillaries, and moves into the interstitial tissue of the lungs. If the condition continues, the fluid can also occupy space in the alveoli themselves.

Pulmonary edema can be a chronic condition. As the previous section discussed, many patients describe ongoing symptoms such as dyspnea on exertion, paroxysmal nocturnal dyspnea, and orthopnea. However, pulmonary edema can also present in a life-threatening acute fashion when fluid is rapidly transferred into the lungs, causing respiratory failure.

TABLE 28-1 Pathophysiologic Changes in Right and Left Heart Failure

	Pathophysiologic Findings
Right heart failure	Right heart fails because of infarction, increased workload, valvular dysfunction, or a combination of these. It results in the congestion of blood in the vena cava, resulting in jugular venous distention, peripheral edema, enlarged liver, clear breath sounds, and probably hypotension.
Left heart failure	Left heart fails also because of infarction, increased workload (systemic hypertension), valvular dysfunction, or a combination of these. It results in the congestion of blood in the lungs, which increases pressure to a point at which fluid escapes into the alveoli, causing respiratory distress and pulmonary edema. Lung sounds often reveal crackles or "cardiac asthma," blood pressure is commonly normal to high, and peripheral congestion is absent.

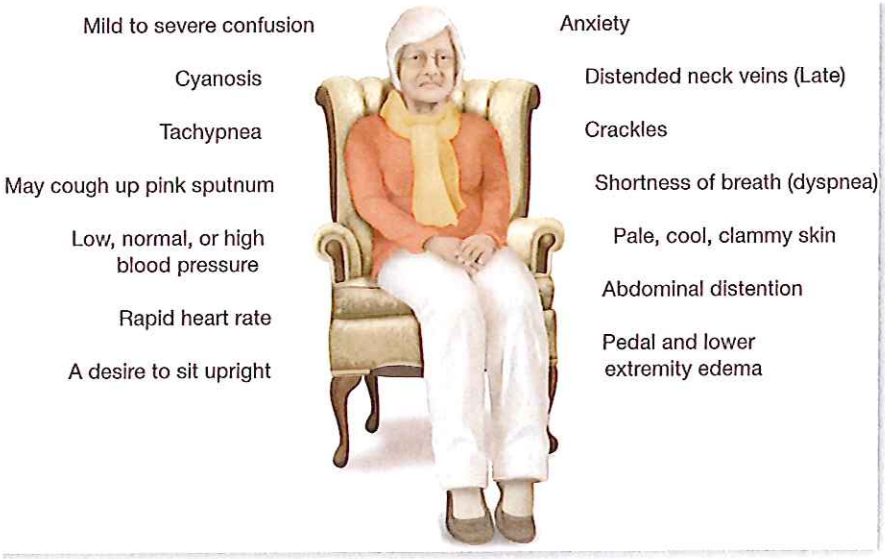

Figure 28-3 Signs and symptoms of heart failure.

The emphasis on assessment should be on the entire patient, and not just on lung sounds.

Again, this is commonly an exacerbation of an ongoing heart failure condition, but it can also be associated with acute disorders such as MI and dysrhythmias.

Acute pulmonary edema poses a specific challenge, as paramedics are faced with both an immediate need for treatment and a disorder that can look very similar to other respiratory ailments. In many cases, acute pulmonary edema can present with wheezes, similar to those seen in asthma, and patients can exhibit other respiratory symptoms, such as those seen with exacerbation of chronic obstructive pulmonary disease (COPD). Although there are no foolproof methods of diagnosing acute pulmonary edema (particularly in the prehospital world), there are some helpful ideas that can lend accuracy to your treatment modality:

- **Listen for crackles.** Crackles (also known as rales) tell a tale of fluid in the alveoli. Although there are other causes of crackles, the most common cause in the acute setting is acute pulmonary edema. Be wary, though, of waiting for crackles. Many patients will present with other symptoms first before presenting with crackles. In fact, fluid in the alveoli is a relatively late sign of acute pulmonary edema, resulting only after nearly one liter of fluid has crossed into pulmonary tissue.
- **Check the monitor.** Often, an electrocardiogram (ECG) can demonstrate the proximal cause for acute pulmonary edema. Look for dysrhythmias and evidence of acute MI.
- **Look for history of heart failure.** Although not every patient in acute pulmonary edema will have a history of heart failure, by far the majority will. As such, inquire about previous cardiac and heart failure history. Look for medications commonly used to treat heart failure. Look for signs of undiagnosed heart failure, such as patients sleeping upright in chairs, history of dyspnea on exertion, and even physical findings such as pedal edema, weight gain, and JVD. Although physical findings such as JVD and pedal edema may not have anything to do with the acute left heart failure, they do demonstrate a prior history of failure.
- **Acute pulmonary edema tends to be acute.** Although pulmonary edema can be chronic in nature, acute episodes tend to develop rapidly with an abrupt onset. Occasionally, there will be a trigger such as exertion, stress, or other conditions that increase cardiac workload. Although onset is not definitive, it can help distinguish acute pulmonary edema from other respiratory disorders such as exacerbations of COPD and pneumonia (which tend to have far more chronic onsets).
- **Look at the whole patient.** No one fact can diagnose any patient. Put all your findings in the context of the larger patient. Crackles are an important finding, but are even more important in the context of a patient with a history of heart failure (and even more important if that patient has had pulmonary edema in the past).

Diagnostic methods are rapidly improving and technology will help make decision making easier. Some EMS systems are now using point-of-care testing to assess for levels of BNP. It is thought that elevated levels of this biochemical marker can indicate the presence of heart failure and can be used as a surrogate marker for acute pulmonary edema.

CRACKLES AND WHEEZING Acute pulmonary edema can cause both crackles and wheezing. Crackles are heard as fluid migrates into the alveoli and small airways. Wheezes are heard as prealveolar sphincters narrow to prevent fluid from escaping the alveolar space. Both these lung sounds can be helpful in diagnosing heart failure, but can also present diagnostic challenges. Because wheezing are commonly associated with other respiratory conditions such as asthma and exacerbated COPD, paramedics can frequently be pulled down an incorrect path.

The emphasis on assessment should be on the entire patient, and not just on lung sounds. Paramedics will have to differentiate based on other findings, including those discussed previously. Unfortunately, many heart failure patients are not simple to diagnose. Many will have a variety of comorbid factors, including those that cause wheezing. Do asthma patients occasionally fall victim to acute pulmonary edema? Yes, they do. The treatment of this undifferentiated patient is very controversial. Of course, if the true etiology of wheezing is heart failure, then you should treat heart failure. Some experts would note that using bronchodilators in a patient with acute pulmonary edema causes increased myocardial oxygen demand (owing to $beta_1$ effects) and therefore can be harmful. Recently, however, the opinion has shifted slightly.

Again, if the origin is known, heart failure should be treated first, but some experts contend that measured bronchodilator use in an undifferentiated acute pulmonary edema patient is not nearly as harmful as we once imagined. These experts contend that most acute heart failure patients are already adrenergically

TABLE 28-2 Clinical Findings and Pathophysiologic Etiology of Heart Failure

Clinical Finding	Pathophysiologic Etiology
Rapid breathing (tachypnea)	Multiple reasons, such as hypoxia, carbon dioxide retention, sympathetic discharge.
Shortness of breath (dyspnea)	Changes in O_2/CO_2 diffusion across alveoli; chemoreceptors in body detect changes in gas levels and cause the perception of dyspnea.
Shortness of breath while lying down (orthopnea)	On lying down, fluid accumulation in lungs from CHF tends to increase, which diminishes gas exchange across alveoli.
Constant waking at night (paroxysmal nocturnal dyspnea)	While lying down, fluid accumulates in the lungs and causes the person to wake up. Patient may state that the dyspnea eases after sitting or standing up from sleep.
Anxiety, tremors, nausea, vomiting	Sympathetic discharge caused by the changes in blood gases and/or cardiac output.
Low pulse oximetry readings	Poor oxygenation from fluid accumulation in lungs diminishes oxygen diffusion into the bloodstream.
Cool, pale, clammy skin	Sympathetic discharge from changes in cardiac output and oxygen/carbon dioxide levels.
Sitting upright and/or tripod positioning	Sitting upright eases dyspnea because of better diaphragmatic function; it also helps to ease fluid accumulation in lungs.
Chest discomfort	Possible angina, infarction in an acute setting.
Inspiratory crackles	Fluid accumulation in the alveoli from backward failure of left ventricle.
Wheezing (cardiac asthma)	Fluid accumulation in alveoli may migrate into bronchioles from breathing, causing stimulation of "irritant receptors" in lung tissue causing bronchoconstriction.
Distended neck veins, enlarged liver, distended abdomen	Increased venous pressure from backward failure of right ventricle.
Changes in blood pressure	Hypotension often caused by failing right ventricle (thereby diminishing left-sided preload). Hypertension often caused by left-sided failure in conjunction with heightened sympathetic tone.
Objective dyspnea findings: nasal flaring, retractions, tachypnea, tripod positioning, mouth breathing, etc.	Most commonly caused by combination of right and left ventricular failure.

stimulated, and the slight increase given by inhaled bronchodilators may actually be mitigated by the increased oxygenation secondary to bronchodilation. The answer is resoundingly unclear, but strong opinions have emerged on both sides. As always, you should follow local protocol.

EMERGENCY MEDICAL CARE

The treatment for the patient with acute heart failure is geared toward improving oxygenation, diminishing fluid accumulation in the lungs, treating the patient for acute coronary syndrome should it concurrently be present, and maintaining normotension. Remember that exacerbation of acute pulmonary edema may be very scary to the patient, so good communication skills and verbal reassurance will also go a long way.

1. **Establish and maintain an open airway.** Airway management should always be goal oriented. Security of the airway may be a high priority in these patients if hypoxia secondary to respiratory failure robs the patient of the ability to maintain an airway. Paramedics should remember that the threshold for endotracheal intubation (ETI) in this population is slightly lower than that for other respiratory disorders. In this group, ETI provides not only airway security, but also therapeutic increased positive pressure. That said, ETI should be reserved for the most serious cases when advanced airway management is necessary.

   In most cases, positive pressure can be achieved noninvasively with the use of continuous positive airway pressure (CPAP). Remember also that many of these patients will be conscious; unless rapid sequence induction is available, ETI will be very difficult, with dangerous side effects associated with sympathetic discharge as a result of fighting the intubation attempt.
2. **Recognize respiratory failure.** Many acute heart failure patients will be breathing, but breathing inadequately. When fluid occupies space in the pulmonary interstitial tissue and in the alveoli, gas exchange is interrupted. Paramedics must recognize the failure of the respiratory system to adequately meet metabolic demands. Assess aggressively for this possibility and initiate positive pressure ventilations when found. Remember again that in acute pulmonary edema patients, positive pressure can help reduce fluid from shifting into the lungs. As such, the threshold to initiate positive pressure ventilation can be slightly lower than that of other respiratory disorder patients.
3. **Provide oxygen.** If the patient is dyspneic or hypoxemic, has obvious signs of heart failure, or has a SpO_2 reading of less than 94 percent. Initiate oxygen therapy via nasal cannula at 4 lpm and titrate oxygen therapy to maintain a SpO_2 reading of 94 percent or greater. If the breathing is inadequate, provide positive pressure ventilation at 10 to 12/min with high-flow supplemental oxygen.

4. **Position the patient.** Semi- or high Fowler positioning will assist the patient in breathing and minimize the negative effects of fluid accumulating in the lungs. If positive pressure ventilation is being provided or frank hypotension is present, the patient may need to be placed supine for ongoing management. Many patients will wish to hang their legs off the stretcher. Surprisingly, this is not just a position of comfort, but probably a survival instinct. Although the evidence is unclear, many experts note that positioning the legs in a dependent posture can help relieve volume pressure by drawing fluid to the lower extremities. These experts suggest that if it can be done safely, patients should be allowed to hang their legs off the stretcher.
5. **Use CPAP if protocol allows.** CPAP can be extremely effective in the fight against acute pulmonary edema. The application of CPAP (▶ Figure 28-4) increases intraalveolar pressure and helps prevent the movement of fluid from the pulmonary capillaries into the lungs. It is most effective when applied early and when combined with aggressive administration of nitrates to help reduce preload. CPAP devices are typically set to provide positive end expiratory pressure (PEEP) at between 5 and 10 cmH_2O. This pressure increases intraalveolar pressure and also pneumatically splints the small airways, preventing atelectasis. As always, follow local protocol.
6. **Initiate an intravenous line of normal saline at a to-keep-open rate.** Most commonly, this access is a medication route, but some heart failure patients may require fluid. In some cases, right-sided MI can cause dramatic right heart failure and may need a measured fluid challenge to increase preload. This therapy is controversial; you should always follow local protocol.
7. **Administer nitroglycerin.** Nitrates are most commonly the most important medication used to treat acute heart failure. Nitroglycerine causes venous dilation and therefore venous pooling, leading to decreased cardiac preload. This reduction of preload decreases cardiac workload and can help decrease failure and backward pressure.

   It is important to remember that in the event of acute heart failure, nitrates should be administered more aggressively than in the case of acute coronary syndrome (although these two conditions are frequently linked). Nitrate levels must be maintained to sustain a continued preload reduction, and as a result, dosing frequency is typically more rapid than the traditional acute coronary syndrome (ACS) regimen.

   Most commonly, sublingual nitroglycerine is administered every 2 to 3 minutes as opposed to every 5 minutes in ACS. Some systems prefer nitrates to be administered intravenously to enable precise titration of blood pressure and maximum therapeutic effect. Nitrates are also administered transdermally via a paste. This can be particularly helpful in patients wearing a CPAP mask, but this route can be somewhat unpredictable in terms of onset and challenging to control in the event of hypotension. Always follow local protocol.
8. **Consider additional pharmacology.** If the failure is secondary to a cardiac dysrhythmia, treat the underlying cause. Both bradycardias and tachycardias can cause acute pulmonary edema. Follow advanced cardiac life support (ACLS) guidelines to address any rhythm disturbance. Some EMS systems use ACE inhibitors such as captopril to decrease afterload and therefore diminish cardiac workload. This treatment is somewhat controversial, but can be effective if used appropriately. Furosemide, once the standard pharmacologic agent in acute pulmonary edema, is quickly losing favor.

   Although diuretics can still be useful in acute heart failure, it has been found that their use should be limited only to those heart failure patients who are hypervolemic. In more than 60 percent of the cases of acute pulmonary edema, patients are not in fact hypervolemic, but rather are normovolemic and suffering only from a pump problem. When these patients are giving diuretics, they become hypovolemic, thereby adding yet another physiologic challenge to an already sick patient. Furthermore, recent studies have shown that when furosemide is given in the case of a misdiagnosis to pneumonia and exacerbated COPD patients, mortality actually increases. Of course, paramedics should always follow local protocols, but they should also keep in mind that diuresis occurs naturally as nitrates and CPAP help restore perfusion to the kidneys.
9. **Ensure appropriate transport to the emergency department.** Notify the receiving ED as early as possible.

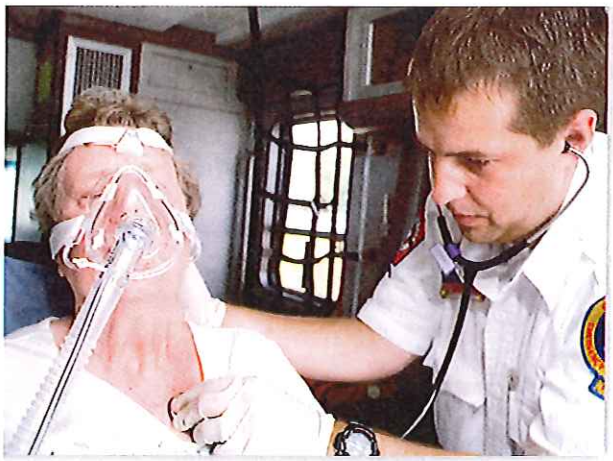

Figure 28-4 CPAP is a form of noninvasive positive pressure ventilation used in the awake and spontaneously breathing patient who needs ventilatory support. (© *Ken Kerr*)

Continually assess the patient and be prepared for respiratory failure and cardiac arrest. Should either of these occur, follow appropriate treatment and local protocol for supporting this lost function.

Patients suffering from exacerbation of heart failure can be among the most challenging patients encountered by the paramedic. They may present anywhere on the continuum from being alert and oriented with only minimal symptoms to being unresponsive and just moments from complete cardiopulmonary arrest. Compounding this picture is that the heart failure patient may go from one extreme to the other very quickly, without much warning. Therefore, the paramedic must always maintain a high degree of suspicion that these patients may deteriorate into arrest very suddenly.

TRANSITIONING

REVIEW ITEMS

1. During your interview, your patient tells you that she has "water on the lungs." This statement most closely reflects what pathophysiologic change in heart failure?
   a. backward failure of the left ventricle
   b. frontward failure of the right ventricle
   c. biventricular failure
   d. elevation in central venous pressure
2. Of the choices below, which is most likely a determining factor causing the development of right-sided heart failure?
   a. high diastolic pressure
   b. preexisting left heart failure
   c. stenosis of the tricuspid valve
   d. history of diabetes mellitus
3. A patient presents with JVD, hypotension, clear breath sounds, and peripheral edema. These findings would be most representative of what type of heart failure?
   a. acute right ventricular failure
   b. acute left ventricular failure
   c. gradual right ventricular failure
   d. gradual left ventricular failure
4. Pulmonary edema would be a manifestation of what type of ventricular failure?
   a. acute right ventricular failure
   b. acute left ventricular failure
   c. gradual right ventricular failure
   d. gradual left ventricular failure
5. Diffusion of excessive alveolar fluid back into the perialveolar capillary bed would be best accomplished by what treatment intervention for the heart failure patient?
   a. placing the patient supine
   b. providing high-flow oxygen
   c. administration of sublingual nitroglycerin
   d. continuous positive airway pressure (CPAP)

APPLIED PATHOPHYSIOLOGY

A 76-year-old female patient has a history of hypertension, insulin-dependent diabetes, and heart failure. She summoned an ambulance because of weakness, mild chest pressure, and progressively worsening dyspnea over the past two days. The patient states that her trouble breathing got worse with physical exertion and when lying down at night to go to bed. Currently her vital signs are blood pressure 180/100, heart rate 102 beats per minute, respiratory rate 24, SpO_2 92 percent on room air. During your assessment of the breath sounds, you note bilateral wheezing.

1. Identify whether you believe this patient has right or left ventricular failure. Support your answer with the appropriate clinical findings.
2. Why might the administration of nitroglycerin to this patient be beneficial to the dyspnea?
3. Identify and discuss why the patient's wheezing should *not* be treated as it would be in an asthmatic or allergic-reaction patient.
4. Discuss the differences in pathophysiology and presentation of right versus left heart failure according to the following three assessment findings:
   a. Breath sounds
   b. Systolic blood pressure
   c. Vital sign changes
5. Differentiate the pathophysiologic changes between *backward* and *forward* ventricular failure.

CLINICAL DECISION MAKING

Late one hot summer night, you are called for a patient suffering from acute respiratory distress. On arrival at the patient's home, you are escorted to an elderly female patient's bedroom on the second floor. As soon as you walk in, you see the patient lying on her back in bed with four or five pillows behind her head and shoulders, helping her to "sit up." The patient is alert and oriented; displays nasal flaring, retractions, and tachypnea; and is speaking in full sentences. On the nightstand beside the bed, you see four or five prescription bottles of medication.

1. Based on the scene size-up characteristics, identify the clues that point to the field impression of heart failure or acute pulmonary edema.
2. What would be common medications you might find on the patient's nightstand to support the field impression of heart failure?

The primary assessment reveals the patient to be alert and well oriented. Her airway is clear, her breathing is rapid at 30/minute, respirations are slightly shallow, vesicular sounds are present and diminished, and slight inspiratory crackles are noted. Peripheral pulses are also present and noted to be rapid and slightly irregular. The neck veins are obviously engorged, and the SpO_2 reading on room air is 87 percent.

3. What are the life threats, if any, to the patient of which you are currently aware?

After managing and supporting the situation described, further assessment reveals the pupils to be equal and reactive, blood glucose level is 193 mg/dL, and the SpO_2 has increased to 94 percent with high-flow oxygen. Breath sounds are unchanged; the JVD is still present. The patient's blood pressure is 100/79, heart rate is 112 and irregular, respirations are fast at 30/minute. The patient is now starting to complain of "chest pressure" as well.

4. From what etiology of heart failure is this patient likely suffering?

5. Explain the pathophysiologic cause for the following:
   a. Breath sounds
   b. Low pulse oximeter reading
   c. Jugular venous distention

6. During the management of the patient, you elect to administer the following therapies. For each one, discuss in specific detail (1) the reason the intervention is warranted, (2) the expected outcome of that intervention, and (3) how you would assess to determine whether the desired effect is occurring (i.e., the treatment worked).
   a. Position patient in high Fowler position
   b. Administer oxygen
   c. Administration nitroglycerin
   d. Application of CPAP at 10 cmH_2O pressure