## Medical Emergencies

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Respiratory Emergencies

Content Area: Medicine

Advanced EMT Education Standard: Applies fundamental knowledge to provide basic and selected advanced emergency care and transportation based on assessment findings for an acutely ill patient.

Objectives

After reading this chapter, you should be able to:

20.1 Define key terms introduced in this chapter.
20.2 Explain the importance of being able to quickly recognize and treat patients with respiratory emergencies.
20.3 Obtain an appropriate history for a patient with a respiratory problem.
20.4 Conduct an appropriate examination for a patient with a respiratory problem.
20.5 Explain the relationship between dyspnea and hypoxia.

Key Terms

- acute respiratory distress syndrome (ARDS) (p. 524)
- atelectasis (p. 523)
- carpopedal spasm (p. 526)
- chronic bronchitis (p. 519)
- chronic obstructive pulmonary disease (COPD) (p. 515)
- cor pulmonale (p. 519)
- croup (p. 527)
- cystic fibrosis (CF) (p. 530)
- emphysema (p. 519)
- expectoration (p. 521)
- hantavirus pulmonary syndrome (HPS) (p. 527)
- hemoptysis (p. 523)
- hyperventilation syndrome (HVS) (p. 526)
- laryngitis (p. 527)
- laryngotracheobronchitis (p. 527)
- leukotriene inhibitors (p. 522)
- paresthesia (p. 526)
- pharyngitis (p. 527)
- pleuritic (p. 523)
- right-sided heart failure (p. 519)
- sinusitis (p. 527)
- spontaneous pneumothorax (p. 525)
- status asthmaticus (p. 522)
- sympathetic beta2 receptors (p. 512)
- tension pneumothorax (p. 526)
- upper respiratory infection (URI) (p. 527)
- ventilation-perfusion (VQ) mismatch (p. 523)

To access Resource Central, follow the directions on the Student Access Card provided with this text. If there is no card, go to www.bradybooks.com and follow the Resource Central link to Buy Access. Under Media Resources, you will find:

- Chronic Obstructive Pulmonary Disease (COPD). Watch and learn about the causes and treatment of COPD.
- Understanding Respiratory Failure. Learn about V-Q mismatch, shunting, and pulse oximetry monitoring and their role in understanding respiratory failure.
- The Spontaneous Pneumothorax. View a short video on signs, symptoms, and prehospital management of a spontaneous pneumothorax.

Au/Ed: too long, edit to fit
20.6 Describe the pathophysiology by which each of the following conditions leads to inadequate oxygenation:
- Asthma
- Cystic fibrosis
- Hyperventilation syndrome
- Lung cancer
- Obstructive pulmonary diseases (emphysema and chronic bronchitis)
- Pneumonia
- Poisonous/toxic exposures
- Pulmonary edema
- Pulmonary embolism
- Spontaneous pneumothorax
- Viral respiratory infections

20.7 Use patient histories and clinical presentations to differentiate among causes of respiratory emergencies.

20.8 Engage in effective clinical reasoning in order to recognize indications for the following interventions in patients with respiratory complaints/emergencies:
- Establishing an airway
- Administration of oxygen

(continued)
Introduction

Patients who present in respiratory distress can deteriorate rapidly into respiratory failure and respiratory arrest. No matter what the underlying cause, death follows quickly unless measures are taken to restore ventilation and oxygenation.

A number of problems arising in the respiratory system can interfere with delivery of oxygen to the tissues. The oxygen deficit is exacerbated by two factors that increase oxygen demand when its supply is already jeopardized. First, the sensation of not being able to breathe is terrifying; and the associated stress response increases the demand for oxygen. Second, increased use of respiratory muscles creates an even higher need for cellular oxygen in the face of the decreased supply. Cellular metabolism with inadequate oxygen results in inefficient energy production and respiratory acidosis. The combination of the uncorrected underlying problem, exhaustion, and acidosis can overwhelm the body’s attempts to compensate and restore homeostasis.

As an Advanced EMT, you must be able to quickly recognize patients with difficulty breathing and intervene. You must ensure an open airway, adequate ventilation, and circulation of oxygenated blood to the tissues. In some cases, you will implement specific treatment measures for the underlying cause of respiratory distress. In other cases, the best prehospital care for the patient is to continue to ensure an open airway and provide support for ventilation and oxygenation while transporting the patient without delay for definitive care.

Understanding the anatomy and physiology of ventilation and respiration, and the pathophysiology of respiratory problems assists you in giving the best care possible to patients with difficulty breathing. There are treatments within the Advanced EMT scope of practice that you can use to treat patients with specific causes of respiratory distress. You must understand when specific medications should be considered, as well as when IV fluids may help and when they may result in harm.

Anatomy and Physiology Review

Cellular energy production, and thus life itself, depends on oxygen from the atmosphere reaching each individual cell. Oxygen from the air that enters the lungs is carried through the blood. It then moves into cells from the network of capillaries that provide circulation to body tissues. This process is more complex than it seems on the surface. It depends on the particular way that the organs of the respiratory and circulatory system are structured and the precise way in which they work together.

For oxygen to reach the microscopic alveoli of the lungs, where gases are exchanged with the circulatory system, there must be an adequate amount of oxygen in the atmosphere. The upper and lower portions of the airway must be open to allow that air to reach the alveoli. Each alveolus must be in close contact with a network of capillaries so that oxygen and carbon dioxide can be exchanged between the lungs and the blood (Figure 20-1).

The red blood cells must contain an adequate amount of hemoglobin to carry oxygen to the cells. The body’s temperature, acid–base balance, and other factors must be in the proper ranges for hemoglobin to accept oxygen from the lungs (external respiration) and release it to the cells (internal respiration).

The right side of the heart must be able to receive deoxygenated blood that is high in carbon dioxide and pump it through the pulmonary artery and into the lungs. The left side of the heart must be able to receive oxygenated blood that is low in carbon dioxide and pump it through the arterial and capillary systems to the cellular level.

Anything that interferes with this complex set of conditions can lead to hypoxia, cell dysfunction, and death. (See Chapters 8 and 10 for more in-depth review.)

The Need for Oxygen

Cells must produce energy to carry out their functions. The functions of brain cells, muscle cells, or liver, kidney,
Respiratory System

Transports air to and from lungs

Air passageways inside the lung

Cleanses, warms, and humidifies inhaled air

Site of gas exchange between air and blood

Carries air to the trachea and produces sound

FIGURE 20-1

Components of the respiratory system.
or cardiac cells all include maintaining their own cell membranes and internal structures and performing their specialized functions for the body. Energy production in the presence of oxygen (aerobic metabolism) is efficient and results in byproducts that are easily eliminated by the body.

In the process of producing energy, hydrogen ions (H\(^+\)) are produced. An increase in H\(^+\) concentration decreases the body’s pH, leading to acidosis. Cells need oxygen (O\(_2\)) to bind to the hydrogen ions (H\(^+\)) that are produced in energy metabolism. By doing so, two products, water (H\(_2\)O) and carbon dioxide (CO\(_2\)), are formed, which can be easily eliminated.

Without oxygen, in anaerobic metabolism, energy production is severely limited and H\(^+\) accumulates in the form of lactic acid. Anaerobic metabolism is a compensatory mechanism when the body’s need for oxygen is greater than its supply, but it is a short-term compensatory mechanism. Unless oxygenation is restored, death will occur.

**Structure and Function of the Lungs**

The lungs are spongy tissues with millions of microscopic air sacs, called alveoli, which allow the exchange of gases between the internal environment of the body and the atmosphere. Air enters the lungs through the nose or mouth in the upper airway, travels through the pharynx, and moves into the larynx and trachea. The trachea divides at the carina into right and left main-stem bronchi, which enter the right and left lungs, respectively, at their hilum.

Once inside the lungs, the bronchi divide into smaller branches that serve the two lobes of the left lung and three lobes of the right lung. The bronchi divide into bronchi into smaller and smaller branches until they become microscopic bronchioles that enter each alveolus.

The trachea and bronchi are composed of sturdy cartilage rings that keep them from collapsing. The bronchioles, however, have a substantial amount of smooth muscle that allows their diameter to change in response to the amount of alveolar ventilation required. The smooth muscle has sympathetic beta\(_2\) receptors that respond to epinephrine from the body, as well as to drugs with beta\(_2\) properties. The effect of beta\(_2\) receptor stimulation is smooth muscle relaxation, which increases bronchodilation (the diameter of the bronchioles).

The lining of the respiratory tract contains cells that secrete mucus, which traps contaminants that enter the respiratory system along with air. Microscopic, hairlike cellular projections called cilia sweep the mucus upward, along with trapped contaminant particles, so they can be expelled (mucociliary clearance). The cilia are paralyzed by the nicotine in cigarette smoke, leaving the smoker unable to clear the lungs of toxins.

The walls of the distal (terminal) bronchioles and alveoli are a single cell-layer thick. A network of capillaries, which also are a single cell-layer thick, surrounds each alveolus. The alveoli and capillaries are in close contact with each other, separated by a small amount of extracellular fluid.

The alveolar and capillary walls together are called the respiratory membrane. Because oxygen and carbon dioxide can diffuse only short distances, this respiratory membrane must remain thin. An increase in extracellular fluid between the capillary and alveolar walls, for example, or fluid or pus in the alveoli, increase the distance between the red blood cells in the capillaries and the contents of the alveoli.

The direction of diffusion of gases depends on their relative concentrations on each side of the cell membrane. Gases diffuse from where they are higher in concentration to where they are lower in concentration.

For gas exchange to occur, deoxygenated blood from the right side of the heart must reach the lungs. Blood from the right ventricle is pumped into the pulmonary artery, which divides into right and left branches to deliver deoxygenated blood to both lungs. The pulmonary arteries divide into smaller and smaller branches until they form the network of capillaries that surround each alveolus.

At the arterial end of the capillary bed, blood is lower in oxygen and higher in carbon dioxide. At the venous end, blood is higher in oxygen and lower in carbon dioxide. Oxygenated blood enters the pulmonary venous system and returns to the left atrium of the heart through the pulmonary vein (Figure 20-2).

**Ventilation**

Chemically, ventilation is stimulated primarily by an increased level of carbon dioxide in the blood and in cerebrospinal fluid. A secondary stimulus is a decreased level of oxygen. Those chemical changes stimulate the inspiratory center in the brain, located in the medulla of the brainstem. The inspiratory center sends nervous impulses to the diaphragm and intercostal muscles, causing them to contract. Muscular contraction flattens and lowers the diaphragm and lifts the ribs upward and outward, increasing the volume of the thoracic cavity.

The increase in thoracic volume creates a vacuum in the potential space between the parietal and visceral pleura, causing the lungs to expand. There is an inverse relationship between the volume of a gas and its pressure. The increased intrapulmonary (within the lung) volume results in intrapulmonary pressure that is lower than atmospheric pressure. Because air moves from areas of higher pressure to areas of lower pressure, air moves from the environment into the lungs (Figure 20-3).

Under normal conditions, the average amount of air that moves into the lungs on inspiration (and then out of the lungs on expiration) is 5–7 mL/kg, or about 500 mL in an average-sized adult. This is known as the tidal volume. Of this 500 mL, 150 mL remains in the conduction portion of the airway (trachea and bronchi), unavailable for gas exchange. This is known as anatomical dead space air. The amount of air available for alveolar ventilation is
350 mL. The volume of anatomical dead space does not change. If tidal volume decreases to 300 mL, 150 mL remains in the dead space and alveolar ventilation is decreased to 150 mL.

Expiration is stimulated by the Hering–Breuer reflex. When stretch receptors in the lungs are activated, nervous signals stimulate the expiratory center and inhibit the inspiratory center in the brainstem. As the diaphragm and intercostal muscles relax, the volume of the thoracic cavity, and therefore the lungs, decreases. This results in higher intrapulmonary pressure with respect to the environment, so air flows out of the lungs (Figure 20-4).

**FIGURE 20-2**

Relationship between pulmonary and systemic circulation.

**FIGURE 20-3**

The diaphragm and intercostal muscles contract, increasing the volume of the thoracic cavity, which lowers intrathoracic (and intrapulmonary) pressure, allowing inspiration.

**IN THE FIELD**

Anything that decreases tidal volume decreases alveolar ventilation. Shallow breathing means that the amount of oxygen delivered to the cells is decreased.
Patients with respiratory problems present in varying degrees of distress (Table 20-1). The patient’s chief complaint may be mild dyspnea, which is often described as being “short of breath” or “having difficulty breathing.” Patients with severe dyspnea may barely be able to offer a chief complaint between gasps for air. Dyspnea may be accompanied by signs of respiratory distress, such as tripoding, wheezing, coughing, and use of accessory muscles of respiration (Figure 20-5). Signs of hypoxia and exhaustion such as cyanosis, altered mental status, and weak respiratory effort indicate respiratory failure and impending respiratory arrest. Patients in respiratory arrest present with ineffective respiratory effort, or apnea. Cardiac arrest will follow quickly without intervention (Figure 20-6).

All patients with dyspnea must receive supplemental oxygen. The method for delivering oxygen and the amount

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<th>Signs of Respiratory Distress, Respiratory Failure, and Respiratory Arrest</th>
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<tr>
<td><strong>Respiratory rate</strong></td>
<td>Normal Breathing</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>12–20 per minute</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>Free movement of air, adequate depth</td>
</tr>
<tr>
<td>Breath sounds</td>
<td>No abnormal sounds, breaths sounds present and equal in all lung fields</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>Normal</td>
</tr>
<tr>
<td>Patient appearance</td>
<td>Good skin color</td>
</tr>
<tr>
<td>Necessary interventions</td>
<td>Oxygen by nasal cannula if indicated by complaints, history, clinical findings</td>
</tr>
</tbody>
</table>
provided depends on the patient’s degree of distress, adequacy of ventilation, pulse oximetry value, and suspected underlying condition. Patients with chronic obstructive pulmonary disease (COPD) who present with a mild increase in dyspnea and few signs of respiratory distress may do well with oxygen by nasal cannula. Patients with severe dyspnea but adequate ventilation require oxygen by nonrebreather mask to maintain an SpO₂ of 95 percent or higher. Other supportive measures include allowing the patient to assume a position of comfort. For most patients, sitting upright and perhaps leaning forward will provide the greatest level of comfort, as long as other conditions (decreased level of responsiveness or hypotension, for instance) do not contraindicate it. Reassure the patient by telling him that you are there to help.

All patients with inadequate ventilation require supplemental oxygen and assistance with ventilation by bag-valve mask device. None of the general signs and symptoms described earlier gives information about the specific underlying cause of the problem. Maintaining the patient’s airway, breathing, oxygenation, and circulation are critical, but those measures are not directed at reversing the underlying cause of the problem. Unless the underlying problem is corrected, the supportive measures only buy a little time. They do not provide a solution. The patient’s history is often the key to determining the underlying cause of the problem.

Scene Size-Up

Begin, as always, with a scene size-up. Occasionally, a respiratory emergency may not be obvious. Hypoxic patients can behave irrationally because of cerebral dysfunction. Even as you must always ensure your own safety, always consider that there may be a medical cause of the patient’s behavior.

Next, form a general impression before deciding how to proceed with the primary assessment. Respiratory failure and respiratory arrest may present as a decreased level of responsiveness. In this case, respiratory effort may be decreased or absent. For these patients, once you have assessed scene safety, take immediate steps to establish the level of responsiveness and check a carotid pulse. If the pulse is absent after checking for 10 seconds, begin chest compressions and prepare to apply an AED. If the carotid pulse is present, ensure an open airway, and provide or assist ventilations with a bag-valve mask device and supplemental oxygen.

In more responsive patients, the presence of respiratory distress or failure may be obvious from your first glimpse of the patient. Note the patient’s position. Often, patients with respiratory distress are sitting upright and may be leaning forward, supporting themselves with their arms (tripod position). You may see exaggerated chest and abdominal movement and use of accessory muscles, indicating labored breathing. The patient may appear drowsy or confused. You
FIGURE 20-6
(A) Progression from respiratory distress (B) to respiratory failure (C) and respiratory arrest.

may see cyanosis, especially of the lips, ears, and nail beds. You may hear wheezing, coughing, crackles (rales), stridor, or coughing.

If the patient is on oxygen, you will have an immediate indication that the patient has a serious chronic illness. The presence of a nebulizer and a metered-dose inhaler (MDI) also provides a clue that the patient has a respiratory illness (Figure 20-7).

FIGURE 20-7
A patient’s medications, including inhalers and nebulizers, can provide an important clue to a history of respiratory problems. (A) Metered-dose inhaler. (B) Small-volume nebulizer. (C) Advair Diskus. (© Carl Leet, YSU)
Make an initial determination of the patient’s priority for transport. Because dyspnea is an indication of a problem with breathing, patients with this complaint are a high priority for transport.

Respiratory distress can quickly progress to respiratory failure and respiratory arrest. However, effective prehospital treatment can substantially improve the patient’s condition. Be prepared to change the patient’s priority based on reassessment findings. Consider early communication with the receiving hospital, if it appears that the patient will need endotracheal intubation.

Secondary Assessment

In the secondary assessment, focus on things that will provide you with the most relevant information first. That includes auscultation of breath sounds, vital signs, pulse oximetry, capnometry and cardiac monitoring if available, and the patient’s medical history.

The history may prompt you to check for additional signs and symptoms, such as edema in the lower extremities. The patient’s medications and history yield important clues (Table 20-2). Whether or not the patient has a history of respiratory disease, heart disease, allergic reactions, recent surgery, or other medical problems plays a crucial role in clinical reasoning.

Clinical Reasoning Process

Understanding the pathophysiology of various causes of difficulty breathing will help you know what questions to ask as you begin to develop and test hypotheses about the underlying cause of a respiratory emergency. A history of emphysema, for example, tells you not only that the patient has a chronic respiratory disease, but also that he is at risk of heart failure, cardiac dysrhythmia, and complications related to long-term corticosteroid use. On the other hand, sudden respiratory distress in a patient with no history of respiratory disease should lead you to think of acute emergencies, such as pulmonary embolism or spontaneous pneumothorax.

Treatment

In addition to basic treatment aimed at maintaining the patient’s airway, breathing, oxygenation, and circulation, other treatments may be indicated. Patients who are unresponsive and without a gag reflex may require the use of a Combitube® or supraglottic airway device. Continuous positive airway pressure (CPAP) may be indicated to provide ventilatory support for patients with pulmonary edema (Figure 20-9).

IV fluids are important in patients with asthma and pneumonia, and bronchodilators can assist patients with asthma or COPD. If protocols allow, patients with pulmonary edema from heart failure can benefit from
### TABLE 20-2
**Patient Medications That May Indicate a Respiratory Illness**

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<tr>
<th>Drug Category</th>
<th>Examples</th>
<th>Actions</th>
</tr>
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<tbody>
<tr>
<td>Antibiotics</td>
<td>Amoxicillin, azithromycin (Zithromax), ciprofloxin (Cipro), erythromycin</td>
<td>Antibiotics work by various mechanisms to treat bacterial infections such as pneumonia and bronchitis</td>
</tr>
<tr>
<td>Anti-inflammatory (steroids/corticosteroids)</td>
<td>Prednisone, fluticasone (Flovent), triamcinolone (Azmacort), beclomethasone (Beclovent)</td>
<td>Reduces the inflammatory component of asthma and COPD</td>
</tr>
<tr>
<td>Anti-inflammatory (mast cell stabilizers)</td>
<td>Cromolyn (Intal)</td>
<td>Reduces inflammation by preventing mast cells from releasing chemical mediators of inflammation, such as histamine</td>
</tr>
<tr>
<td>Anti-inflammatory (leukotriene inhibitors)</td>
<td>Montelukast (Singulair)</td>
<td>Inhibits the release of leukotrienes, which are chemical mediators of inflammation</td>
</tr>
<tr>
<td>Bronchodilators (short-acting beta2 adrenergic agonists)</td>
<td>Albuterol (Proventil), levalbuterol (Xopenex)</td>
<td>Fast acting, but short duration; cause smooth muscle relaxation in the bronchioles</td>
</tr>
<tr>
<td>Bronchodilators (long-acting beta2 adrenergic agonists)</td>
<td>Terbutaline (Brethine), salmeterol (Serevent), formoterol</td>
<td>Slower acting with a longer duration of action, especially when taken in tablet form; cause smooth muscle relation in the bronchioles</td>
</tr>
<tr>
<td>Bronchodilators (anticholinergics)</td>
<td>Ipratropium (Atrovent), tiotropium (Spiriva)</td>
<td>Inhibits bronchoconstriction through inhibition of parasympathetic (cholinergic) action on the bronchioles</td>
</tr>
<tr>
<td>Bronchodilators (xanthines)</td>
<td>Theophylline</td>
<td>Stimulates respiratory drive and causes bronchodilation; associated with cardiac dysrhythmias</td>
</tr>
<tr>
<td>Cough suppressants (antitussives)</td>
<td>Codeine, hydrocodone, dextromethorphan</td>
<td>Act on the central nervous system to suppress dry coughs</td>
</tr>
<tr>
<td>Expectorants/mucolytics</td>
<td>Guaifenesin (expectorant), acetylcysteine (mucolytic)</td>
<td>Thin mucus to allow it to be more easily expectorated</td>
</tr>
<tr>
<td>Pancreatic enzymes</td>
<td>Pancrelipase (Pancrease)</td>
<td>Used in CF to support pancreatic digestive enzymes that are blocked by mucus secretions</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Portable or fixed oxygen cylinder or oxygen concentrator used with a nasal cannula</td>
<td>Provides supplemental oxygen to patients with advanced chronic respiratory diseases</td>
</tr>
</tbody>
</table>

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**Reassessment**

Ongoing respiratory distress, acidosis, and hypoxia can lead to exhaustion. Patients with respiratory distress can progress quickly to respiratory arrest and failure, even when you are doing everything that can be done in the prehospital setting. Frequent reassessment of the patient’s mental status. A change in mental status is a sign of hypoxia. Ensure that the patient can maintain his airway. Be ready to use suction, positioning, and basic airway adjuncts as needed. Monitor the effectiveness of ventilations, assisting with a bag-valve mask device or implementing CPAP as indicated.

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**FIGURE 20-9**

CPAP can provide significant improvement in gas exchange in patients with some respiratory problems, including pulmonary edema.
Auscultate the breath sounds for changes and communicate with the patient to determine whether treatment has relieved his symptoms. Recheck the vital signs and SpO₂. If cardiac monitoring and capnometry have been implemented, reassess their results frequently, as well. Be prepared to change treatment, if needed, based on the results of reassessment.

**Chronic Obstructive Pulmonary Disease**

Chronic obstructive pulmonary disease (COPD) includes both **emphysema** and **chronic bronchitis**, and is the fourth leading cause of death in the United States (American Lung Association, 2010b). Emphysema and chronic bronchitis typically occur in middle age and are almost exclusively caused by cigarette smoking. Smoking causes 85–90 percent of COPD deaths; most other cases can be attributed to secondhand smoke exposure, occupational exposure, and air pollution. Rarely (2–3 percent of cases), emphysema is caused by a genetic disorder in which there is a deficiency of a lung-protective protein.

Although emphysema and chronic bronchitis have some different features, most patients with COPD have some degree of both diseases, with the features of one or the other predominating. Difficulty breathing is caused by progressive destruction of lung tissue with one or more of the following: decreased diameter of the small airways, loss of elasticity of the airways, obstruction because of inflammation and increased mucus production, and decreased alveolar surface area for gas exchange (National Heart, Blood, and Lung Institute, 2010a).

Increased resistance to blood flow through the pulmonary vasculature means that the right ventricle of the heart must work harder to circulate blood through the lungs. This can result in enlargement of the right ventricle and **right-sided heart failure**, as well as pulmonary artery hypertension. Damage to the right side of the heart can lead to the cardiac dyssrhythmia atrial fibrillation, which is common in patients with COPD. Atrial fibrillation is an irregular heart rhythm and it is not unusual to detect an irregular pulse in patients with a history of COPD. Right-sided heart failure results in edema, particularly of the lower extremities and within the abdomen. Right-sided heart failure caused by pulmonary disease is called **cor pulmonale** (Figure 20-10).

Chronic hypoxia leads to a condition called clubbing of the fingers (Figure 20-11). Some patients with COPD become accustomed to having an increased level of carbon dioxide, making their primary respiratory drive a low oxygen level (hypoxic drive) instead of a high carbon dioxide level.

**Chronic Bronchitis**

In patients with chronic bronchitis, the mucus-producing cells in the bronchi are increased in size and produce more mucus than normal, resulting in a persistent “smoker’s cough.” Destruction of the cilia that line the airway makes it more difficult to rid the airway of the mucus, which allows bacteria to become trapped in the lungs (Figure 20-12). The patient is stable for periods of time but has episodes of decompensation called **acute exacerbation**, often caused by infection.

**IN THE FIELD**

Hypoxic drive, the reliance on decreased oxygen levels as the stimulus to breathe, may occur in some COPD patients. In such cases, prolonged administration of high levels of oxygen may result in respiratory depression. However, you must never withhold oxygen from a patient who needs it. Monitor the patient’s ventilations and provide assistance by bag-valve mask device if respiratory effort becomes inadequate.
Chronic bronchitis is characterized by a cough that produces sputum for a total of three months during two consecutive years (Fayyaz, Hmidi, Nascimento, Olade, & Lessnau, 2010). Patients with chronic bronchitis are typically over 50 years old. As the disease progresses, the patient may take on the classic appearance referred to as a “blue bloater.” Patients with chronic bronchitis become cyanotic during exacerbations, and are prone to right-sided heart failure, which leads to peripheral edema. During acute exacerbations, the patient’s cough may be more frequent and more productive, there may be a change in the character of sputum, and wheezing may occur as a result of bronchoconstriction. Patients with chronic bronchitis are prone to hypercapnia, which can lead to confusion, drowsiness, and headache.

Emphysema

Patients with emphysema have extensive destruction of the walls of the alveoli, resulting in reduced surface area for gas exchange (Figure 20-12). Because of chronic hypoxia, the body compensates by increasing production of red blood cells to carry oxygen. The increased red blood cell level allows the patient to have good skin color, despite being short of breath. The classic presentation of a patient with emphysema is thus called the “pink puffer.”

Patients with emphysema also are prone to acute exacerbations, which may be triggered by exposure to smoke, cold air, or other irritants. The level of respiratory distress increases, and narrowed airways result in wheezing. Patients with emphysema tend to be thin, with well-developed accessory muscles of respiration (Figure 20-13). Air trapped within the damaged alveoli leads to a “barrel-chested” appearance.

The tendency of the damaged alveoli to collapse causes patients to compensate by breathing through pursed lips. This provides increased resistance to expiration and keeps pressure in the lungs higher. This reflex results in conditions similar to positive end-expiratory pressure (PEEP) ventilation used in automatic ventilator settings.

**FIGURE 20-12**
Pathophysiology of chronic bronchitis and emphysema.

**FIGURE 20-13**
Classic appearance of a patient with emphysema.
COPD Management

In both types of disease—chronic bronchitis and emphysema—patients’ quality of life can be severely affected. Both diseases are progressive and cannot be reversed. However, stopping smoking is an important step in slowing the progress of the disease.

In mild cases, patients suffer from dyspnea on exertion. As the disease progresses, dyspnea is more frequent and more severe.

Pneumonia and spontaneous pneumothorax are complications of COPD. Patients are typically treated with corticosteroids or other anti-inflammatory drugs to reduce inflammation. However, the drugs have significant side effects, including increased risk of infection. Long-acting bronchodilators are used to prevent bronchoconstriction, and short-acting bronchodilators (sometimes called rescue inhalers) are used to treat episodes of increased dyspnea.

Bronchodilators include beta2 agonists and parasympatholytic (anticholinergic) drugs. Acute exacerbations of chronic bronchitis are often treated with antibiotics. As the disease progresses, patients may rely on supplemental oxygen part or all of the time.

Despite some differences in the diseases, the goals of prehospital treatment are the same: to improve ventilation and oxygenation. In addition to providing supplemental oxygen and (if needed) assisted ventilation, sympathetic beta2 agonists (albuterol, for example) can be used to treat bronchoconstriction in patients who are wheezing. In some EMS systems, a combined beta2 agonist/parasympatholytic agent, such as ipratropium (Atrovent), is preferred.

Thick, dehydrated mucus in the lungs can lead to obstruction of the bronchi. Hydration decreases mucus plugging. Keep in mind, though, that patients with COPD are prone to heart failure, and fluid overload can be detrimental. Follow protocols for IV fluid administration, and check with medical direction if you have questions about the rate of fluids for specific patients.

Check lung sounds frequently for both improvement in wheezing and development of crackles (rales). The saline used to nebulize bronchodilators will help with expectoration, and humidified oxygen should be used if available.

If transport times are long or for interfacility transport, the physician may order a Venturi mask at a specific oxygen setting. CPAP is used with caution because the increased pulmonary pressures in the weakened lung tissues can lead to pneumothorax.

Asthma

Twenty-three million people in the United States suffer from asthma, a chronic inflammation of the airways with reversible episodes of obstruction (American Lung Association, 2010a). Asthma affects people of all ages, and untreated asthma can lead to death.

Asthma is thought to have a significant genetic component, but can be caused by certain respiratory illnesses in childhood and early exposure to certain viruses and environmental contaminants when the immune system is developing.

Asthma rates are higher in children in urban areas. One reason is thought to be the increased exposure to antigens from cockroaches and other urban contaminants (National Heart, Lung, and Blood Institute, 2010b). Asthma triggers include cigarette smoke, pet dander, pollutants, exercise, respiratory infections, and other irritants.

Asthma Pathophysiology

There are two components to asthma. Patients have chronic inflammation of the bronchioles, which can be exacerbated, causing an “asthma attack.” During an asthma attack, the airways narrow because of smooth muscle constriction, and the underlying inflammation increases (Figure 20-14).

One of the body’s normal responses to lung irritants is constriction of the bronchioles to limit exposure. This reaction is especially pronounced on expiration, because the bronchioles normally dilate on inspiration and constrict on expiration (Martini, Bartholomew, & Bledsoe, 2008). The result is expiratory wheezing and overinflation of the alveoli (air trapping) (Table 20-3).

Even when bronchiolar smooth muscle constriction is treated by beta2 agonists, the underlying increase in inflammation may just be starting. The increased inflammation leads to swelling of the bronchioles and increased mucus production. Additional medications are needed to treat the inflammatory component.
Asthma Management

Two types of medications are used to treat asthma. Anti-inflammatory agents such as corticosteroids, cromolyn sodium, and leukotriene inhibitors are used to decrease inflammation and prevent asthma attacks. Bronchodilators, usually beta₂ agonists and anticholinergics, are used to prevent asthma attacks and to relax bronchiolar smooth muscle to treat acute attacks. In severe asthma attacks that do not respond well to the short-acting beta₂ agonists, additional anti-inflammatory medication may be needed.

Often, an asthma patient or those around him will call 911 after his attempts to reverse an attack with a rescue inhaler have failed. A patient may successfully use his rescue inhaler to treat the initial bronchoconstriction that occurs after exposure to a trigger, but the inflammatory phase that develops hours after exposure to the trigger will cause recurrence of symptoms that cannot be treated by beta₂ agonists. Status asthmaticus is a severe, prolonged, life-threatening asthma attack that does not respond to treatment with bronchodilators. The patient may be approaching or in respiratory failure by the time the ambulance arrives.

Prehospital treatment of asthma patients begins with an assessment of the patient’s level of responsiveness, airway, breathing, oxygenation, and circulation. Altered mental status and signs of exhaustion, cyanosis, and diminished air movement are signs that the patient’s condition is immediately life threatening.

Immediately establish an airway, assist with ventilation, and provide supplemental oxygen. The patient must be transported without delay with an IV established en route. Contact the receiving facility for notification and consult with medical direction about the possibility of using SC or IM epinephrine to treat status asthmaticus.

For asthma patients who are awake and breathing adequately, begin oxygen administration as you complete your assessment and obtain a history. Base oxygen administration on the patient’s level of distress, vital signs, and SpO₂. Obtain as much information as possible about what the patient has done to treat the asthma attack. Patients may already have used more than the recommended amount of their rescue inhaler without significant relief. They may be experiencing side effects associated with the medications, including increased anxiety, palpitations, and tachycardia. If this is case, consult with medical direction before...
administering additional beta₂ agonists. Otherwise, follow your protocol in administering a beta₂ agonist or combined beta₂ agonist/anticholinergic by small-volume nebulizer.

Patients with asthma benefit from hydration with IV fluids. Follow protocols in the amount of fluids to administer. Frequently reassess the asthma patient, including mental status, airway, breathing, vital signs, breath sounds, level of distress, and SpO₂. Keep in mind the possibility of deterioration and be prepared to provide an airway and start ventilations by bag-valve mask device.

**Pulmonary Embolism**

Pulmonary embolism (PE) is a condition in which there is an obstruction to blood flow in the pulmonary arterial system by a blood clot (embolus). This means that part of the lung is not able to participate in gas exchange. Early in the process, air enters the alveoli of the affected part of the lung, but the absence of circulation in the surrounding capillaries means that oxygen does not enter the blood. This imbalance in ventilation and perfusion in the lung is called a ventilation–perfusion (VQ) mismatch.

Because part of the lung is not available for gas exchange, hypoxia can result. The degree of distress suffered by the patient depends on the degree to which lung perfusion is affected. A small embolus affecting circulation to a relatively small number of alveoli may cause mild shortness of breath.

It is not uncommon for patients to present with vague signs and symptoms up to a week before being diagnosed with PE. However, many pulmonary emboli are larger, obstructing large areas of blood flow.

In some cases, a number of smaller emboli may obstruct many different branches of pulmonary circulation. In such cases, patients generally experience a sudden onset of severe dyspnea. This may be accompanied by hypotension and signs of severe respiratory distress and hypoxia. In some cases, the patient will also complain of a sudden onset of sharp chest pain (Table 20-4).

Patients with significant PE can deteriorate quickly, getting worse despite all attempts to oxygenate and ventilate him and increase the blood pressure through fluid administration. Definitive treatment requires anticoagulation to prevent further embolus formation, fibrinolytic therapy to breakdown the existing clot, or less frequently, embolectomy by surgery or catheterization.

Patients at greatest risk for a pulmonary embolism usually have existing medical conditions that predispose them to abnormal blood clot formation. A common source of the embolus is deep vein thrombosis (DVT) of the pelvis or lower extremities.

Although blood clots are the most common cause of pulmonary embolism, air, fat, bone marrow, and amniotic fluid in the circulation also can cause it. Some risk factors include recent surgery, cancer, immobilization (from a fracture, major injury, illness, or obesity), estrogen use (hormone replacement or contraceptives), pregnancy (all trimesters and up to 6–12 weeks postpartum), and older age.

Suspect pulmonary embolism in patients with otherwise unexplained dyspnea and hypoxia (SpO₂ < 95 percent) (Sutherland, 2010). Early in the process, breath sounds are clear and equal, although the nonperfused portion of the lung will undergo atelectasis 24–72 hours after the onset.

The patient’s history may reveal risk factors that increase your suspicion for pulmonary embolism. Other signs and symptoms may exist, but their absence cannot rule it out. Additional signs and symptoms include chest, back, shoulder, or upper abdominal pain. Chest pain may be pleuritic. The patient may experience syncope (fainting), and may have hypotension, tachycardia, hemoptysis, or swelling of one leg. Severely hypoxic patients may present with cyanosis or mottling; cool, diaphoretic skin; altered mental status; respiratory failure; or respiratory arrest.

Treat patients with suspected pulmonary embolism with oxygen by nonrebreather mask, if ventilations are adequate, or bag-valve mask device if ventilations are inadequate. Start an IV. If the patient is hypotensive, administer fluids. Patients with pulmonary embolism who have hypotension are likely to be severely hypoxic and acidotic. Be prepared for respiratory and cardiac arrest. As with any critical patient, notify the receiving facility and if questions about treatment arise, consult with medical direction.

**TABLE 20-4** Signs and Symptoms of Pulmonary Embolism

- Unexplained shortness of breath
- Tachypnea
- Tachycardia
- Hypotension
- Feeling of dread, anxiety
- Syncope
- Diaphoresis
- Chest pain (pleuritic)
- Coughing, hemoptysis
- New cardiac dysrhythmia
- Swollen, tender lower extremity (calf)

**IN THE FIELD**

Although wheezing is a common sign of an asthma attack, keep two things in mind. First, all that wheezes is not asthma. Other conditions, which may require different treatment, also cause wheezing. Second, wheezing can occur only if a certain amount of air is moving through the bronchioles. In a severe asthma attack, little air is moving and wheezing may not be heard. Silence is an ominous sign when auscultating the chest of a patient with respiratory distress.
Pulmonary Edema

Pulmonary edema occurs when there is an increase in interstitial fluid that increases the distance of gas diffusion between the alveoli and pulmonary capillaries. Pulmonary edema is classified as cardiogenic or noncardiogenic.

Cardiogenic Pulmonary Edema

In cardiogenic pulmonary edema, congestion of the pulmonary capillaries from left-sided heart failure can force fluid into the alveoli, where it mixes with air, producing crackles (rales) on auscultation and frothy sputum (Figure 20-15). (See Chapter 21 for the pathophysiology of heart failure.)

Because cardiogenic pulmonary edema is caused by increased afterload (amount of resistance the heart has to overcome to eject blood from the ventricles) and pulmonary vascular congestion, medications that cause vasodilation are important in its treatment. One such medication carried by Advanced EMTs is nitroglycerin. Nitroglycerin may be administered sublingually, according to your protocol or in consultation with medical direction.

Noncardiogenic Pulmonary Edema

Noncardiogenic causes of pulmonary edema include acute respiratory distress syndrome (ARDS) and delayed toxin-induced lung injury (National Association of EMTs, 2007). ARDS is a complication of severe illnesses and is rarely seen in the prehospital setting. Some factors that can directly or indirectly injure the lungs and result in ARDS are shock, sepsis, drug overdose, severe pneumonia, severe trauma or illness, aspiration of stomach contents, and exposure to inhaled toxins. ARDS carries a high mortality rate.

There are measures that you can take to decrease a patient’s risk of developing ARDS. They include recognizing and managing shock by controlling bleeding and ensuring adequate oxygenation, and preventing aspiration of stomach contents through proper airway management, including positioning the patient and having suction readily available.

A number of toxic substances that reach the lungs either by inhalation or blood circulation can lead to noncardiogenic pulmonary edema and other damage to the lungs that may not be evident immediately after exposure. This is sometimes referred to as delayed toxin-induced lung injury (National Association of EMTs, 2007). You should maintain a high index of suspicion for later complications in patients who have a history of exposure to toxins, even when they appear noncritical immediately following the incident.

Toxic causes of noncardiogenic pulmonary edema by inhalation of fumes include smoke produced by fires, chlorine gas, anhydrous ammonia, acid fumes or vapors (such as hydrogen fluoride or sulfur dioxide), hydrogen sulfide (from sewage treatment or farming operations), phosgene, and others (Mullen, 2007).

Toxins introduced to the circulation through injection or oral administration include heroin, methadone, and other opioid drugs; narcotic antagonists such as naloxone (rarely);...
aspirin overdose; calcium channel blocker overdose; some herbal remedies; and scorpion envenomation. In those cases, obtaining and communicating a complete history, including that of herbal medications, occupational exposure to substances, and illicit drug use is critical. Prehospital treatment includes management of ventilation and oxygenation.

**Spontaneous Pneumothorax**

Pneumothorax is a condition in which air has accumulated within the pleural cavity, outside the lung, interfering with the ability of the lung to expand during inspiration (Figure 20-16). In lay terms, this is referred to as a collapsed lung. The degree of impaired ventilation and hypoxia depend on how much air has accumulated within the pleural space.

Although pneumothorax is often the result of trauma, medical patients can have risk factors for pneumothorax. When pneumothorax occurs without trauma, it is referred to as a spontaneous pneumothorax. Lung diseases, such as lung cancer and COPD, result in weakened areas of the lung that can rupture spontaneously or in response to coughing (Figure 20-16). Some otherwise healthy individuals are at higher risk of spontaneous pneumothorax, possibly due to an inherent weakness in the connective tissues of the lung. Those individuals are likely to be young, tall, thin males. In addition to coughing, any other activity that leads to an increase in intrapulmonary pressure, such as exhaling against a closed glottis when lifting something heavy, may result in pneumothorax in these individuals.

When an area of the lung ruptures, air leaks out and accumulates in the pleural space. In most cases, the defect in the lung is small and self-sealing. This limits the amount of air that escapes from the lung. This condition is called simple pneumothorax. The term “simple” can be deceiving, because the amount of air in the pleural cavity on the affected side can be substantial, leading to respiratory distress and hypoxia.

Spontaneous pneumothorax may present with pain, and patients often present with a sudden onset of dyspnea, which may have been preceded by coughing or an activity that would increase intrapulmonary pressure. The amount of dyspnea varies from mild to severe, and may be stable or increasing (Table 20-5).

Lung sounds will often be absent in the affected area, but this can be difficult to detect unless the pneumothorax is large. Breath sounds from the functioning area of the lung can be transmitted to the chest wall over the nonfunctioning portion. You may detect this as diminished, rather than absent, breath sounds. Further complicating assessment of breath sounds, the accumulation of air in the pleural cavity will collect at the top of the pleural cavity. The aspect of the chest that is “at the top” depends on the patient’s position.

Other indications of simple spontaneous pneumothorax depend on the severity of the pneumothorax. The patient may have tachypnea, tachycardia, and a decreased SpO₂. Your suspicion should increase in patients with a history that includes risk factors for spontaneous pneumothorax.

Provide the patient with oxygen. His level of distress and SpO₂ guide dosage and administration. Allow the
patient to assume a position of comfort, which will often be sitting up. Provide reassurance.

If ventilations are inadequate, assist using a bag-valve mask device. Be aware that positive pressure ventilation from a bag-valve mask can increase the leakage of air from the affected lung. Use the minimum amount of force needed to achieve adequate ventilation. If resistance to bagging increases, suspect tension pneumothorax, described in the next section. Never use CPAP or administer nitrous oxide to a patient with a suspected pneumothorax.

Use judgment in starting an IV. A patient in mild distress whose condition is stable may not require an IV in the prehospital setting. A patient in moderate to severe distress should have an IV. Reassess the patient frequently, monitoring mental status, breathing, lung sounds, oxygenation, and vital signs.

**Tension Pneumothorax**

In some cases, the defect in the lung is large and cannot seal itself. This may occur when the rupture of the lung includes a larger bronchus. Air continues to accumulate and cannot escape. Pressure increases within the thorax, first compressing the lung on the affected side, then pushing the structures in the mediastinum toward the other lung, and finally collapsing the opposite lung. This is called a tension pneumothorax. It is a critical, life-threatening emergency.

In addition to signs and symptoms of simple pneumothorax, indications of tension pneumothorax include increasingly severe dyspnea, respiratory failure, cyanosis, distended neck veins, and hypotension. Very late in the condition, the trachea may be deviated away from the affected side. The poor outcome associated with tension pneumothorax is due to obstructive shock. The high intrathoracic pressure impedes blood return to the heart, reducing cardiac output.

Paramedics can perform a life-saving procedure called needle thoracostomy (needle chest decompression) to decompress the chest. If you suspect tension pneumothorax, request ALS. If ALS is not available, transport the patient as quickly as possible while assisting with ventilation. Be aware, however, that ventilation can increase the amount of air escaping from the damaged lung. Use the least amount of pressure needed to ventilate the patient. Start an IV and infuse fluids for hypotension. Notify the receiving facility as early as possible during transport.

**Hyperventilation Syndrome**

Hyperventilation syndrome (HVS) is a condition in which the patient’s minute ventilation exceeds his metabolic demands (Kern & Rosh, 2009). When this occurs, the patient’s arterial pCO₂ may drop, although in some cases the patient’s pCO₂ level is not low enough to explain the signs and symptoms. Patients with HVS may present with dyspnea, anxiety, chest pain, dizziness, near-syncope, weakness, paresthesia (particularly around the mouth and in the hands), and carpopedal spasm.

Patients often have a feeling of suffocation despite an increased respiratory rate and volume and a normal SpO₂. The patient may report mental changes, such as a feeling of unreality. Often, an emotionally distressing event preceding the onset of symptoms can be identified, and the patient may have a past history of similar episodes. HVS occurs more frequently in females, and usually occurs in patients between the ages of 15 and 55 (Kern & Rosh, 2009).

There is a high degree of overlap between HVS and panic disorder. However, there are still physiological mechanisms at work. A number of electrolyte changes are

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**TABLE 20-5** Signs and Symptoms of Simple and Tension Pneumothorax

<table>
<thead>
<tr>
<th>Feature</th>
<th>Simple Pneumothorax</th>
<th>Tension Pneumothorax</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea</td>
<td>Mild to severe</td>
<td>Increasingly severe, progressing to respiratory failure</td>
</tr>
<tr>
<td>Hypoxia</td>
<td>Mild to severe</td>
<td>Increasingly severe</td>
</tr>
<tr>
<td>Lung sounds (sounds from the unaffected lung may be transmitted to the affected side)</td>
<td>Decreased on affected side</td>
<td>Decreased first on affected side, later on both sides</td>
</tr>
<tr>
<td>Chest pain</td>
<td>May be present</td>
<td>Present in 90 percent of cases</td>
</tr>
<tr>
<td>Circulation</td>
<td>May have tachycardia related to anxiety and dyspnea</td>
<td>Hypotension, tachycardia, impaired return of blood to heart results in jugular venous distension; if untreated, cardiac arrest with pulseless electrical activity or asystole</td>
</tr>
<tr>
<td>Other possible physical findings (these signs may rarely be seen and may be subtle; be aware of their meaning, but do not rely on their presence to identify tension pneumothorax)</td>
<td></td>
<td>Pulsus paradoxus, tracheal deviation away from affected side, hyperexpansion of the chest, hyperresonance of the chest to percussion</td>
</tr>
</tbody>
</table>
IN THE FIELD

Do not have the patient breathe into a bag over his face, and do not apply a nonrebreather mask without oxygen in an attempt to have the patient rebreathe his exhaled carbon dioxide.

induced by hypcapnia, and the resulting symptoms are very real. Therefore, you should recognize that the patient cannot simply “calm down.”

It is thought that increased levels of lactate or carbon dioxide can induce hyperventilation in some patients, especially those with panic disorder. Other theories are that patients with panic disorder–induced HVS have an exaggerated fight or flight response.

Do not assume that a patient with increased ventilation has HVS. Hypoxia also causes increased ventilation and anxiety. Death from HVS is rare, but death from hypoxia can occur quickly if not corrected. Consider spontaneous pneumothorax and pulmonary embolism as differential diagnoses.

Although chest pain associated with HVS is usually sharp in nature, it can present similarly to the chest pain of acute coronary syndrome (ACS). HVS also induces ECG changes that can appear similar to those of ACS. If in doubt, older patients should be treated for ACS, but nitroglycerin will not relieve chest pain from HVS. However, hypcapnia from HVS can result in coronary vasospasm, which would be expected to respond to nitroglycerin. Death can occur in severe HVS as a result of vasospasm induced by low carbon dioxide levels and impaired dissociation of oxygen from hemoglobin at the cellular level.

Although you may suspect HVS in the field, it cannot be diagnosed in the field. Conditions leading to serious hypoxia may be present, and HVS itself can have significant complications. Do not take the condition lightly. Supplemental oxygen will not harm, and may help, the patient. Reassurance is important, but do not become frustrated if it seems to have minimal effect. Coaching the patient to use “diaphragmatic” breathing may help. Instruct the patient to focus on using his abdominal muscles to breathe.

Infectious Respiratory Diseases

Viruses, fungi, and bacteria can all cause infection of the upper or lower respiratory system. An upper respiratory infection (URI) may be sinusitis (sinus infection), pharyngitis (sore throat), and rhinovirus infection—the common cold. URIs are rarely life threatening. One exception is epiglottitis, a bacterial infection that causes swelling of the epiglottis, which can lead to airway obstruction. Fortunately, widespread vaccination against Haemophilus influenza has all but eradicated this disease, which now is mostly seen in unvaccinated adults. Laryngitis can occasionally result in respiratory distress as evidenced by stridor. Laryngotracheobronchitis, or croup, produces stridor and a characteristic “seal bark” cough. Both epiglottitis and croup are primarily seen in children and are discussed in Chapter 44.

Lower respiratory infections can lead to impairments of oxygenation and become life-threatening. Pneumonia, influenza, and acute bronchitis are most common and occur in patients of all ages. Respiratory syncytial virus (RSV), which causes inflammation of the bronchioles (bronchiolitis), is most often seen in children and can require hospitalization. (See Chapter 44.) Metapneumovirus is the second most common cause of bronchiolitis.

Two less common but very serious conditions are severe acute respiratory syndrome (SARS), caused by a coronavirus, and hantavirus pulmonary syndrome (HPS), which is spread by deer mice and is typically concentrated in the Four Corners region of the United States (Arizona, Colorado, New Mexico, Utah). Pertussis (whooping cough), which was once under control due to widespread vaccination, is now on the increase and is of particular concern in infants and small children. (See Chapters 28 and 44.)

Pneumonia

Pneumonia is an infectious disease that results in inflammation of the lungs. The causative agent and the products of the body’s inflammatory response to the infection infiltrate the tissues. Pneumonia may be community-acquired or hospital-acquired (nosocomial). Pneumonia can be fatal in individuals with weakened immune systems, including the elderly. Patients with asthma, COPD, heart failure, and other medical problems, as well as smokers, are at increased risk of pneumonia, which exacerbates underlying respiratory conditions.

Pneumonia is quite common in patients who are immobile (for example, because of stroke, surgery, or other problems) or who reside in extended-care facilities. Pneumonia also occurs as a complication of other lower respiratory infections, such as influenza.

Patients with pneumonia can be quite ill, presenting with a cough, difficulty breathing, shaking chills, fever, and malaise. In the elderly, in whom the immune response is diminished, fever may or may not be a prominent sign. Elderly patients may present with altered mental status, such as confusion, because of infection and associated hypoxia.

Dyspnea and respiratory distress range from mild to severe. The patient’s cough is usually productive and the sputum may be yellow or tinged with a rust color.

Pneumonia occurs in one or more lobes of the lung. Breath sounds will be diminished in the affected areas on auscultation. Pneumonia presents a VQ mismatch, in which part of the lung is not ventilated. The blood circulating around the affected alveoli is therefore not oxygenated before circulating back to the tissues.

Differentiating between pneumonia and pulmonary edema can be difficult (Table 20-6). Basic treatment of hypoxia and impaired ventilation is the same. However, some treatments indicated for one condition are not appropriate for the other. Both patients require administration of supplemental oxygen, usually by nonrebreather mask.
In both cases, your protocol may allow you to treat wheezing caused by bronchospasm with an inhaled beta₂ agonist. However, by failing to identify cardiogenic pulmonary edema, the patient may not receive the benefit of treatment with nitroglycerin in the field. Nitroglycerin, however, may cause hypotension in the fluid-depleted patient with pneumonia.

### Acute Bronchitis

Acute bronchitis causes inflammation of the bronchi, with increased mucus production. The presence of mucus in the larger airways produces the rhonchi characteristically heard on auscultation of the lungs. Although anyone can get bronchitis, smokers and patients with lung disease are at increased risk. Acute bronchitis can be caused by viruses (including influenza), bacteria, and irritants.

Patients with acute bronchitis may experience wheezing, coughing, shortness of breath, fever, chills, and malaise.
Lung Cancer

Lung cancer is a common and deadly disease, with 222,520 new cases and 157,300 deaths predicted in 2010 (National Cancer Institute, 2010). The primary cause of lung cancer is smoking, although some types of lung cancer are a result of occupational exposure to asbestos and some have an underlying genetic predisposition. Exposure to radon gas and air pollution also can play a role. Radon gas is produced from the natural decay of uranium in the soil. It seeps into homes through cracks in the foundation and can accumulate. Inexpensive radon detection kits are available, and vent pipe systems can be installed to reduce radon levels.

Lung cancer is often not diagnosed early enough for a cure. The five-year survival rate after diagnosis is just 15.8 percent, in part due to the presence of few signs and symptoms until the disease has progressed.

There are two types of lung cancer: small cell and non-small cell. Small cell lung cancer is less common (13 percent of cases) but spreads very quickly. Non-small-cell lung cancer accounts for the remaining 87 percent of cases.

A patient may inform you of what stage his cancer is in. Staging is based on the degree to which the cancer has metastasized. Stages of lung cancer range from stage 0, in which the cancerous cells are localized to the innermost
Cystic Fibrosis

Cystic fibrosis (CF) is a relatively rare (affecting 30,000 people in the United States) genetic disease of the secretory glands (Cystic Fibrosis Foundation, 2009). Many organs are affected, including the lungs and digestive tract. The presence of two defective genes, one inherited from each parent, results in the production of extremely viscous mucus. In the respiratory tract, the thick secretions can obstruct the airways and lead to life-threatening infection. In the pancreas, mucus blocks the ducts of the exocrine glands that secrete digestive enzymes into the digestive tract.

Until recent years, patients with CF died as children or very early in adulthood. Advances in treatment are allowing CF patients to live into middle age, meaning it is no longer a disease that affects only children. The median life expectancy for patients with CF is in the mid-30s (Cystic Fibrosis Foundation, 2009). Some patients now survive into their 40s and 50s.

You are most likely to encounter CF patients with an acute respiratory emergency, but they are also prone to electrolyte disturbances, bowel obstruction, pancreatitis, dehydration, osteoporosis, and diabetes.

Treat the patient for his signs and symptoms. Administering oxygen without humidification may aggravate the tenacious mucus in the lungs, making it harder to expectorate. However, do not withhold oxygen. IV fluids may assist in hydrating the mucus. Consult with medical direction for the type of fluid and rate of administration. CPAP may be useful for patients with impending respiratory failure (Leder & Dorkin, 2010). Administer a nebulized bronchodilator for wheezing, if allowed by medical direction.

Clinical Reasoning Process

Advanced EMTs Toby Marshall and Brent Croft are providing care for Mr. Emerson, a 60-year-old with a history of COPD who presents today with respiratory distress, fever, confusion, and a productive cough. Mr. Emerson is severely hypoxic; his breath sounds include wheezes, rhonchi, and crackles (rales); and he has bilateral pedal edema.

Toby and Brent realize that they must immediately begin to correct Mr. Emerson’s hypoxia. They administer oxygen by nonrebreather mask, with plans to start an albuterol treatment for the bronchospasm indicated by wheezing.

Toby knows that he must carefully observe Mr. Emerson for signs of respiratory failure and be ready to intervene. En route to the hospital, while Mr. Emerson is receiving an albuterol treatment by small-volume nebulizer, Toby starts an IV of normal saline at 30 mL/hour.

Once the albuterol treatment is finished, Mr. Emerson’s wheezing is reduced, but he remains short of breath with rhonchi and crackles (rales). Despite oxygen and the albuterol treatment, his SpO₂ is 87 percent, and he is becoming more exhausted. Toby begins treatment with CPAP and notifies the hospital emergency department of Mr. Emerson’s condition. He continues to reassess Mr. Emerson’s condition en route.
CHAPTER REVIEW

Chapter Summary

Acute and chronic respiratory problems can be life threatening because of impaired ventilation and oxygenation. Respiratory distress can quickly progress to respiratory failure and respiratory arrest as the underlying problem progresses and the patient becomes exhausted, hypoxic, and acidotic.

Problems include COPD and lung cancer, which are highly associated with smoking, as well as asthma, pulmonary edema, hyperventilation syndrome, infectious diseases, and cystic fibrosis.

Your ability to recognize signs and symptoms, obtain a relevant history, and develop a clinical impression of the problem are important in deciding how to best manage the patient. Quick recognition of the patient’s level of distress and intervention to restore and maintain ventilation and oxygenation can be life saving.

In some cases, you will administer specific treatments, such as bronchodilators, aimed at treating the underlying cause of respiratory distress. In all cases, your ability to empathize and calmly interact with the patient is critical.

Review Questions

Multiple-Choice Questions

1. The reason sympathetic beta2 agonists are indicated in asthma is because they:
   a. reduce inflammation.
   b. increase expectoration.
   c. relax bronchiolar smooth muscle.
   d. stimulate respiratory drive.

2. An average-size adult who has taken a narcotic overdose has a tidal volume of 250 mL. The amount of air reaching the alveoli for gas exchange is about _____ mL.
   a. 250
   b. 200
   c. 150
   d. 100

3. The primary chemical stimulus to breath is a(n) _____ level.
   a. increased carbon dioxide
   b. decreased carbon dioxide
   c. increased carbon dioxide
   d. decreased oxygen

4. The most important medication for any patient with respiratory distress is:
   a. albuterol.
   b. ipatroprium.
   c. oxygen.
   d. epinephrine.

5. COPD includes which one of the following conditions?
   a. Emphysema, chronic bronchitis
   b. Acute bronchitis, chronic bronchitis
   c. Emphysema, cystic fibrosis
   d. Asthma, cystic fibrosis

6. The two components of asthma are:
   a. infection and inflammation.
   b. infection and bronchospasm.
   c. inflammation and bronchospasm.
   d. loss of alveolar surface area and bronchospasm.

7. Which one of the following best describes the underlying problem in cystic fibrosis?
   a. Secretion of thick, sticky mucus
   b. Smooth muscle spasms
   c. Abnormal cell growth
   d. Exposure to toxins

8. Which one of the following would you expect in the early stages of a pulmonary embolism?
   a. Absent breath sounds on the affected side
   b. Fever
   c. Sudden onset of dyspnea
   d. Rhonchi

9. The primary cause of lung cancer is:
   a. asbestos exposure.
   b. smoking.
   c. genetic.
   d. radon gas.

10. Which one of the following is contraindicated in suspected pneumothorax?
    a. Oxygen by nonrebreather mask
    b. Nitrous oxide
    c. IV fluids
    d. Bag-valve mask device
Critical-Thinking Questions

11. How would you recognize a patient with a tension pneumothorax?

12. What are some risk factors for pulmonary embolism?

13. What are some circumstances in which you would suspect noncardiogenic pulmonary edema?

14. Compare and contrast expected findings in cardiogenic pulmonary edema and pneumonia.

References


