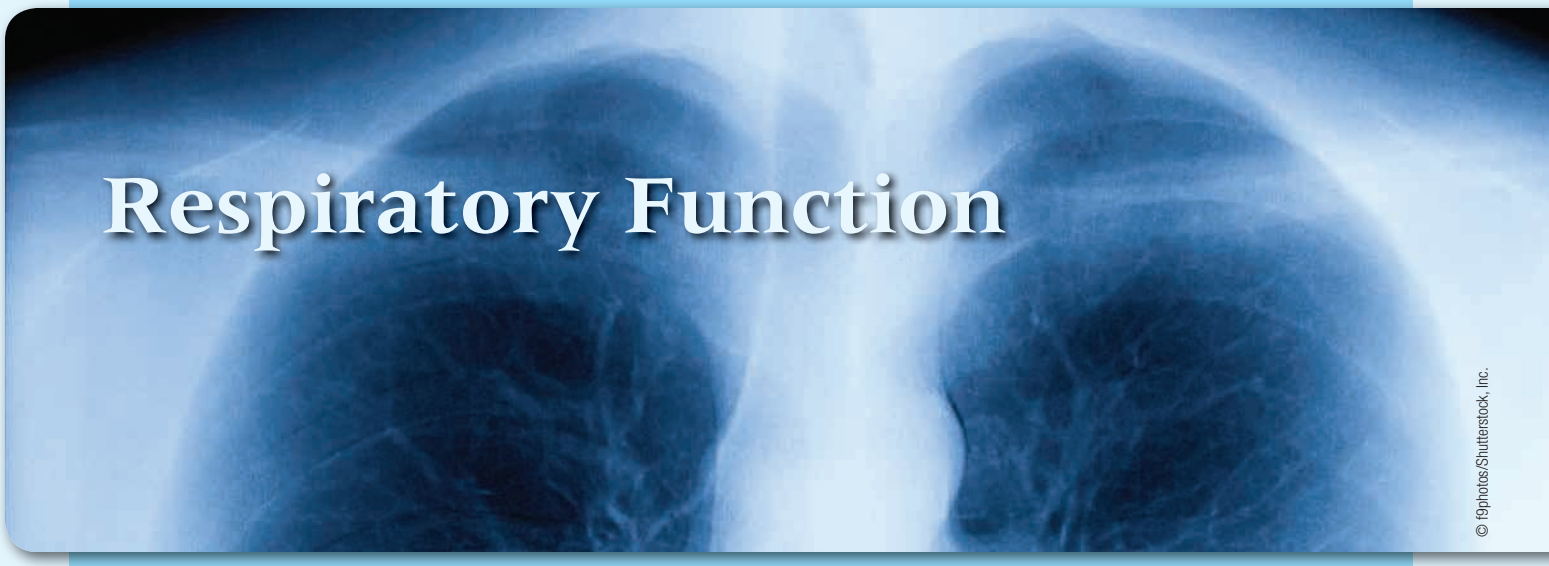
CHAPTER 5

Respiratory Function



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LEARNING OBJECTIVES

- Discuss normal respiratory anatomy and physiology.
- Describe and compare infectious disorders of the respiratory system.
- Describe and compare obstructive diseases of the respiratory system.
- Describe and compare restrictive diseases of the respiratory system.

KEY TERMS

active infection
acute bronchitis
acute lung injury (ALI)
acute respiratory distress syndrome (ARDS)
acute respiratory failure (ARF)
alveolus
aspiration pneumonia
asthma
atelectasis
bacterial pneumonia
blue bloaters
bronchiole
bronchiolitis
bronchopneumonia
bronchus
chronic bronchitis
chronic obstructive pulmonary disease (COPD)
cilium
community-acquired pneumonia
cystic fibrosis
diaphragm
drug-induced asthma
emphysema
epiglottis
epiglottitis
exercise-induced asthma
expiration
expiratory reserve volume
extrinsic asthma
forced expiratory volume in 1 second
forced vital capacity
infectious rhinitis
influenza
inspiration
inspiratory reserve volume
interstitial pneumonia
intrinsic asthma
laryngitis
laryngotracheobronchitis
larynx
Legionnaires' disease
lobar pneumonia
lung cancer
Middle East respiratory syndrome
minute respiratory volume
mucus
Mycoplasma pneumoniae
nocturnal asthma
non-small-cell carcinoma
nosocomial pneumonia
occupational asthma
perfusion
pharynx
pink puffers
pleural effusion
pleurisy
Pneumocystis jiroveci pneumonia
pneumonia
pneumothorax
primary TB infection
residual volume
secondary TB infection
severe acute respiratory syndrome (SARS)
sinusitis
small-cell carcinoma
spontaneous pneumothorax
status asthmaticus
surfactant
tension pneumothorax
tidal volume
trachea
traumatic pneumothorax
tuberculosis (TB)
type A influenza
type B influenza
type C influenza
ventilation
ventilation/perfusion ratio (VQ ratio)
viral pneumonia
vital capacity

The respiratory system includes the organs and structures associated with breathing and gas exchange. The structures of the respiratory system are grouped into two branches—the upper respiratory tract (mouth, nasal cavity, pharynx, and larynx) (FIGURE 5-1) and the lower respiratory tract (trachea, bronchi, bronchioles, and alveoli). This chapter focuses on normal and abnormal states of the lungs. The respiratory tract functions automatically to provide cells with oxygen and to remove carbon dioxide waste. Disorders of the respiratory tract can become serious quickly because of the body's critical need for oxygen. Patients with these disorders will need astute nurses who respond quickly yet thoughtfully.

Anatomy and Physiology

The respiratory system provides vital oxygen and removes toxic carbon dioxide through the act of breathing. The respiratory tract allows a person to breathe in and out approximately 23,000 times each day. In fact, if you had a dollar for each breath, you would be a millionaire in a month and a half. The act of breathing allows for gas exchange of oxygen and carbon dioxide. Oxygen is necessary for cells to produce energy through cellular metabolism; carbon dioxide is the waste product of this process. Through these functions, the respiratory system plays a pivotal role in maintaining homeostasis.

The respiratory system consists of two basic functional divisions—an air-conducting portion and a gas-exchanging portion (**TABLE 5-1**). The air-conducting portion delivers air to the lungs, while the gas-exchanging portion allows gas exchange to occur between the air and the blood (Figure 5-1). The gas-exchanging portion of the respiratory tract includes the lungs with their millions of **alveoli** and capillaries (FIGURE 5-2).

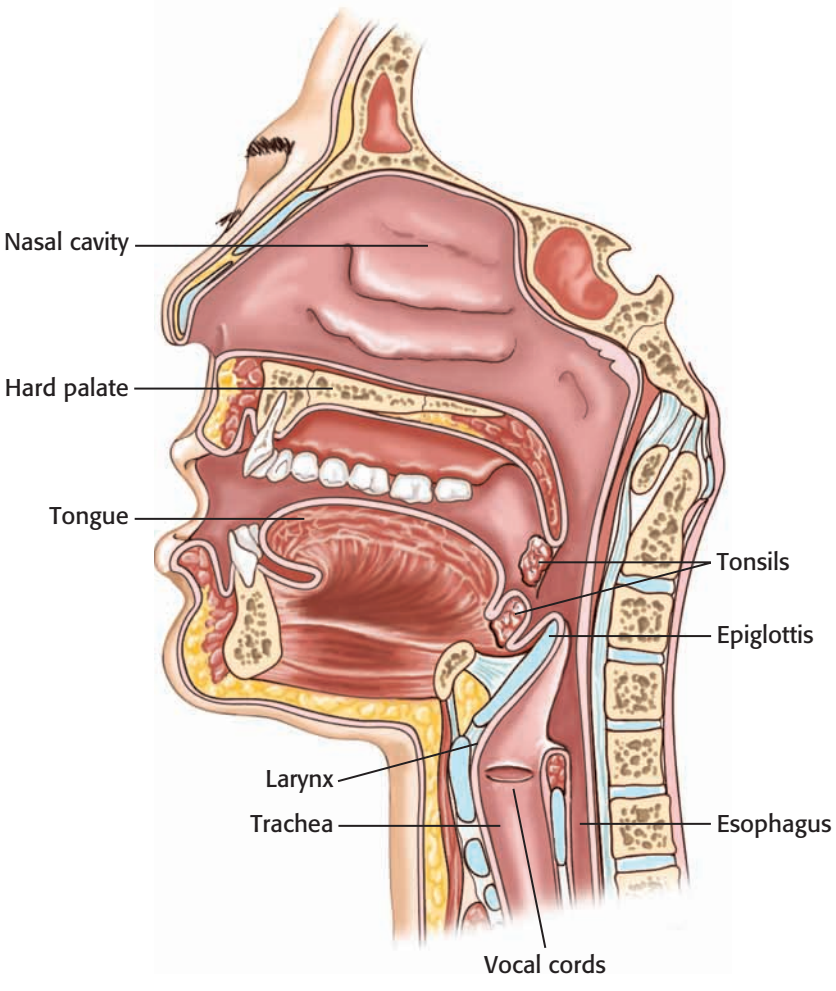

FIGURE 5-1 The upper respiratory tract.

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TABLE 5-1 Summary of the Respiratory System

Organ	Function
Air Conducting	
Nasal cavity	Filters, warms, and moistens air; also transports air to pharynx
Oral cavity	Transports air to pharynx; warms and moistens air; helps produce sounds
Pharynx	Transports air to larynx
Epiglottis	Covers the opening to the trachea during swallowing
Larynx	Produces sounds; transports air to trachea; helps filter incoming air; warms and moistens incoming air
Trachea and bronchi	Warm and moisten air; transport air to lungs; filter incoming air
Bronchioles	Control air flow in the lungs; transport air to alveoli
Gas Exchange	
Alveoli	Provide area for exchange of oxygen and carbon dioxide

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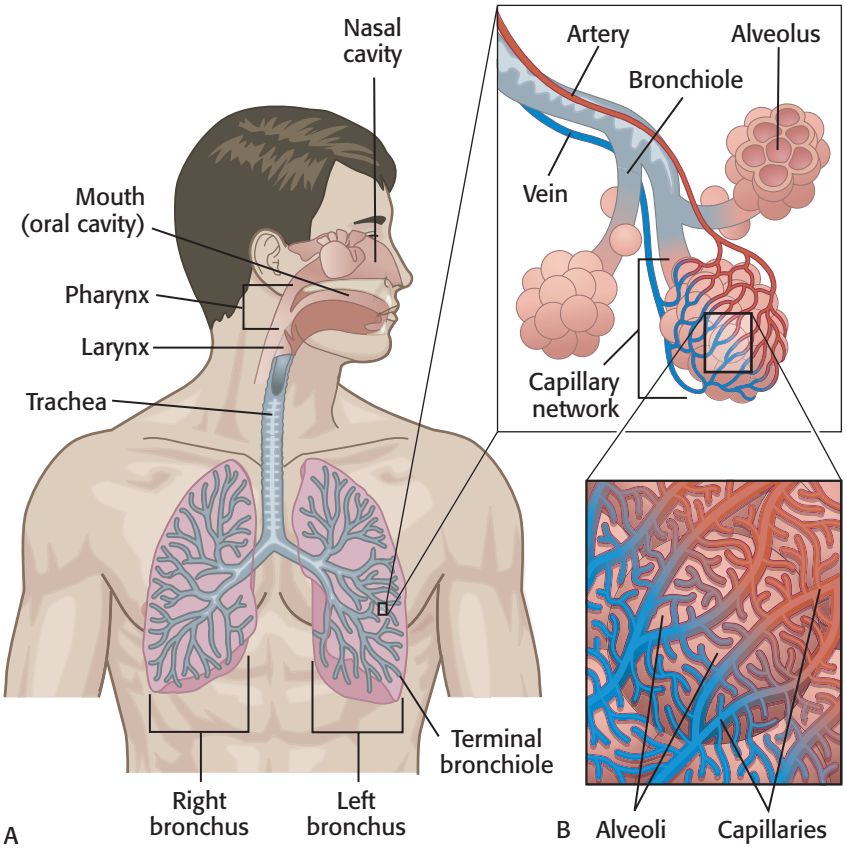

FIGURE 5-2 The respiratory system. (a) This illustration shows the air-conducting portion and the gas-exchange portion of the human respiratory system. The insert shows a higher magnification of the alveoli where oxygen and carbon dioxide exchange occurs. (b) A scanning electron micrograph of the alveoli, showing the rich capillary network surrounding them.

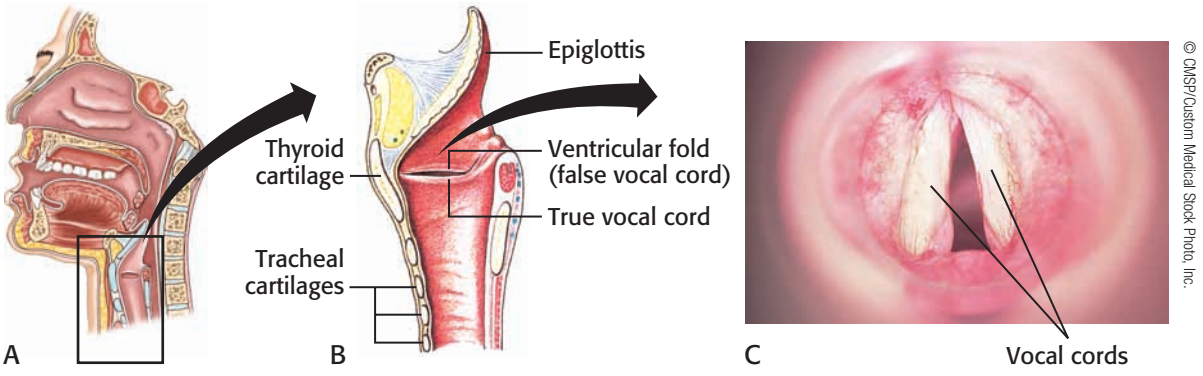

FIGURE 5-3 The vocal cords. (a) Uppermost portion of the respiratory system, showing the location of the vocal cords. (b) Longitudinal section of the larynx showing the location of the vocal cords. Note the presence of the false vocal cord, so named because it does not function in phonation. (c) View into the larynx of a patient showing the true vocal cords from above.

Air enters the respiratory system through the nose and mouth, traveling to the **pharynx**. The pharynx joins the **larynx**, or voice box (FIGURE 5-3). The larynx is made of cartilage and plays a central role in swallowing and talking. When food is swallowed, the larynx rises so that it is closed by the **epiglottis**. This process prevents food and liquids from entering the lungs, where they would cause severe irritation. Food occasionally enters the lungs, often triggering the cough reflex (a primitive protective reflex). The larynx works much like the strings of a guitar or violin to produce sound—tightening and loosening to change pitch. It opens up into the **trachea**, or windpipe. From the trachea, the air travels to the mainstem **bronchi**, where it branches into the right and left bronchi, one for each lung (FIGURE 5-4). The left bronchus is narrow and positioned more horizontally than the right bronchus; the right bronchus is shorter and wider than the left bronchus and extends downward more vertically. Because of the difference in size between the two, objects are more easily inhaled (aspirated) into the right bronchus.

Inside the lungs, the bronchi branch extensively into smaller and smaller tubes, or **bronchioles**, until they reach the alveoli. This branching from larger to smaller mimics the vessels in the cardiovascular system. The walls of the bronchioles are also like the vessel walls in that they mostly consist of smooth muscle. The smooth muscle allows for constriction and dilation of the bronchioles to control air flow. When oxygen needs are higher (e.g., during exercise or stress), the airways open more (dilate) to allow more air to enter the lungs. In times of normal or decreased oxygen needs (e.g., during sleep), the airways may narrow (constrict) slightly. Disease processes may cause constriction to the point of impeding air flow—a dangerous development.

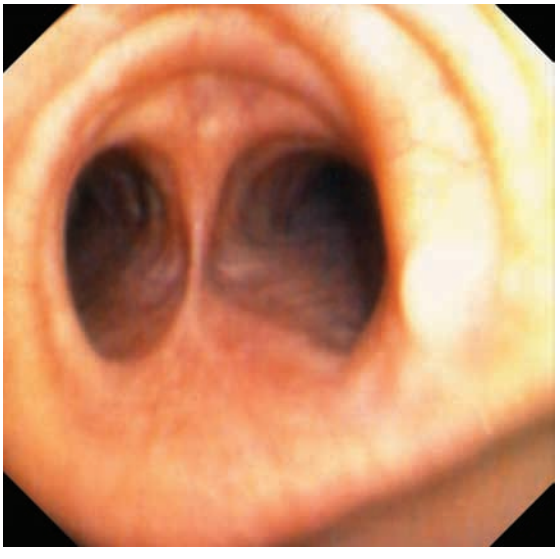

FIGURE 5-4 The bifurcation of the trachea at the carina into the right and left mainstem bronchi.

The air entering the respiratory tract often contains particles that can be harmful. These particles may include infectious organisms (e.g., bacteria, viruses, and fungi) and environmental agents (e.g., dust, pollen, and pollutants). The respiratory system is equipped to filter out some of these particles as well as to protect against the body those that gain entry. The air-conducting portion of the respiratory tract filters many particles by trapping them in

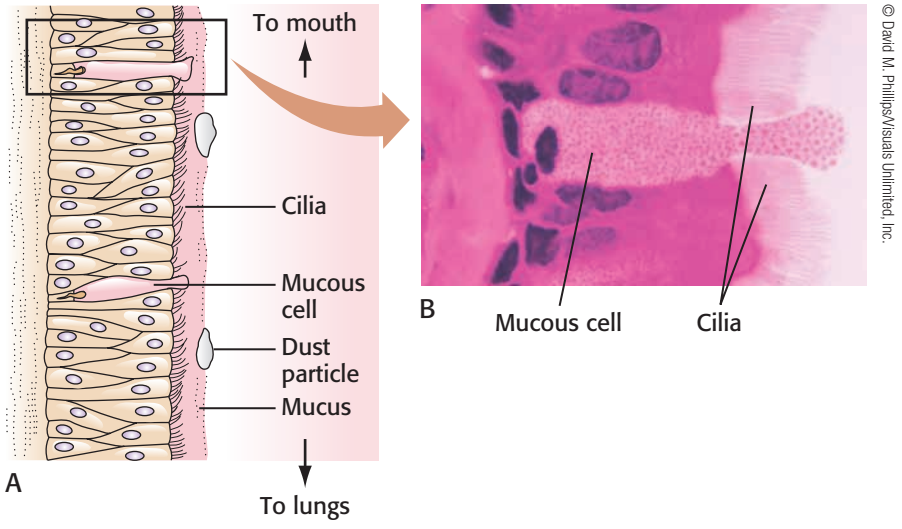

FIGURE 5-5 Mucous trap. (a) Drawing of the lining of the trachea. Mucus produced by the mucous cells of the lining of much of the respiratory system traps bacteria, viruses, and other particulates in the air. The cilia transport the mucus toward the mouth. (b) Higher magnification of the lining showing a mucous cell and ciliated epithelial cells.

the mucous layer (FIGURE 5-5). **Mucus** is a thick, sticky substance produced by the goblet cells in the epithelial lining of the nose, trachea, and bronchi. This epithelial lining also contains many **cilia**, or hair-like projections, that move in a wavelike motion to propel the mucus and trapped particles upward to the mouth, where they can be expectorated (spit out). Cigarette smoking and air pollution can decrease mucus production and destroy cilia, increasing the risk of respiratory infections. Alcohol consumption can paralyze cilia, also increasing infection risk.

Additionally, the immune system is outfitted with IgA cells that prevent the attachment and invasion of bacteria and viruses on mucous membranes (see the *Body Defenses* chapter). Macrophages are also present around the alveoli in the lungs; they keep the lungs clean by phagocytizing particles that gain access to this area (FIGURE 5-6). Once the macrophages fill with particulates, they move into the surrounding connective tissue. When an unusually large number of particulates are present (e.g., with cigarette and marijuana smoking and when breathing in heavy pollution), the lungs become blackened by the accumulation of the particles.

The air-conducting portion of the respiratory system also moistens and warms incoming air. An extensive network of capillaries lies beneath the epithelium of the respiratory tract. These capillaries release moisture into the incoming air, humidifying it to prevent drying of the respiratory tract. The warm blood circulating through the capillaries warms this air prior to entering the lungs, protecting the lungs from cold temperatures. As the air leaves the respiratory tract, much of the water that has been added to the air condenses on the slightly cooler lining of the nasal passages. The condensation is recycled for the next inhalation to conserve water, and contributes to runny noses on cold days.

Alveoli are the site for gas exchange with the bloodstream (FIGURE 5-7). Oxygen is delivered to the alveoli by the air-conducting portion of the respiratory system, and carbon dioxide is brought to the lungs by the circulatory system. Each human lung contains approximately 150 million alveoli that create a surface area approximately the size of a tennis court for gas exchange. The alveoli and capillaries are often a single cell layer thick, which further facilitates gas exchange. The amount of gas exchanged depends on the total surface area available and the thickness of the alveoli and capillary walls. The more surface area and the thinner the layers, the more rapidly gas diffuses.

Gas exchange in the alveoli requires adequate **ventilation** of air and **perfusion** of blood flow. The **ventilation/perfusion ratio**, or **VQ ratio**, is a measurement of the efficacy and adequacy of these two processes. In the ideal lung, inspired air reaches all the alveoli and all the alveoli have the same blood supply. Actually,

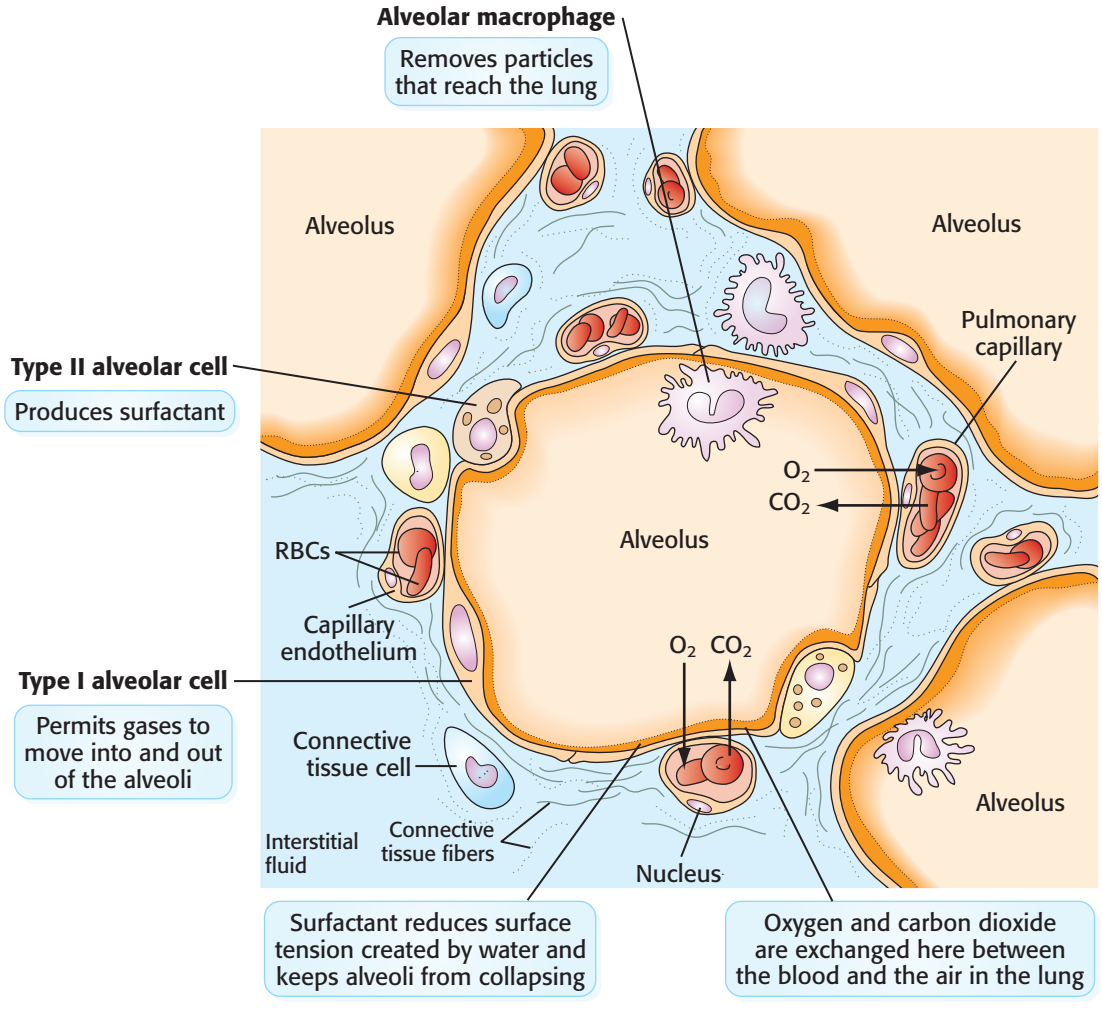

FIGURE 5-6 The alveolar macrophages.

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neither alveolar ventilation nor capillary blood flow is uniform. The supply of air and blood never match perfectly, even in healthy persons. Because of gravity, the lower parts of the lungs have greater blood flow than the upper parts. Distribution of alveolar ventilation from the top to the bottom of the lungs is also uneven. Normal ventilation is 4 liters of air per minute, and normal perfusion is 5 liters of blood per minute. This makes the expected VQ ratio 4/5, or 0.8. A VQ ratio higher than 0.8 indicates that ventilation exceeds perfusion, and a VQ ratio less than 0.8 indicates poor ventilation. Some respiratory disorders involve ventilation problems, whereas others result from perfusion issues. In any event, the result is impaired gas exchange.

When air is inhaled, gases are exchanged between the alveoli and the capillaries; carbon dioxide is removed through expiration, and oxygen is delivered to cells by the cardiovascular system (FIGURE 5-8). Hemoglobin carries oxygen to the cells, then releases it there. The rate at which hemoglobin binds and releases oxygen is affected by several factors, including temperature and pH, among other (FIGURE 5-9).

The surface of the alveoli contains a substance called **surfactant**. Surfactant is a lipoprotein produced by alveoli cells that has a detergent-like quality. This watery substance produces surface tension on the alveoli, which enhances pulmonary compliance (elasticity) and prevents the alveoli from collapsing. Because the pressure in the lungs is negative compared with the atmospheric pressure, the walls of the alveoli tend to draw inward, making them collapse. This pressure is much like that seen with a vacuum-sealed pack of coffee. The pressure and, therefore, the risk of collapse further increase at the end of expiration. Surfactant promotes reinflation of the alveoli during

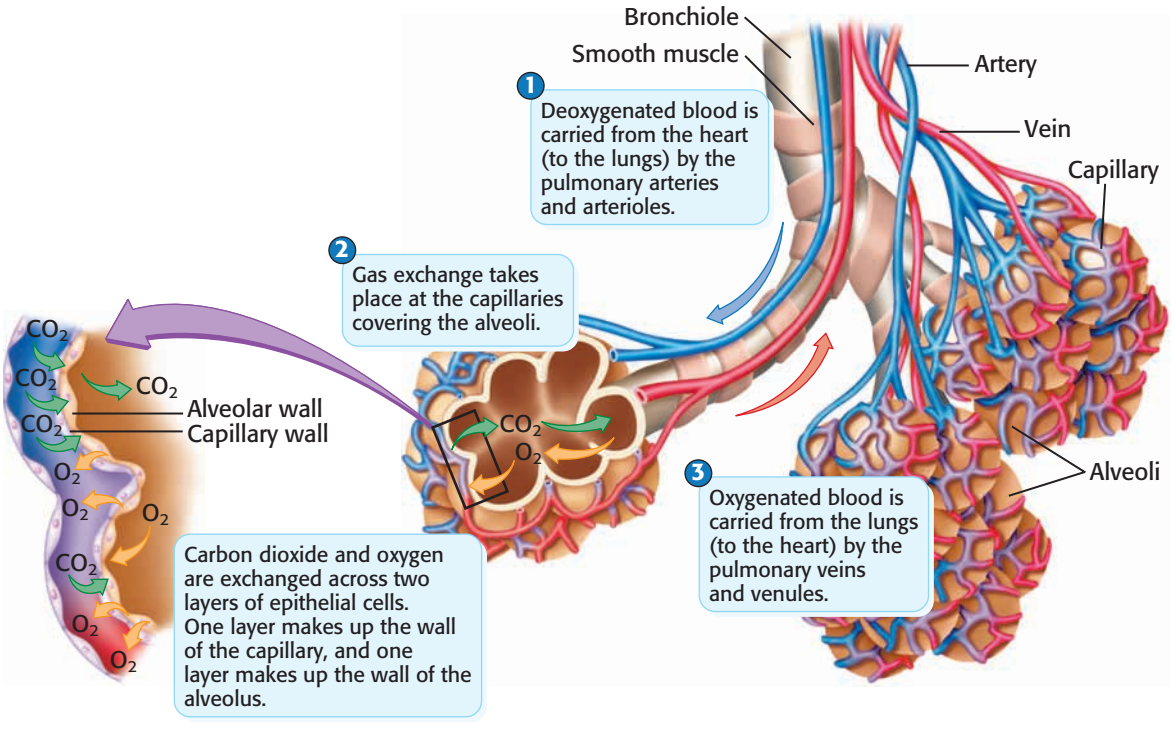

FIGURE 5-7 Gas exchange in the lungs.

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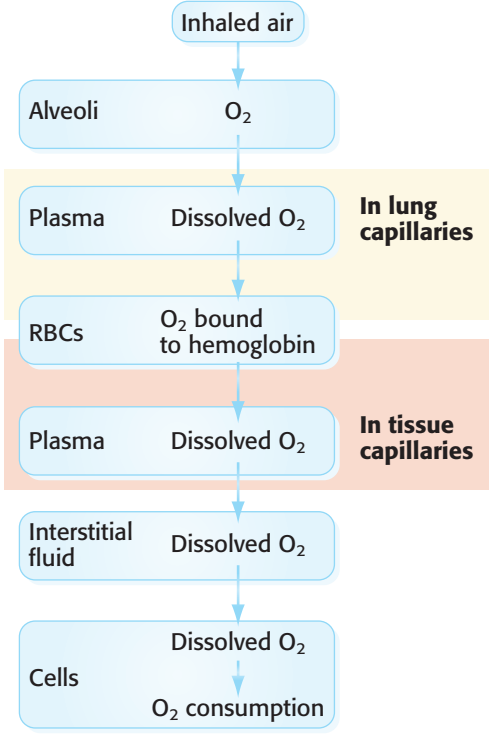

FIGURE 5-8 Oxygen diffusion. Oxygen travels from the alveoli into the blood plasma, then into the RBCs, where much of it binds to hemoglobin. When the oxygenated blood reaches the tissues, oxygen is released from the RBCs and diffuses into the plasma, then into the interstitial fluid and body cells.

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inspiration. Disease states and other conditions can decrease surfactant, leading to the collapse of the alveoli (called atelectasis). For example, premature infants lack surfactant, and smoking alters surfactant production. Synthetic surfactant may be administered to overcome any inadequacies in production.

The process of breathing is largely involuntary and controlled by the medulla oblongata in the brain. This center is located in the brain stem, which controls many vital functions in the body (e.g., heart rate, blood pressure, and temperature). Breathing includes two phases: **inspiration** (inhalation—moving air in) and **expiration** (exhalation—moving air out). Inspiration is an active neural process that begins with nerve impulses traveling from the brain to the **diaphragm**, a dome-shaped muscle that separates the thoracic and abdominal cavities (FIGURE 5-10). These impulses cause the diaphragm to contract, lower, and flatten, which draws air into the lungs. Inspiration also involves the intercostal muscles between the ribs. Nerve impulses cause the intercostal muscles to contract, lifting the ribs up and out. Contraction of the diaphragm and intercostal muscles changes the intrapulmonary pressure, causing air to naturally flow into the lungs. In contrast, expiration is passive—it does not require

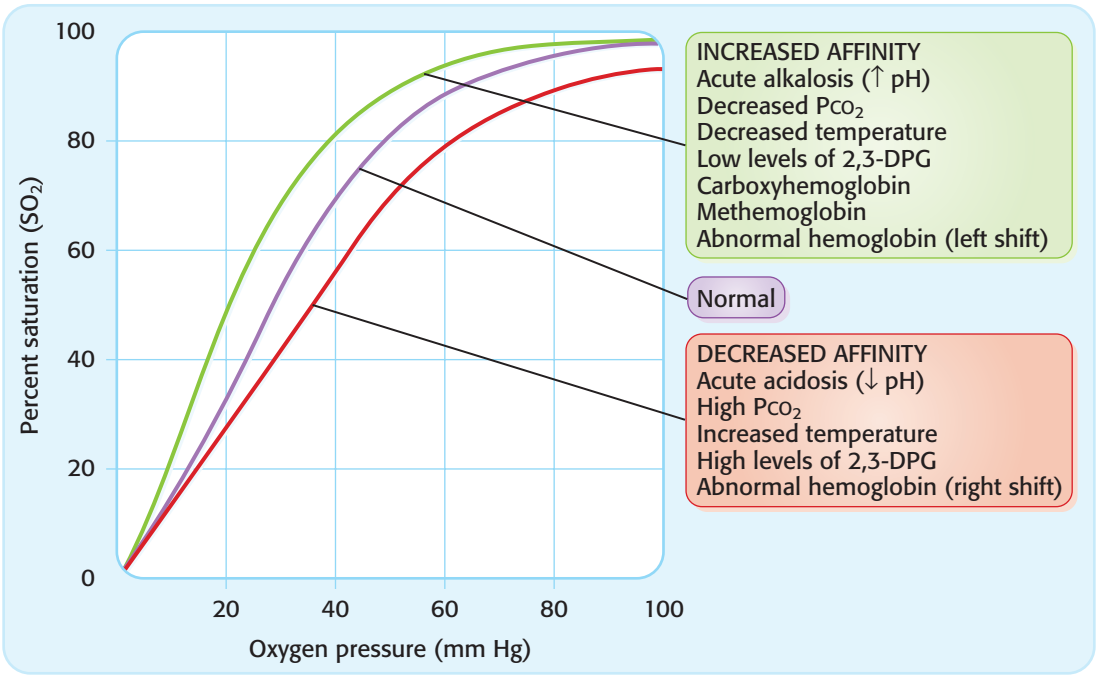


FIGURE 5-9 Oxyhemoglobin dissociation curve.

muscle contraction. As the lungs fill with air, the diaphragm and intercostal muscles relax, returning to their previous position. Returning to their natural position decreases thoracic volume and increases intrapulmonary pressure. This greater pressure forces air out of the lungs. Elastic fibers in the lungs aid in passive expiration by causing the lungs to recoil. Expiration can also be active—that is, created by contracting the chest and abdominal muscles. Air flow, both inspiratory and expiratory, can be measured to aid in diagnosis of respiratory disorders (FIGURE 5-11).

The pulmonary function test evaluates lung volumes and capacities. **Tidal volume** is the amount of air involved in one normal inhalation and exhalation. The average tidal volume is 500 mL, but is smaller in shallow breathing. The **minute respiratory volume** is the amount inhaled and exhaled in 1 minute. This volume is calculated by multiplying the tidal volume by the number of respirations per minute; the average is 6 liters per minute. The **inspiratory reserve volume** is the amount of air beyond the tidal volume that can be taken in with the deepest inhalation. Inspiratory reserve volume averages 2–3 liters. The **expiratory reserve volume** is the amount of air beyond tidal volume that can be forcibly exhaled beyond the normal passive exhalation. The average expiratory reserve volume is 1–1.5 liters. The **vital capacity** is the sum of the tidal volume and reserves. Some air is always present in the lungs, which is called **residual volume**. Even after the most forceful exhalation, 1–1.5 liters of air remains in the lungs, which ensures efficient and consistent gas exchange. The **forced expiratory volume in 1 second** is compared to the **forced vital capacity** to diagnose pulmonary disease.

The medulla controls breathing through nerve cells that generate nerve impulses to the respiratory muscles. When the lungs are full, these impulses cease, allowing the muscles to relax. Chemoreceptors inside the brain and arteries also regulate breathing. These receptors detect carbon dioxide levels and send messages to the medulla. Carbon dioxide levels normally drive breathing (FIGURE 5-12). When these levels go up, respiration depth and rate increase to excrete the excess carbon dioxide, and vice versa. In some disease states, this drive becomes altered, and oxygen levels drive breathing. Additionally, stretch receptors in the lungs aid in breathing by detecting when the lungs are full. In such a case, the stretch receptors in the lungs send a message to the medulla to cease firing. This effect, which is called Hering-Breuer reflex,

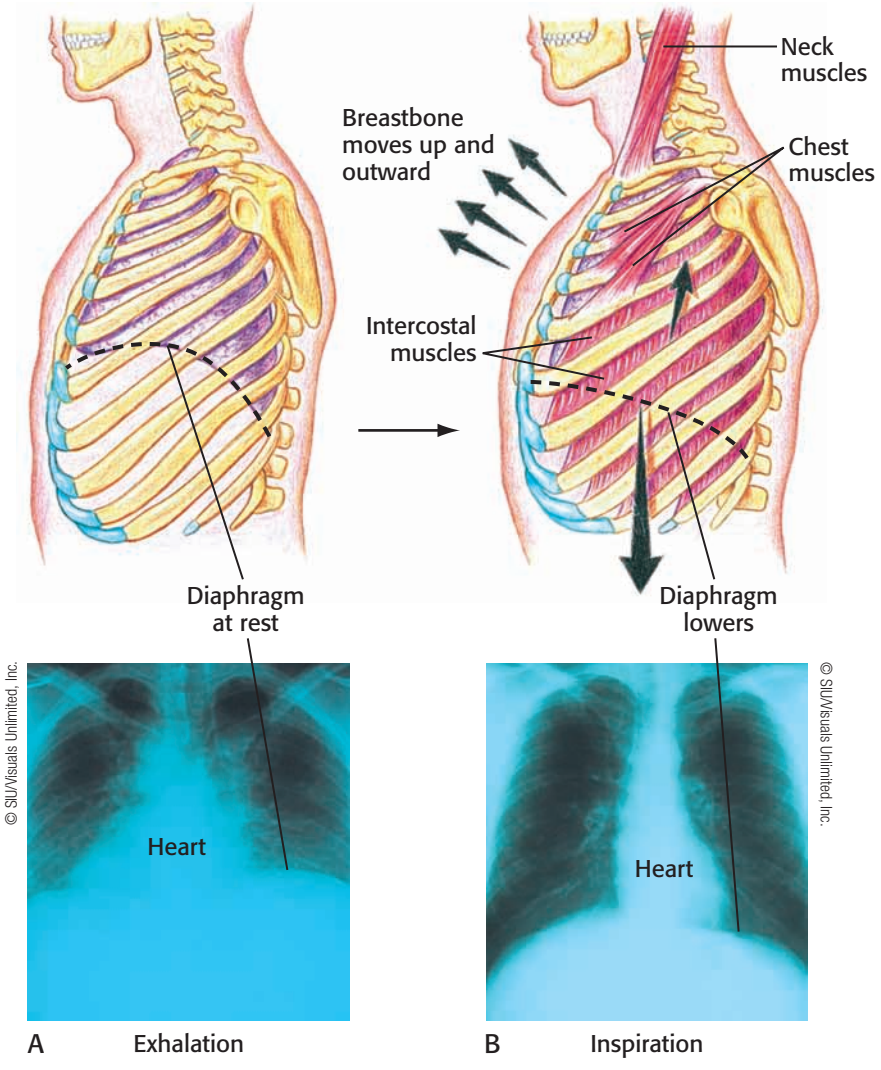


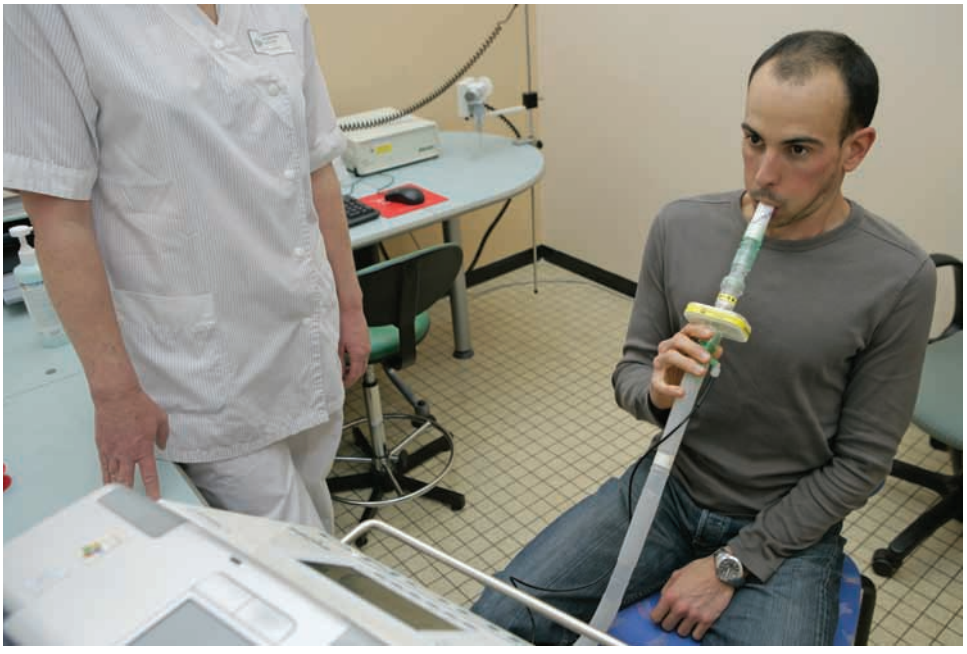
FIGURE 5-10 Breathing. The rising and falling of the chest wall through the contraction of the intercostal muscles (muscles between the ribs) is shown in the diagram, illustrating the bellows effect. Inspiration is assisted by the diaphragm, which lowers. Like pulling a plunger out on a syringe, the rising of the chest wall and the lowering of the diaphragm draw air into the lungs. Illustrations and X-rays showing the lungs in full exhalation (a) and full inspiration (b).

prevents overinflation of the lungs. The body also has oxygen receptors, but they are not very sensitive. These receptors do not generate impulses until oxygen levels fall to a critical point.

In addition to regulating oxygen and carbon dioxide levels, the lungs aid in regulating pH by altering breathing rate and depth. Carbon dioxide is a source of acid in the body. Increasing the respiratory rate and depth will lead to excretion of more carbon dioxide, making the blood less acidic. Conversely, decreasing the respiratory rate and depth will cause retention of more carbon dioxide, making the blood more acidic. This compensatory mechanism allows for a quick fix of pH imbalances to reestablish homeostasis (also see the *Fluid, Electrolyte, and Acid–Base Homeostasis* chapter).

LEARNING POINTS

Carbon dioxide is the normal driving force for breathing. This means that breathing is controlled by carbon dioxide levels. How does this translate into action? As carbon dioxide levels rise, the lungs will exhale to expel the excess carbon dioxide. To understand how strong this drive is, consider holding your breath. If you take a deep breath and try to hold it, eventually you have to let the air out. No matter how hard you try, you cannot hold your breath forever. The body can be trained to hold a breath longer and longer (as swimmers and divers do), but no matter the training, you will eventually have to let it out.



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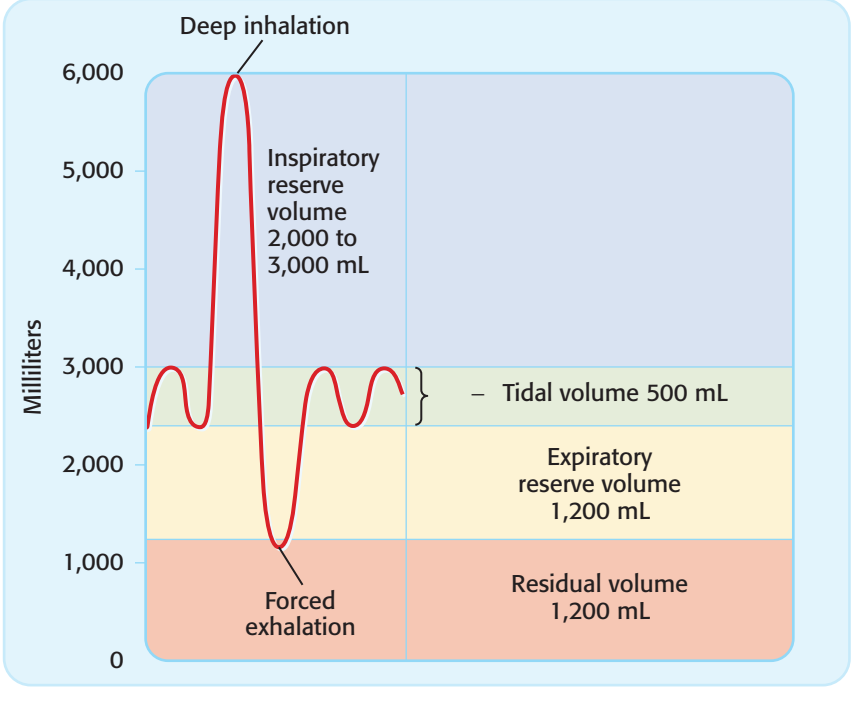


B

FIGURE 5-11 Measuring air flow. (a) This machine allows healthcare workers to determine tidal volume, inspiratory reserve volume, and other lung-capacity measurements to determine the health of an individual's lung. (b) This graph shows several common measurements.

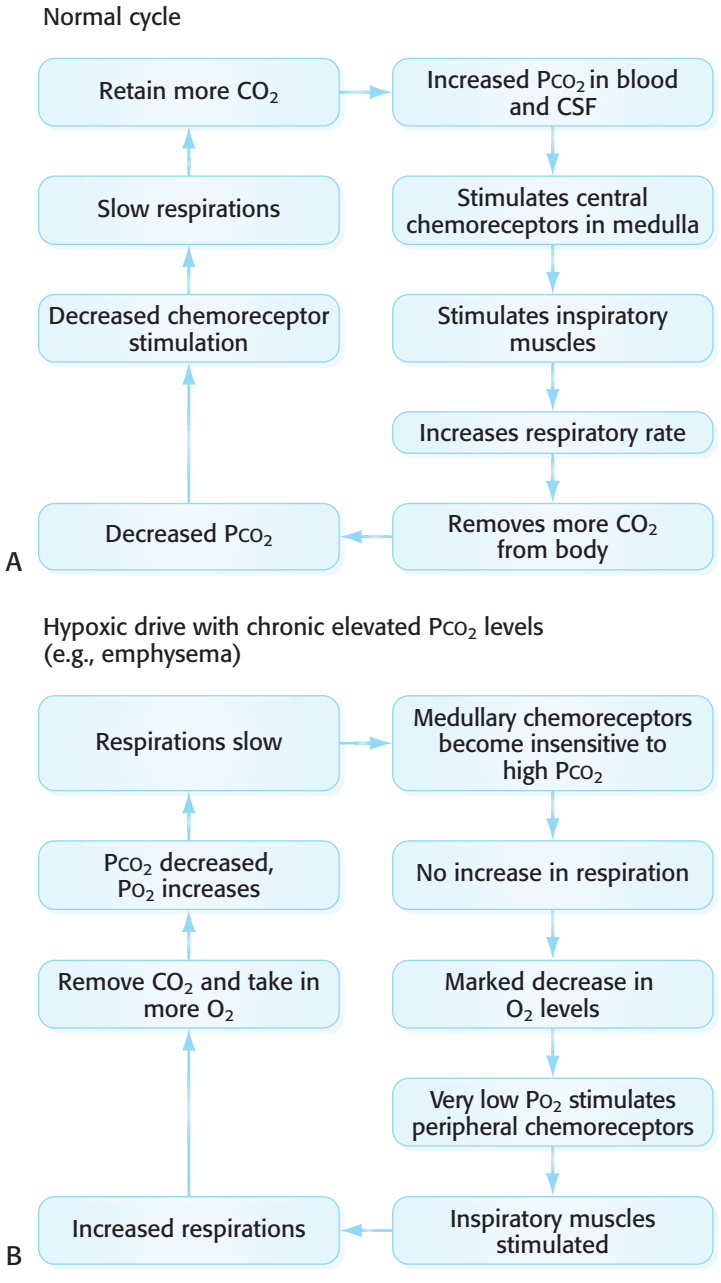


FIGURE 5-12 Normal respiratory control and hypotoxic drive.

UNDERSTANDING CONDITIONS THAT AFFECT THE RESPIRATORY SYSTEM

When considering alterations in the respiratory system, organize them based on their basic underlying pathophysiology to increase understanding. Those pathophysiological concepts include those conditions that are infectious in nature, problems with ventilation, and issues with perfusion. Infectious conditions trigger the inflammatory

response, explaining many of the symptoms associated with these conditions. The respiratory system requires both ventilation and perfusion to function properly. Problems in either area will impair the respiratory system's ability to meet the body's needs. Ventilation conditions result from problems with moving air in or out of the lungs. Perfusion conditions result from problems that prevent gas exchange; these conditions are generally cardiovascular in nature (e.g., pulmonary embolism).

When considering these conditions, also determine which respiratory-related nursing diagnosis is in play—altered gas exchange or ineffective airway clearance. Altered gas exchange includes conditions that decrease gas exchange in the lungs (e.g., asthma, emphysema, and chronic bronchitis). Impaired airway clearance includes conditions in which secretions are stagnant (e.g., pneumonia and cystic fibrosis).

Infectious Disorders

Upper Respiratory Tract Infections

Infectious Rhinitis

Infectious rhinitis, or the common cold, is a viral upper respiratory infection. The most frequent culprit is the rhinovirus, but many viruses (e.g., adenovirus, coronavirus, and influenza) can cause this illness. In fact, there are more than 100 causative organisms, making it difficult to develop immunity.

The infectious organism invades the epithelial lining of the nasal mucosa. Mild cellular inflammation leads to nasal discharge, mucus production, and shedding of the epithelial cells. This break in the first line of defense increases vulnerability to bacterial invasions. As a consequence, secondary bacterial infections (e.g., otitis media, sinusitis, bronchitis, and pneumonia) are common with viral infections (FIGURE 5-13). The risk for secondary bacterial infections is further increased in individuals who smoke because of the chronic damage done by smoke to the mucosa and cilia. Despite popular misconceptions, wet and cold conditions do not cause or increase occurrences of infectious rhinitis. Close physical contact with the virus transmits the infection through exchanges with other humans (e.g., shaking hands) and surfaces (e.g., doorknobs and telephones). Transmission may occur through both inhalation and contact (e.g., hand to hand or hand to mucous membrane). Hands are virally contaminated 60% of the time. The apparent increase in occurrence of the infectious rhinitis during rainy and cold weather is due to increased congregation in confined spaces. Those persons in closer contact with other people will be at higher risk for developing the infection (e.g., children in daycare centers, healthcare providers, and teachers). The virus is highly contagious because it is shed in large numbers from the nasal mucosa, and the virus can survive for several hours outside the body.

An individual who contracts infectious rhinitis usually experiences an incubation period between the invasion of the virus and the onset of symptoms that usually lasts about 2–3 days, but can be as long as 7 days. Clinical manifestations include the following signs and symptoms:

- Sneezing
- Nasal congestion or stuffiness
- Clear nasal discharge
- Sore throat
- Lacrimation (eye tearing)
- Nonproductive cough
- Malaise
- Myalgia
- Low-grade fever
- Hoarseness
- Headache
- Chills

MYTH BUSTERS

A common misconception is that you get colds from being cold or wet. The fuel for this myth is the increased occurrence of colds during cold and wet weather. In fact, the weather conditions themselves do not make you sick—but the weather *does* increase congregation of people indoors to avoid those weather conditions. The congregation of people in close, closed spaces is responsible for the spread of the cold. The infectious rhinitis virus is virulent and highly contagious through close contact.

Misconceptions are hard to change; it usually takes multiple efforts. So do your part to educate the public about the truth of cold transmission and prevention!

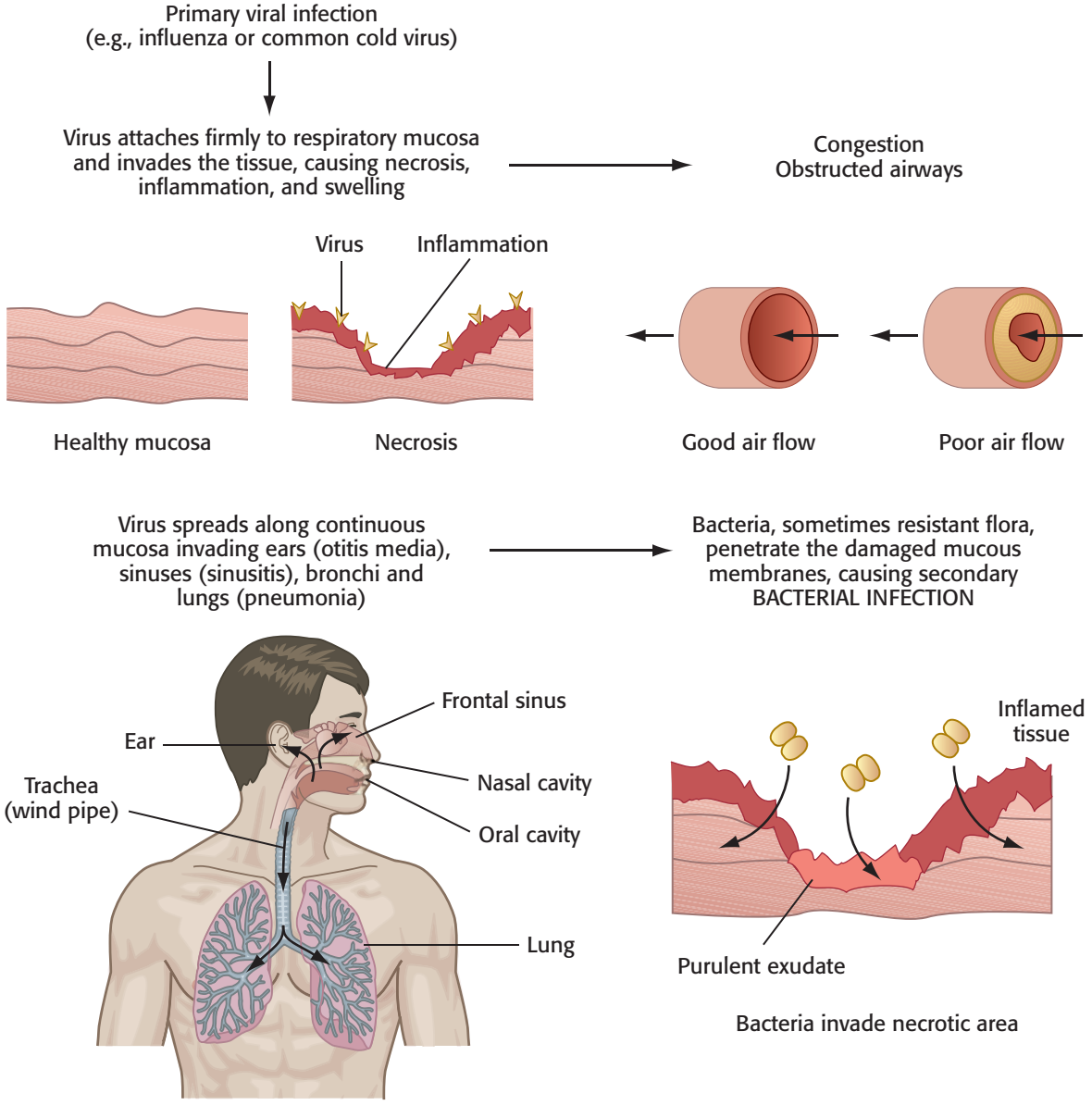


FIGURE 5-13 Complications of viral respiratory infections.

Diagnosis of infectious rhinitis is primarily made based on the presence of symptoms. Treatment is symptomatic. Most over-the-counter cold preparations are ineffective in shortening the course of the infection, and they should be avoided in children younger than 4 years. Pharmacologic therapies that may be used include antipyretics (for fever), analgesics (for discomfort), decongestants (for nasal symptoms), cough suppressants or expectorants, and antibiotics (only if a bacterial infection is present). Humidifiers can liquefy secretions to aid in expectoration. Maintaining adequate hydration can also liquefy secretions and help manage fevers. Nasal saline can ease nasal discomfort. The benefit of vitamin C in prevention and treatment remains controversial. Proper hand washing remains the long-standing cornerstone of prevention because the hands are a significant source of transmission. Other measures will limit the spread of active infections to others:

- Covering one's mouth when coughing and sneezing, using tissue or the upper sleeve of one's shirt
- Disposing of tissue immediately after use

Sinusitis

Sinusitis is an inflammation of the sinus cavities most often caused by a viral infection (e.g., rhinovirus, influenza, or adenovirus). Other causative agents include bacteria (e.g., *Streptococcus pneumoniae* or *Haemophilus influenzae*) and fungi (e.g., *Aspergillus* or mucormycosis). Environmental irritants (e.g., smoke exposure and air pollutants), being immunocompromised (e.g., HIV), conditions that increase mucus production (e.g., cystic fibrosis or asthma), and nasal structural abnormalities (e.g., nasal polyps) can further increase risk. Sinusitis can be a result of a secondary bacterial infection associated with infectious rhinitis or allergic rhinitis in which the drainage from the sinus cavity has become blocked (FIGURE 5-14). The drainage accumulation provides a supportive medium for bacterial growth. *Streptococcus pneumoniae* and *Haemophilus influenzae* are commonly found in the upper airways of healthy people.

Sinusitis can present in several different types:

- Acute, which last up to 4 weeks
- Subacute, which lasts 4–12 weeks

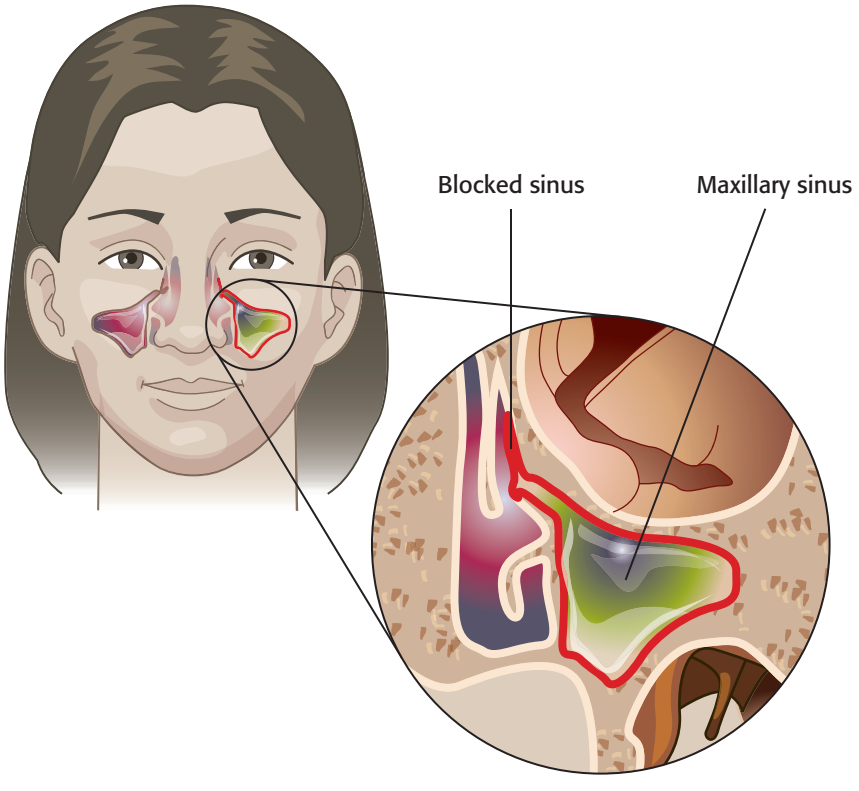


FIGURE 5-14 Blocked sinus.

- Chronic, which lasts more than 12 weeks and can continue for months or even years
- Recurrent, with several attacks occurring within a year

As exudate accumulates, pressure builds in the sinus cavity, which causes facial bone pain and headache. The location of pain can indicate which sinus is affected. Other clinical manifestations of sinusitis may already be present, such as nasal congestion, purulent nasal discharge, halitosis, mouth breathing, fever, sore throat, and malaise. Complications can include orbital cellulitis, meningitis, osteomyelitis, and abscess.

Diagnostic procedures for sinusitis include a history, physical examination, nasal cultures, sinus X-ray, sinus computerized tomography (CT), and sinus transillumination (FIGURE 5-15). Treatment usually includes decongestants (to shrink swollen nasal membranes), analgesics, and nasal corticosteroid spray (to shrink swollen nasal membranes) until the sinuses begin draining. Bacterial infections require antibiotic therapy to resolve. Other measures may include humidifiers (to liquefy secretions and aid in drainage) and avoidance of irritants (e.g., smoke, chemicals, and other allergens). In children with recurrent sinusitis and other upper respiratory infections, removing the adenoids may resolve the issue.

Epiglottitis

Epiglottitis is a life-threatening condition of the epiglottis, the protective cartilage lid covering the trachea opening. In the past, *Haemophilus influenzae* type B (Hib) was the most common cause in the United States, but widespread use of the Hib vaccine has dramatically decreased the rate of *H. influenzae* infections in recent years. With the Hib vaccine, epiglottitis is now very uncommon. Common culprits now include Group A beta-hemolytic *Streptococcus*, *Streptococcus pneumoniae*, and *Staphylococcus aureus*. Other causes include throat trauma from events such as drinking hot liquids, swallowing a foreign object, a direct blow to the throat, or smoking crack or heroin. The inflammatory response is triggered by these events, causing the epiglottis to quickly swell and block the air entering the trachea, leading to respiratory failure. The bacteria can also travel to the bloodstream, leading to sepsis, which is also life threatening because it activates a massive immune response. Epiglottitis was once most often seen in children ages 2–6, but rarely seen in adults. This trend may change with the impact of the Hib vaccine.

The onset of clinical manifestations is typically rapid and includes the following signs and symptoms:

- High fever
- Chills and shaking
- Sore throat and hoarseness

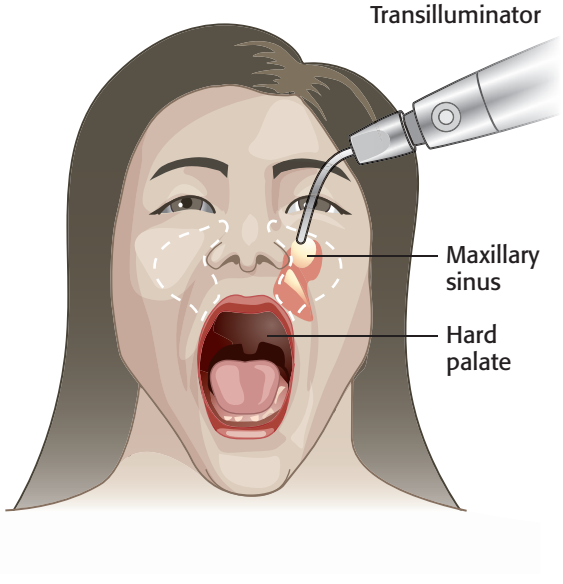


FIGURE 5-15 Transillumination of the sinuses.

- Dysphagia (difficulty swallowing)
- Drooling with the mouth open
- Mild inspiratory stridor (a harsh, high-pitched sound made as a result of air turbulence)
- Respiratory distress
- Central cyanosis (blue discoloration of the mouth and lips)
- Anxiety, irritability, or restlessness (a result of hypoxia)
- Pallor
- Assuming a sitting tripod position, often leaning slightly forward (a subconscious attempt to facilitate breathing)

If epiglottitis is suspected, maintaining the airway and stabilizing respiratory status is a priority before diagnostic procedures are performed. Efforts to preserve respiratory function include humidified oxygen therapy (likely delivered via mask), endotracheal intubation with mechanical ventilation, and tracheotomy. Additional efforts to minimize oxygen consumption should include keeping the patient calm and controlling fever (e.g., with antipyretics and hydration). Aerosolized epinephrine and systemic steroids (often treatments for croup) should be avoided because they may worsen the condition. Additionally, a tongue depressor should not be used to examine the patient. Once the patient is stabilized, diagnostic procedures include visualization of the epiglottis through a fiber-optic camera, X-rays (throat and chest), cultures (throat and blood), arterial blood gases (ABGs), and a complete blood count (CBC).

Intravenous antibiotics will be used to treat infections quickly. Hib vaccinations should be encouraged for children, the elderly, and immunocompromised persons so that transmission rates will continue to be low. Other prevention strategies include proper hand washing, avoiding crowds, cleaning objects (e.g., toys), and not sharing objects (e.g., pacifiers and bottles).

Laryngitis

Laryngitis is an inflammation of the larynx that is usually a result of an infection, increased upper respiratory exudate, irritants (e.g., stomach acid, inhaled smoke, or chemicals), or overuse. Viral infections (e.g., influenza, rhinovirous, and adenovirus) are the most common cause. With laryngitis, the vocal cords become irritated and edematous because of the inflammatory process. This inflammation distorts sounds, leading to hoarseness and in some cases making the voice undetectable. Occasionally, the airway can become blocked. Laryngitis can also be associated with croup and epiglottitis.

LEARNING POINTS

Epiglottitis is often misdiagnosed as croup. One distinct difference in manifestations can assist in moving to appropriate life-saving treatment more quickly. With epiglottitis, patients "look worse than they sound." With croup, they "sound worse than they look" due to the seal-like barking cough. Additionally, cough is not generally seen with epiglottitis. Although severe croup can cause complete airway obstruction, it is rarely life threatening. Picking up on this significant manifestation can save the patient's life!

Clinical manifestations of laryngitis usually last less than a week and include the following signs and symptoms:

- Hoarseness
- Weak voice or voice loss
- Tickling sensation and raw feeling in the throat
- Sore, dry throat
- Dry cough
- Swollen nodes of the neck
- Leukocytosis (if bacterial)
- Difficulty breathing (in children)

Diagnostic procedures for laryngitis include a history, physical examination, CBC, and laryngoscopy. A biopsy may be conducted if symptoms persist, because throat cancer can mimic acute laryngitis. Treatment depends on the cause, and many times the laryngitis will improve without treatment. Treatment strategies aim to increase comfort or decrease the duration of the inflammation:

- Warm humidity
- Resting the voice
- Increasing fluid intake
- Treating the underlying cause (e.g., infection or gastric reflux)
- Analgesics
- Throat lozenges
- Gargling with salt water
- Avoidance of decongestants (they dry out the mucous membranes)

Laryngotracheobronchitis

Laryngotracheobronchitis, or croup, is a common viral infection in children 3 months to 3 years of age. Other children and adults may also contract it. Routine causative agents include parainfluenza viruses, adenoviruses, and respiratory syncytial virus. Additional causes include bacterial infections, allergens, and irritants (e.g., stomach acid, inhaled smoke, or chemicals). Outbreaks and epidemics occur in autumn to early winter, but cases can occur sporadically

year-round. Croup was once a deadly disease caused by diphtheria bacteria, but the introduction of antibiotics and immunizations have improved its prevention and treatment. Today, most cases of croup are mild. Nevertheless, this disease can still be dangerous.

Croup usually begins as an upper respiratory infection with nasal congestion and cough. The larynx and surrounding area swell, leading to airway narrowing and obstruction. This swelling can lead to respiratory failure. Clinical manifestations worsen at night and usually resolve in about a week. These manifestations include the following signs and symptoms:

- Low-grade fever
- Nasal congestion
- Seal-like barking cough (because of laryngeal swelling)
- Hoarseness
- Inspiratory stridor
- Mild expiratory wheezing
- Dyspnea
- Anxiety
- Cyanosis

Diagnostic procedures for croup consist of a history, physical examination, X-rays (throat), throat cultures, ABGs, and CBC. A throat X-ray will reveal a narrowing of the trachea (often referred to as the steeple sign) in 50% of cases (FIGURE 5-16).

Croup is usually self-limiting but can be life threatening without supportive therapy. Treatment strategies include cool humidity, corticosteroids (to decrease edema), humidified oxygen (e.g., an oxygen tent over an infant's crib), bronchodilators (to open the airway), hydration (to combat fever, moisten the airway, and liquefy secretions), and aerosolized epinephrine (in severe respiratory distress). Cool humidity may decrease edema, but no studies have supported this treatment. This intervention should be avoided in patients with asthma because it may trigger bronchial constriction. Cool humidity can be accomplished with a cool-mist humidifier, exposure to cool outside air especially at night, and exposure to cold shower mist in a closed bathroom. Strategies to decrease oxygen consumption include keeping the patient calm and avoiding unnecessary procedures. Additionally, educating the public regarding diphtheria vaccination compliance is critical to manage this once life-threatening condition.

Acute Bronchitis

Acute bronchitis is an inflammation of the tracheobronchial tree or large bronchi. This inflammation is most commonly caused by a wide range of viruses (e.g., influenza, rhinovirus, respiratory syncytial virus, and adenovirus). Bacterial invasions (e.g., *Streptococcus pneumoniae* and *Haemophilus influenzae*), irritant inhalation (e.g., smoke, marijuana, pollution, and ammonia), and allergic reactions are less frequent causes. Young children, the elderly, and smokers are at the highest risk for developing acute bronchitis.

In this condition, the airways become irritated and narrowed due to the results of the inflammatory process (e.g., capillary dilation, edema, and exudate). Clinical manifestations of acute bronchitis are usually mild and resolve in 7–10 days, but coughing may linger for several weeks after the infection is resolved. These manifestations include the following:

- Productive or nonproductive cough
- Dyspnea
- Wheezing
- Abnormal lung sounds (usually rales or rhonchi)
- Low-grade fever
- Pharyngitis

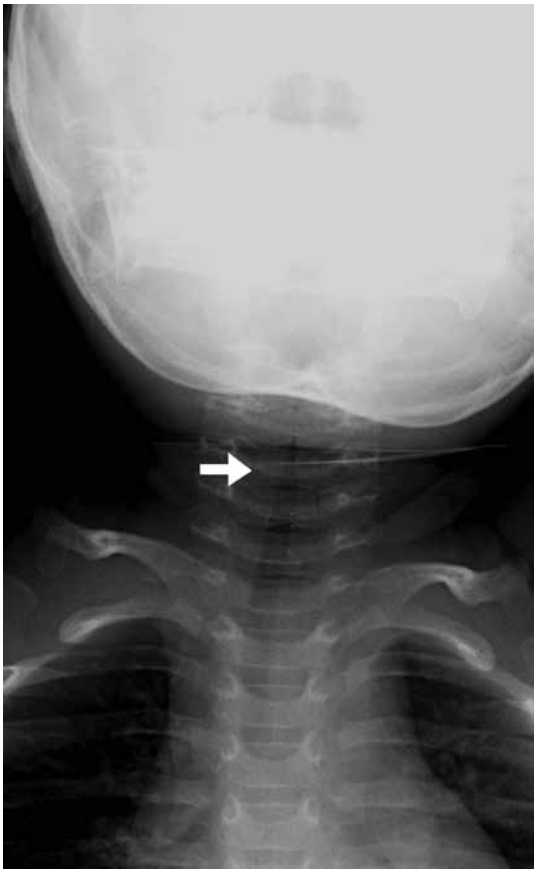

Courtesy of Hugh Dainer, MD, PhD

FIGURE 5-16 Steeple sign.

- Malaise
- Myalgia
- Chest discomfort

Diagnosis of acute bronchitis is usually based on symptoms. Additionally, a CBC and chest X-ray may be performed for differential diagnosis purposes.

Acute bronchitis is generally self-limiting; therefore, treatment is often supportive. Pharmacologic treatment may include antipyretics, analgesics, antihistamines, decongestants, cough suppressants, bronchodilators, and antibiotics (if bacterial). Use cough suppressants cautiously. Coughing mobilizes secretions and prevent pneumonia. Other strategies include increasing fluid intake, avoiding smoke, and humidifying air.

Influenza

Influenza, or flu, is a viral infection that may affect the upper and lower respiratory tract. Three types are distinguished—A, B, and C. These influenza viruses are highly adaptive and constantly mutate, preventing the development of any long-term immune defense.

- **Type A influenza**, which includes several subtypes, is the most common type of influenza virus. This type is usually responsible for the most serious epidemics and global pandemics, such as those that occurred in the United States in 1918, 1957, 1968, and 2009. A subgroup of type A is H1N1, colloquially referred to as the swine flu, which was responsible for a serious pandemic that started in the United States and Mexico in 2009.
- **Type B influenza** outbreaks can also cause regional epidemics, but the disease is generally milder than that caused by type A.
- **Type C influenza** causes sporadic cases and minor, local outbreaks. Type C has never been connected with a large epidemic.

Type A influenza viruses are found in humans and many animals (e.g., ducks, chickens, pigs, and whales). Type B is isolated primarily in humans, whereas Type C is found in humans, pigs, and dogs.

Type A influenza has been found in aquatic birds for years without causing harm to them, but recently this frequently mutating flu virus has shown that it can jump the species barrier from wild birds to domesticated poultry and swine. Pigs can be infected by avian and human flu, especially in areas where human contact is frequent. If a pig becomes infected with the avian and human flu simultaneously, the two types may exchange genes. This "reassorted" flu can sometimes spread from pigs to humans and may cause a more or less severe strain. In 1997, for the first time, scientists found that a form of avian H5N1 flu skipped the pig step and infected humans directly. Alarmed health officials feared a worldwide epidemic (a pandemic). Fortunately, the virus could not pass from person to person, so it did not spark an epidemic. As of August 2012, there had been 608 confirmed cases of this avian flu virus, which resulted in 359 deaths.

Millions of Americans contract the flu each year. The flu season in the United States, when the incidence is the highest, is typically between November and March. The virus is transmitted through the inhalation or contact with respiratory droplets. Children are two to three times more likely than adults to contract the flu, and children frequently spread the virus to others. The 2012–2013 U.S. flu season saw mostly influenza type A (H3N2), but influenza type B and influenza type A (H1N1) were also reported (Centers for Disease Control and Prevention [CDC], 2013b). This flu season was moderately severe, when compared to the mild 2011–2012 season, but did not reach the pandemic levels of the 2009–2010 season. The 2012–2013 flu season saw a higher percentage of outpatient visits for influenza-like illness, higher rates of hospitalization, and more reported deaths attributed to pneumonia and influenza (mortality exceeded the epidemic threshold at times) compared with recent years. Persons at greater risk for having negative outcomes because of the flu include children, elderly, individuals who are immunocompromised, pregnant women, and individuals with preexisting chronic diseases. Often deaths associated with the flu are a result of secondary bacterial pneumonia.

The influenza virus has an incubation period of 1–4 days, with peak transmission risk starting at approximately 1 day before onset of symptoms and lasting 4–7 days afterward in adults. Children can be infectious for more than 10 days, and young children can spread the virus for 6 days before the onset of symptoms occurs. Severely immunocompromised persons can spread the virus for weeks or months. Flu differs from the common cold in that the flu usually has a sudden onset of symptoms (**TABLE 5-2**). Clinical manifestations of the flu include the following signs and symptoms:

- Fever
- Headache

TABLE 5-2 Comparison of Infectious Rhinitis and Influenza Manifestations

Symptoms	Infectious Rhinitis	Influenza
Fever	Rarely	Usual; high (100°F to 102°F, but occasionally higher, especially in young children); lasts 3–4 days
Headache	Rare	Common
Myalgia	Slight	Usual; often severe
Malaise	Sometimes	Usual; can last up to 2–3 weeks
Exhaustion	Never	Usual; at the beginning of the illness
Stuffy nose	Common	Sometimes
Sneezing	Usual	Sometimes
Sore throat	Common	Sometimes
Chest discomfort, cough	Mild to moderate; nonproductive cough	Common; can be severe

Adapted from National Institutes of Allergy and Infectious Disease. (2008). Is it the cold or the flu? Retrieved from http://www.niaid.nih.gov/topics/Flu/Documents/sick.pdf

- Chills
- Dry cough
- Body aches
- Nasal congestion
- Sore throat
- Sweating
- Malaise
- Vomiting and diarrhea (more common in children than in adults)

Typically, fever and body aches last 3–5 days, while cough and fatigue may last for 2 or more weeks.

Diagnostic procedures for influenza consist of a history, physical examinations, rapid flu screen, and flu culture (a nasal culture that tests for the presence of the virus). Treatment is symptomatic and supportive unless a secondary bacterial infection is present. Antiviral medications can reduce the severity and the duration of the symptoms. These antivirals can also be given on a postexposure basis to decrease the likelihood of developing the flu. Other strategies include increasing fluids, rest, antipyretics, and analgesics. Prevention strategies are similar to those used to prevent the common cold (e.g., hand washing and avoiding crowds), but also include vaccinations.

Currently, vaccinations exist for the seasonal flu and H1N1 flu (if warranted). Four types of seasonal flu vaccinations are produced—regular seasonal flu vaccine (the form most commonly administered to people 6 months and older), high-dose vaccine (for people aged 65 and older), intradermal vaccine (for people aged 18–64), and intranasal flu vaccine (produced from a live, weakened virus, unlike the other vaccines that are produced from a killed virus; approved for healthy, nonpregnant people aged 2–49). Prior to each flu season (usually before the previous season is over), the CDC develops a seasonal flu vaccine based on predictions of the likely strain to be encountered. In the United States, the seasonal flu vaccine should be administered each year in October and is recommended to everyone age 6 months and older, especially those in high-risk groups (e.g., persons with a chronic medical condition, age 65 or older, pregnant, living in a community setting, healthcare providers, and household contacts). When outbreaks of other types of the flu occur, as with the H1N1 influenza in 2009, the CDC develops vaccinations specific for those strains.

Vaccine development can be a lengthy process. In many cases, the vaccines are grown in fertilized chicken eggs for approximately 10 months. Therefore, flu vaccines should not be administered to persons with egg allergies. Additionally, children younger than 6 months and people with a history of Guillain-Barré syndrome should not be vaccinated. People with an active febrile illness should wait to be vaccinated until after the illness resolves.

MYTH BUSTERS

A common misconception is that you can get the flu from the flu vaccine. What fuels this myth is that some people may experience very mild flulike symptoms (e.g., low-grade fever, aches, malaise) after receiving the vaccination. These symptoms are not because the individual has a mild case of the flu; it is due to the immune system developing antibodies against the virus. An additional factor fueling this myth is the possibility that people may still have the flu even after receiving the vaccination. This infection is not caused by the flu vaccine; rather, it occurs because the person encountered a strain of the flu that was not covered by the vaccination. Remember, the vaccination is based on predictions. Negative outcomes from the flu vaccine are rare and minimal. So get vaccinated and encourage others to do the same!

Lower Respiratory Tract Infections

Bronchiolitis

Bronchiolitis is a common viral infection of the bronchioles, which is most frequently caused by the respiratory syncytial virus (RSV). The infection most often occurs in children younger than 1 year of age, and incidence increases in the fall and winter months. According to the CDC (2010b), 2.1 million children younger than the age of 5 years seek medical attention annually for RSV, and nearly all children will have an RSV infection by the time they are 2 years old. Bronchiolitis can also be caused by parainfluenza, influenza, adenoviruses, and metapneumoviruses (an emerging paramyxovirus).

When the virus infects the bronchioles, these small airways become inflamed and swollen. As a result of the inflammatory process, mucus collects in these airways. The combination of edema and mucus prevents air flow into the alveoli. Transmission of RSV occurs through contact with or inhalation of infected respiratory droplets. Factors contributing to the development of bronchiolitis include neonatal prematurity, asthma family history, and cigarette smoke exposure.

Clinical manifestations of bronchiolitis vary in severity but include the following signs and symptoms:

- Nasal drainage
- Nasal congestion
- Cough
- Wheezing
- Abnormal lung sounds (e.g., rhonchi or rales)
- Rapid, shallow respirations
- Labored breathing (e.g., chest retractions, nasal flaring, and grunting)
- Dyspnea or tachypnea
- Fever
- Tachycardia
- Malaise

Diagnostic procedures include a history, physical examination, chest X-ray, mucus swab, CBC, and ABGs. Bronchiolitis can progress to atelectasis (collapse of the alveoli) and respiratory failure without aggressive and early treatment; therefore, airway management and respiratory stability are the treatment foci. Hospitalization is often required, and intubation may be necessary if the child decompensates or respiratory failure occurs. Other treatment strategies include oxygen therapy, cool humidity, suctioning secretions, increased fluids (either by mouth or intravenously), keeping the child calm, bronchodilators, and corticosteroids (not recommended for children). No specific treatment for RSV infection is available. Prevention strategies are the same as those previously discussed for other infectious respiratory conditions (e.g., hand washing and avoiding crowds). Palivizumab (Synagis) can be given to at-risk infants during RSV season to prevent and minimize infection. A vaccine for RSV is in development but is not yet commercially available.

Pneumonia

Pneumonia is an inflammatory process caused by numerous infectious agents (e.g., bacteria, viruses, and fungi) and injurious agents or events (e.g., aspiration and smoke). According to the CDC (2010a), pneumonia accounts for 1.1 million hospitalizations and nearly 50,000 deaths annually. *Streptococcus pneumoniae* is responsible for 75% of all cases of pneumonia, and the most common viral causes are influenza, parainfluenza, and RSV. **Viral pneumonia** and **bacterial pneumonia** have some notable differences (**TABLE 5-3**). Viral pneumonia is usually mild and heals without intervention, but it can lead to virulent bacterial pneumonia.

Irritating agents or events can also lead to pneumonia—for example, aspiration of gastric contents, endotracheal intubation, respiratory

TABLE 5-3 Comparison of Viral and Bacterial Pneumonia

	Viral	Bacterial
Cough	Nonproductive	Productive
Fever	Low grade	Higher
WBC	Normal (low)	Elevated
X-ray	Minimal change	Infiltrates
Severity	Less	More
Antibiotics	No	Yes

suctioning, and inhalation of smoke or chemicals. **Aspiration pneumonia** frequently occurs when the gag reflex is impaired because of a brain injury or anesthesia. Aspiration can also occur because of impaired lower esophageal sphincter closure secondary to nasogastric tube placement or disease (e.g., gastroesophageal reflux disease). Additionally, inappropriate tube-feeding placement can lead to tube-feeding formulas entering the lungs rather than the stomach. Gastric contents and tube-feeding formulas irritate the lung tissue, triggering the inflammatory response. The inflammatory response increases mucus production, which can in turn lead to atelectasis and pneumonia. Tube-feeding formulas also contain sugar and protein, creating a superior medium in which bacteria can grow and flourish. Finally, pneumonia can develop from stasis of pulmonary secretions. Activities such as movement, talking, and coughing normally keep pulmonary secretions moving, and adequate hydrations keep secretions thin. When these secretions become thick and stagnate, ciliary action cannot remove the bacteria-laden mucus, leading to pneumonia.

Pneumonia is classified based on the causative agents or events previously discussed and its location in the lung (**TABLE 5-4**). **Lobar pneumonia** is confined to a single lobe and is described based on the affected lobe (e.g., right upper lobe). **Bronchopneumonia** is the most frequent type and is a patchy pneumonia spread throughout several lobes. **Interstitial pneumonia**, or atypical pneumonia, occurs in the areas between the alveoli. Interstitial pneumonia is routinely caused by viruses (e.g., influenza type A and B) or by uncommon bacteria (e.g., *Legionella pneumophilia* and *Mycoplasma pneumoniae*).

Legionnaires' disease is a specific type of pneumonia that is caused by *Legionella*

TABLE 5-4 Types of Pneumonia

	Lobar Pneumonia	Bronchopneumonia	Interstitial Pneumonia
Distribution	All of one or two lobes	Scattered small patches	Scattered small patches
Cause	*Streptococcus pneumoniae*	Multiple bacteria	Influenza virus; *Mycoplasma*
Pathophysiology	Inflammation of the alveolar wall and leakage of cells, fibrin, and fluid into alveoli, causing consolidation	Inflammation and purulent exudates in alveoli, often developing from pooled secretions or irritation	Interstitial inflammation around alveoli Necrosis of bronchial epithelium
Onset	Sudden and acute	Insidious	Variable
Signs	High fever Chills Productive cough of rusty sputum Rales progressing to absent breath sounds in affected lobes	Mild fever Productive cough of yellow-green sputum Dyspnea	Variable fever Nonproductive hacking cough Headache Myalgia

pneumophilia. These bacteria thrive in warm, moist environments (e.g., air-conditioning systems, standing freshwater, respiratory therapy equipment, and whirlpools). Legionnaires' disease is not contagious. Instead, most people acquire this type of pneumonia from inhaling the bacteria as they are spread by an air-conditioning system or spa. Persons with a weakened immune system are at highest risk for developing Legionnaires' disease. Although most people with this type of pneumonia recover without incident, the disease can be fatal if untreated. Symptoms are similar to other types of pneumonia and usually appear 10–14 days post exposure. Additional symptoms may include nausea, vomiting, and diarrhea. In addition to the usual pneumonia diagnostic procedures, a urine test can be performed to identify the presence of *Legionella* antigens. Treatment of Legionnaires' disease follows the usual pneumonia treatment protocol.

Mycoplasma pneumoniae is a common type of pneumonia that usually affects people younger than 40 years of age. People who live or work in crowded places such as schools, homeless, and prisons are at higher risk of developing this type of pneumonia. *Mycoplasma* infection is usually mild and responds well to antibiotics, but it can be serious. Skin rash, arthralgia, and hemolysis may also be present.

Severe acute respiratory syndrome (SARS) is a rapidly spreading respiratory illness that presents similarly to atypical pneumonia. First identified in China, its prevalence rates remain higher in Asian countries. SARS is caused by a coronavirus, SARS-CoV. Transmission occurs through inhalation of respiratory droplets or close contact, although oral–fecal contact may also be a mechanism of transmission. SARS has high mortality and morbidity rates. The incubation period for this disease is 2–7 days. The first stage presents as a flulike syndrome (e.g., fever, chills, headache, myalgia, anorexia, and diarrhea) that lasts 3–7 days. Several days later, a dry cough and dyspnea develop as the lungs become damaged and the patient moves into the second stage of the disease. Interstitial congestion and hypoxia progress rapidly. Additionally, liver damage can occur. If the patient continues to the third stage, severe and sometimes fatal respiratory distress can develop. Diagnostic procedures for SARS consist of a history, physical examination, and chest X-ray. Treatment focuses on maintaining oxygenation and respiratory status. Strategies include oxygen therapy, bronchodilators, and antiviral drugs. Endotracheal intubation with mechanical ventilation support may be required as hypoxia worsens.

An emerging illness in the same coronavirus family is **Middle East respiratory syndrome** (MERS-CoV). Since its discovery in April 2012 through July 2013, 77 cases and 42 deaths from this disease had been reported. The virus is currently isolated to four countries in the Arabian Peninsula. It seems to spread through close contact, but the CDC is still working on better understanding the virus (CDC, 2013c).

Pneumonia is also classified according to where it is acquired. **Nosocomial pneumonia** refers to pneumonia that develops more than 48 hours after a hospital admission. Ventilator-associated pneumonia (VAP) is an example of nosocomial pneumonia. With this disease, the endotracheal tube breaks the body's first line of defense and provides a portal of entry for bacteria. In contrast, **community-acquired pneumonia** is acquired outside the hospital or healthcare setting.

Most healthy people do not develop pneumonia from fungal exposure. Many of these illnesses occur instead as opportunistic infections, which can be fatal in immunocompromised individuals (e.g., children and persons with AIDS or cancer). ***Pneumocystis jiroveci* pneumonia**, formerly known as *Pneumocystis carinii* pneumonia, is a specific type of pneumonia that is caused by yeastlike fungus. Its diagnosis is accomplished through identification of the fungus through a sputum culture. Aggressive and early treatment will improve outcomes in these vulnerable patients. Other fungal-related pneumonias include histoplasmosis, coccidiomycosis, and cryptococcal pneumonia. In addition to previously discussed risk factors, persons at risk for developing pneumonia and having serious complications include children, the elderly, immunocompromised individuals, those with existing chronic disease conditions, smokers, and alcoholics. Otherwise-healthy patients usually recover completely from pneumonia when treated properly. Those high-risk persons are more likely to develop complications including septicemia, pulmonary edema, lung abscess, pleural effusion, and acute respiratory distress syndrome.

Clinical manifestations of pneumonia include the following signs and symptoms:

- Productive or nonproductive cough
- Fatigue
- Pleuritic pain
- Dyspnea

- Fever
- Chills
- Abnormal lung sounds (e.g., crackles or rales)
- Pleural rub
- Tachypnea
- Mental status changes (especially in the elderly)
- Leukocytosis

Early diagnosis and treatment of pneumonia are paramount to have positive outcomes. Diagnostic procedures may include a history, physical examination, chest X-ray, sputum cultures, CBC, ABGs, and bronchoscopy. Endotracheal intubation may be necessary to provide ventilation support and maintain oxygenation. Additional treatment strategies include antibiotics (if bacterial infection is present), bronchodilators, corticosteroids, antipyretics, analgesics, humidified oxygen therapy, chest physiotherapy, increased fluids (either by mouth or intravenously), and rest. If aspiration is the cause of the pneumonia, additional treatment includes eliminating the causes and not giving the patient anything by mouth until swallowing studies can be performed. Pneumonia prevention strategies include hand washing, avoiding crowds, vaccinations (e.g., for pneumococcus, influenza, and Hib), mobilizing secretions (e.g., turning, coughing, deep breathing), and smoking cessation.

Tuberculosis

Tuberculosis (TB), an ancient disease, is one of the world's deadliest conditions. Although on the decline, TB remains a major cause of illness, with one-third of the world's population being infected. According to the World Health Organization (WHO), TB was responsible for approximately 1.4 million deaths worldwide in 2011, and it is the leading cause of death for persons infected with HIV (CDC, 2012). In the United States, TB rates are higher among Asians, Native Hawaiians, and other Pacific Islanders.

TB is caused by *Mycobacterium tuberculosis*, a slow-growing aerobic (requires oxygen) bacillus that is somewhat resistant to the body's immune efforts. Person-to-person transmission occurs through the inhalation of tiny infected aerosol droplets (FIGURE 5-17). Only people with active TB can spread the disease to others. The bacillus is capable of surviving in dried sputum for weeks. Ultraviolet light, heat, alcohol, glutaraldehyde, and formaldehyde destroy the bacillus. Many people contract TB but do not develop the disease because of an intact, healthy immune system or early treatment. A growing number of multidrug-resistant TB strains are emerging, however, increasing treatment concerns and prevalence rates. Fifteen percent of persons infected with TB in the United States have a multidrug-resistant strain.

Although TB most frequently involves the lungs, it can also affect other organs and tissues (e.g., liver, brain, and bone marrow). TB is often considered an opportunistic infection because it is more likely to become active in someone with a weakened immune system. Therefore, at-risk persons include those with immune deficiency (e.g., AIDS and cancer), malnutrition, diabetes mellitus, and alcoholism. Poverty, overcrowding, homelessness, and drug abuse also increase the risk for acquiring TB.

There are two stages of TB pathogenesis—primary and secondary infection. **Primary TB infection** occurs when the bacillus first enters the body. In this phase, macrophages engulf the microbe, causing a local inflammatory response. Some bacilli travel to the lymph nodes, activating the type IV hypersensitivity reaction (see the *Body Defenses* chapter). Lymphocytes and macrophages congregate to form a granuloma (an epithelial nodule). The granuloma contains some live bacilli, forming a tubercle. Caseous necrosis, a cottage cheese–like material, develops in the center of the tubercle (see the *Cellular Function* chapter). An intact immune system can resist this development, so the lesions— referred to as Ghon complexes (FIGURE 5-18)—remain small, become walled off by fibrous tissue, and calcify. The bacilli can remain dormant and viable in the tubercle for years as long as the immune system is intact. In this phase, the individual has been infected by the bacilli and remains asymptomatic. When the primary infection can no longer be controlled, the infection progresses to the **secondary**, or **active**, **infection** phase. During this phase, TB can spread throughout the lungs and to other organs.

Clinical manifestations begin to appear in the secondary infection phase:

- Productive cough
- Hemoptysis (coughing up blood or bloody sputum)
- Night sweats
- Fever
- Chills
- Fatigue
- Unexplained weight loss

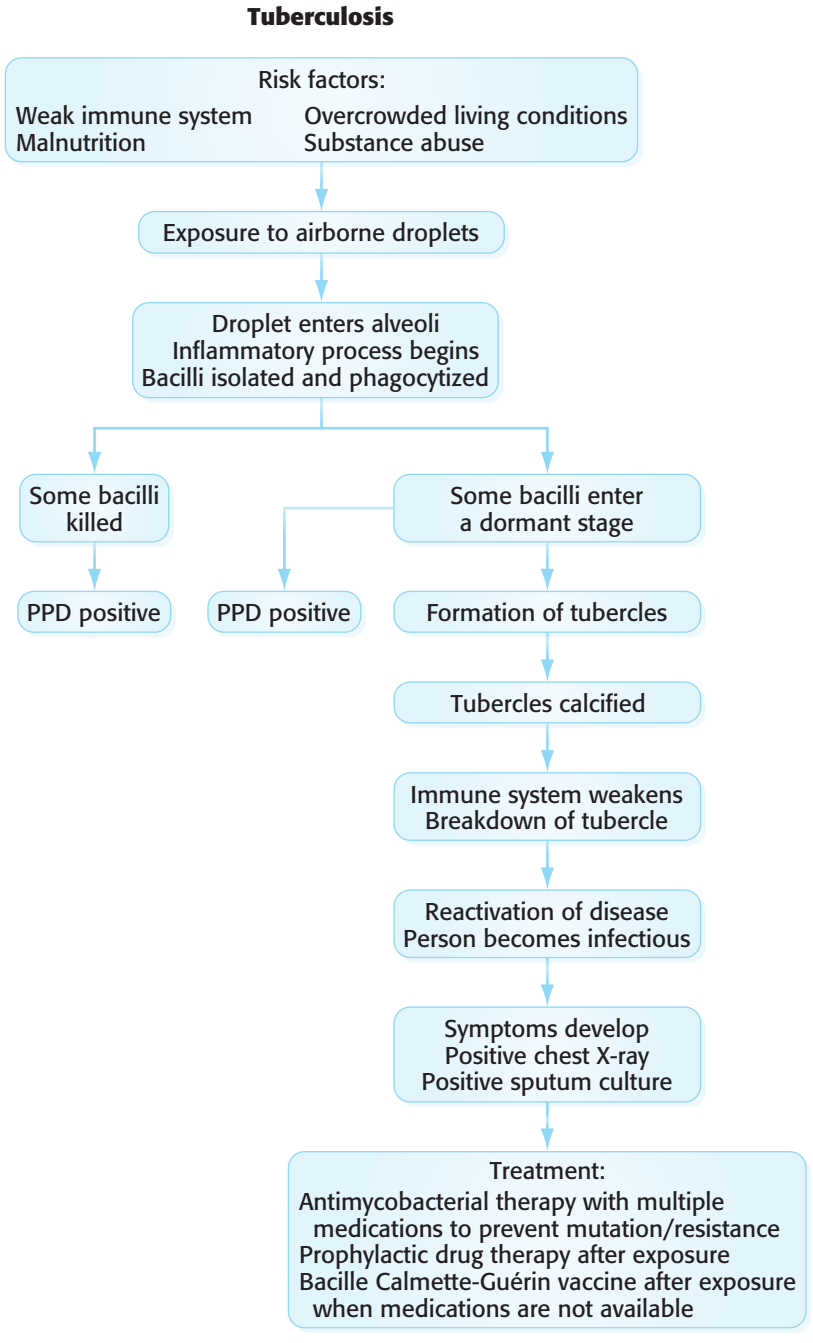


FIGURE 5-17 Tuberculosis.

Madara, M., & Pomarico-Denino, V. (2008). *Quick look nursing: Pathophysiology* (2nd ed.). Sudbury, MA: Jones & Bartlett.

LEARNING POINTS

TB skin testing is only useful as a screening tool to identify new TB exposure cases. Once a person's immune system has developed antibodies against TB, the person tests positive. This immunity reaction happens after the first exposure and vaccination administration. A person can be treated for TB, and he or she will continue to test positive because the antibodies are still present. Chest X-rays and sputum cultures are better diagnostic procedures once someone has tested positive. So remember . . . once positive, always positive!

- Anorexia
- Miscellaneous symptoms depending on other organ involvement

Diagnostic procedures for TB are multifaceted, beginning with a TB skin test (Mantoux test). For the TB skin test, a small amount of a purified protein derivative tuberculin is injected just below the dermis. If the person has been infected by the bacilli, a local reaction (e.g., redness and induration) will occur (FIGURE 5-19). Persons will test positive once the bacilli trigger

Courtesy of Benjamin M. Marais

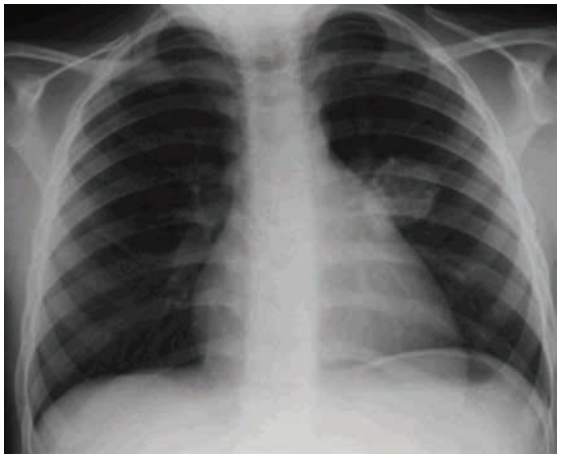


FIGURE 5-18 Ghon complexes.

© Phototake, Inc./Alamy

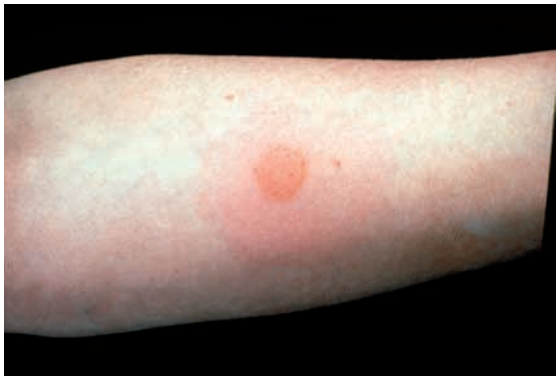


FIGURE 5-19 Positive TB skin test.

the inflammatory response (Figure 5-18). A history of bacillus Calmette-Guérin (BCG) vaccination will produce a false-positive reaction. Additionally, previously treated TB will generate a false-positive reaction. Conversely, persons with immature (e.g., children) or compromised (e.g., AIDS or cancer) immune systems may not generate enough of a response to test positive. Because of the uncertainty that the TB skin test creates, chest X-rays and sputum cultures (most definitive) are used after a positive TB skin test is noted (either to confirm an original case or to assess reinfection). A computerized tomography (CT) scan can also be used to visualize TB lesions because this imaging modality is more sensitive than an X-ray. Nucleic acid amplification may be performed on the sputum to detect the presence of resistant strains.

TB is often successfully treated in the home setting; however, treatment requires diligence to eradicate the disease. Treatment requires an average of 6–9 months of antimicrobial therapy. Combination therapy (consisting of two or more drugs) is recommended to prevent the emergence of resistant strains. The slow-growing bacilli have a high mutation rate, with those mutations often appearing when the pathogen is exposed to monotherapy. Because TB is a public health risk, antituberculin medications are provided free of charge by the U.S. Public Health Service. In some states, therapy noncompliance is unlawful, and imprisonment may be used to ensure adherence when other measures fail (e.g., direct observed therapy). Compliance is a common problem in treating TB because of the length of therapy and the medication side effects (e.g., nausea, paresthesias, and discolored bodily secretions). Patient education, including an emphasis on taking the entire regimen of drugs as ordered, is crucial to maximize therapy success and prevent resistance. Strategies to prevent the transmission of TB include respiratory precautions (e.g., TB-approved masks, covering one's mouth when coughing, and disposing of tissues), adequate ventilation (if the patient is at home), placing the patient in a negative-pressure isolation room (if he or she is hospitalized), and the bacillus Calmette-Guérin vaccination (primarily used in children and in developing countries).

Alterations in Ventilation

Asthma

Asthma is a chronic pulmonary disease that produces intermittent, reversible airway obstruction. It is characterized by acute airway inflammation, bronchoconstriction, bronchospasm, bronchiole edema, and mucus production (FIGURE 5-20). Asthma is one of the most common chronic illness in children in the United States. Diagnosis, hospitalizations, and death rates associated with asthma all increased from 1996 to 2006 (CDC, 2009). These rate increases may be a result of surging urbanization and pollution. Another theory, referred to as the hygiene hypothesis, is that the modern Western lifestyle's focus on hygiene and sanitation limits early childhood allergen and infection exposure,

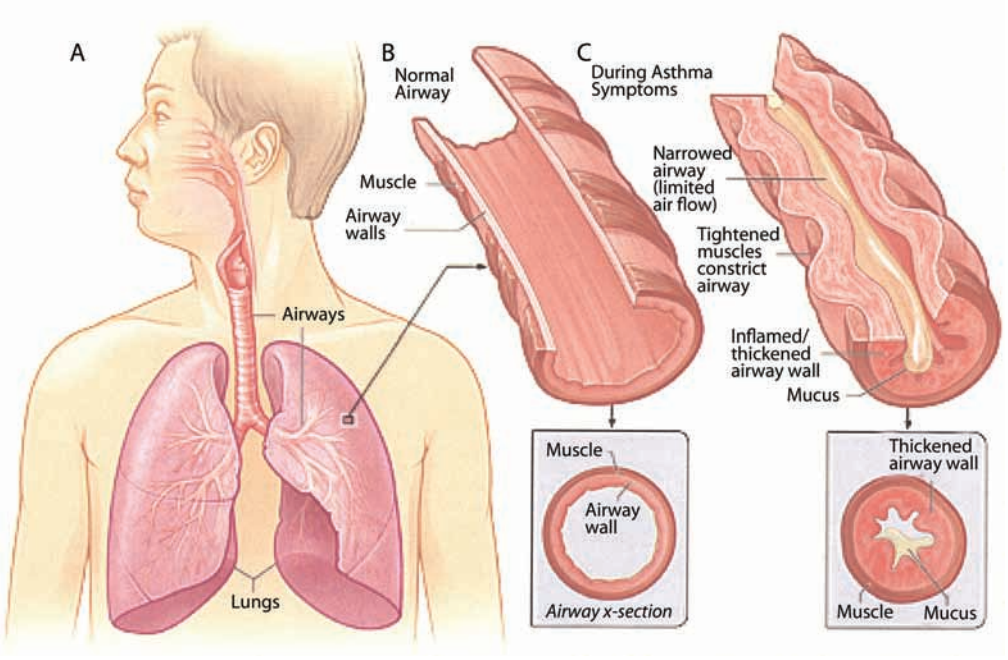


FIGURE 5-20 Asthma. (a) Location of the lungs and airways in the body. (b) Cross section of a normal airway. (c) Cross section of an airway during asthma symptoms.

National Heart, Lung, and Blood Institute (www.nhlbi.nih.gov)

thereby rendering individuals more vulnerable when they do finally encounter allergens and infectious agents. Women are more likely to have asthma than men, but in children, boys are more likely to have asthma than girls. Mixed-race and African American adults and children are more likely to have asthma than Caucasian adults and children. Other risk factors include lower socioeconomic status, obesity, smoke exposure, and family history.

Asthma is usually classified according to cause (extrinsic, intrinsic, nocturnal, exercise-induced, occupational, or drug-induced) and by severity (mild intermittent, mild persistent, moderate persistent, and severe persistent) (**TABLE 5-5**). **Extrinsic asthma** is a result of increased IgE synthesis and airway inflammation, which leads to mast cell destruction and inflammatory mediator release. Extrinsic triggers include allergens such as food, pollen, dust, and medications. The release of the inflammatory mediators in response to these triggers causes bronchoconstriction, increased capillary permeability, and mucus production. Extrinsic asthma generally presents in childhood or adolescence. **Intrinsic asthma** is not an allergic reaction and usually presents after age 35 years. Intrinsic triggers include upper respiratory infections, air pollution, emotional stress, smoke, exercise, and cold exposure.

Nocturnal asthma usually occurs between 3:00 and 7:00 a.m. and is thought to be related to circadian rhythms. At night, cortisol and epinephrine levels decrease, while histamine levels increase. Changes in these naturally occurring substances lead to bronchoconstriction.

Exercise-induced asthma is common and usually occurs 10–15 minutes after physical activity ends. Symptoms can linger for an hour with this type of asthma. The airways can become cool and dry during exercise, and asthmatic symptoms may be a compensatory mechanism to warm and moisten the airways. Following each episode of exercise-induced asthma, a refractory (symptom-free) period begins within 30 minutes and can last 90 minutes. During this time, little or no bronchospasm can be induced even if the person is rechallenged with vigorous exercise. Athletes often take advantage of this fact by warming up vigorously to induce a refractory period prior to competition.

TABLE 5-5 Classification of Asthma Severity

Step/Classification*	Daytime Symptoms	Nighttime Symptoms	PEF or FEV1†	PEF Variability
Step 1: Mild intermittent	≤ 2/wk	≤ 2/wk	≥ 80%	< 20%
Step 2: Mild persistent	> 2/wk, but < daily	> 2 nights/mo	> 80%	20–30%
Step 3: Moderate persistent	Daily	> 1 night/wk	60–80%	> 30%
Step 4: Severe persistent	Continual	Frequent	≤ 60%	> 30%

*Classification is based on symptoms and lung function before treatment. Patients should be assigned to the most severe step in which any feature occurs.

†Percentage of predicted function.

PEF = peak expiratory flow (rate); FEV1 = forced expiratory volume in 1 second.

National Heart, Lung, and Blood Institute (www.nhlbi.nih.gov).

Occupational asthma is caused by a reaction to substances encountered at work (e.g., plastic or formaldehyde). Symptoms develop over time, worsening with each exposure and improving when away from work (e.g., on weekends or during vacations).

Drug-induced asthma is frequently caused by aspirin and can be fatal. Reactions can be delayed up to 12 hours after drug ingestion. Aspirin and other drugs (e.g., nonsteroidal anti-inflammatory drugs) prevent the conversion of prostaglandins, which stimulate leukotriene release—a powerful bronchoconstrictor.

Regardless of the classification system used, asthma attacks are the body's response to bronchial inflammation. Stage 1 of an acute asthma attack is primarily related to bronchospasm, and is usually signaled by coughing. Peaking within 15 to 30 minutes, the inflammatory mediators responsible for this stage include leukotrienes, histamine, and some interleukins. Stage 2 of an asthma attack peaks within 6 hours of symptom onset. This stage is a result of airway edema and mucus production. The alveolar hyperinflation causes air trapping. Bronchospasm, smooth muscle contraction, inflammation, and mucus production combine to narrow the airways.

Clinical manifestations of asthma include the following:

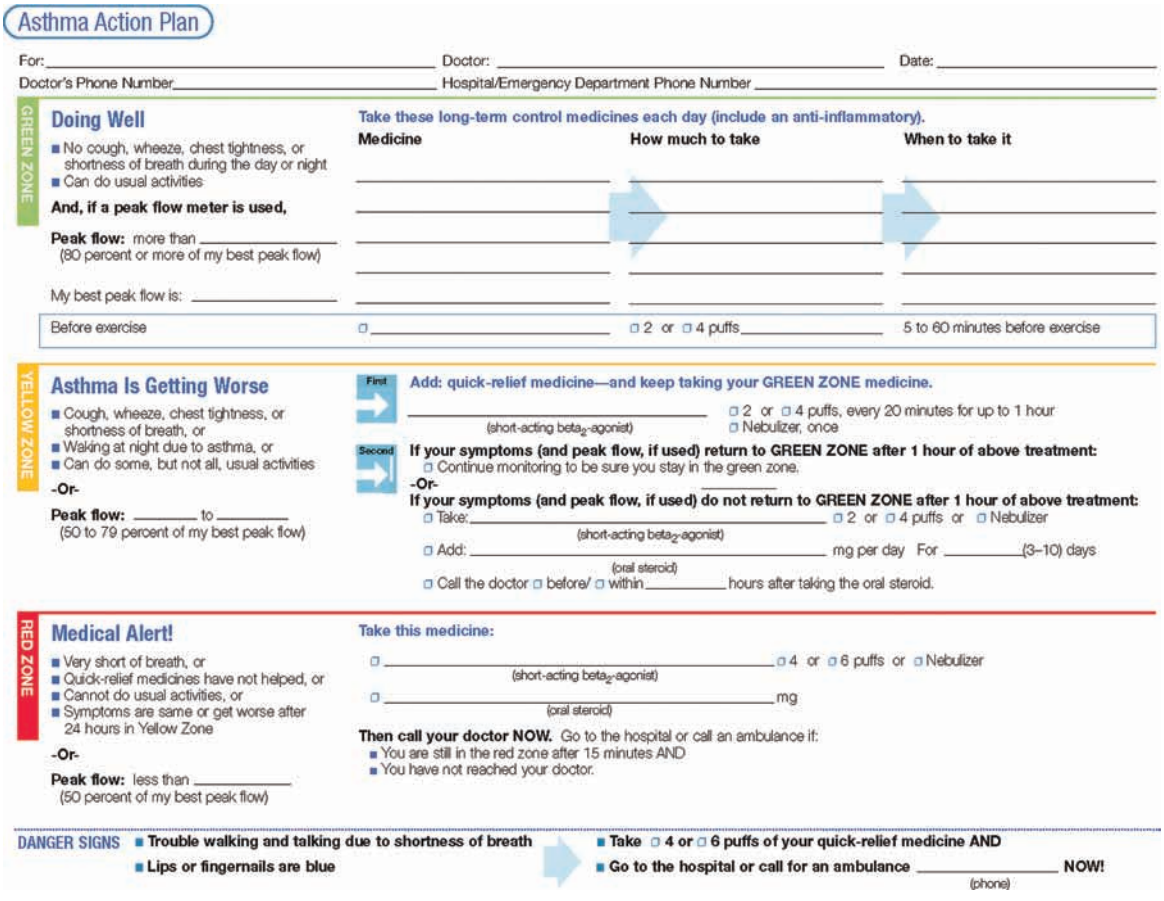
- Wheezing
- Shortness of breath
- Dyspnea
- Chest tightness
- Cough
- Tachypnea
- Anxiety

Status asthmaticus is a life-threatening, prolonged asthma attack that does not respond to usual treatment. Maintaining a patent airway is critical in such cases, and endotracheal intubation with ventilation support may be necessary. In addition, acid–base imbalances—specifically respiratory alkalosis (from expelling too much carbon dioxide because of tachypnea)—can develop. Treatment of these conditions will be crucial to improve outcomes.

Diagnostic procedures can be used to identify those persons with asthma as well as to track progression of the disease. These diagnostic procedures include a history, physical examination, pulmonary function tests (Figure 5-11), chest X-ray, ABGs, CBC, challenge testing, and allergen testing.

Asthma cannot be cured, but its symptoms can be controlled. Unless treated promptly, asthma attacks can lead to impaired gas exchange and death. Left untreated, long-term asthma can result in bronchial damage and scarring. The goals of treatment are to minimize the occurrence and severity of asthma attacks. Pharmacologic treatment includes inhaled and systemic corticosteroids, bronchodilators, beta agonists, nebulizer treatments, leukotriene mediators, mast cell stabilizers, and anticholinergics. Additional strategies include the following measures:

- Develop an asthma plan (FIGURE 5-21) and teach it to all caregivers
- Avoid triggers



Asthma Action Plan

For: _____ Doctor: _____ Date: _____

Doctor's Phone Number _____ Hospital/Emergency Department Phone Number _____

GREEN ZONE

Doing Well

- No cough, wheeze, chest tightness, or shortness of breath during the day or night
- Can do usual activities

And, if a peak flow meter is used,

Peak flow: more than _____ (80 percent or more of my best peak flow)

My best peak flow is: _____

Take these long-term control medicines each day (include an anti-inflammatory).

Medicine	How much to take	When to take it
Before exercise	☐ _____	☐ 2 or ☐ 4 puffs _____ — 5 to 60 minutes before exercise

YELLOW ZONE

Asthma Is Getting Worse

- Cough, wheeze, chest tightness, or shortness of breath, or
- Waking at night due to asthma, or
- Can do some, but not all, usual activities

-Or-

Peak flow: _____ to _____ (50 to 79 percent of my best peak flow)

First: **Add: quick-relief medicine—and keep taking your GREEN ZONE medicine.**

_____ (short-acting beta$_2$-agonist) ☐ 2 or ☐ 4 puffs, every 20 minutes for up to 1 hour ☐ Nebulizer, once

Second: **If your symptoms (and peak flow, if used) return to GREEN ZONE after 1 hour of above treatment:**

☐ Continue monitoring to be sure you stay in the green zone.

-Or-

If your symptoms (and peak flow, if used) do not return to GREEN ZONE after 1 hour of above treatment:

☐ Take: _____ (short-acting beta$_2$-agonist) ☐ 2 or ☐ 4 puffs or ☐ Nebulizer

☐ Add: _____ (oral steroid) mg per day For _____ (3–10) days

☐ Call the doctor ☐ before/ ☐ within _____ hours after taking the oral steroid.

RED ZONE

Medical Alert!

- Very short of breath, or
- Quick-relief medicines have not helped, or
- Cannot do usual activities, or
- Symptoms are same or get worse after 24 hours in Yellow Zone

-Or-

Peak flow: less than _____ (50 percent of my best peak flow)

Take this medicine:

☐ _____ (short-acting beta$_2$-agonist) ☐ 4 or ☐ 6 puffs or ☐ Nebulizer

☐ _____ (oral steroid) mg

Then call your doctor NOW. Go to the hospital or call an ambulance if:

- You are still in the red zone after 15 minutes AND
- You have not reached your doctor.

DANGER SIGNS

- **Trouble walking and talking due to shortness of breath**
- **Lips or fingernails are blue**

- **Take ☐ 4 or ☐ 6 puffs of your quick-relief medicine AND**
- **Go to the hospital or call for an ambulance _____ (phone) NOW!**

FIGURE 5-21 Example of asthma action plan.

National Heart, Lung, and Blood Institute (www.nhlbi.nih.gov).

- Keep the environment clean
- Limit environmental fabrics
- Filter indoor air
- Maintain a healthy immune system (e.g., exercise, get adequate nutrition)

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) describes a group of chronic respiratory disorders characterized by irreversible, progressive tissue degeneration and airway obstruction. These debilitating conditions can impair an individual's ability to work and function independently. Severe hypoxia and hypercapnia can lead to respiratory failure. The chronic hypercapnia shifts the normal breathing drive from the need to expel excess carbon dioxide to the need to raise oxygen levels (Figure 5-12). Additionally, COPD can lead to cor pulmonale, right-sided heart failure due to lung disease (see the *Cardiovascular Function* chapter).

The most significant contributing factor to developing COPD is cigarette smoking. Other contributing factors include the inhalation of pollution and chemical irritants. Groups at higher risk of developing COPD also include Caucasians, women, individuals of lower socioeconomic status, and persons with a history of asthma.

Prevalence rates are likely underestimated because COPD is often asymptomatic in its early stages or is masked by smoking symptoms. According to the CDC (2013b), COPD was the third leading cause of death in the United States in 2011. Twelve million Americans are currently diagnosed with COPD, and an additional 12 million Americans may have this disease and not know it. Symptoms usually present around 60 years of age. A rare familial type of COPD (emphysema only), alpha-1 antitrypsin deficiency, presents much earlier—in the 30s or 40s.

COPD is often one of or a mixture of two diseases—chronic bronchitis and emphysema (FIGURE 5-22). These two diseases are discussed next.

Chronic Bronchitis

Chronic bronchitis is an obstructive respiratory disorder characterized by inflammation of the bronchi, a productive cough, and excessive mucus production. This disorder differs from

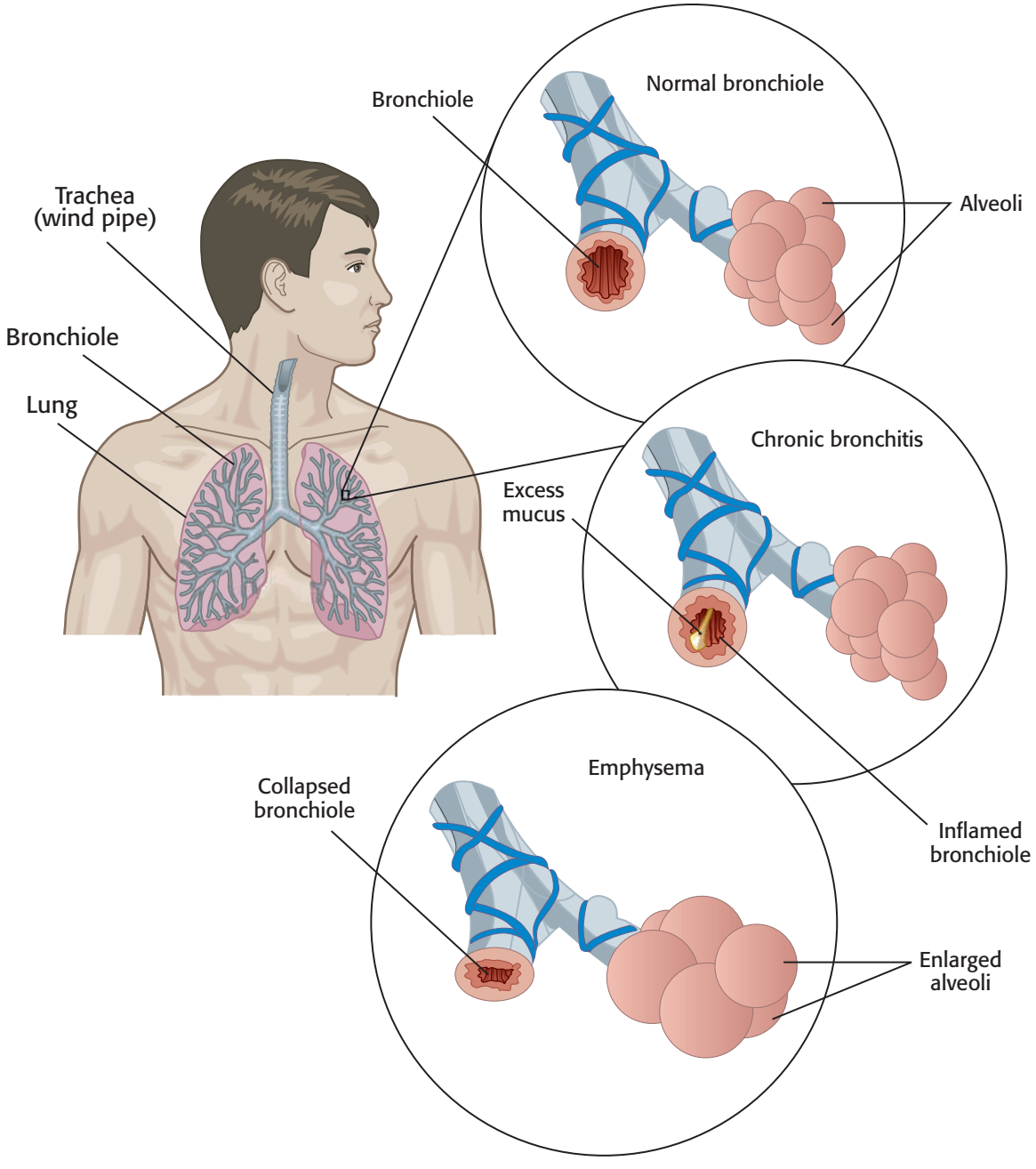


FIGURE 5-22 Chronic obstructive pulmonary disease (COPD) is often one disease or a mixture of two diseases—chronic bronchitis and emphysema.

acute bronchitis in that the chronic type is not necessarily caused by an infection and symptoms persist longer. As previously mentioned, cigarette smoking is the greatest contributing factor for chronic bronchitis. The inflammatory response results in mucous gland hyperplasia, edema, excessive mucus production, bronchoconstriction, and cough in defense against inhaled irritants. Airway resistance affects inspiratory and expiratory air flow. Impaired pulmonary defenses (e.g., cilia damage and decreased phagocytic activity) result in frequent respiratory infections and, in some cases, respiratory failure.

Airway resistance results in hypoventilation, hypoxemia, cyanosis, hypercapnia, polycythemia, clubbing of fingers, and dyspnea at rest. Additional clinical manifestations include the following signs and symptoms:

- Abnormal lung sounds (e.g., wheezing and rhonchi)
- Edema
- Weight gain

LEARNING POINTS

Patients with chronic bronchitis are unable to increase ventilatory effort to maintain adequate gas exchange; therefore, they eventually develop cyanosis. This cyanosis coupled with the edema that develops gives these patients the nickname "**blue bloaters**." Additionally, chronic bronchitis pathogenesis can be remembered by these three S's—Sputum, Swelling, and Spasm.

- Malaise
- Chest pain
- Fever

Diagnostic procedures for chronic bronchitis consist of a history (persistent, productive cough for at least 3 months in a year for 2 consecutive years), physical examination, chest X-ray, pulmonary function tests (Figure 5-11), ABGs, and CBC. The goal of treatment is to maintain airway patency. Treatment strategies include oxygen therapy (in limited amounts, because too much will knock out the newly oxygen-centered drive for breathing), bronchodilators, corticosteroids, antibiotics (if bacterial infection is present), postural drainage, chest physiotherapy, and increased hydration.

Emphysema

Emphysema is an obstructive respiratory disorder that results in the destruction of the alveolar walls, leading to large, permanently inflated alveoli. Lung tissue normally remodels during periods of growth and repair related to infections and inflammation. Enzymes are involved in this process to prevent excessive tissue damage. Enzyme deficiency, however, may result from genetic predisposition (less than 2% of cases) and smoking. Smoking initiates inflammation, causing changes in these enzyme levels and leading to structural changes. Emphysema gradually turns the alveoli into large, irregular pockets with gaping holes, which in turn limits the amount of oxygen entering the bloodstream. The elastic fibers and surfactant that normally keep the alveoli open are slowly destroyed, so the alveoli collapse during expiration, trapping air in the lungs. The loss of elastic recoil and hyperinflation of the alveoli narrow the terminal bronchioles, but inspiration is not affected.

Coughing is usually not a symptom. Instead, emphysema is characterized by the following clinical manifestations:

- Dyspnea upon exertion
- Diminished breath sounds
- Wheezing
- Chest tightness
- Tachypnea
- Hypoxia
- Hypercapnia
- Activity intolerance
- Anorexia
- Malaise

Diagnosis and progress monitoring are accomplished through the same procedures thatare used for chronic bronchitis. Treatment strategies include those identified for chronic bronchitis, plus pursed-lip breathing and lung reduction surgery. Pursed-lip breathing increases expiratory resistance and produces airway backpressure, preventing collapse of the alveoli.

Cystic Fibrosis

Cystic fibrosis is a common inherited respiratory disorder (about 1,000 patients diagnosed per year in the United States) that presents at birth (Cystic Fibrosis Foundation, n.d.). Caucasians are more likely to develop cystic fibrosis than other ethnic groups, followed by Latinos and American Indians. This life-threatening condition causes severe lung damage and nutrition deficits. Cystic fibrosis changes the cells that produce mucus, sweat, saliva, and digestive secretions. As a result, these normally thin secretions become thick and tenacious. Instead of lubricating the respiratory tract, the secretions occlude airways, ducts, and passageways. The genetic defect that leads to cystic fibrosis has been isolated to chromosome 7, and transmission follows an autosomal recessive pattern (see the *Cellular Function* chapter). More than 10 million Americans are carriers of this faulty gene, and many do not know they are carriers. The genetic deficit is related to a protein involved in sodium, chloride, and water cellular transport. The lungs and pancreas are primarily affected, but other organs can also be involved (e.g., liver, intestines, sinuses, and reproductive organs).

Atelectasis develops as airways are obstructed, leading to permanent damage (FIGURE 5-23). Mucus stagnates, becoming a prime medium for bacterial growth. Infections are recurrent and contribute to the progressive lung

LEARNING POINTS

Patients with emphysema often hyperventilate, creating a pink appearance to their skin. This panting-like breathing pattern coupled with the pink skin has earned emphysema patients the nickname "**pink puffers**." Additionally, the pathogenesis of emphysema can described as "lung dry rot."

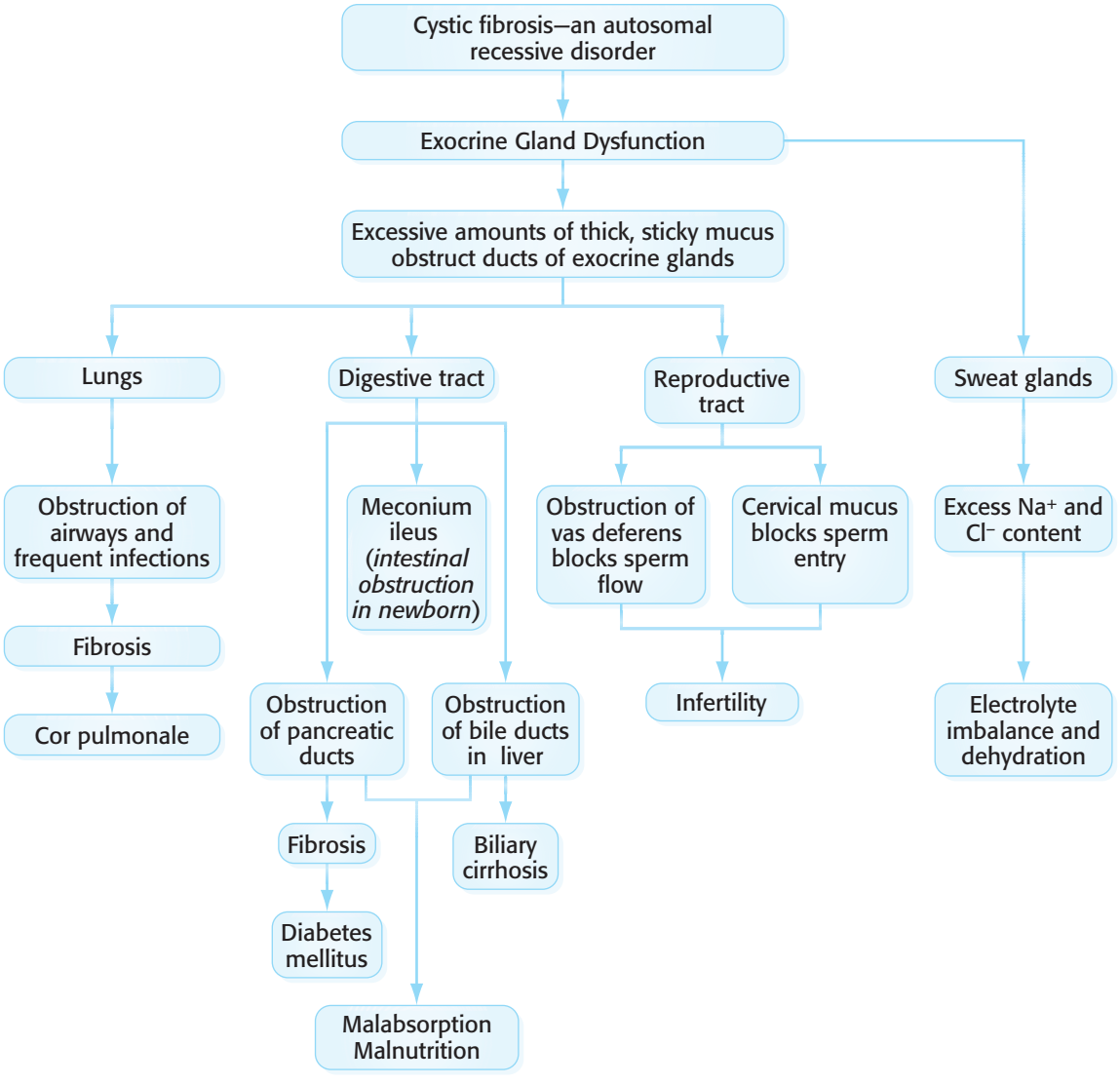


FIGURE 5-23 Cystic fibrosis.

destruction. Bronchiectasis and emphysema-like changes are common as fibrosis and obstructions advance. Respiratory failure is the most common cause of death in people who have cystic fibrosis. Ultimately, cor pulmonale (right-sided heart failure) or respiratory failure occurs. In the digestive tract, the mucus blocks the intestines, producing a meconium ileus in the newborn. It also blocks pancreas ducts, leading to a pancreatic enzyme excretion deficit. Without these digestive enzymes, malabsorption and malnutrition develop. The trapped digestive enzymes damage pancreatic tissue, contributing to the development of diabetes mellitus and osteoporosis. Blocked bile ducts add to the malabsorption issues and increase risk for developing cirrhosis. Salivary glands are only mildly affected by blockages. Sweat glands produce sweat high in sodium chloride, which can cause electrolyte imbalances in times of excessive loss (e.g., during exercise or hot weather). Obstructions in the reproductive system can lead to sterility and infertility.

Clinical manifestations of cystic fibrosis may appear at birth and progressively worsen throughout the life span. Lung function often starts declining in early childhood. These manifestations, which vary in severity, include the following signs and symptoms:

- Meconium ileus
- Salty skin (parents may notice that their baby's skin tastes salty when they kiss the child)
- Steatorrhea (fatty, foul-smelling stools)
- Fat-soluble vitamin deficiency (vitamins A, D, E, and K)

- Voracious appetite
- Chronic cough with tenacious sputum (may include hemoptysis)
- Frequent respiratory infections
- Hypoxia
- Audible rhonchi and wheezing
- Dyspnea
- Fatigue
- Activity intolerance
- Digital clubbing
- Delayed growth and development
- Complication development (e.g., chronic bronchitis, failure to thrive, cardiomegaly, diabetes mellitus, pancreatitis, rectal prolapse, liver disease, cholelithiasis, osteoporosis, hyponatremia, metabolic alkalosis, and infertility)

Diagnosis of cystic fibrosis can be accomplished prenatally when family history warrants testing. Sweat analysis can be conducted at about 2–3 weeks of age to detect electrolyte abnormalities. Some states include cystic fibrosis testing as a part of their newborn screening. The Delta F508 test can identify the chromosome 7 mutation, and it is often used if a false-negative sweat test is suspected or to identify carrier status of siblings. In addition, stool can be evaluated for the presence of pancreatic content. Other tests that assess lung function include chest X-rays, pulmonary function tests, and ABGs.

Cystic fibrosis treatment requires diligent family involvement and an interdisciplinary approach because of the progressive, multisystem nature of the disease. Improved treatment regimens have improved the life expectancy for patients who have cystic fibrosis, with some people living into their 40s or 50s. Treatment often requires a multidisciplinary approach involving cystic fibrosis specialists, respiratory therapists, dieticians, and psychological counselors. Strategies include the following measures:

- Pancreatic enzyme replacement
- Bile salt replacement
- A well-balanced, high-protein, low-fat, high-sodium diet
- Fat-soluble vitamin replacement
- Increased fluid intake
- Intensive chest physiotherapy (includes chest clapping or percussion, which can be done by hand or with a mechanical percussor)
- Postural drainage
- Coughing exercises
- Humidified air
- Bronchodilators
- Regular, moderate exercise
- Early, aggressive treatment of infections with antibiotics
- Oxygen therapy
- Avoidance of respiratory irritants (e.g., smoke, pollution, dust, and allergens)
- Heart–lung transplant

Lung Cancer

Lung cancer is the second most often diagnosed cancer in men and women and the leading cause of cancer in the United States (U.S. Cancer Statistics Working Group, 2012). After increasing for decades, lung cancer incidence and mortality rates are now decreasing in parallel with decreases in rates of cigarette smoking in this country. Frequently, other cancers—such as breast and liver, to name a few—metastasize (spread) to the lung tissue. Smoking contributes to the majority (80–90%) of lung cancer cases. The more than 4,000 chemicals in cigarette smoke include carcinogens and chemicals that paralyze cilia. The risk for developing lung cancer is directly related to the length of time a person smokes and the number of cigarettes smoked. Secondhand smoke can also be a significant contributing factor, and in fact, some research has indicated that it may be worse than firsthand smoking. Smoking cessation or removing the smoke exposure will gradually decrease risk. Inhalation of other chemicals (e.g., asbestos, radon gas, tar, and pollution) and chronic lung disease can also increase risk (FIGURE 5-24).

The lungs provide an optimal environment for tumor development and growth. Carcinogens can seek refuge in the many nooks and crannies of the air passages, having an opportunity to cause cellular changes (usually metaplasia) there. The scores of blood vessels supplying the lungs serve as entrance points for distant cancer cells to gain access, and those vessels furnish the cancer with a rich blood source to facilitate its growth.

Lung cancers are divided into two types—small cell and non–small cell. **Small-cell carcinoma**, often referred to as oat-cell carcinoma, occurs almost exclusively in heavy smokers and is less frequent than non-small-cell cancers. **Non-small-cell carcinoma**, often referred to as bronchogenic carcinoma, is the most common type of malignant lung cancer, accounting for 75% of all cases of lung cancer. This very aggressive lung cancer is classified into several subgroups—squamous cell carcinoma, adenocarcinoma, and bronchioalveolar carcinoma.

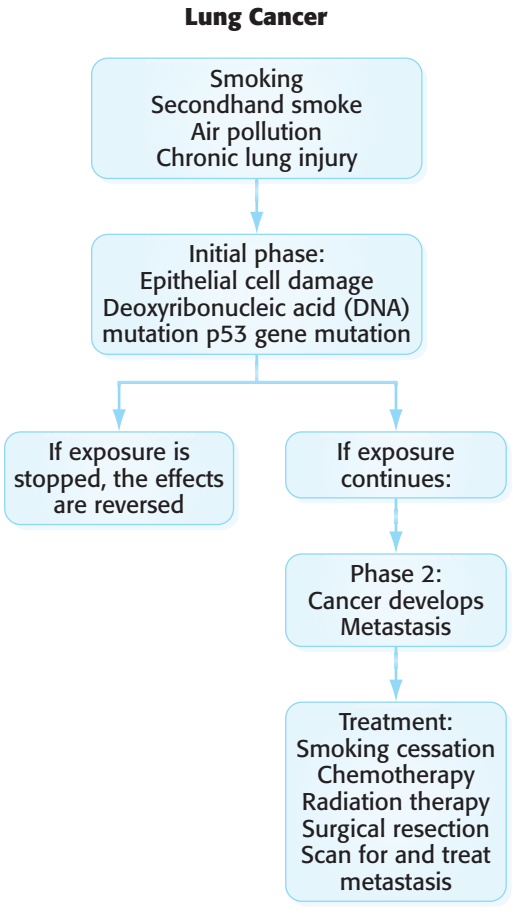


FIGURE 5-24 Lung cancer.

Madara, M., & Pomarico-Denino, V. (2008). *Quick look nursing: Pathophysiology* (2nd ed.). Sudbury, MA: Jones & Bartlett.

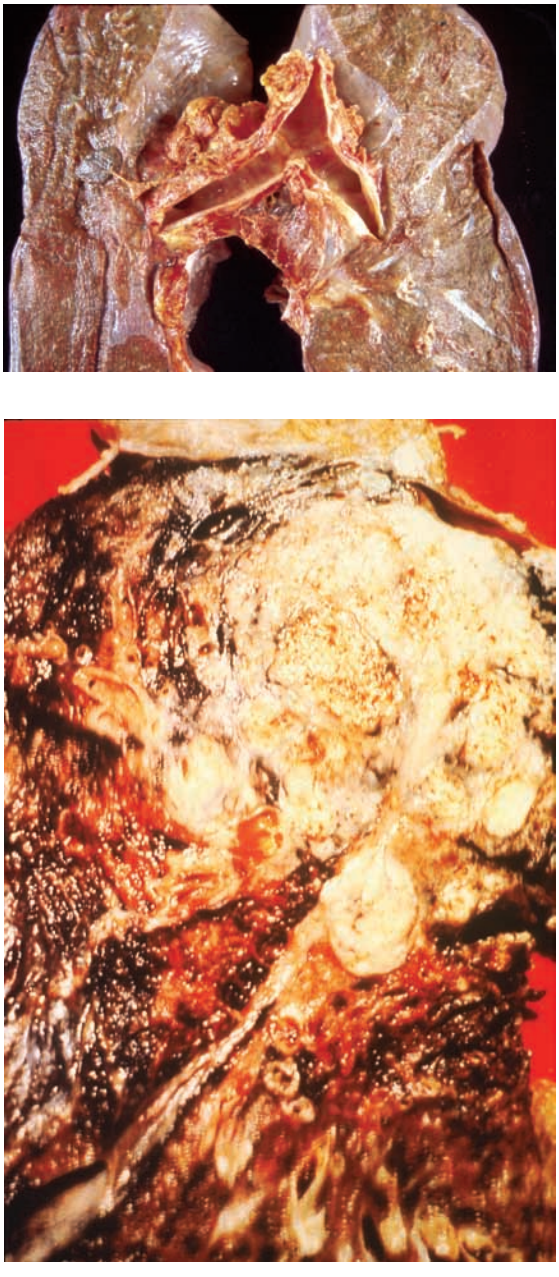
Upon exposure to the carcinogen, irreversible oncogene DNA mutations and inactivation of tumor suppressor genes occur. If carcinogen exposure continues, cancer develops (FIGURE 5-25).

Tumors in the lungs lead to several issues, including the following:

- Airway obstruction
- Inflammation of lung tissue, eliciting coughing and contributing to infections
- Fluid accumulation in the pleural space (e.g., pleural effusion, hemothorax, and pneumothorax)
- Paraneoplastic syndrome (endocrine dysfunction associated with hormone secretion from the tumor)

Clinical manifestations of lung cancer are insidious because they mimic signs of smoking:

- Persistent cough or a change in usual cough
- Dyspnea
- Hemoptysis
- Frequent respiratory infections



Courtesy of National Cancer Institute

FIGURE 5-25 The normal (top) and cancerous (bottom) lung.

© University of Alabama at Birmingham Department of Pathology PEIR Digital Library (http://peir.net)

- Chest pain
- Hoarseness
- Weight loss
- Anorexia
- Anemia
- Fatigue
- Other symptoms specific to the site(s) of metastasis

Diagnostic procedures for lung cancer include a history, physical examination, chest X-ray, CT, MRI, bronchoscopy, sputum studies,

TABLE 5-6 Staging and Treatment of Non-Small-Cell Lung Cancer

Stage	Description	Usual Treatment Plan
Stage I	Cancer has invaded the underlying lung tissue but has not spread to the lymph nodes.	Surgery
Stage II	Cancer has spread to neighboring lymph nodes or invaded the chest wall.	Surgery, radiation, and chemotherapy
Stage IIIA	Cancer has spread from the lung to lymph nodes in the center of the chest.	Combined chemotherapy and radiation, sometimes surgery based on results of treatment
Stage IIIB	Cancer has spread locally to areas such as the heart, blood vessels, trachea, and esophagus—all within the chest—or to lymph nodes in the area of the collarbone or to the tissue that surrounds the lungs within the rib cage (pleura).	Chemotherapy, sometimes radiation
Stage IV	Cancer has spread to other parts of the body, such as the liver, bones, or brain.	Chemotherapy, targeted drug therapy, clinical trials, supportive care

TABLE 5-7 Staging and Treatment of Small-Cell Lung Cancer

Stage	Description	Usual Treatment Plan
Limited	Cancer is confined to one lung and to its neighboring lymph nodes.	Combined chemotherapy and radiation, sometimes surgery
Extensive	Cancer has spread beyond one lung and nearby lymph nodes, and may have invaded both lungs, more remote lymph nodes, or other organs.	Chemotherapy, clinical trials, supportive care

biopsy, positron emission tomography, bone scans, and pulmonary function tests. Treatment is based on staging and follows usual cancer treatment—chemotherapy, surgery, and radiation (**TABLE 5-6**; **TABLE 5-7**). The treatment is generally palliative because the tumor does not usually respond favorably to treatment. Early diagnosis and treatment will improve this prognosis. Other strategies include those to maintain optimal respiratory function—oxygen therapy, bronchodilators, and antibiotics (if bacterial infections are present).

Pleural Effusion

A **pleural effusion** is the accumulation of excess fluid in the pleural cavity. Normally, a very small amount of fluid drained from the lymphatic system is present in this space to lubricate the constantly moving lungs. Excessive fluid in the pleural cavity can compress the lung and limit expansion during inhalation. Effusions vary in nature and may affect both lungs or one lung. Fluid that can accumulate to create the effusion includes exudates (due to inflammation), transudate (due to increased hydrostatic pressure), blood (due to trauma), and pus (due to infection). The consequence of this effusion depends on its type, location, amount, and fluid accumulation rate. Large amounts of fluids can cause the pleural membranes to separate, preventing their cohesion during inhalation (FIGURE 5-26; FIGURE 5-27). This lack of cohesion impedes full expansion, leading to atelectasis and a pneumothorax. Large effusions can also impair venous return in the inferior vena cava and cardiac filling by putting pressure on those structures.

Pleurisy, or pleuritis, can precede or follow the effusion, or it may occur independently. Pleurisy refers to inflammation of the pleural membranes, which leads to swollen and irregular tissue. This inflammation is often associated with pneumonia and creates friction in the pleural membranes.

Clinical manifestations of pleural effusion include the following signs and symptoms:

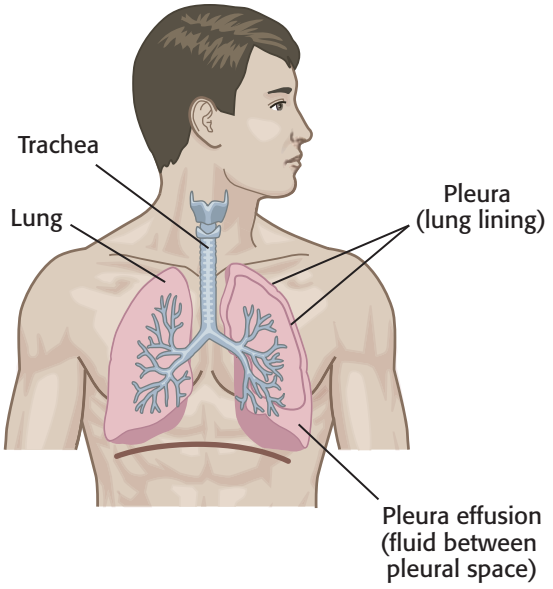


FIGURE 5-26 Pleural effusion is a buildup of fluid in the lining of the lungs.

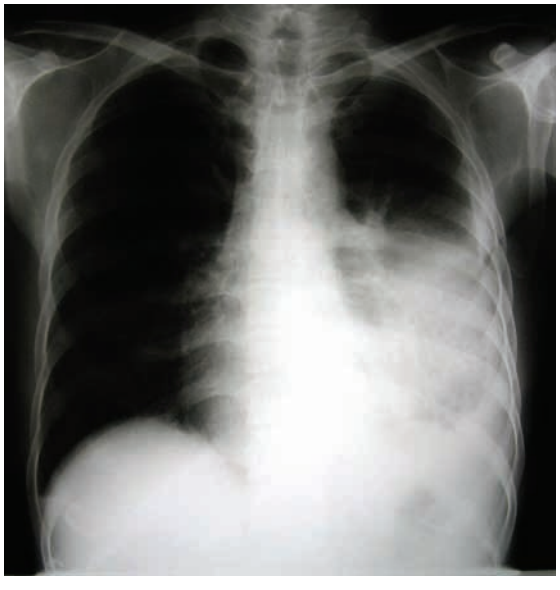


FIGURE 5-27 X-ray of pleural effusion.

- Dyspnea
- Chest pain (usually sharp and worsening with inhalation)
- Tachypnea
- Tracheal deviation (toward the unaffected side)
- Diminished or absent lung sounds over the affected area
- Dullness to percussion over affected area
- Tachycardia
- Pleural friction rub (pleurisy)

Diagnostic procedures for pleural effusion include a history, physical examination, chest X-ray, CT, ABGs, CBC, and thoracentesis (needle aspiration of fluid) with subsequent examination of fluid. Treatment focuses on addressing the underlying cause. Nevertheless, regardless of etiology, removal of the fluid is necessary to promote full expansion of the lungs. Strategies to do so may include thoracentesis, placement of a chest drainage tube, and antibiotics.

Pneumothorax

Pneumothorax refers to air in the pleural cavity. The presence of atmospheric air in the pleural cavity and the separation of pleural membranes can lead to atelectasis. The resulting pressure can cause a partial or complete collapse of a lung (FIGURE 5-28; FIGURE 5-29). A small pneumothorax causes mild symptoms and may heal on its own. A larger pneumothorax generally requires aggressive treatment to remove the air and reestablish pulmonary negative pressure. Risk factors for developing pneumothorax include smoking, tall stature, and history of lung disease or previous pneumothorax.

Several types of pneumothorax are distinguished, based on their cause. A **spontaneous pneumothorax** develops when air enters the pleural cavity from an opening in the internal airways. Primary spontaneous pneumothorax occurs when a small air blister (bleb) on the top of the lung ruptures. Blebs are caused by a weakness in the lung tissue and can rupture from changes in air pressure, such as occurs in scuba diving, flying, mountain climbing, or listening to extremely loud music. Additionally, a primary spontaneous pneumothorax may happen while smoking marijuana—a deep inhalation, followed by slow breathing out against partially closed lips, forces the smoke deeper into the lungs. Most commonly, these blebs rupture for no obvious reason, although genetic factors may play a role. A primary spontaneous pneumothorax is usually mild because pressure from the collapsed portion of the lung may, in turn, collapse the bleb. A secondary spontaneous pneumothorax develops in people with preexisting lung disease (e.g., emphysema, pneumonia, cystic fibrosis, or lung cancer). In these cases, the pneumothorax occurs because the diseased lung tissue is weakened. Secondary spontaneous pneumothorax can be more severe and even life threatening because diseased tissue can create a larger opening, allowing more air to enter the pleural space. Additionally, pulmonary disease reduces lung reserves, making any further reduction in lung function more serious.

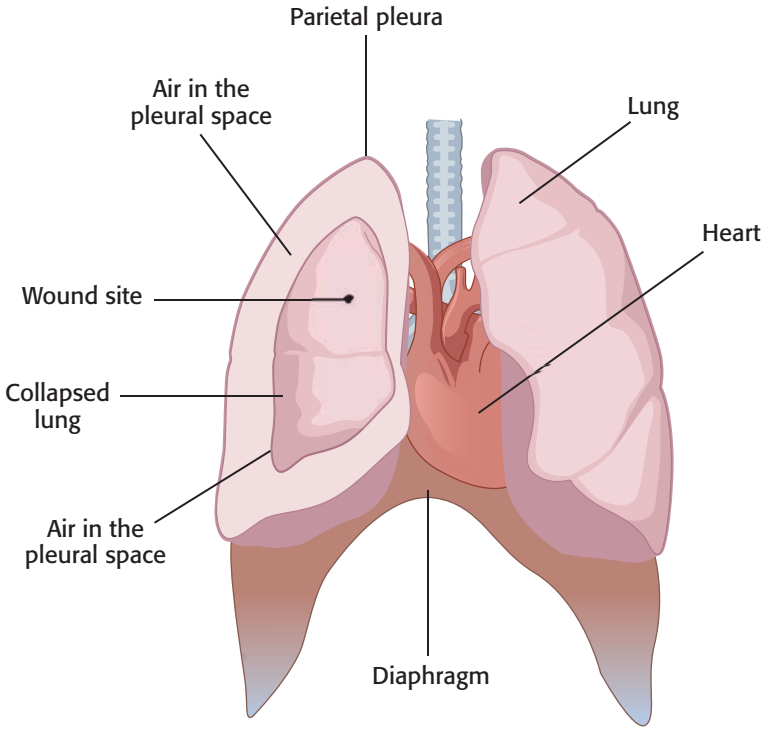


FIGURE 5-28 A pneumothorax occurs when air leaks into the pleural space between the parietal and visceral pleura. The lung collapses as air fills the pleural space and the two pleural membranes are no longer in contact with each other.

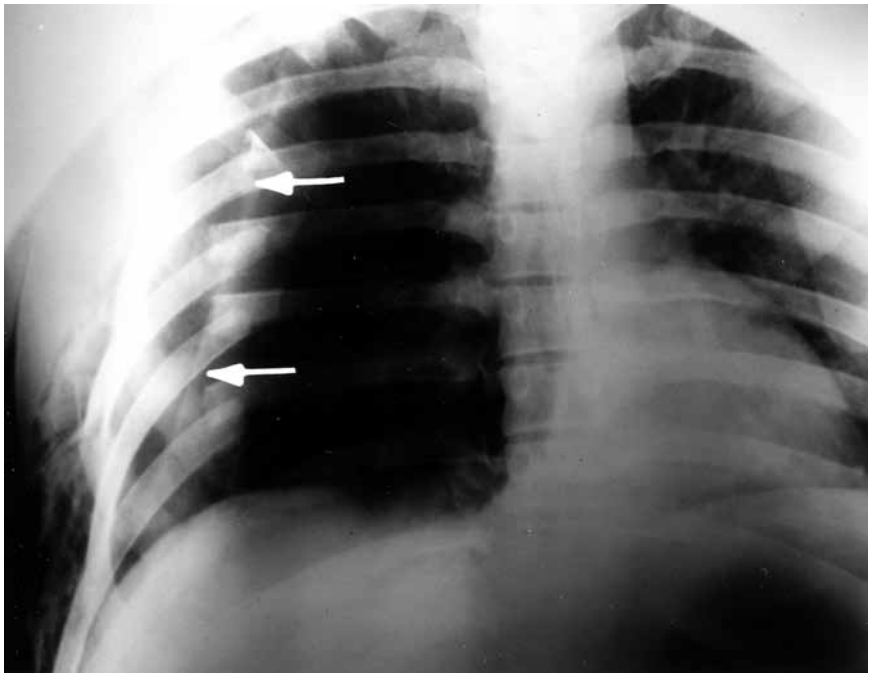


FIGURE 5-29 X-ray of pneumothorax.

Courtesy of Leonard V. Crowley, MD, Century College.

A **traumatic pneumothorax** stems from any blunt trauma (e.g., vehicle air bag deployment) or penetrating injury (e.g., knife or gunshot wounds) to the chest. These injuries can inadvertently occur during certain medical procedures, such as chest tube insertion, cardiopulmonary resuscitation, and lung or liver biopsies.

A **tension pneumothorax** is the most serious type of pneumothorax; it occurs when the pressure in the pleural space is greater than the atmospheric pressure. This increased pressure arises due to trapped air in the pleural space or entering air from a positive-pressure mechanical ventilator. The force of the air can cause the affected lung to collapse completely and shift the heart toward the uncollapsed lung (called a mediastinal shift), compressing the unaffected lung and the heart (FIGURE 5-30). Tension pneumothorax progresses rapidly and is fatal if not treated quickly.

Clinical manifestations vary in severity depending on the type of pneumothorax. These manifestations include the following signs and symptoms:

- Sudden chest pain over the affected lung
- Chest tightness
- Dyspnea
- Tachypnea
- Decreased breath sounds over the affected area
- Asymmetrical chest movement
- Trachea and mediastinum deviation toward the unaffected side
- Anxiety
- Tachycardia
- Pallor
- Hypotension

Diagnostic procedures for pneumothorax consist of a history, physical examination, chest X-ray, CT, and ABGs. Treatment usually involves removal of the air and reestablishment of negative pressure, allowing for full expansion of the lungs. Such strategies may include a thoracentesis and placement of a chest drainage tube with suction (which removes fluid and reestablishes negative pressure). Surgery may be required in some cases to correct and prevent future episodes. During surgery, the leak is repaired and a chemical may be used to scar the area (pleurodesis).

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is a sudden failure of the respiratory system, often as a result of fluid accumulation in the alveoli. ARDS has many other names, such as shock lung, wet lung, and stiff lung.

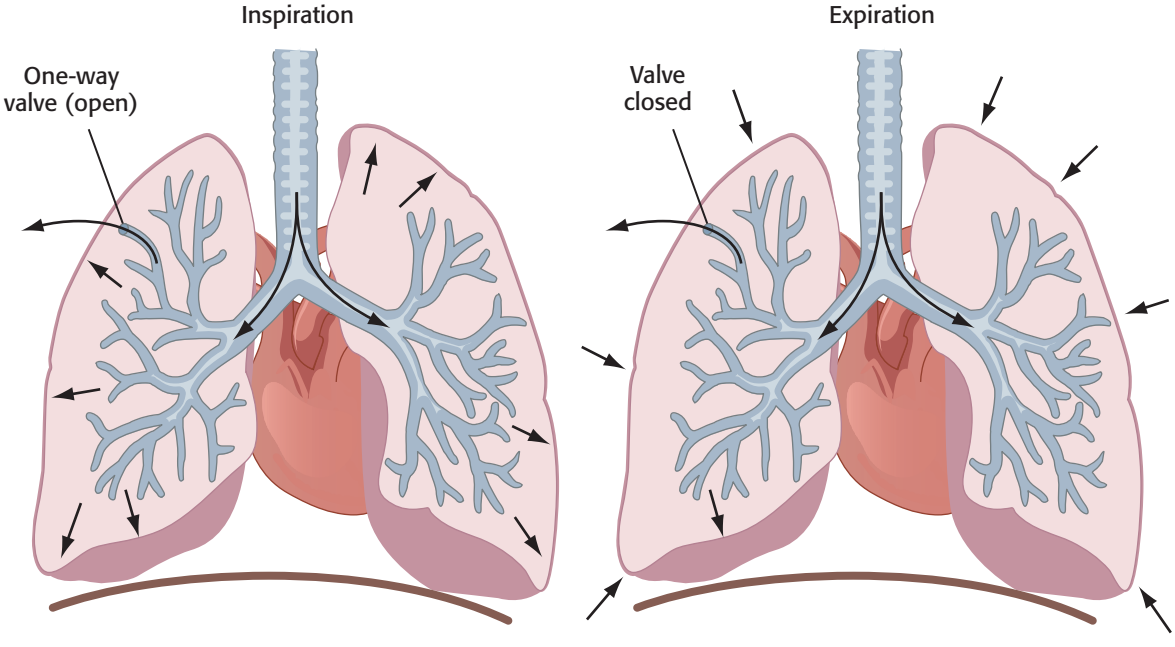


FIGURE 5-30 Tension pneumothorax. A one-way valve allows air into the pleural space during inspiration, but not out during expiration.

Acute lung injury (ALI) refers to a slightly less severe form of ARDS. Multiple conditions can precipitate ARDS, including prolonged shock, burns, aspiration, and smoke inhalation. This condition involves an acute hypoxemia resulting from a systemic event (e.g., trauma, septicemia, acute pancreatitis, drug overdose, cardiopulmonary bypass, or transfusion reaction) or pulmonary event (e.g., illicit drug and toxic gas inhalation, pneumonia, RSV, gastric acid aspiration, near drowning, and fat embolism) that is not cardiac in origin. ARDS develops

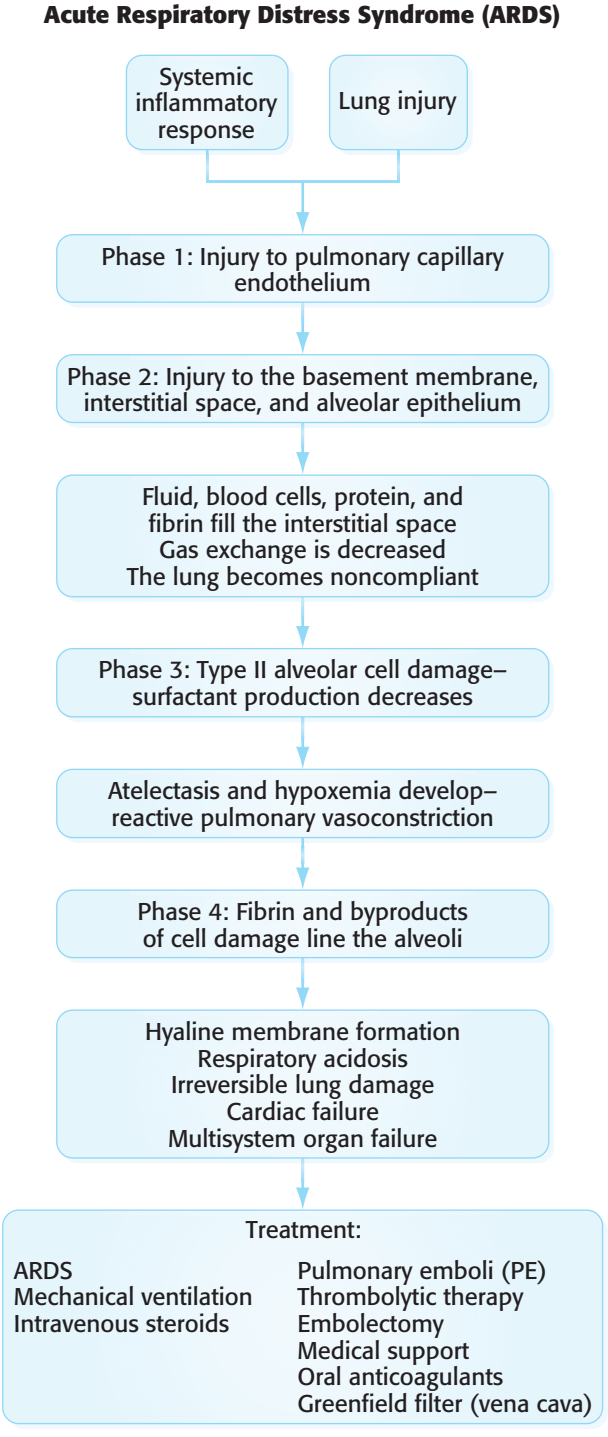


FIGURE 5-31 Acute respiratory distress syndrome (ARDS).

Madara, M., & Pomarico-Denino, V. (2008). *Quick look nursing: Pathophysiology* (2nd ed.). Sudbury, MA: Jones & Bartlett.

rapidly, often within 90 minutes of a systemic inflammatory response or within 48 hours of a lung injury. Other risk factors include the presence of a chronic lung disease (e.g., emphysema, chronic bronchitis, and asthma), alcoholism, age greater than 65 years, and mechanical ventilation. ARDS is fatal in one-third of cases. Individuals who survive will fully recover, but it may take as long as a year for them to regain complete lung function.

In ARDS, injury to the alveoli and the capillary membranes leads to the release of chemical inflammatory mediators (FIGURE 5-31; FIGURE 5-32). These mediators increase capillary permeability, promote fluid and protein accumulation in the alveoli, and damage surfactant-producing cells. These events result in decreased gas exchange, reduced pulmonary blood flow, and limited lung expansion. Diffuse atelectasis and reduced lung capacity ensue. Lung damage progresses as neutrophils migrate to the site, releasing proteases and other mediators once there. A hyaline membrane, or a thin layer of tissue, forms in the alveoli and causes them to become stiff. Additionally, increased platelet aggregation promotes microemboli development. If the patient survives, scattered necrosis and fibrosis are apparent throughout the lungs. ARDS is similar in pathogenesis to disseminated intravascular coagulation (DIC) (see the *Hematopoietic Function* chapter).

ARDS is a serious condition that can lead to several complications:

- Respiratory failure
- Respiratory and metabolic acidosis
- Pulmonary fibrosis
- Pneumothorax
- Bacterial lung infections (e.g., stasis pneumonia and VAP)
- Decreased lung function
- Renal failure
- Stress ulcer
- Thromboembolism
- Muscle wasting
- Memory, cognitive, and emotional issues (due to brain damage as a result of the hypoxia)

Clinical manifestations of ARDS can develop suddenly and include the following:

- Dyspnea
- Labored (requiring the use of accessory muscles), shallow respirations
- Abnormal lung sounds (e.g., rales and rhonchi)
- Productive cough with frothy sputum

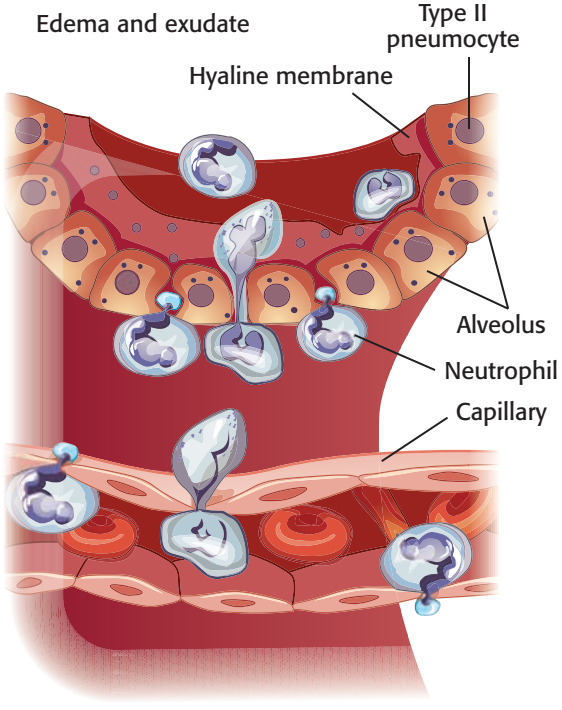


FIGURE 5-32 Acute respiratory distress syndrome.

- Hypoxia
- Cyanosis
- Fever
- Hypotension
- Tachycardia
- Restlessness
- Confusion
- Lethargy
- Anxiety

Diagnostic procedures for ARDS consist of a history, physical examination, ABGs, chest X-ray, CT, and CBC. The main goal of treatment is to maintain adequate oxygenation and respiratory status. Such strategies include endotracheal intubation with a mechanical ventilator (including positive end-expiratory pressure [PEEP]), high-dose oxygen therapy, corticosteroids, inhaled beta agonists, and antibiotics (if bacterial infections are present), as well as prevention and treatment of emboli (e.g., embolectomy, anticoagulants, and antiplatelet agents). Other strategies include nutritional support (e.g., enteral feedings), conservative fluid therapy, sedation, and stress ulcer prophylaxis.

Alterations in Ventilation and Perfusion

Atelectasis

Atelectasis refers to incomplete alveolar expansion or collapse of the alveoli. It occurs when the walls of the alveoli stick together. Atelectasis may be caused by the following conditions:

- Surfactant deficiencies (surfactant is the lipoprotein that coats the inside of the alveoli, allowing them to remain open at the end of expiration)
- Bronchus obstruction (e.g., foreign objects, mucus plugs, and tumors)
- Lung tissue compression (e.g., tumor, pneumothorax, and pleural effusion)
- Increased surface tension (e.g., pulmonary edema)
- Lung fibrosis (e.g., emphysema)

When alveoli are not filled with air, they shrivel much like a raisin. This ventilation issue can, in turn, impair blood flow through the lung. Ineffective ventilation and perfusion impair gas exchange. Surgery and immobility increase the risk for developing atelectasis for this reason.

Atelectasis can occur in smaller or larger areas. If only a small area is affected, the respiratory rate will increase to control carbon dioxide levels (increasing the respiratory rate will increase the excretion of carbon dioxide). The larger the area affected, the more severe the symptoms experienced. Necrosis, infection (e.g., pneumonia), and permanent lung damage can occur if the alveoli are not reinflated quickly.

The clinical manifestations of atelectasis are due to impaired ventilation and perfusion:

- Diminished breath sounds
- Dyspnea
- Tachypnea
- Asymmetrical lung movement
- Anxiety
- Restlessness
- Tracheal deviation
- Tachycardia

Diagnostic procedures for atelectasis include a history, physical examination, chest X-ray (FIGURE 5-33), CT, bronchoscopy, ABGs, and CBC. Treatment of atelectasis focuses on treating the underlying causes (e.g., antibiotics, thoracentesis) and reinflating the alveoli. Incentive spirometry (a device to promote ventilation) is effective in reinflating the alveoli. For more severe cases, continuous positive airway pressure or endotracheal intubation may be necessary for ventilation support. Prevention strategies include increasing mobility (e.g., turning and ambulating), coughing, and deep breathing exercises (e.g., incentive spirometry) every 1–2 hours. Effective pain management and postoperative incisional splinting increase the likelihood that these interventions will be performed adequately.

Acute Respiratory Failure

Acute respiratory failure (ARF) is a serious, life-threatening condition that can be the result of many disorders (e.g., COPD, asthma, ARDS,

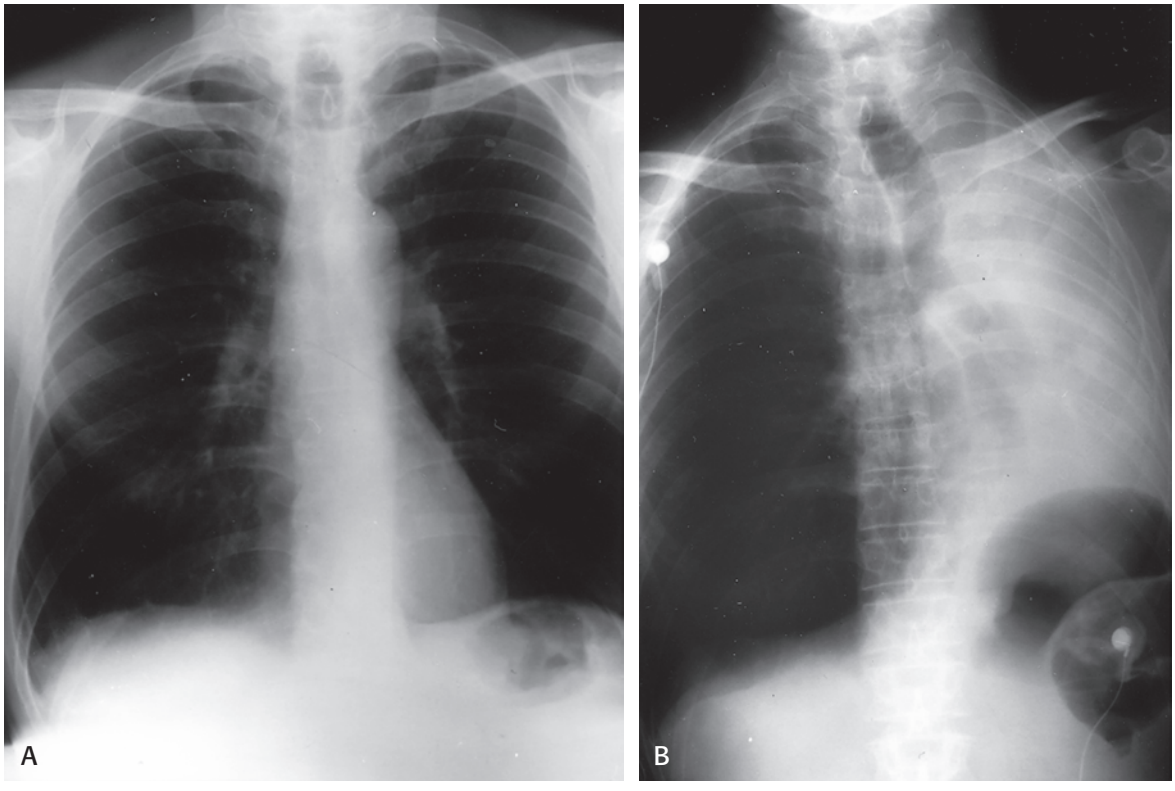


FIGURE 5-33 X-ray of atelectasis (a) Normal; (b) Atelectasis.
Courtesy of Leonard V. Crowley, MD, Century College.

amyotrophic lateral sclerosis, alcohol or drug overdose, and spinal cord injury). In ARF, the oxygen levels become dangerously low (less than 50 mm Hg) or carbon dioxide levels become dangerously high (greater than 50 mm Hg). Normally, oxygen levels are in the range of 80–100 mm Hg and carbon dioxide levels are in the range of 35–45 mm Hg. The low oxygen levels observed in ARF are not sufficient to meet the body's metabolic needs, and the nervous system quickly becomes affected by the shortage of oxygen. This gas level becomes progressively worse as the patient's condition worsens. Respiratory acidosis develops as the carbon dioxide levels rise (see the *Fluid, Electrolyte, and Acid–Base Homeostasis* chapter). The hypoxia and acidosis trigger a reflex pulmonary vasoconstriction, further impairing gas exchange and increasing cardiac workload. The heart decompensates from the lack of oxygen, which could lead to cardiac arrest. Respiratory arrest may occur as the respiratory system ceases all activity from the strain.

Clinical manifestations are usually evident and result from the impaired gas exchange:

- Shallow respirations
- Headache
- Tachycardia
- Dysrhythmias
- Lethargy
- Confusion

Diagnostic procedures for ARF consist of a history, physical examination, ABGs, chest X-ray, electrocardiogram (EKG), and CBC. Treatment focuses on resolving the cause and maintaining adequate respiratory status. Strategies include oxygen therapy, endotracheal intubation with ventilation support (may require a tracheostomy), bronchodilators, antibiotics (if bacterial infection is present), corticosteroids, and treatment of emboli (e.g., embolectomy and anticoagulants). Cardiac support is usually inevitable as the heart arrests under the strain (e.g., cardiopulmonary resuscitation, sympathomimetic medications, and inotropic agents).

application to practice

Now that we have learned what can go wrong in the respiratory system, let's put that knowledge into practice. While working in the emergency department, the following patients need to be triaged. Which patient would have the highest priority?

- 6-month-old female with fever, audible inspiratory stridor, and restlessness
- 25-year-old male with nasal drainage and hoarseness
- 40-year-old female with fever, severe body aches, and nasal congestion
- 59-year-old male with stage III bronchiogenic carcinoma complaining of nausea and pain that measures a 6 on a 0–10 scale

Once again, you go through the usual thought process—who would die first, acute versus chronic conditions, Maslow's hierarchy of needs, and patient safety. Starting with the 6-month-old, the combination of manifestations reflects the possibility of epiglottitis or some other acute infectious respiratory process. Inspiratory stridor indicates that the child may have limited air entering the lungs due to an obstruction. The restlessness could be an indication of hypoxia. Additionally, the patient's young age increases the likelihood of her decompensating quickly. This patient definitely needs to stay on your radar. The 25-year-old is exhibiting manifestations likely due to laryngitis, which is not usually life threatening. Additionally, there is no indication that this patient is in any respiratory distress. The 40-year-old likely has the flu. Although extremely uncomfortable, the flu is not usually life threatening in otherwise healthy adults. Also, this patient is not displaying any respiratory distress. Finally, consider the 59-year-old with lung cancer. Lung cancer is often life threatening, but this patient was admitted with pain and nausea. Although nausea and pain are uncomfortable, they are not life-threatening conditions. Furthermore, this patient is not exhibiting any manifestations indicating respiratory distress. After considering all the patients to be triaged, it becomes clear that the 6-month-old should take priority and be seen first because she is exhibiting signs of respiratory distress.

application to practice

Emma is a 7-year-old girl who has been admitted to the intensive care unit with severe respiratory distress. Her parents report that 3 days ago she developed a fever, aches, and nasal discharge, at which time she was taken to her pediatrician. Her pediatrician diagnosed her with the H1N1 strain of influenza. Emma was prescribed antiviral drugs, and her parents were given instructions on fever and hydration management. Twenty-four hours ago, her parents said she was improving—her fever was minimal, she was drinking more, and she was beginning to play. Emma's parents brought her into the emergency department because her symptoms suddenly worsened. They reported that over the course of a few hours, her breathing became more and more labored, her fever spiked, and coughing started. The emergency department healthcare provider diagnosed her with a secondary bacterial pneumonia that had quickly progressed to acute respiratory distress syndrome.

In the emergency department, Emma was intubated, placed on ventilator support, given bronchodilators, and started on intravenous antibiotics. Upon admission to the intensive care unit, she was stable but fragile. The following are her latest laboratory findings:

- ABG: pH 7.32, PaO_2 72 mm Hg, $PaCO_2$ 48 mm Hg, HCO_3 23 mm Hg
- CBC: WBC 16,000 mm^3, neutrophils 8,000 mm^3

1. What are the priority nursing interventions for Emma?
2. Describe the progression of this patient's condition from the simple flu to the life-threatening ARDS.
3. What is the significance of her lab findings?
4. What do you think this patient's prognosis is? Give your rationale.
5. What do you expect the treatment plan to include?

CHAPTER SUMMARY

The respiratory system plays a crucial role in supplying oxygen essential for cellular metabolism as well as excreting the carbon dioxide waste product of that metabolism. Because of this vital function, respiratory disorders can cause extensive and devastating problems throughout the body. Often the healthcare team has a limited amount of time to identify and respond to some of these respiratory disorders so as to control their negative consequences. Additionally, many of these diseases are preventable; therefore, identifying those persons at increased risk and implementing prevention strategies can limit the severity or halt the development of these debilitating conditions. Prevention, early detection, and prompt treatment will improve outcomes of persons suffering from these conditions, and nurses are uniquely positioned to have a positive influence on their health.

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