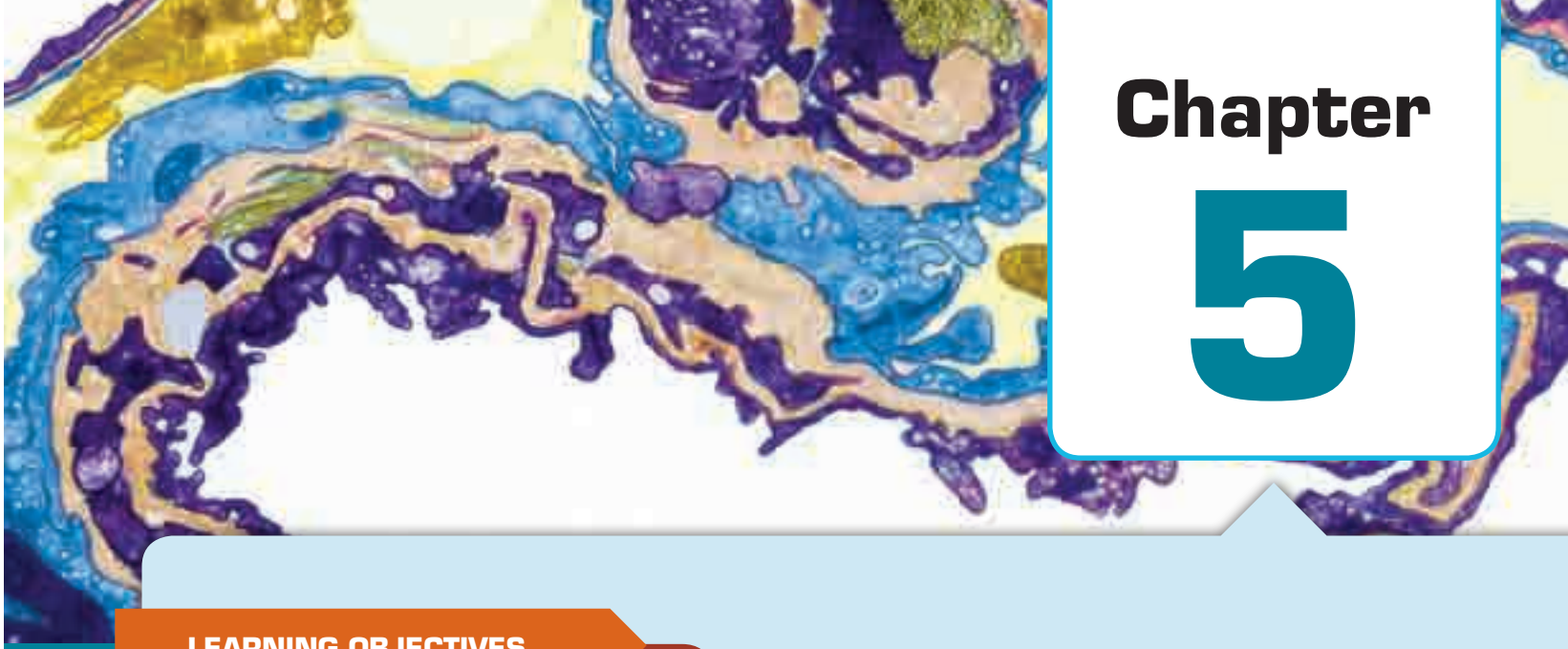

Chapter

5

Respiratory Function

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 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 one second
forced vital capacity
infectious rhinitis
influenza
inspiration
inspiratory reserve volume
interstitial pneumonia
intrinsic asthma
laryngitis
laryngotracheobronchitis
larynx
legionnaires' disease
lobar pneumonia
lung cancer
minute respiratory volume
mucus
nocturnal asthma
non–small cell carcinoma
nosocomial pneumonia
occupational asthma
perfusion
pharynx
pink puffers
pleural effusion
pleurisy
Pneumocystis carinii 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 will focus on normal and abnormal states of the lungs. The respiratory tract functions automatically to provide cells oxygen and 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 one to breathe in and out approximately 23,000 times a 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-exchange portion (**Table 5-1**). The air-conducting portion delivers air to the lungs while the gas-exchange 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**).

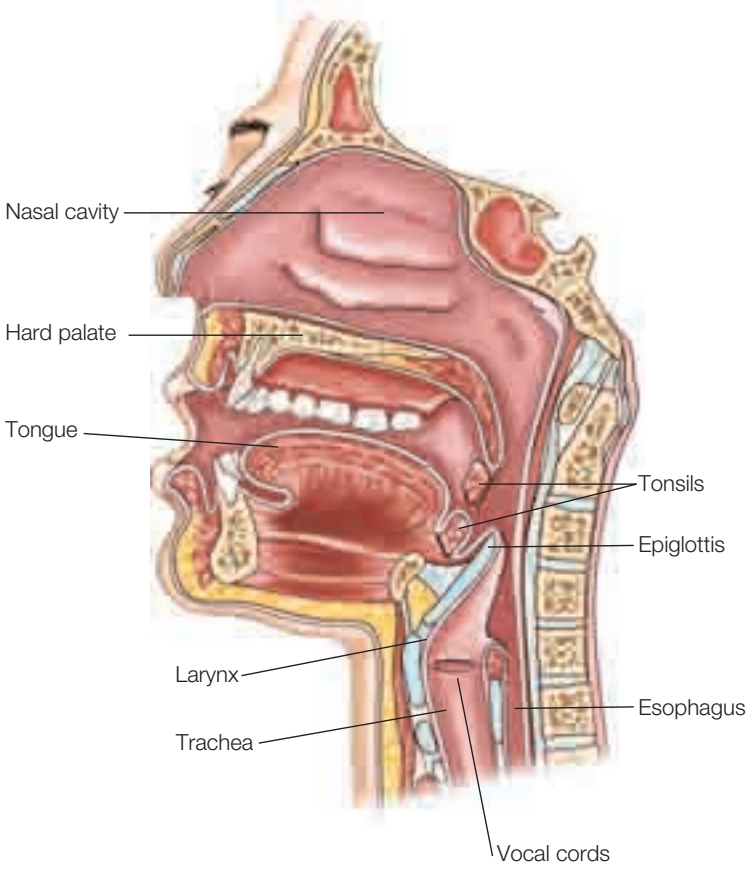

Figure 5-1

The upper respiratory tract.

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

Source: Chiras, D. (2008). *Human biology* (6th ed.). Sudbury, MA: Jones & Bartlett Learning.

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 to be 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. The larynx 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. The right bronchus is shorter and wider than the left 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 reaching 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 are mostly smooth muscle. The smooth muscle allows for constriction and dilatation of the bronchioles to control airflow. 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 airflow, which becomes dangerous.

The air entering the respiratory tract often contains particles that can be harmful. These particles 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 protect against those that gain entry. The air-conducting portion of the respiratory tract filters many particles by trapping them in 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

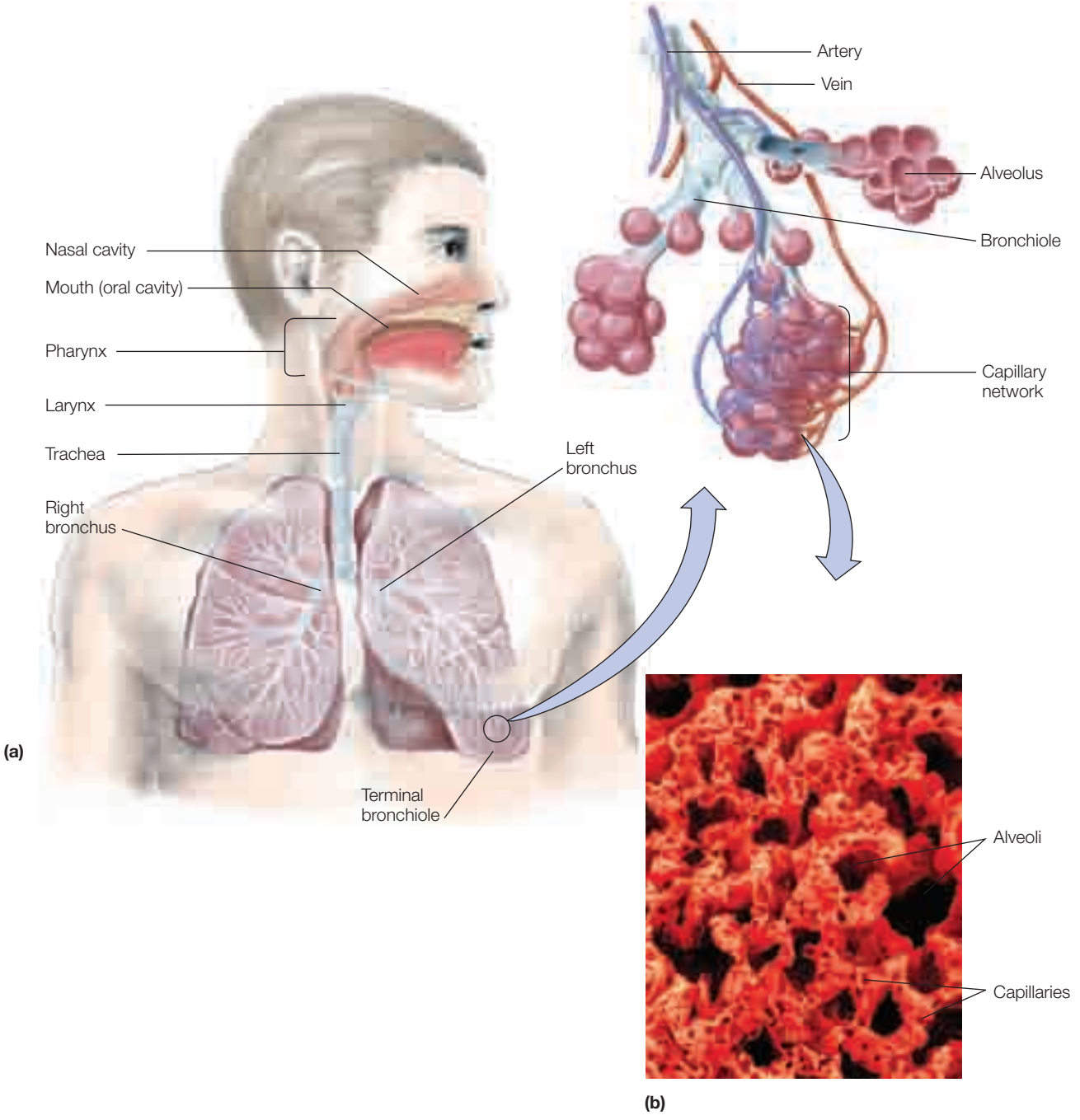

Figure 5-2

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.

epithelial lining also contains many **cilia**, hair-like projections, that move in a wavelike motion to propel the mucus and trapped particles upwards 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 Chapter 3). Macrophages are also present around the alveoli in the lungs to keep the lungs clean by phagocytizing particles that gain access (**Figure 5-6**). Once the macrophages fill with particulates, they reside in the surrounding connective tissue. In situations where there are an unusually high number of particulates (e.g., situations that occur with cigarette and marijuana smoking and pollution), the lungs become blackened by the accumulation of the particles.

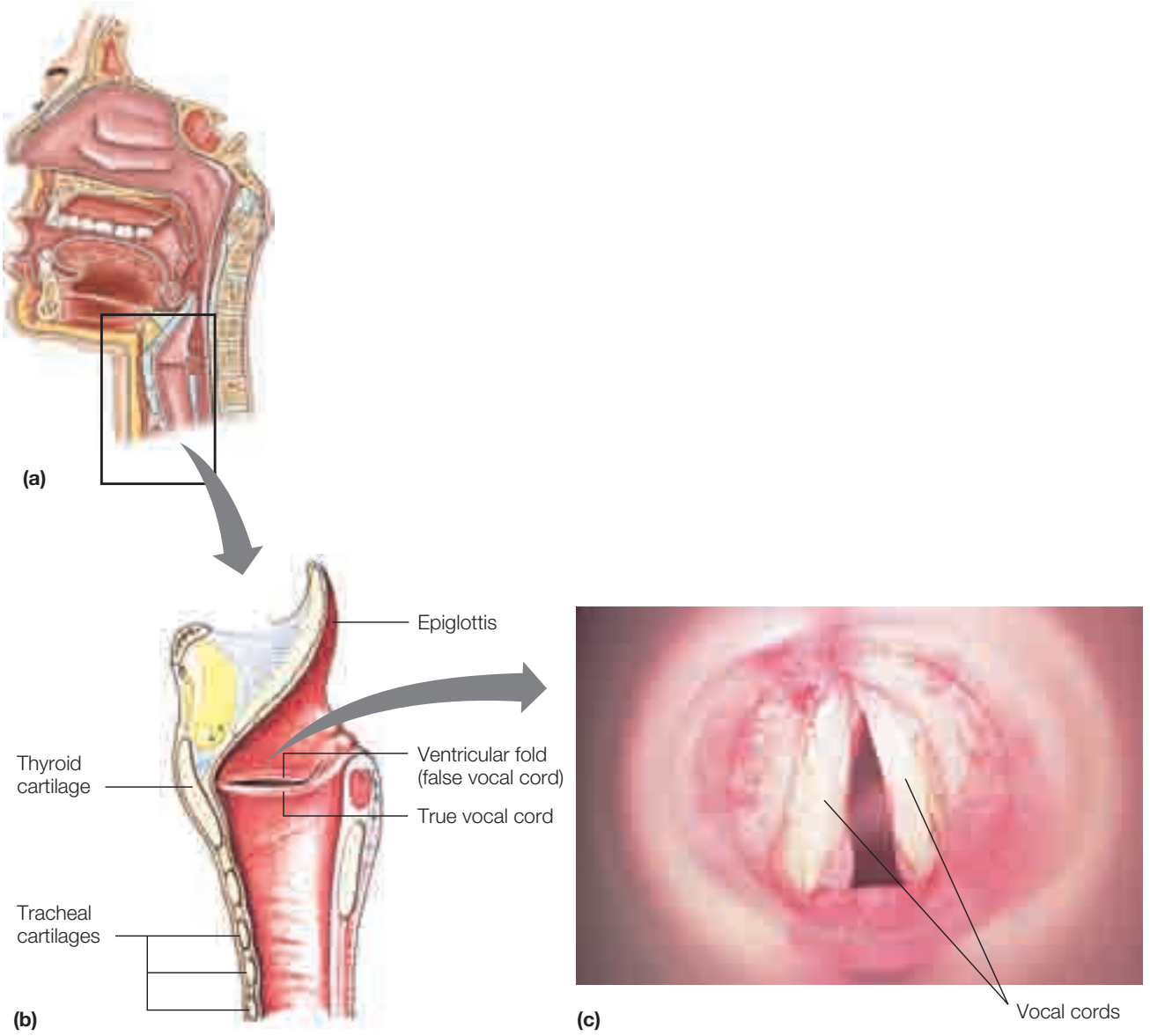

Figure 5-3

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.

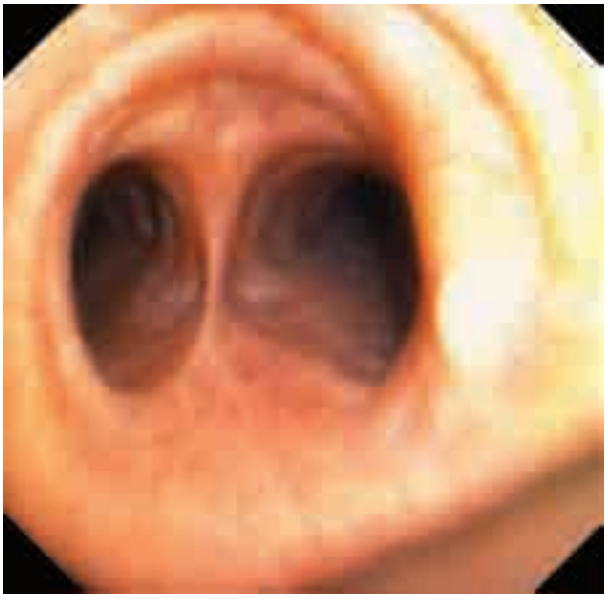

Figure 5-4

The bifurcation of the trachea at the carina into the right and left mainstem bronchi.

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 that is about 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 is dependent upon the total surface area and the thickness of the alveoli and capillary walls. The more surface area and the thinner the layers, the more rapidly gas is diffused. 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 used

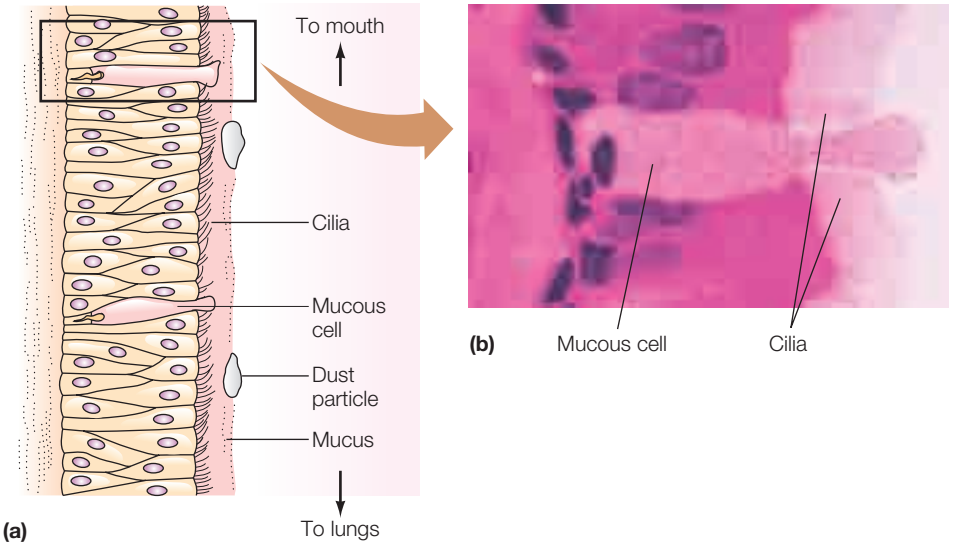

Figure 5-5

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.

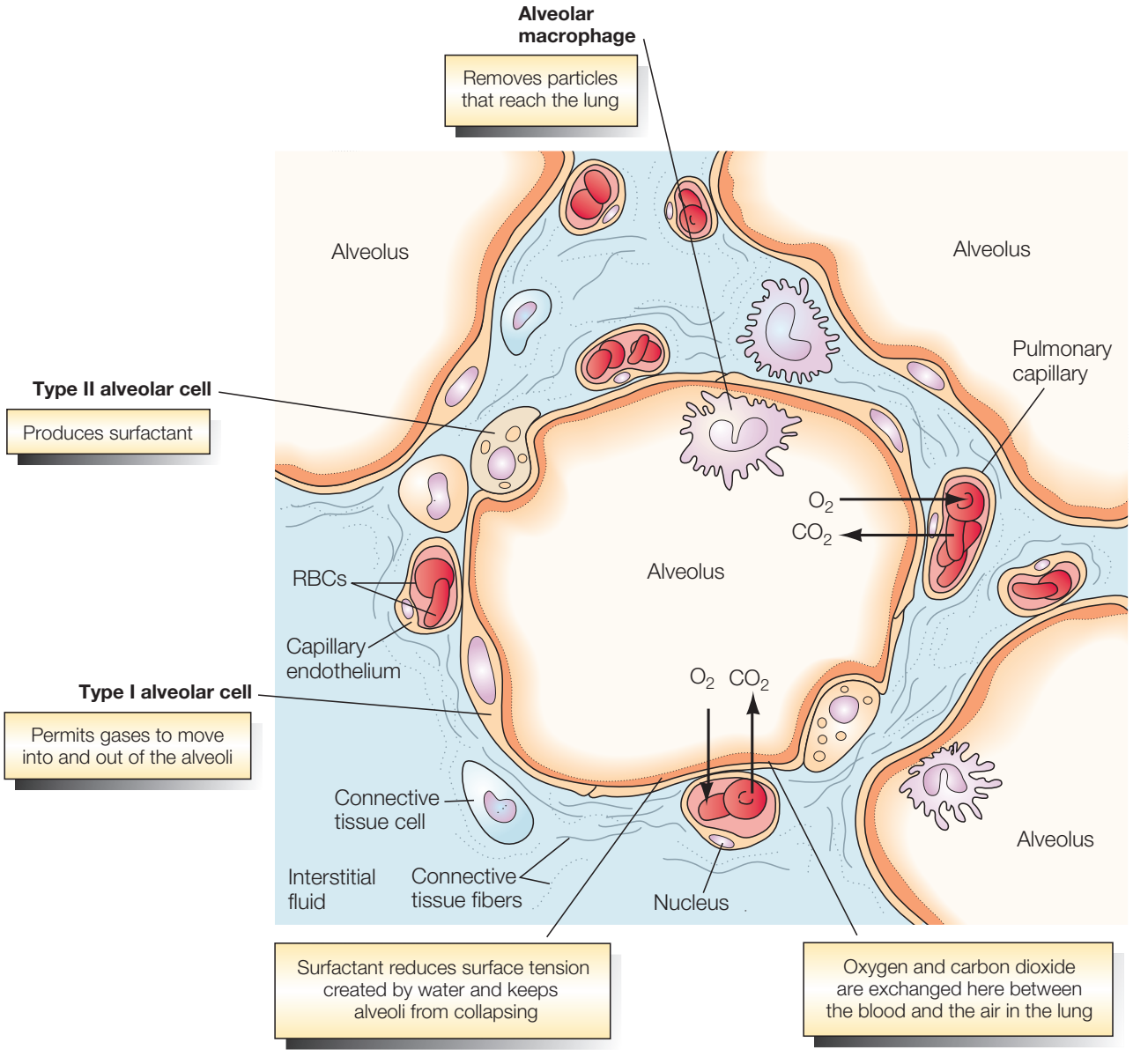

Figure 5-6

The alveolar macrophages.

to assess 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, neither alveolar ventilation nor capillary blood flow is uniform. The supply of air and blood is never equally matched, 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 is exceeding perfusion, and a VQ ratio less than 0.8 indicates poor ventilation. Some respiratory disorders are issues with ventilation while others are with perfusion, but regardless, the result is impaired gas exchange.

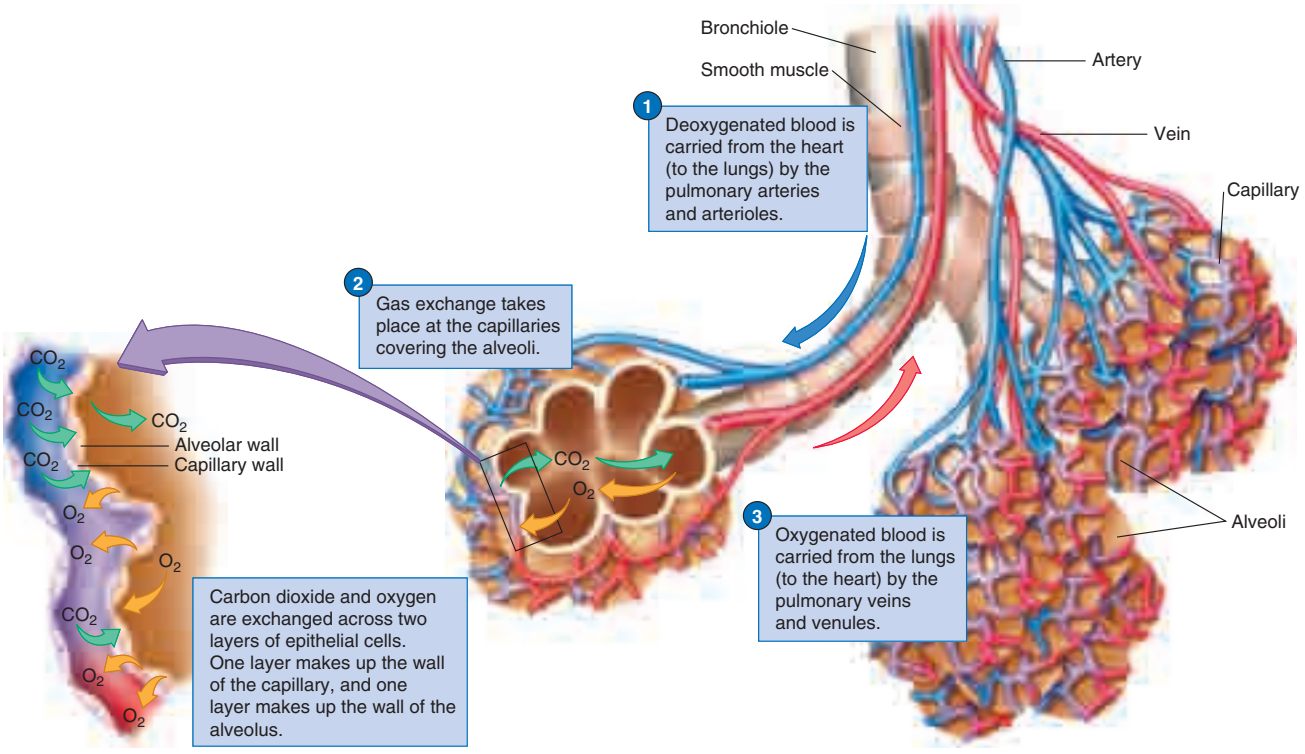

Figure 5-7

Gas exchange in the lungs.

Once 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**). Once hemoglobin carries oxygen to the cells, hemoglobin releases the oxygen. The rate at which hemoglobin binds and releases oxygen is affected by several factors such as temperature, pH, and others (**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. Surfactant is a watery substance that 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 as compared to atmospheric pressure, the walls of the alveoli tend to draw inward, making them collapse. This pressure is much like a vacuum-sealed pack of coffee. This pressure and, therefore, risk of collapse further increase at the end of expiration. Surfactant promotes reinflation of the alveoli during 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. Synthetically made surfactant may be given to replace 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 brainstem, 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 intrapulmonary pressure,

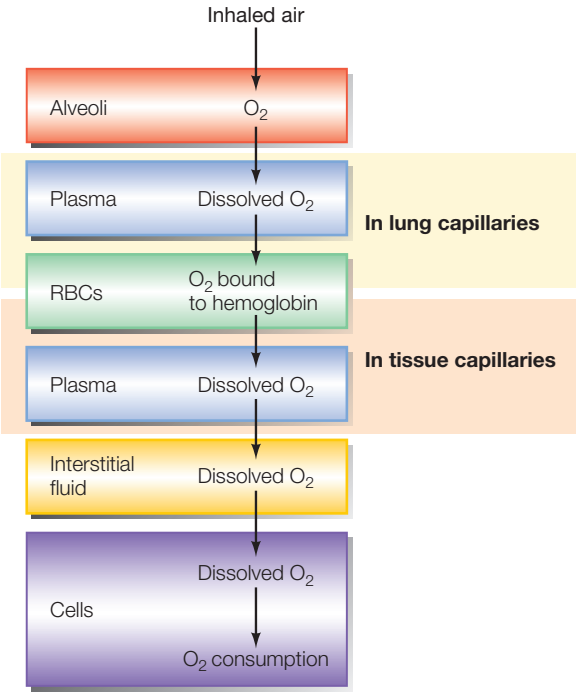

Figure 5-8

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.

causing air to naturally flow into the lungs. In contrast, expiration is passive—it does not require 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. Increasing 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 by contracting the chest and abdominal muscles. Airflow, 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 less in shallow breathing. The **minute respiratory volume** is the amount inhaled and exhaled in 1 minute. It is determined by the tidal volume multiplied by the respirations per minute, and 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.

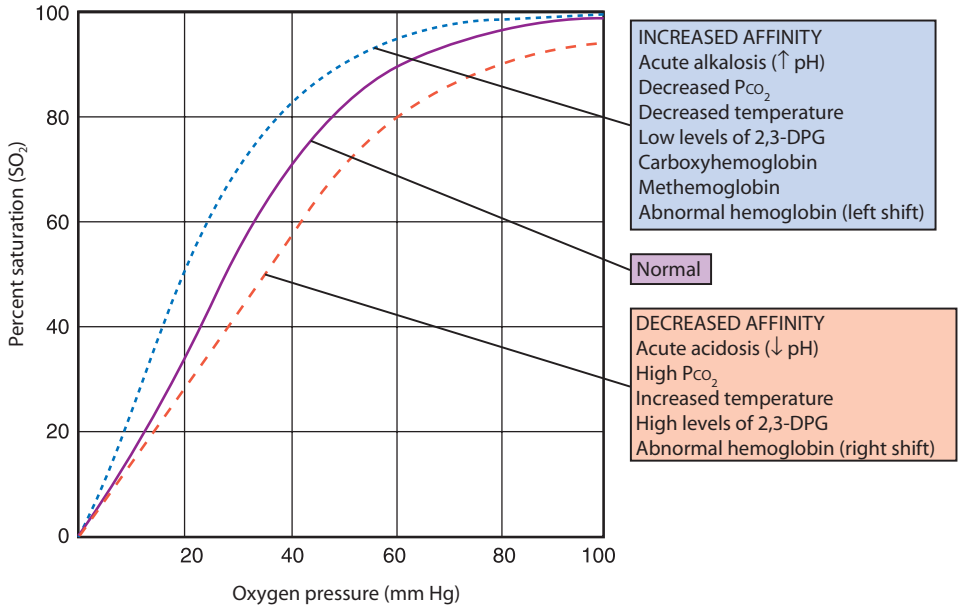

Figure 5-9

Oxyhemoglobin dissociation curve.

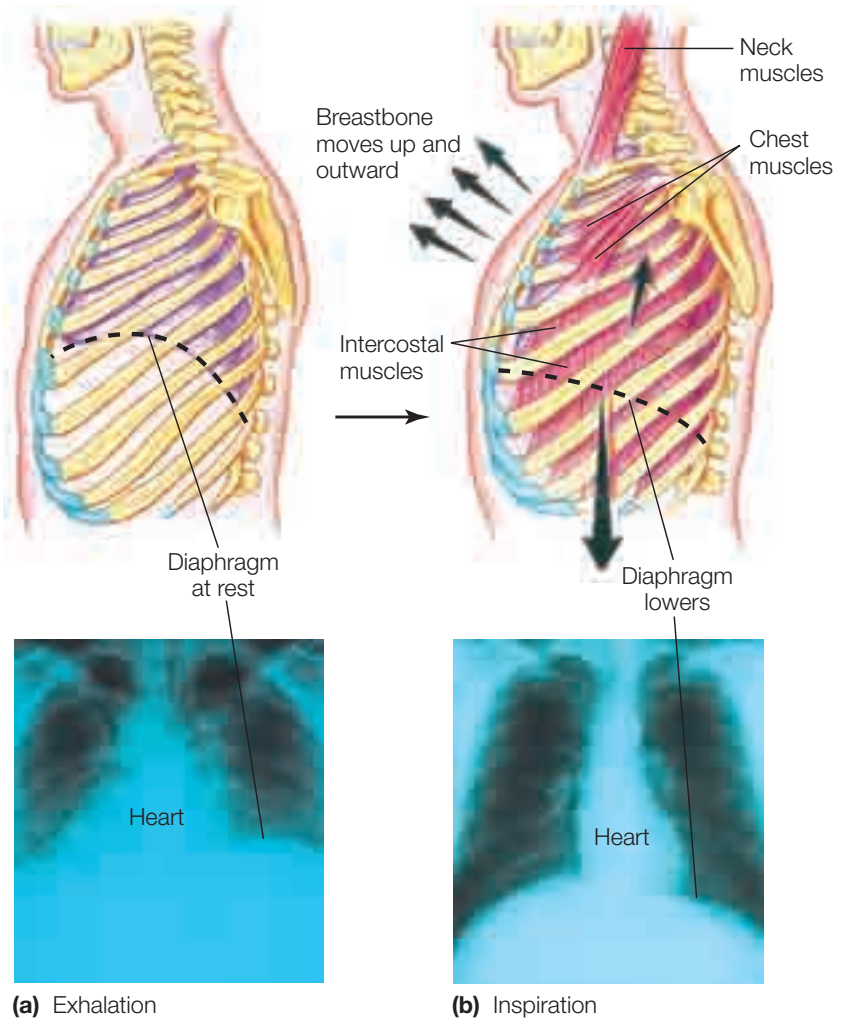

Figure 5-10

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).

The average expiratory reserve volume is 1–1.5 liters. The **vital capacity** is the sum of the tidal volume and reserves. There is always air in the lungs, which is called **residual volume**. Even after the most forceful exhalation, 1–1.5 liters of air remain in the lungs. This ensures efficient and consistent gas exchange. The **forced expiratory volume in one 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 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. When the lungs are full, the stretch receptors in the lungs send a message to the medulla to cease firing. These stretch receptors prevent overinflation of the lungs (this is called the Hering-Breuer reflex). The body

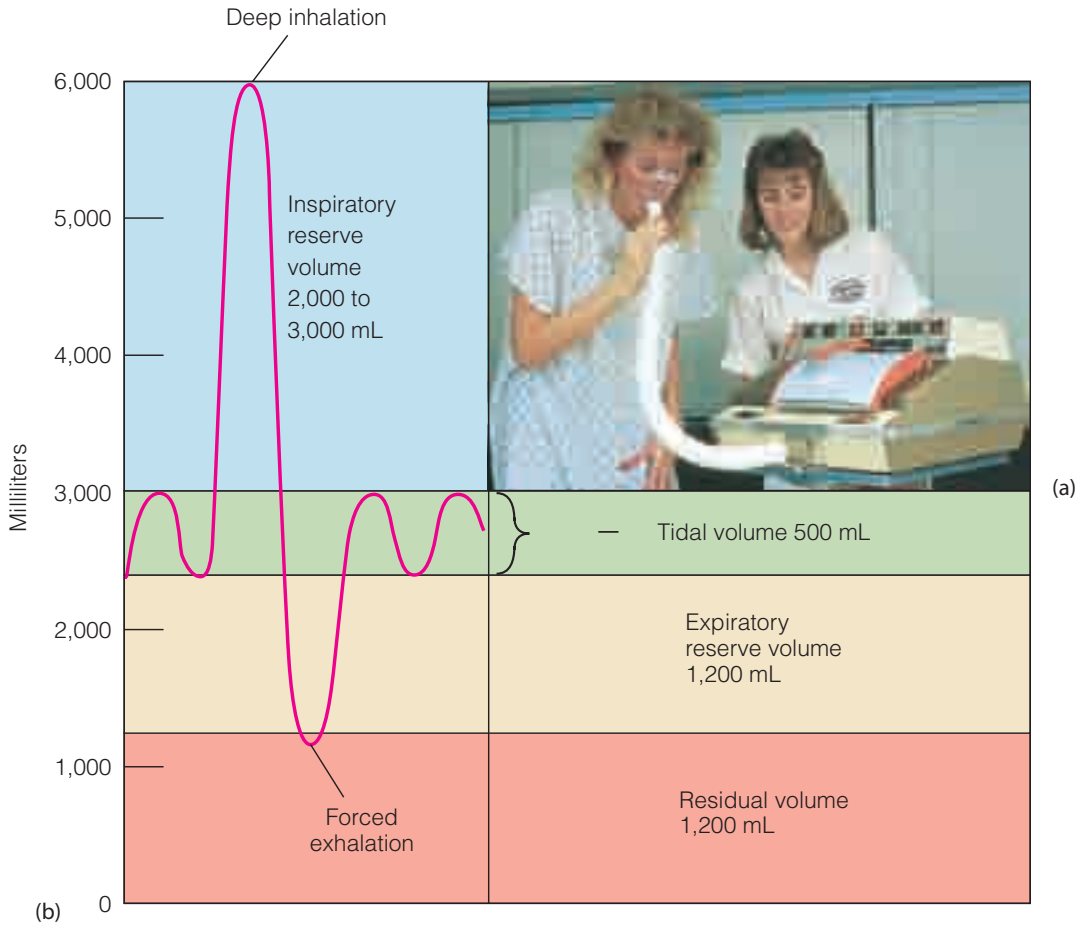

Figure 5-11

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.

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.

also has oxygen receptors, but they are not very sensitive. These receptors do not generate impulses until oxygen levels fall to critical levels.

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 excrete more carbon dioxide, making the blood less acidic, and decreasing the respiratory rate and depth will retain more carbon dioxide, making the blood more acidic. This compensatory mechanism allows for a quick fix to pH imbalances to reestablish homeostasis.

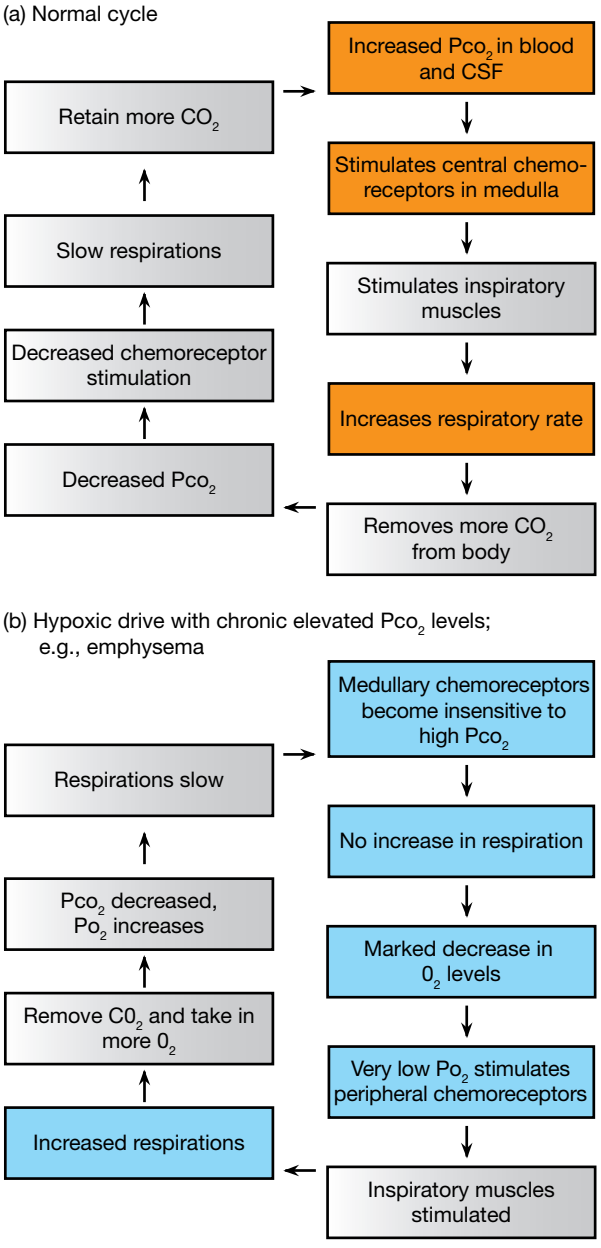

Figure 5-12

Normal respiratory control and hypoxic drive.

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 it. There are more than 100 causative organisms, making it difficult to develop immunity. The 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. Therefore, secondary bacterial infections (e.g., otitis media, sinusitis, and pneumonia) are common with viral infections (**Figure 5-13**). Despite popular misconceptions, wet and cold conditions do not cause or increase occurrences. Close physical contact with the virus causes the infection through exchanges with other humans (e.g., shaking hands) and surfaces (e.g., doorknobs and telephones). Transmission occurs through inhalation and contact (e.g., hand to hand or hand to mucous membrane). The apparent increase in occurrence of the infectious rhinitis during rainy and cold weather is due to the 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 the virus 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 and the onset of symptoms that usually lasts about 2–3 days, but it can last up to 7 days. Clinical manifestations include:

- Sneezing
- Nasal congestion
- Nasal discharge
- Sore throat
- Nonproductive cough
- Malaise
- Myalgia
- Low-grade fever
- Hoarseness
- Headache
- Chills

Diagnosis is primarily made by the presence of symptoms. Treatment is symptomatic. Most over-the-counter cold preparations are ineffective in shortening the course of the infection. Pharmacologic therapies that may be used include antipyretics (for fever), analgesics (for discomfort), antihistamines (for nasal symp-

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. The weather conditions themselves do not make you sick. 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 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!

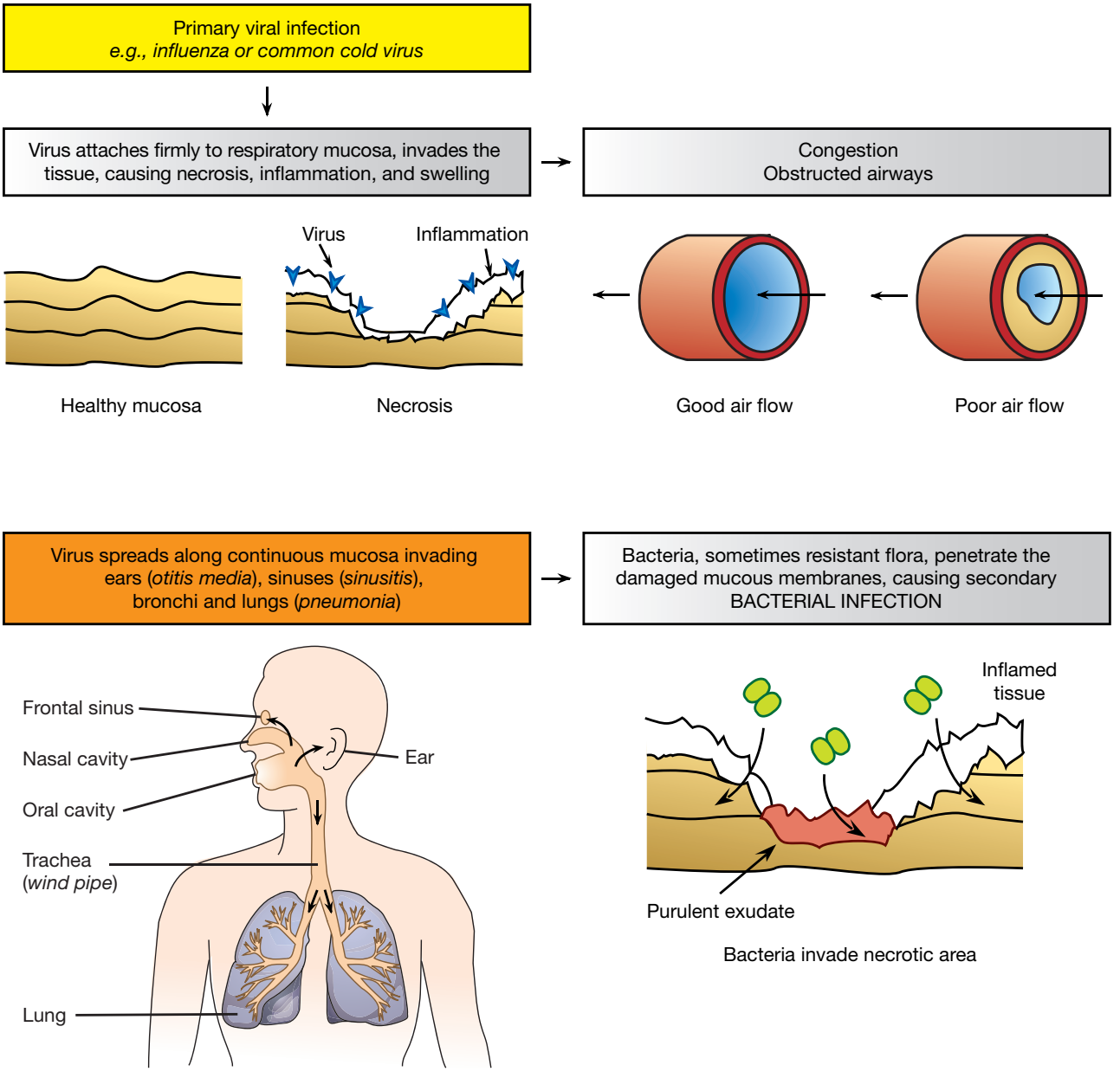

Figure 5-13

Complications of viral respiratory infections.

toms), decongestants, and antibiotics (only if a bacterial infection is present). Humidifiers can liquefy secretions to aid in expectoration. The benefit of vitamin C in prevention and treatment remains controversial. Proper hand washing remains the long-standing cornerstone of prevention. Other measures will limit the spread of active infections to others, including:

- 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. Other causative agents include bacteria and fungi. 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**). As exudate accumulates, pressure builds in the sinus cavity, which causes facial bone pain. Other clinical manifestations of sinusitis may already be present, such as nasal congestion, fever, and sore throat. Diagnostic procedures for sinusitis include a history, physical

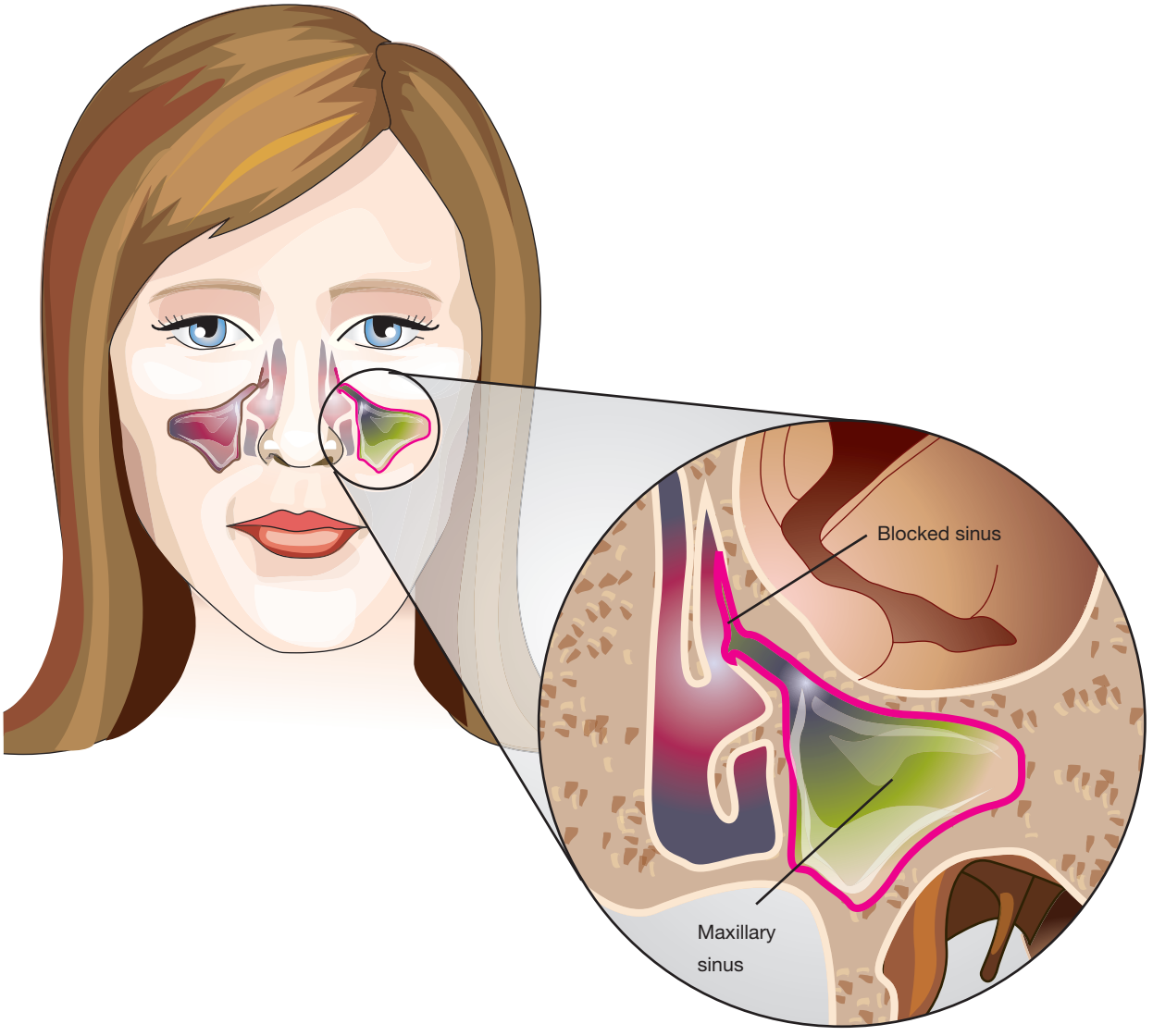

Figure 5-14

Blocked sinus.

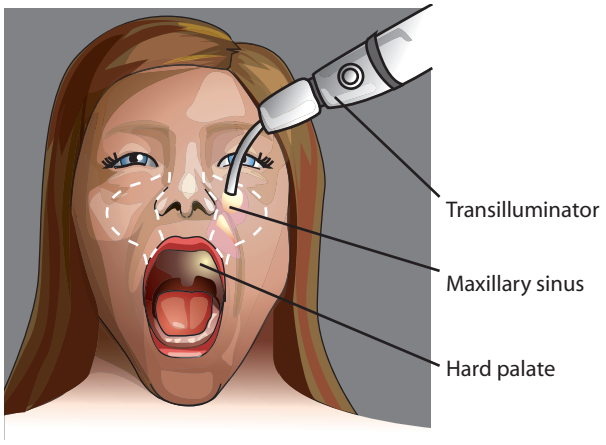

Figure 5-15

Transillumination of the sinuses.

examination, sinus X-ray, and transillumination (**Figure 5-15**). Treatment usually includes decongestants and analgesics until the sinuses begin draining. Bacterial infections require antibiotic therapy to resolve.

Epiglottitis

Epiglottitis is a life-threatening condition of the epiglottis, the protective cartilage lid covering the trachea opening. Haemophilus influenza type B (Hib) is the most common cause. Hib is a routine infection in children 3–7 years of age, especially those children in daycare centers. 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 Hib invasion triggers the inflammatory response, causing the epiglottis to quickly swell and block the air entering the trachea, leading to respiratory failure. Hib can also travel to the bloodstream, leading to sepsis, which is also life-threatening because it activates a massive immune response.

The onset of clinical manifestations is typically rapid and includes:

- Fever
- Sore throat
- Difficulty swallowing
- Drooling with mouth open
- Inspiratory stridor (harsh, high-pitched sound made as result of air turbulence)
- Respiratory distress
- Central cyanosis (blue discoloration of the mouth and lips)
- Anxiety (a result of hypoxia)
- Pallor
- Assuming a sitting position (subconscious attempt to facilitate breathing)

If epiglottitis is suspected, maintaining airway and stabilizing respiratory status is a priority before diagnostic procedures are performed. Efforts to preserve respiratory function include oxygen therapy (likely via mask), endotracheal intubation with mechanical ventilation, and a tracheotomy. 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 are available for prevention and should be administered to children, the elderly, and immune-compromised persons. 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, or overuse. 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.

Clinical manifestations of laryngitis usually last less than a week and include:

- Hoarseness
- Weak voice or voice loss
- Tickling sensation and raw feeling in the throat
- Sore, dry throat
- Dry cough
- 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. Strategies aim to increase comfort or decrease the duration. These strategies include:

- Warm humidity
- Resting the voice
- Increasing fluid intake
- Treating the underlying cause (e.g., infection or gastric reflux)
- 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 1–2 years of age. Other children and adults may also contract it. Routine causative agents include parainfluenza viruses and adenoviruses. 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 of croup include:

- Nasal congestion
- Seallike barking cough (because of laryngeal swelling)
- Hoarseness
- Inspiratory stridor
- Dyspnea
- Anxiety
- Cyanosis

Diagnostic procedures for croup consist of a history, physical examination, X-rays (throat and chest), throat cultures, ABGs, and CBC. Croup is usually self-limiting but can be life threatening without supportive therapy. Treatment strategies include cool humidity, corticosteroids, and bronchodilators.

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, coronavirus, and adenovirus). Bacterial invasions, irritant inhalation (e.g., smoke, chlorine, and bromine), and allergic reactions are less frequent causes. Young children, the elderly, and smokers are at the highest risk for developing acute bronchitis. In acute bronchitis, the airways become irritated and narrowed due to the results of the inflammatory process (e.g., capillary dilatation, edema, and exudate).

Clinical manifestations of acute bronchitis are usually mild and include:

- Productive and nonproductive cough
- Dyspnea
- Wheezing
- Low-grade fever
- Pharyngitis
- Malaise
- Chest discomfort

Diagnosis of acute bronchitis is usually based on symptoms. Additionally, a CBC and chest X-ray may be performed for differential diagnosis. A throat X-ray reveals a narrowing of the trachea often referred to as the steeple sign (**Figure 5-16**). Acute bronchitis is generally self-limiting; therefore, treatment is often supportive. Pharmacologic treatment may include antipyretics, analgesics, antihistamines, decongestants, cough suppressants, and bronchodilators. 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. There are three types—A, B, and C. The 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, and 1968. 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 it produces is generally milder than that caused by type A. **Type C influenza** causes sporadic

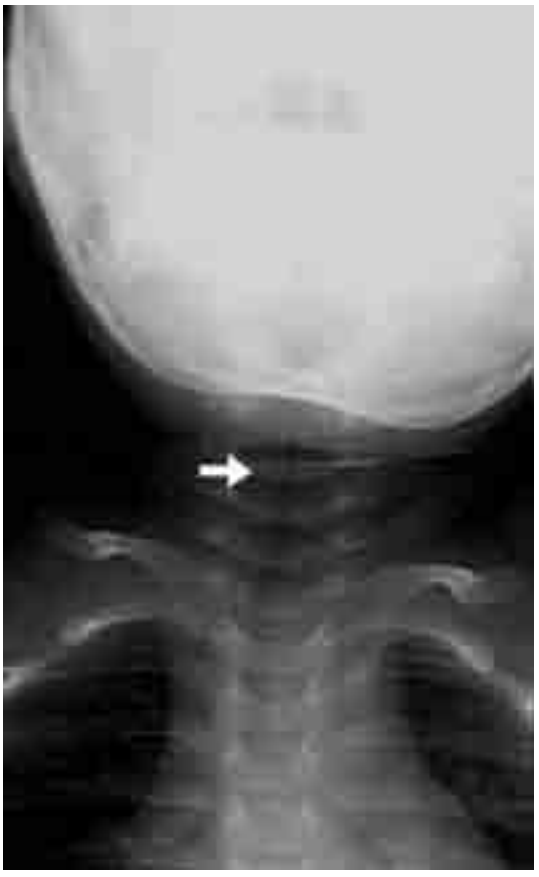

Figure 5-16

Steeple sign.

cases and minor, local outbreaks. Type C has never been connected with a large epidemic.

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. Although many people recover from the flu, it does account for about 200,000 hospitalizations and about 36,000 deaths per year (CDC, 2008). Persons at risk for having negative outcomes because of the flu are children, elderly, those who are immune compromised, and those 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 6 days before onset of symptoms. 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. Clinical manifestations of the flu include:

- Fever
- Headache
- Chills
- Dry cough
- Body aches
- Nasal congestion
- Sore throat
- Sweating
- Malaise

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 postexposure to decrease the likelihood of developing the flu. Other strategies include increasing fluids, rest, antipyretics, and analgesics. Prevention strategies involve those to prevent the common cold (e.g., hand washing and avoiding crowds) and vaccina-

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. An additional factor fueling this myth is that people may still have the flu even after receiving the vaccination. This infection is not because of the flu vaccine; rather, it is because they 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!

tions. Currently, vaccinations exist for the seasonal flu and H1N1 flu. Prior to each flu season (usually before the previous season is over), the Centers for Disease Control and Prevention (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. When outbreaks of other types of the flu occur, like with the H1N1 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 those persons with egg allergies.

Lower Respiratory Tract Infections

Bronchiolitis

Bronchiolitis is a common viral infection of the bronchioles most frequently caused by the respiratory syncytial virus. The infection most often occurs in children under 1 year of age, and incidence increases in the fall and winter months. 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 airflow into the alveoli. Transmission of the respiratory syncytial virus occurs through contact with or inhalation of infected respiratory droplets. Contributing factors to developing bronchiolitis include neonatal prematurity, asthma family history, and cigarette smoke exposure.

Clinical manifestations of bronchiolitis vary in severity and include:

- Nasal drainage
- Nasal congestion
- Cough
- Wheezing
- Rapid, shallow respirations
- Chest retractions
- Dyspnea
- Fever
- Tachycardia
- Malaise

Diagnostic procedures include a history, physical examination, chest X-ray, mucous 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, increased fluids (either by mouth or intravenously), keeping the child calm, bronchodilators, and corticosteroids. Prevention strategies are the same as those previously discussed for other infectious respiratory conditions (e.g., hand washing and avoiding crowds).

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). The sixth leading cause of death in the United States, pneumonia can be a primary or secondary infection (CDC, 2008). *Streptococcus pneumoniae* is responsible for 75% of all cases of pneumonia. **Viral pneumonia** and **bacterial pneumonia** have some notable differences (**Table 5-2**). In contrast to bacterial pneumonia, viral pneumonia is usually mild and heals without intervention, but viral pneumonia can lead to a virulent bacterial pneumonia. Irritating agents or events can also lead to pneumonia. Some of these agents or events include aspiration of gastric contents, endotracheal intubation, respiratory 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

Table 5-2 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

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 that can lead to atelectasis and pneumonia. Tube-feeding formulas also contain sugar and protein, creating a superior medium for bacteria to 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 5-3**). **Lobar pneumonia** is confined to a single lobe and is described by that affected lobe (e.g., right upper lobe). **Bronchopneumonia** is the most frequent type and is a patchy pneumonia throughout several lobes. **Interstitial pneumonia**, or atypical, 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*). 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. In contrast, **community-acquired pneumonia** is acquired outside the hospital or healthcare setting.

SPECIAL CASES

Legionnaires' disease is a specific type of pneumonia that is caused by *Legionella pneumophila*. The bacteria thrive in warm, moist environments, particularly air conditioning systems and spas. Legionnaires' disease is not contagious. Most people acquire this type of pneumonia from inhaling the bacteria as they are spread by an air conditioning system or spa. Those persons with a weakened immune system are at highest risk for developing legionnaires' disease. Although most people with legionnaires' disease recover without incident, this type of pneumonia can be fatal if untreated. Symptoms are similar to other types of pneumonia and usually appear 10–14 days postexposure. 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.

In addition to previously discussed risk factors, persons at risk for developing pneumonia include children, the elderly, those with immune-compromised states, those with existing chronic disease conditions, smokers, and alcoholics. Otherwise healthy patients

Table 5-3 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

SPECIAL CASES

***Pneumocystis carinii* pneumonia** is a specific type of pneumonia that is caused by yeastlike fungus, *Pneumocystosis jiroveci*. This type of pneumonia occurs as an opportunistic infection and can be fatal to the immune compromised (e.g., children or those with AIDS or cancer). Diagnosis is accomplished through identification of the fungus through a sputum culture. Aggressive and early treatment will improve outcomes in these vulnerable 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, and acute respiratory distress syndrome.

Clinical manifestations of pneumonia include:

- Productive or nonproductive cough
- Fatigue
- Pleuritic pain
- Dyspnea
- Fever
- Chills
- Crackles or rales
- Pleural rub
- Tachypnea
- Mental status changes (especially in the elderly)

Early diagnosis and treatment will be 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, 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 and influenza), mobilizing secretions (e.g., turning, coughing, deep breathing), and smoking cessation.

Tuberculosis

Once on the decline, **tuberculosis (TB)** is a potentially serious infectious disease that is increasing globally (**Figure 5-17**). Significant advances have been made in TB treatment, yet many new cases are detected each year, particularly among AIDS patients in Africa. TB remains a major cause of illness and death worldwide, killing more than 2 million people each year. Person-to-person transmission occurs through the inhalation of tiny infected aerosol droplets. 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, increasing treatment concerns and prevalence rates. Fifteen percent of persons infected with TB in the United States have a multidrug-resistant strain (CDC, 2008).

TB is caused by *Mycobacterium tuberculosis*, a slow-growing aerobic (requires oxygen) bacillus that is somewhat resistant to the body's immune efforts. The bacillus is capable of surviving in dried sputum for weeks. Ultraviolet light, heat, alcohol, glutaraldehyde, and formaldehyde destroy the bacillus. Although TB most frequently involves the lungs, it can also affect other organs and tissue (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 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 Chapter 2). 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

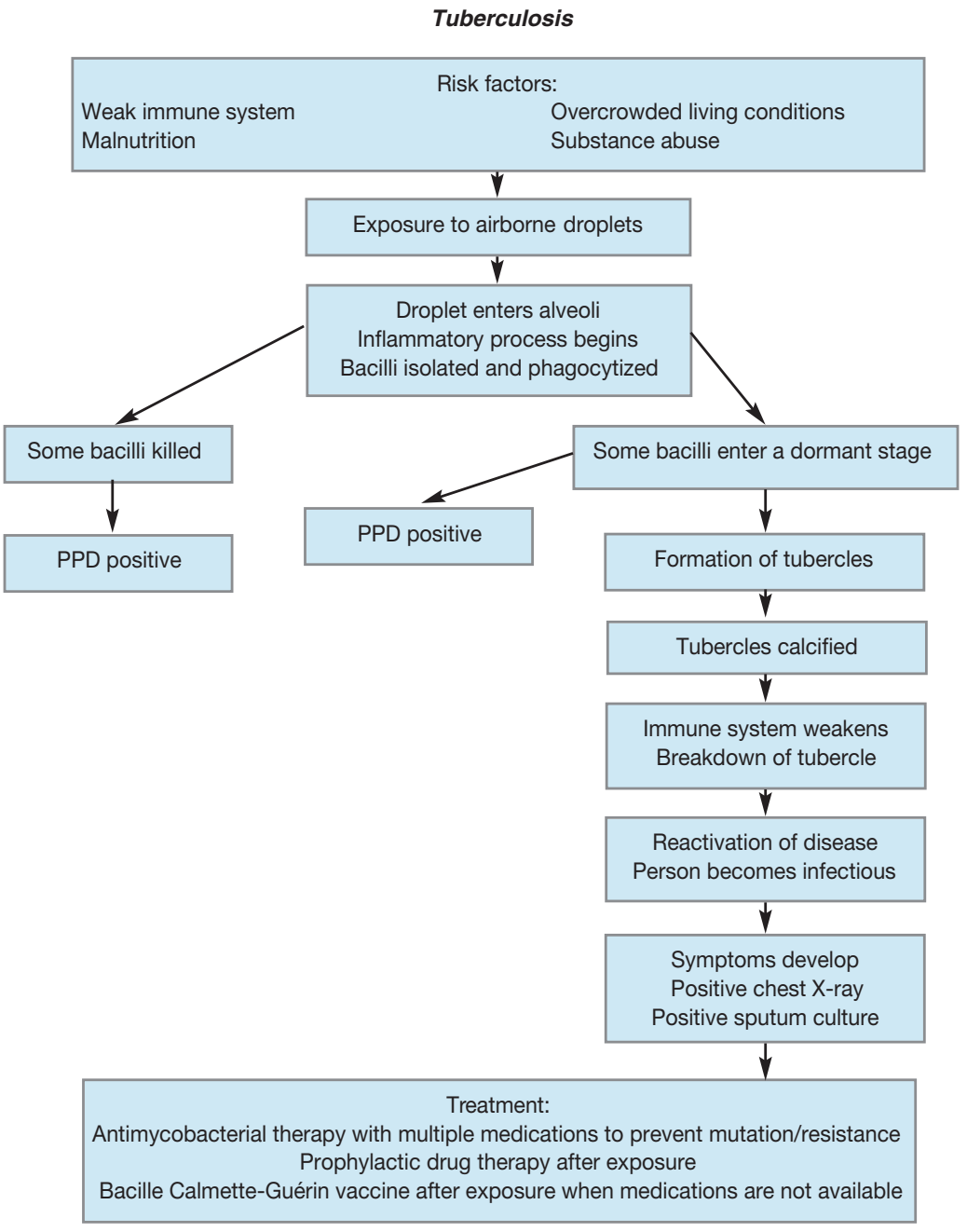

Figure 5-17

Tuberculosis.

(see Chapter 1). An intact immune system can resist this development, so the lesions remain small, become walled off by fibrous tissue, and calcify. These lesions are referred to as Ghon complexes (**Figure 5-18**). 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.

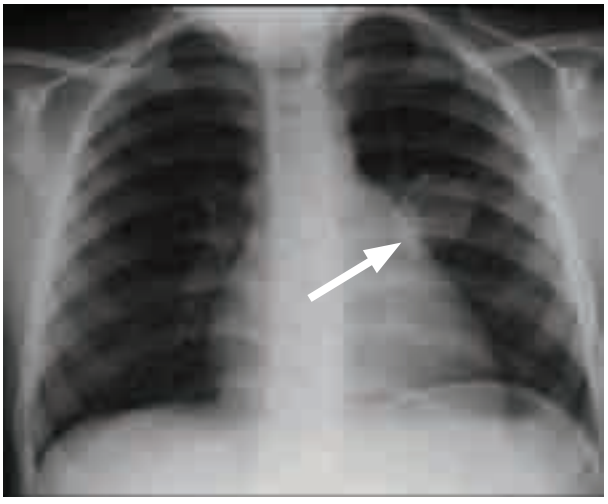

Figure 5-18

Ghon complexes.

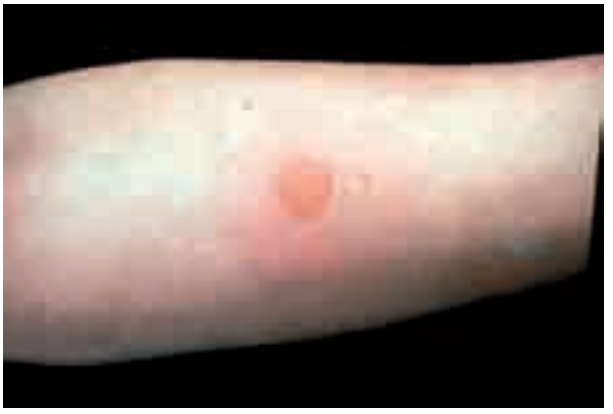

Figure 5-19

Positive TB skin test.

Clinical manifestations begin to appear in the secondary infection phase. These clinical manifestations include:

- Productive cough
- Hemoptysis (coughing up blood or bloody sputum)
- Night sweats
- Fever
- Chills
- Fatigue
- Unexplained weight loss
- 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 the inflammatory response (Figure 5-17). A history of the bacillus Calmette-Guérin (BCG) vaccination will produce a false-positive reaction. Additionally, previously treated TB will generate a false-positive reaction. On the other hand, those 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 are used after a positive TB skin test is noted (whether to confirm an original case or to assess reinfection). A computerized tomography (CT) scan can also be used to visualize TB lesions because it 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 resistant strains. The slow-growing bacilli have a high mutation rate and develop those mutations when

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!

exposed to monotherapy. Because TB is a public health risk, antituberculin medications are provided free of charge by the United States 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 medication side effects (e.g., nausea, paresthesias, and discolored bodily secretions). Patient education, including an emphasis on taking an 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, 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 developing countries).

SPECIAL CASES

Severe acute respiratory syndrome (SARS) is a rapidly spreading respiratory illness that presents similarly to atypical pneumonia. First identified in China, 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 SARS 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. 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.

Obstructive Diseases

Asthma

Asthma is a chronic pulmonary disease that produces intermittent, reversible airway obstruction. Asthma is characterized by acute airway inflammation, bronchoconstriction, bronchospasm, bronchiole edema, and mucus production (**Figure 5-20**). Asthma is the most common chronic illness in children in the United States. Diagnosis, hospitalizations, and death rates associated with asthma have increased from 1996 to 2006 (CDC, 2009). These rate increases may be a result of surging urbanization and pollution.

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 5-4**). **Extrinsic asthma** is a result of increased IgE synthesis and airway inflammation, resulting in mast cell destruction and inflammatory mediator release. Extrinsic triggers include allergens such as food, pollen, dust, and medications. The release of the inflammatory mediators cause 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 activity ends. Symptoms can linger for an hour with exercise-induced 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 rechallenged with vigorous exercise. Athletes often take advantage of this fact by warming up vigorously in order to induce a refractory period prior to competition. **Occupational asthma** is caused by a reaction to substances encountered at work (e.g., plastic or formaldehyde). Symptoms

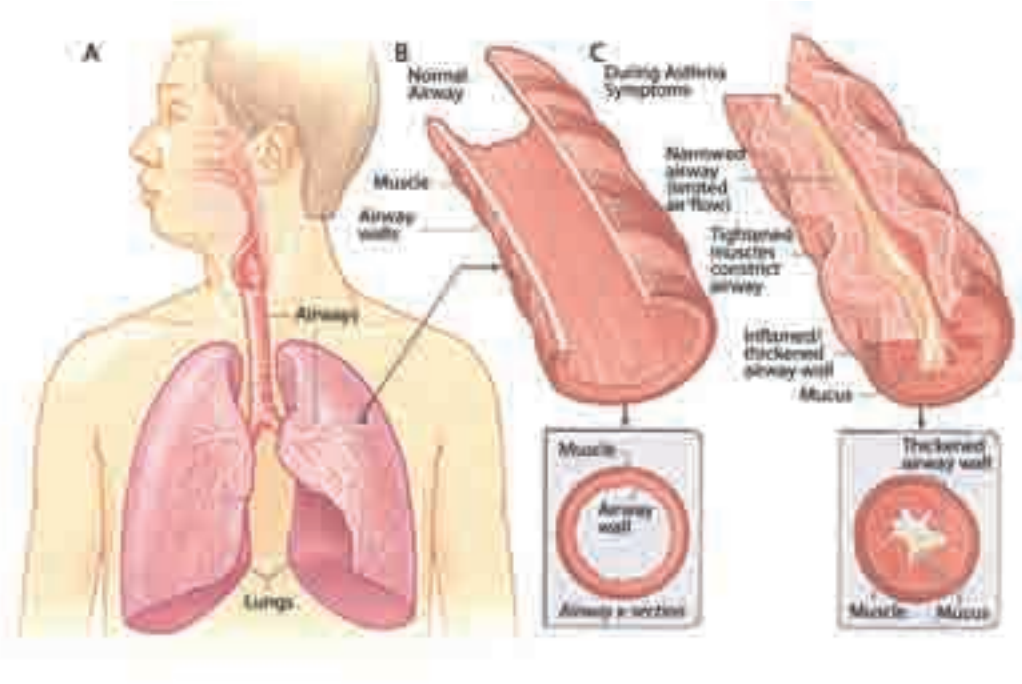

Figure 5-20

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.

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 classification, asthma attacks are the body's response to bronchial inflammation. Stage one of an acute asthma attack is primarily related to bronchospasms, and it is usually signaled by cough-

Table 5-4 Classification of Asthma Severity

Step/Classification*	Daytime Symptoms	Nighttime Symptoms	PEF or FEV_1†	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); FEV_1 = forced expiratory volume in 1 second.
Source: National Heart, Lung and Blood Institute (www.nhlbi.nih.gov).

ing. Peaking within 15 to 30 minutes, inflammatory mediators responsible for this stage include leukotrienes, histamine, and some interleukins. Stage two 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:

- 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, 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 track progression. These diagnostic procedures include a history, physical examination, pulmonary function tests (Figure 5-11), ABGs, CBC, challenge testing, and allergen testing.

Asthma cannot be cured, but 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:

- Develop an asthma plan (**Figure 5-21**) and teach it to all caregivers
- Avoid triggers
- Keep 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 Chapter 4). The most significant contributing factor to developing COPD is cigarette smoking. Other contributing factors include the inhalation of pollution and chemical irritants. Prevalence rates are likely underestimated because COPD is often asymptomatic in early stages or masked by smoking symptoms. 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 in the upcoming sections.

Chronic Bronchitis

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

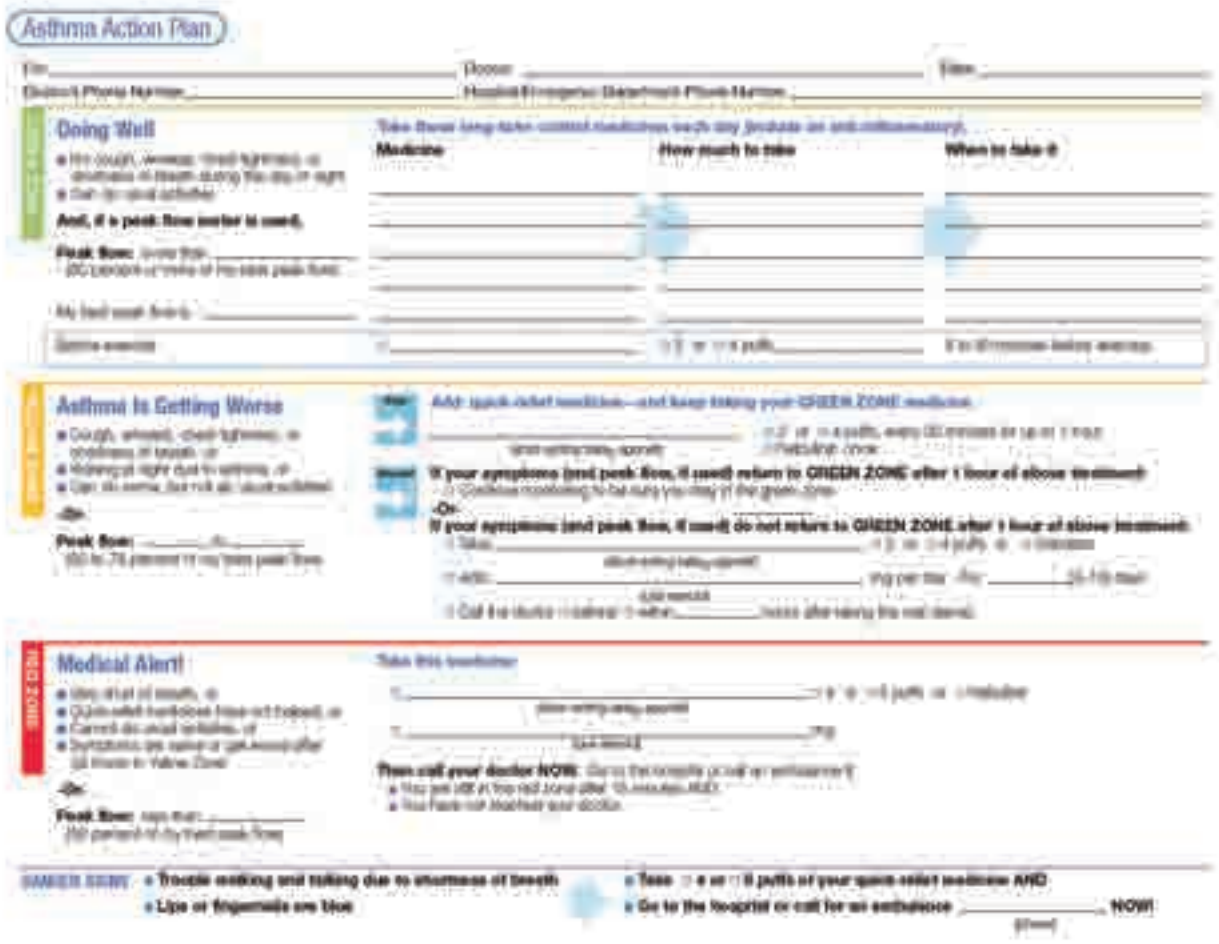

Figure 5-21

Example of asthma action plan.

clubbing of fingers, and dyspnea at rest. Additional clinical manifestations include:

- Wheezing
- Edema
- Weight gain
- Malaise
- Chest pain
- Fever

Diagnosis 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.

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, the **blue bloaters**.

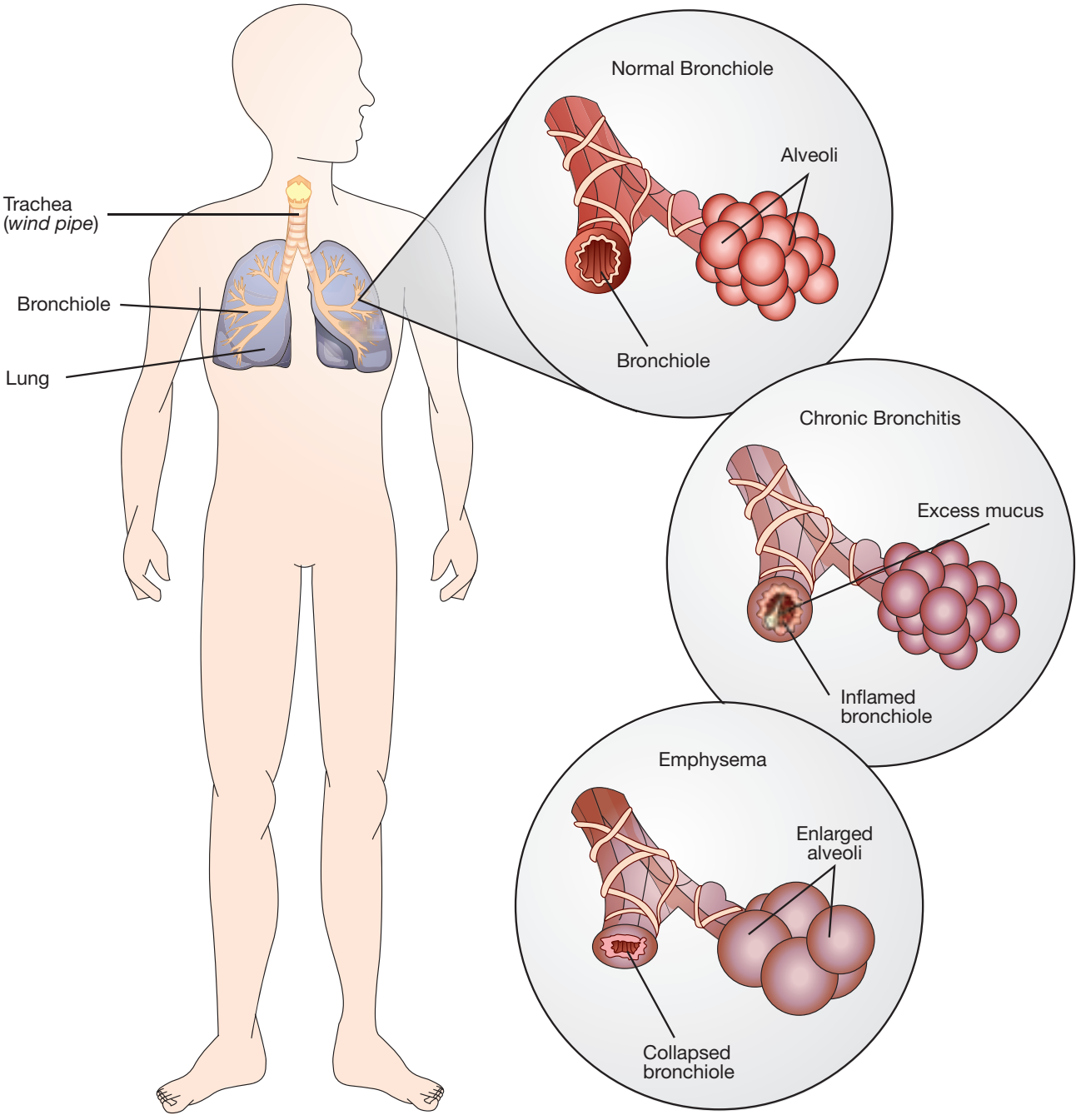

Figure 5-22

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

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 may result from genetic predisposition (less than 2% of cases) and smoking. Smoking initiates inflammation, causing changes in these enzyme levels leading to structural changes. Emphysema gradually turns the alveoli into large, irregular pockets with gaping holes, limiting 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. Clinical manifestation of emphysema includes:

- 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 as with chronic bronchitis. Treatment strategies include those identified for chronic bronchitis with the addition of pursed-lip breathing. Pursed-lip breathing increases expiratory resistance and produces airway back pressure, preventing alveoli collapse.

LEARNING POINTS

Patients with emphysema often hyperventilate, creating a pink appearance to their skin. This pantinglike breathing pattern coupled with the pink skin has earned emphysema patients the nickname, the **pink puffers**.

Cystic Fibrosis

Cystic fibrosis is a common inherited respiratory disorder (about 1,000 diagnosed per year in the United States) that presents at birth (CDC, 2008). This life-threatening condition causes severe lung damage and nutrition deficits. Cystic fibrosis changes cells that produce mucus, sweat, saliva, and digestive secretions. These normally thin secretions become thick and tenacious. Instead of lubricating the respiratory tract, these secretions occlude airways, ducts, and passageways. The genetic defect has been isolated to the seventh chromosome, and transmission follows an autosomal recessive pattern (see Chapter 1). The genetic deficit is related to a protein involved in chloride cellular transport. The lungs and pancreas are primarily affected, but other organs can be involved.

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 destruction. Bronchiectasis and emphysema-like changes are common as fibrosis and obstructions advance. Ultimately, cor pulmonale (right-sided heart failure) or respiratory failure results. In the digestive tract, the mucus blocks the intestines, producing a meconium ileus in the newborn. Mucus 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. 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 manifestation of cystic fibrosis may appear at birth and progressively worsen throughout the life span. These manifestations include:

- Meconium ileus
- Salty skin
- Steatorrhea (fatty, foul-smelling stools)
- Fat-soluble vitamin deficiency (vitamins A, D, E, and K)
- Chronic cough
- Frequent respiratory infections

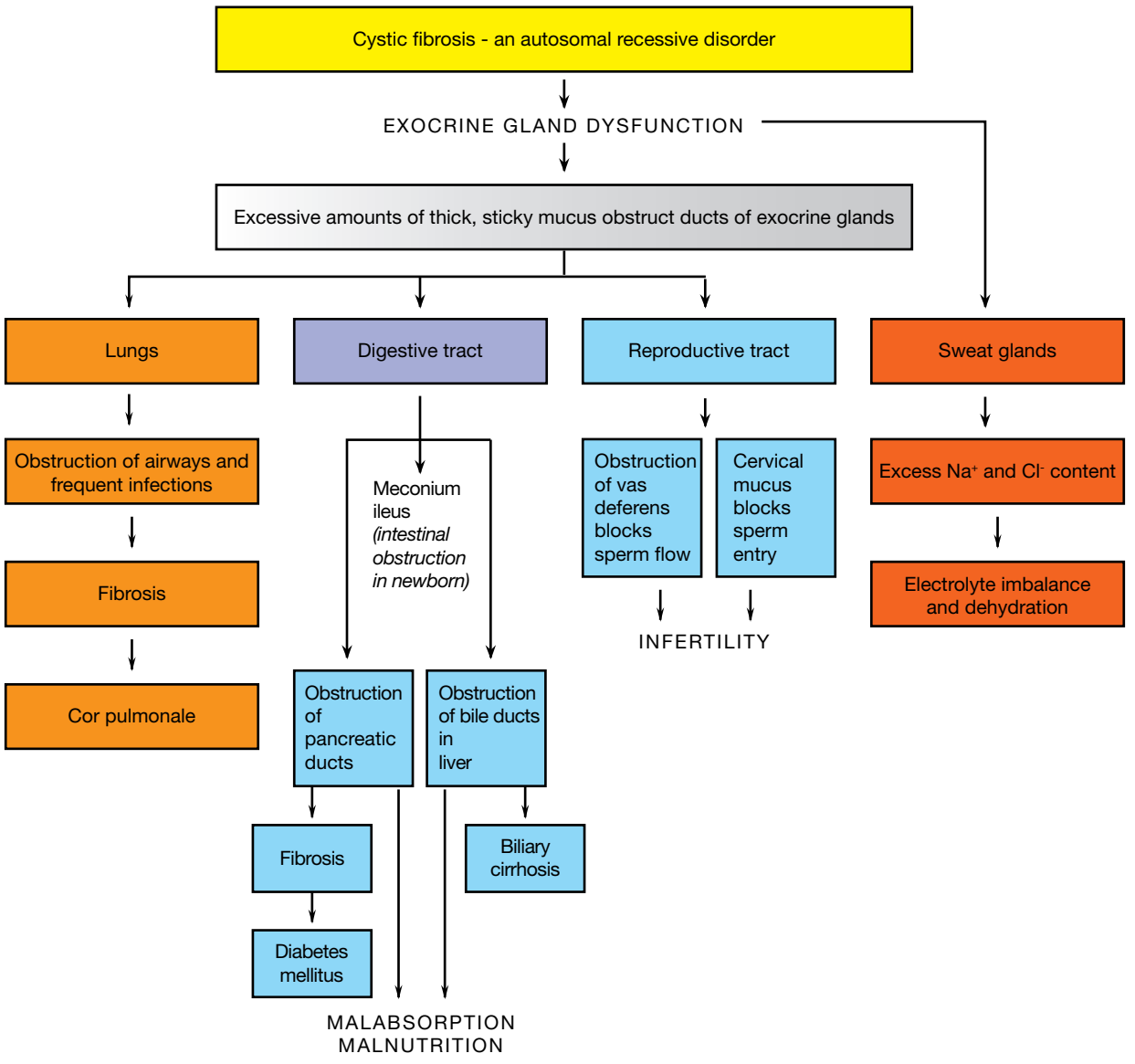

Figure 5-23

Cystic fibrosis.

- Hypoxia
- Fatigue
- Activity intolerance
- Audible rhonchi
- Delayed growth and development

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. 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. With advances in treatment, the life expectancy of children with cystic fibrosis extends into adulthood. Treatment strategies include:

- Pancreatic enzyme replacement
- Bile salt replacement
- A well-balanced, high-protein, low-fat diet
- Fat-soluble vitamin replacement

- Increased fluid intake
- Intensive chest physiotherapy
- Postural drainage
- Coughing exercises
- Humidified air
- Bronchodilators
- Regular, moderate exercise
- Early, aggressive treatment of infections with antibiotics
- Oxygen therapy
- Heart-lung transplant

Lung Cancer

Lung cancer is the third most common neoplasm that can arise as a primary and secondary tumor (approximately 180,000 new cases per year) (CDC, 2008). Frequently, other cancers, such as breast, liver, and lung, to name a few, metastasize (spread) to the lung tissue. Lung cancer is the deadliest of the cancers among men and women—mortality rates are about 90%. Smoking contributes to the majority (80–90%) of 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 one smokes and the number of cigarettes smoked. Second-hand smoke can also be a significant contributing factor, and in fact, some research has indicated that it may be worse than first-hand smoking. Smoking cessation or removing the smoke exposure will gradually decrease risk. Inhalation of other chemicals (e.g., asbestos, tar, and pollution) and chronic lung disease can also increase risk (**Figure 5-24**).

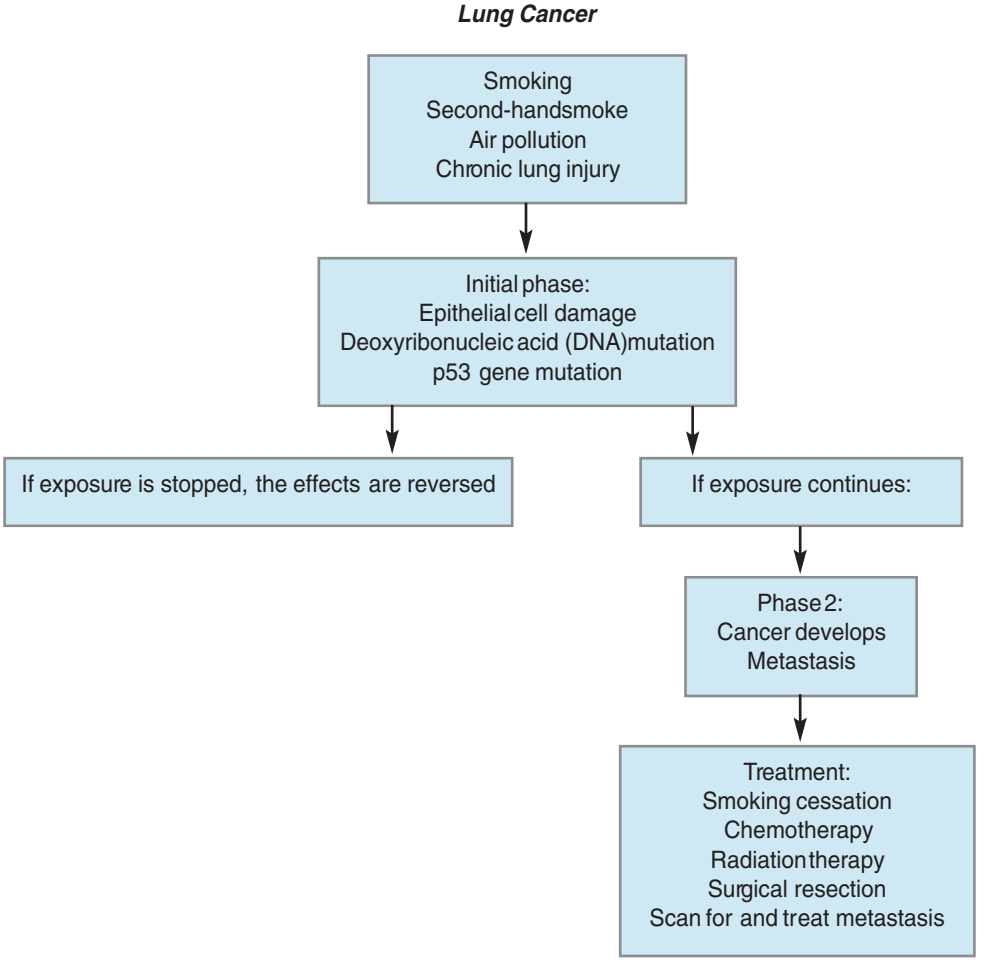

Figure 5-24

Lung cancer.

The lungs provide an optimum environment for tumor development and growth. Carcinogens can seek refuge in the many air passages, having an opportunity to cause cellular changes (usually metaplasia). 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.

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. This very aggressive lung cancer has several subgroups—squamous cell carcinoma, adenocarcinoma, and bronchioalveolar carcinoma. Upon exposure to the carcinogen, irreversible oncogene deoxyribonucleic acid 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. These manifestations include:

- Persistent cough or a change in usual cough
- Dyspnea
- Hemoptysis
- Frequent respiratory infections
- Chest pain
- Hoarseness
- Weight loss
- Anemia
- Fatigue
- Other symptoms specific to site of metastasis

Diagnostic procedures of lung cancer include a history, physical examination, chest X-ray, CT, MRI, bronchoscopy, sputum studies, biopsy, positron emission tomography, bone scans, and pulmonary function tests. Treatment is based on staging and follows usual cancer treatment—chemotherapy, surgery, and radia-

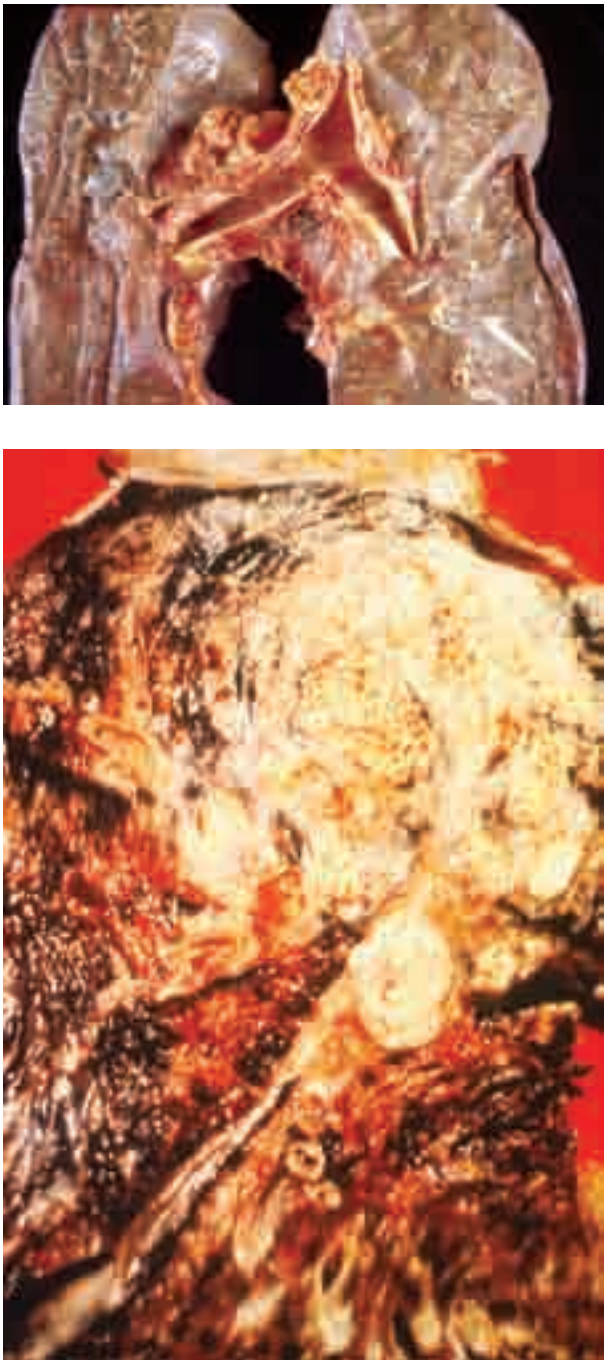

Figure 5-25

The normal (top) and cancerous (bottom) lung.

Table 5-5 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 5-6 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

tion (**Table 5-5; Table 5-6**). 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 optimum respiratory function—oxygen therapy, bronchodilators, and antibiotics (if bacterial infections are present).

Restrictive Diseases

Atelectasis

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

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

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

Clinical manifestations of atelectasis are due to impaired ventilation and perfusion. These manifestations include:

- 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, 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 of performing these interventions adequately.

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 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 pneumothorax. Large

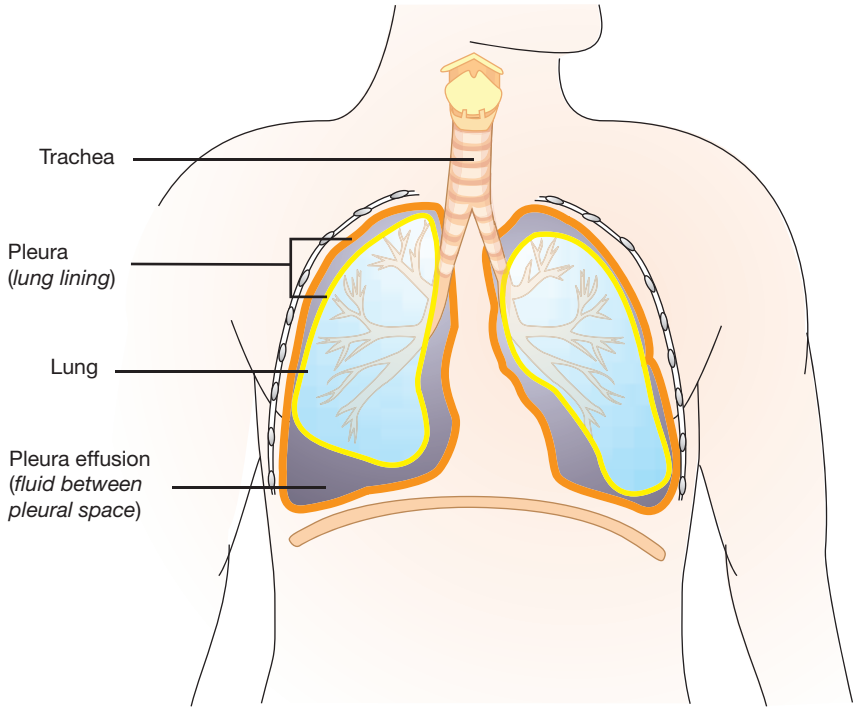

Figure 5-26

Pleural effusion is a buildup of fluid in the lining of the lungs.

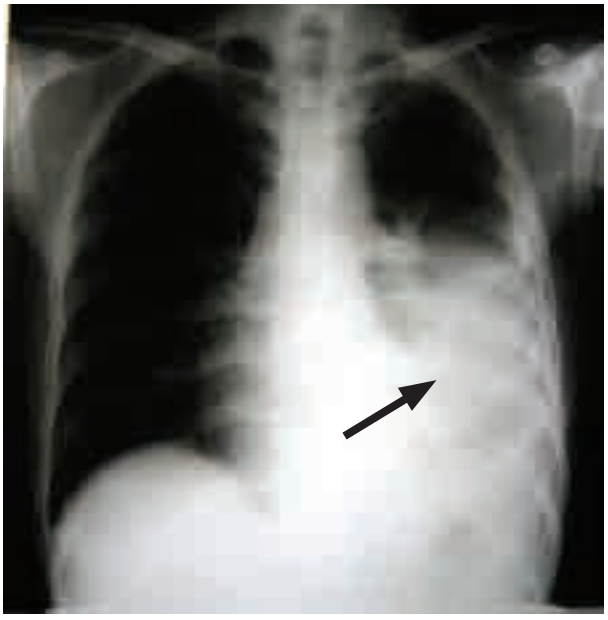

Figure 5-27

X-ray of pleural effusion.

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:

- Dyspnea
- Chest pain (usually sharp and worsening with inhalation)
- Tachypnea
- Tracheal deviation (toward the unaffected side)
- 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 examination of fluid. Treatment focuses on addressing the underlying cause, but regardless of etiology, removal of the fluid is necessary to promote full expansion of the lungs. Strategies may consist of a thoracentesis, 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 to pleural membranes can lead to atelectasis. The pressure can cause a partial or complete collapse of a lung (**Figure 5-28**). 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 a pneumothorax include smoking, tall stature, and history of lung disease or previous pneumothorax.

There are several types of pneumothorax, defined by 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 partial-

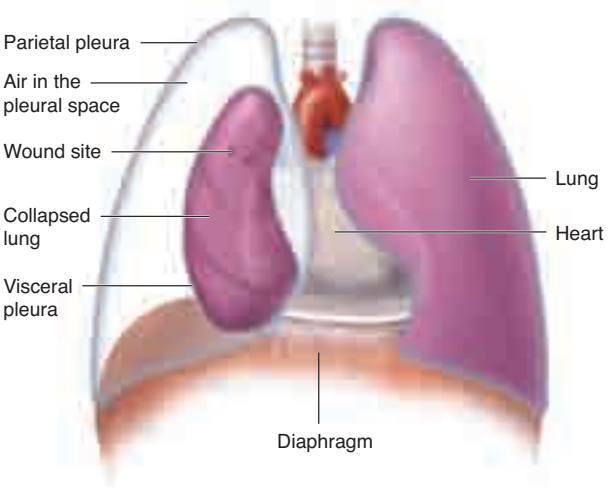

Figure 5-28

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.

ly closed lips forces the smoke deeper into the lungs. Most commonly, these blebs rupture for no obvious reason, but 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, and 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 into the pleural space. Additionally, pulmonary disease reduces lung reserves, making any further reduction in lung function more serious. A **traumatic pneumothorax** stems from any blunt (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 tubes 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 is 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 5-29**). 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:

- 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 reestablishing negative pressure, allowing for full expansion of the lungs. Such strategies may include a thoracentesis and chest drainage tube with suction (which removes fluid and reestablishes negative pressure).

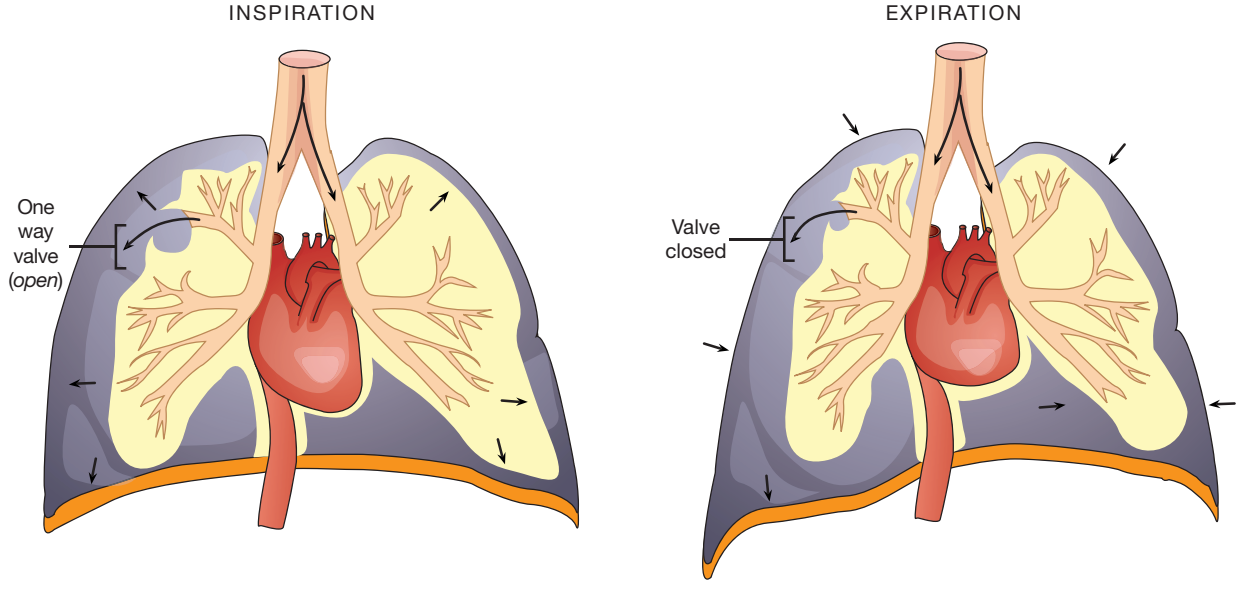

Figure 5-29

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

Acute Respiratory Distress Syndrome

Acute respiratory distress syndrome (ARDS) is a sudden failure of the respiratory system often occurring from fluid accumulation in the alveoli. ARDS has many other names, such as shock lung, wet lung, and stiff lung. Multiple conditions can precipitate ARDS, including prolonged shock, burns, aspiration, and smoke inhalation. ARDS involves an acute hypoxemia resulting from a systemic (e.g., trauma, septicemia, pancreatitis, drug overdose) or pulmonary (e.g., illicit drug and toxic gas inhalation, near drowning, fat embolism) event that is not cardiac in origin. ARDS develops rapidly, often within 90 minutes of a systemic inflammatory response or within 48 hours of a lung

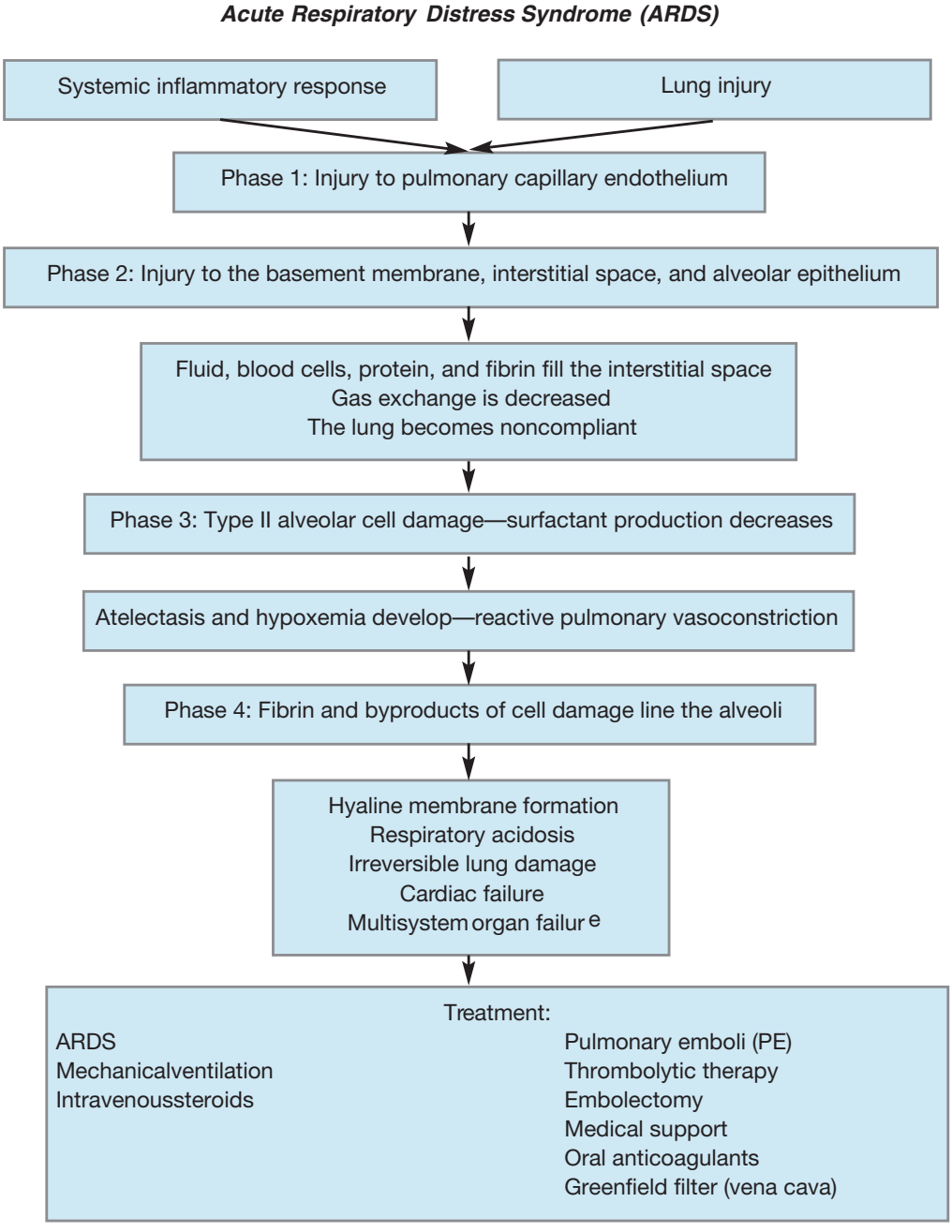

Figure 5-30

Acute respiratory distress syndrome (ARDS).

injury. ARDS is fatal in many cases. Those who survive will fully recover, but it may take up to a year to regain complete lung function.

Injury in the alveoli and the capillary membranes lead to the release of chemical inflammatory mediators (**Figure 5-30; Figure 5-31**). 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. 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 a serious condition that can lead to several complications, including:

- Respiratory failure
- Respiratory and metabolic acidosis
- Pulmonary fibrosis
- Pneumothorax
- Bacterial infections
- Decreased lung function
- Muscle wasting
- Memory, cognitive, and emotional issues

Clinical manifestations of ARDS can develop suddenly and include:

- Dyspnea
- Labored (requiring the use of accessory muscles), shallow respirations
- Rales
- Productive cough with frothy sputum
- Hypoxia
- Cyanosis
- Fever
- Hypotension
- Tachycardia
- Restlessness

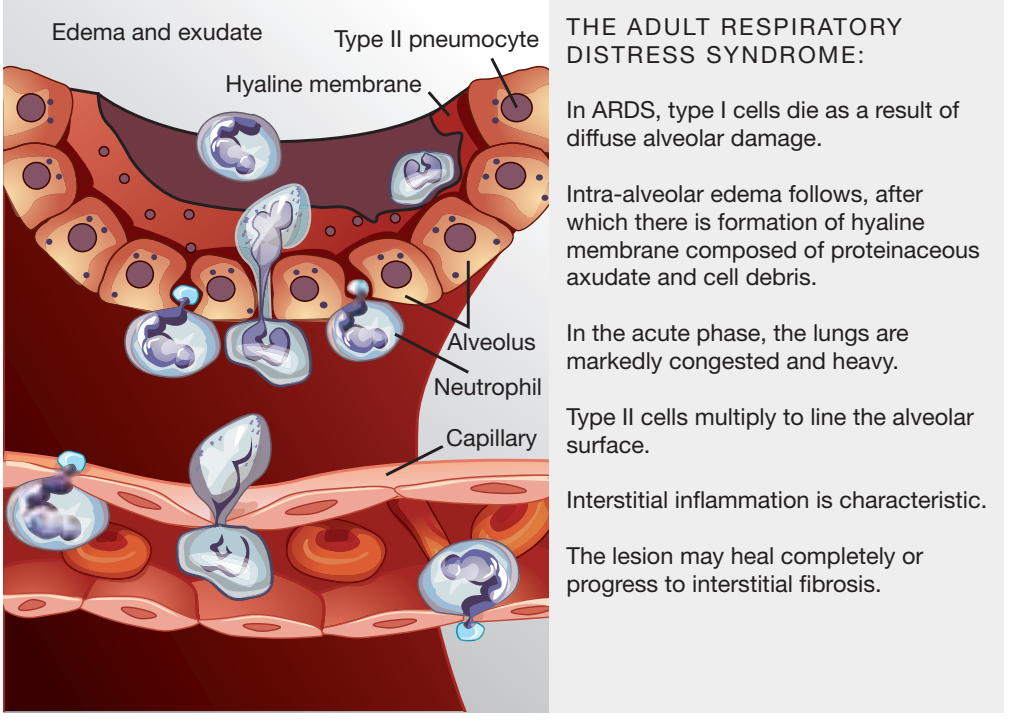

Figure 5-31

Acute respiratory distress syndrome.

- Confusion
- Lethargy
- Anxiety

Diagnostic procedures for ARDS involve 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 mechanical ventilator, oxygen therapy, corticosteroids, and antibiotics (if bacterial infections are present), as well as prevention and treatment of emboli (e.g., embolectomy, anticoagulants, and antiplatelet agents).

Acute Respiratory Failure

Acute respiratory failure (ARF) is a serious, life-threatening condition that can be the result of many pulmonary disorders. 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 80–100 mm Hg and carbon dioxide levels are 35–45 mm Hg. These low oxygen levels are unable to meet the body's metabolic needs, and the nervous system quickly becomes affected. In ARF, these gas levels progressively change as the patient's condition worsens. Respiratory acidosis develops as the carbon dioxide levels rise (see Chapter 6). 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. These manifestations include:

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

Diagnostic procedures for ARF consist of a history, physical examination, ABGs, chest X-ray, and CBC. Treatment focuses on resolving the cause and maintaining adequate respiratory status. Strategies include oxygen therapy, endotracheal intubation with ventilation support, bronchodilators, antibiotics (if bacterial infection is present), corticosteroids, and treating 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).

CASE STUDY

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. She 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, 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 48 $mmHg_2$, 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 to control their negative consequences. Additionally, many of these diseases are preventable; therefore, identifying those at 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.

Case Study Answers

1. Maintaining oxygen therapy and respiratory treatments through the mechanical ventilator and decreasing anxiety to prevent oxygen consumption (e.g., administering sedatives, limiting stimuli, and having consistent caregivers).
2. The viral infection caused damage to the mucosa of the respiratory tract, allowing the opportunity for bacterial invasion. The massive reaction from both the viral and bacterial infections by the immune system led to fluid accumulation in the alveoli and triggered the ARDS cascade.
3. The significance of the lab findings are as follows:
   - The ABG indicates respiratory acidosis (the pH is low, the PaCO is high), which is likely due to decreased gas exchange because of the ARDS.
   - The respiratory acidosis is uncompensated because the HCO_3 is normal (see Chapter 6).
   - The ABG indicates hypoxemia (PaO_2 is low), which is also due to the impaired gas exchange.
   - The CBC finding indicates a significant infection (WBC and neutrophils are high) (see Chapter 3).
4. Though Emma and her family will have a long recovery ahead of them, she will likely survive this condition because of her age, no preexisting conditions, and early intervention.
5. The treatment plan will likely include the following:
   - Continued oxygen and respiratory support
   - Continued intravenous antibiotics
   - Thrombus prevention strategies (e.g., turning, range of motion, anticoagulants, and antiplatelet agents)
   - Emotional support for both child and parents
   - Possible physical therapy depending on the longevity of the condition

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Resources

www.the-abg-site.com

www.cancer.gov

www.cancer.org

www.cdc.gov

www.cff.org

www.lungusa.org

www.medlineplus.gov

www.nih.gov