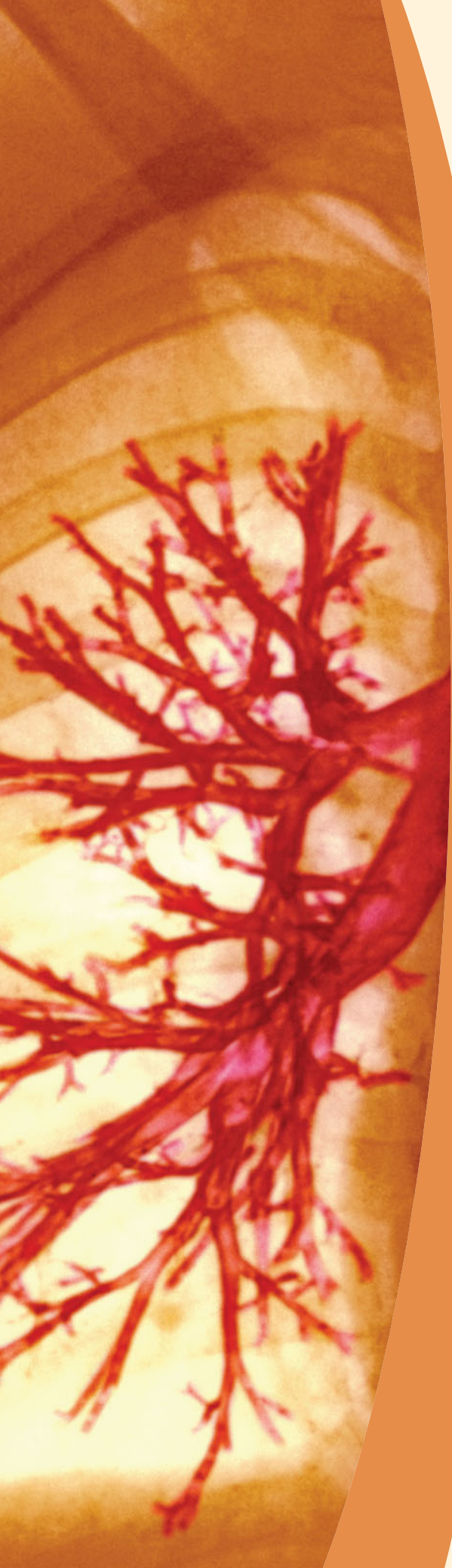
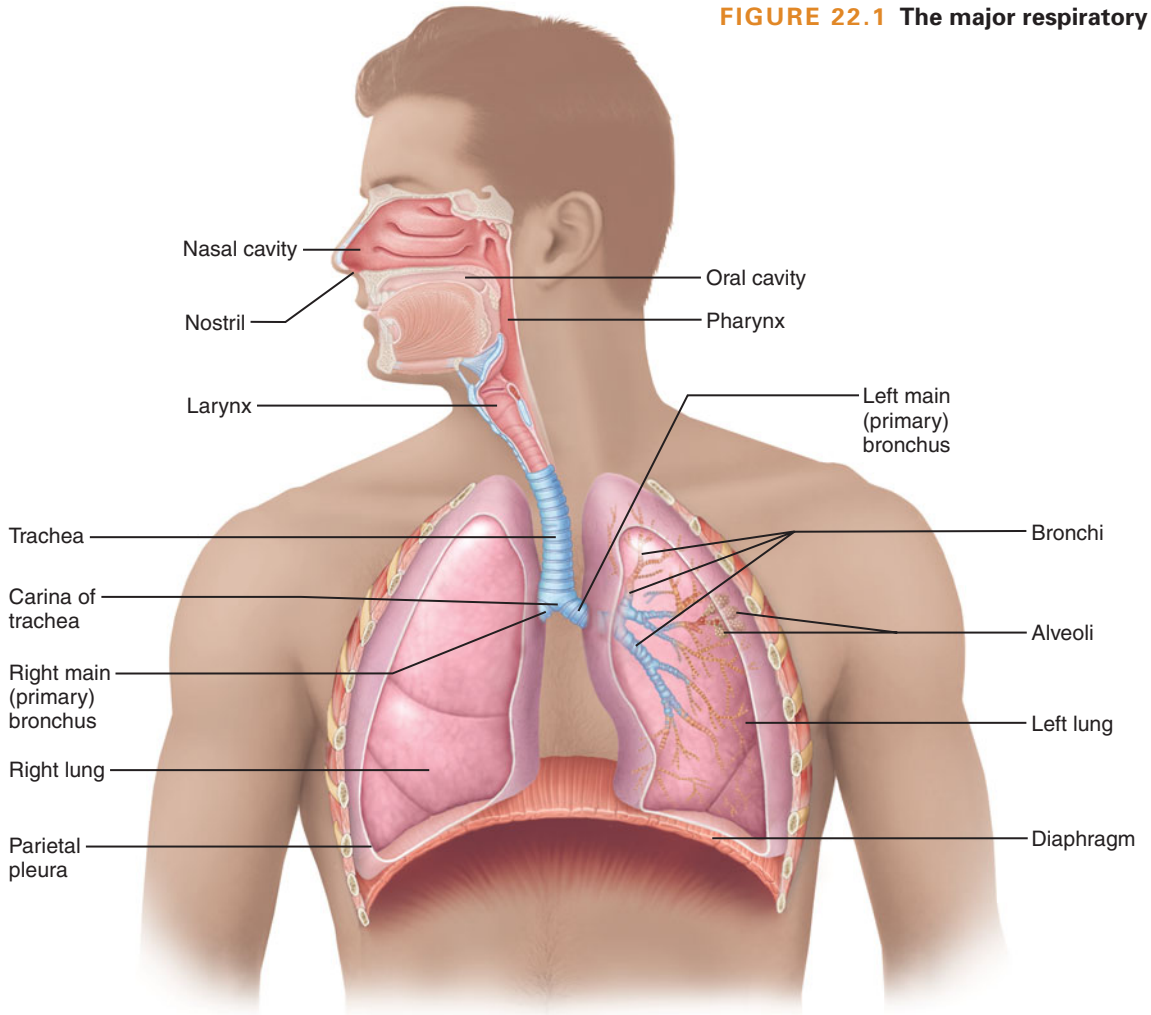


22 The Respiratory System

Humans can live without water for days and without food for weeks, but they cannot live without oxygen for even a few minutes. Breathing is our most urgent need. The trillions of cells in the body need a continuous supply of oxygen to produce the energy needed to carry out their vital functions. Furthermore, as the cells use oxygen, they produce carbon dioxide, a waste product the body must eliminate. The major function of the **respiratory system** is to fulfill these needs—that is, to supply the body with oxygen and dispose of carbon dioxide (CO_2). To accomplish this, the following processes, collectively called **respiration,** must occur:

Bronchial tree of a healthy human lung (colored X ray).

FIGURE 22.1 The major respiratory organs.



1. **Pulmonary ventilation.** Air must be moved into and out of the lungs so that the gases in the air sacs (alveoli) of the lungs are continuously replaced. This movement is commonly called **ventilation,** or breathing.
2. **External respiration.** Gas exchange must occur between the blood and air at the lung alveoli. Oxygen in the air sacs diffuses into the blood; CO_2 in blood diffuses into the air sacs.
3. **Transport of respiratory gases.** Oxygen and carbon dioxide must be transported between the lungs and the cells of the body. This is accomplished by the cardiovascular system, with blood serving as the transporting fluid.
4. **Internal respiration.** At the systemic capillaries, gases must be exchanged between the blood and the tissue cells.

Oxygen is used by the cells and carbon dioxide is produced as a waste product during the chemical process that converts glucose to cellular energy (ATP). This process is called **cellular respiration.** The respiratory processes described above ensure that cellular respiration can occur in virtually all body cells. This chapter focuses on pulmonary ventilation and external respiration, because they alone are the special responsibility of the respiratory system. However, unless gas transport and internal respiration also occur, the respiratory system cannot accomplish its main goal of supplying oxygen to the cells and removing carbon dioxide. Thus, the respiratory and cardiovascular systems are closely coupled, and if either system fails, the body's cells begin to die from oxygen starvation.

Because it moves air into and out of the body, the respiratory system is also involved in the sense of smell and with the vocalizations of speech, which are briefly discussed along with respiration.

FUNCTIONAL ANATOMY OF THE RESPIRATORY SYSTEM

- Identify the respiratory passageways in order, from the nose to the alveoli in the lungs. Distinguish the structures of the conducting zone from those of the respiratory zone.
- List and describe several structures that protect the respiratory system from dust, bacteria, food particles, and other foreign matter.

The organs of the respiratory system include the *nose, nasal cavity,* and *paranasal sinuses;* the *pharynx;* the *larynx;* the *trachea;* the *bronchi* and their smaller branches; and the *lungs,* which contain the terminal air sacs, or *alveoli* **(Figure 22.1)**. Functionally, these respiratory

TABLE 22.1 Principal Organs of the Respiratory System

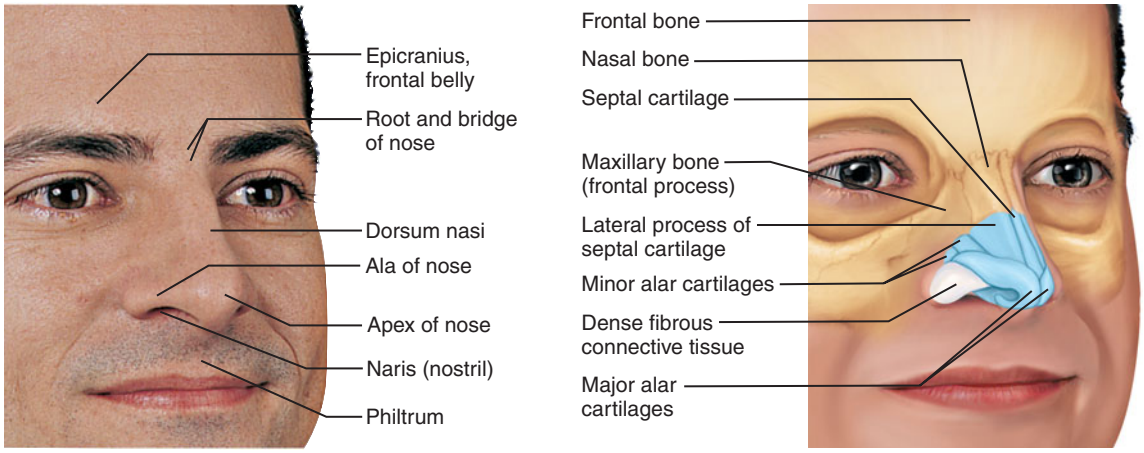
Structure	Description, General and Distinctive Features	Function
Nose	External portion supported by bone and cartilage; internal nasal cavity divided in half by midline nasal septum and lined with respiratory mucosa	Produces mucus; filters, warms, and moistens incoming air; resonance chamber for speech
	Roof of nasal cavity contains olfactory mucosa	Receptors for sense of smell
Paranasal sinuses	Mucosa-lined hollow cavities within the sphenoid, ethmoid, maxillary, and frontal bones	Sinuses function the same as nasal cavity; also lighten skull
Pharynx	Passageway connecting nasal cavity to larynx and oral cavity to esophagus; three subdivisions: nasopharynx, oropharynx, and laryngopharynx	Passageway for air and food
	Houses tonsils	Tonsils respond to inhaled or ingested antigens
Larynx	Connects pharynx to trachea; framework of cartilage and dense connective tissue; opening (rima glottidis) can be closed by epiglottis or vocal folds	Air passageway; prevents food from entering lower respiratory tract
	Houses true vocal cords	Voice production
Trachea	Flexible tube running from larynx and dividing inferiorly into two main (primary) bronchi; walls contain C-shaped cartilages that are incomplete posteriorly where trachealis muscle occurs	Air passageway; filters, warms, and moistens incoming air
Bronchial tree	Consists of right and left main bronchi, which subdivide within the lungs to form lobar (secondary) and segmental (tertiary) bronchi, smaller bronchi, and bronchioles; bronchiolar walls contain complete layer of smooth muscle; constriction of this muscle impedes expiration	Air passageways connecting trachea with alveoli; warms and moistens incoming air
Alveoli	Microscopic chambers at end of bronchial tree; walls of simple squamous epithelium underlain by thin basement membrane; external surfaces intimately associated with pulmonary capillaries	Main sites of gas exchange
	Type II alveolar cells produce surfactant	Surfactant reduces surface tension; helps prevent lung collapse
Lungs	Paired composite organs located within pleural cavities of thorax; composed primarily of alveoli and respiratory passageways; stroma is fibrous elastic connective tissue, allowing lungs to recoil passively during expiration	House passageways smaller than main bronchi
Pleurae	Serous membranes; parietal pleura lines thoracic cavity; visceral pleura covers external lung surfaces	Produce lubricating fluid and compartmentalize lungs

structures are divided into conducting and respiratory zones. The *conducting zone* includes the respiratory passageways that carry air to the sites of gas exchange. The structures of the conducting zone also filter, humidify, and warm the incoming air. Thus, the air reaching the lungs contains much less dust than it did when it entered the nose and is warm and damp. The *respiratory zone,* the actual site of gas exchange in the lungs, is composed of the terminal respiratory passageways that contain alveoli—namely, the *respiratory bronchioles, alveolar ducts,* and *alveolar sacs.* The major organs of the respiratory system are summarized in Table 22.1.

The Nose and the Paranasal Sinuses

The Nose

The **nose** is the only externally visible part of the respiratory system. Although the nose is often referred to in negative connotations, as in "brown-nosing" your instructor or being "nosy," its many important functions make it worthy of higher esteem. The nose (1) provides an airway for respiration, (2) moistens and warms entering air, (3) filters inhaled air to cleanse it of foreign particles, (4) serves as a resonating chamber for speech, and (5) houses the olfactory (smell) receptors.



(a) Surface anatomy **(b) External skeletal framework**

FIGURE 22.2 External nose.

The structures of the nose are divided into the *external nose* and the internal *nasal cavity.* The skeletal framework of the **external nose,** shown in Figure 22.2, consists of the frontal and nasal bones superiorly (forming the root and bridge, respectively), the maxillary bones laterally, and flexible plates of hyaline cartilage inferiorly (the lateral, septal, and alar cartilages). The septal cartilage forms the anterior margin of the nose, called the dorsum nasi. The tip of the nose, its apex, is formed by the major alar cartilages, and the lateral border of the nostril, the ala, is formed from dense fibrous connective tissue. The great variation in nose size and shape is largely due to differences in the nasal cartilages. The skin covering the nose's anterior and lateral surfaces is thin and contains many sebaceous glands that open into some of the largest skin pores on the face.

The Nasal Cavity

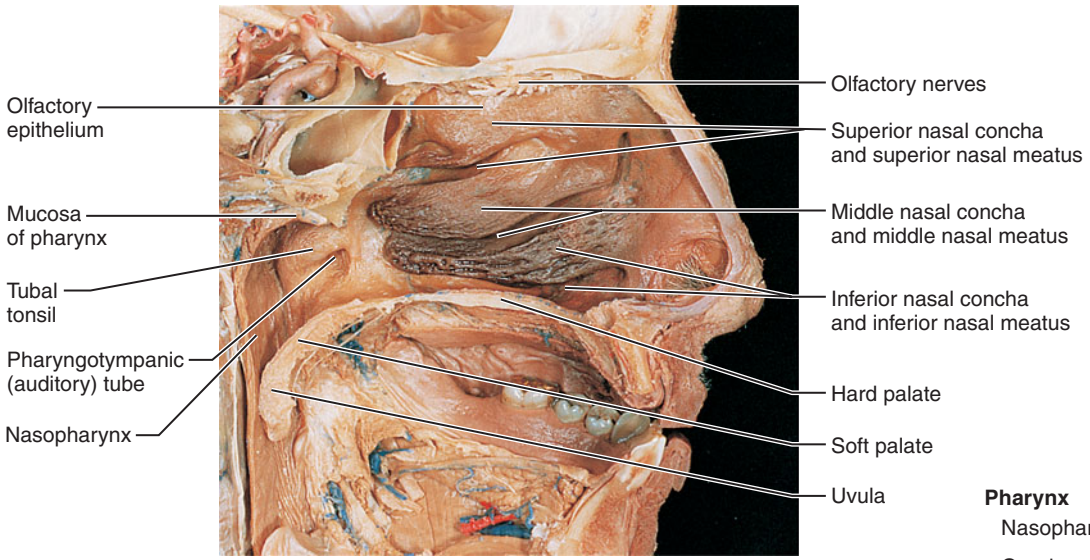
The **nasal cavity** (Figure 22.1 and Figure 22.3) lies in and posterior to the external nose. During breathing, air enters this cavity by passing through the external **nares** (na′rēz), or *nostrils.* The nasal cavity is divided into right and left halves by the *nasal septum* in the midline; this septum is formed by the perpendicular plate of the ethmoid bone, the vomer, and a septal cartilage (see Figure 7.14 on p. 164), all covered by a mucous membrane. Posteriorly, the nasal cavity is continuous with the nasal part of the pharynx (nasopharynx) through the **posterior nasal apertures,** also called the *choanae* (ko-a′ne; "funnels") or *internal nares.*

To review the bony boundaries of the nasal cavity, its roof is formed by the ethmoid and sphenoid bones, and its floor is formed by the *palate* (pal′at), which separates the nasal cavity from the mouth inferiorly and keeps food out of the airways. Anteriorly, where the palate contains the horizontal processes of the palatine bones and the palatine process of the maxillary bone, it is called the **hard palate** (see Figure 7.14, p. 164); the posterior part is the muscular **soft palate** (see Figure 22.3).

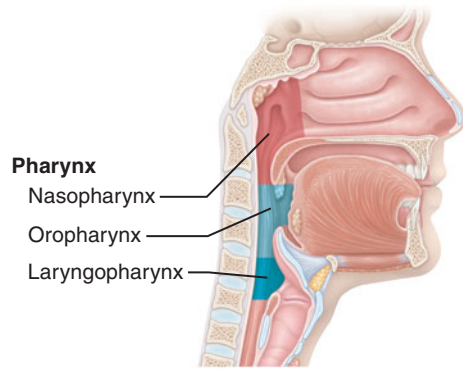
The part of the nasal cavity that lies just superior to the nostrils, within the flared wings of the external nose, is the **vestibule** ("porch, entranceway"). This is lined with skin containing sebaceous and sweat glands and numerous hair follicles. The nose hairs, or *vibrissae* (vi-bris′e) (*vibro* = to quiver), filter large particles, such as insects and lint, from the inspired air. The rest of the nasal cavity is lined with two types of mucous membrane: (1) the small patch of *olfactory mucosa* near the roof of the nasal cavity, which houses the receptors for smell (see pp. 484–485), and (2) the *respiratory mucosa,* a mucous membrane that lines the vast majority of the nasal cavity.

The respiratory mucosa consists of a pseudostratified ciliated columnar epithelium containing scattered goblet cells, and the underlying connective tissue lamina propria. This lamina propria is richly supplied with compound tubuloalveolar glands (see Figure 4.5, p. 74) that contain mucous cells and serous cells. (*Mucous cells* secrete mucus, whereas *serous cells* in glands secrete a watery fluid containing digestive enzymes.) Each day, the nasal glands and the epithelial goblet cells secrete about a quart of mucus containing lysozyme, an enzyme that digests and destroys bacteria. The sticky mucus forms a sheet that covers the surface of the mucosa and traps inhaled dust, bacteria, pollen, viruses, and other debris from the air. Thus, an important function of the respiratory mucosa is to filter the inhaled air. The ciliated cells in the epithelial lining create a gentle current that moves the sheet of contaminated mucus posteriorly to the pharynx, where it is swallowed. In this way, particles filtered from the air are ultimately destroyed by digestive juices in the stomach. In addition, the sheet of mucus is a wet film that moistens the inhaled air.

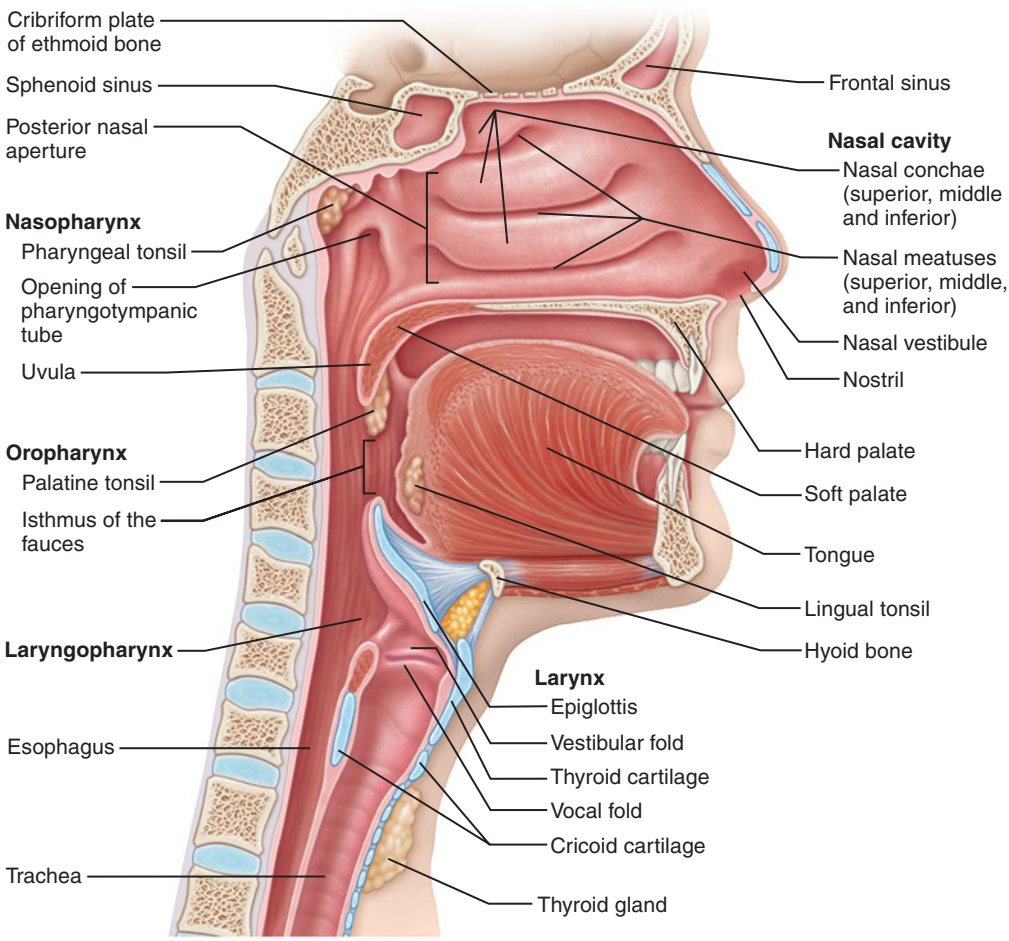
RHINITIS Inflammation of the nasal mucosa, called **rhinitis** (ri-ni′tis; "nose inflammation"), is caused by such things as cold viruses, streptococcal bacteria, and various allergens. This inflammation is accompanied by an excessive production of mucus, which results in nasal congestion, a runny nose, and postnasal drip.



(a) Photograph



(b) Regions of the pharynx



(c) Illustration

FIGURE 22.3 The upper respiratory tract. Midsagittal section of the head and neck. (See *A Brief Atlas of the Human Body,* Second Edition, Figure 47.)

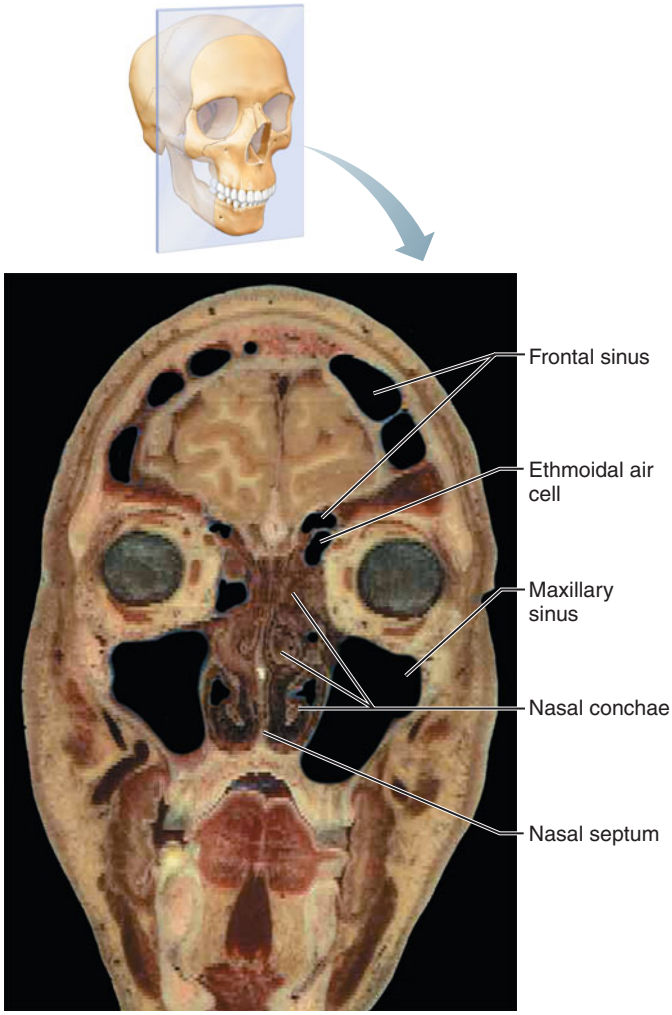


FIGURE 22.4 Nasal conchae and paranasal sinuses.
Frontal section through the face, photograph.

The nasal mucosa is richly supplied with sensory nerve endings. A sneeze reflex is stimulated when irritating particles (dust, pollen, and so on) contact this sensitive mucosa. The resulting sneeze propels air outward in a violent burst, expelling the irritant from the nose.

Rich plexuses of capillaries and thin-walled veins occupy the lamina propria of the nasal mucosa and warm the incoming air that flows across the mucosal surface. When the temperature of inhaled air drops, as when you step outside on a cold day, the vascular plexuses respond by engorging with warm blood, thereby intensifying the air-heating process. Because of the abundance and the superficial location of these vessels, nosebleeds are common and often profuse.

The Nasal Conchae Projecting medially from each lateral wall of the nasal cavity are three mucosa-covered, scroll-like structures: the *superior* and *middle conchae* of the ethmoid bone, and the *inferior concha,* which is a separate bone (**Figure 22.4** and Figure 22.3). The groove inferior to each concha is a *meatus.* As inhaled air rushes over the curved conchae, the resulting turbulence greatly increases the amount of contact between the nasal mucosa and this inspired air. The gases in the inhaled air swirl through the twists and turns of the conchae, but the air's particulate matter is deflected onto the mucus-coated surfaces, where it becomes trapped. As a result, few particles larger than 4 μm get past the nasal cavities.

The conchae and nasal mucosa function during inhalation to filter, heat, and moisten the air. During exhalation, they reclaim this heat and moisture. The inhaled air cools the conchae, and then during exhalation these cooled conchae precipitate moisture and extract heat from the humid air flowing over them. This reclamation mechanism minimizes the amount of moisture and heat lost from the body through breathing, helping people to survive in dry and cold climates.

The Paranasal Sinuses

The nasal cavity is surrounded by a ring of air-filled cavities called *paranasal sinuses* located in the frontal, sphenoid, ethmoid, and maxillary bones (Figure 22.4). Recall from p. 165 that these sinuses open into the nasal cavity, are lined by the same mucosa, and perform the same air-processing functions as does that cavity. Their mucus drains into the nasal cavities, and the suctioning effect caused by nose blowing helps to drain them.

SINUSITIS Inflammation of the paranasal sinuses, a condition called **sinusitis,** is caused by viral, bacterial, or fungal infections. When the passages that connect the paranasal sinuses to the nasal cavity become blocked by swelling of the inflamed nasal mucosa, air in the sinus cavities is absorbed into the blood vessels of the mucosal lining, resulting in a partial vacuum and a sinus headache localized over the inflamed areas. Later, if the infection persists, inflammatory fluid oozes from the mucosa, fills the obstructed sinus, and exerts painful positive pressure. Most cases of sinusitis originate just inferior and lateral to the middle concha, where the openings of the frontal and maxillary sinuses and one of the ethmoidal air cells lie in close proximity and can be blocked simultaneously. Serious cases of sinusitis are treated by promoting drainage and with antibiotics.

check your understanding

1. What respiratory structures are lined with respiratory mucosa? List the functions of this mucous membrane.
2. Differentiate external respiration, internal respiration, and cellular respiration.

For answers, see Appendix B.

The Pharynx

The **pharynx** (far′ingks) is the funnel-shaped passageway that connects the nasal cavity and mouth superiorly to the larynx and esophagus inferiorly (Figure 22.3). It descends from the base of the skull to the level of the sixth cervical vertebra

and serves as a common passageway for both food and air. In the context of the digestive tract, the pharynx is commonly called the *throat.*

On the basis of location and function, the pharynx is divided into (from superior to inferior) the *nasopharynx, oropharynx,* and *laryngopharynx* (lah-ring″go-far′ingks) (Figure 22.3b). The muscular wall of the pharynx consists of skeletal muscle throughout its length, but the nature of the mucosal lining varies among the three pharyngeal regions.

The Nasopharynx

The **nasopharynx** lies directly posterior to the nasal cavity, inferior to the sphenoid bone and superior to the level of the soft palate (Figure 22.3b and c). Because it is superior to the point where food enters the body, the nasopharynx serves only as an air passageway. During swallowing, the soft palate and its pendulous **uvula** (u′vu-lah; "little grape") reflect superiorly, an action that closes off the nasopharynx and prevents food from entering the nasal cavity. When a person giggles, this sealing action fails, and swallowed fluids can spray from the nose.

The nasopharynx is continuous with the nasal cavity through the posterior nasal apertures, and its ciliated pseudostratified epithelium takes over the job of propelling mucus where the nasal mucosa leaves off, such that dusty mucus is moved downward through the nasopharynx. High on the posterior nasopharyngeal wall is the **pharyngeal tonsil,** or *adenoids,* a lymphoid organ that destroys pathogens entering the nasopharynx in the air.

INFECTION OF THE ADENOIDS Infected and enlarged adenoids, a condition especially common in children, can obstruct the flow of air through the nasopharynx. Because the nasal airway is blocked, breathing through the mouth becomes necessary.

A *pharyngotympanic (auditory) tube,* which drains the middle ear, opens into each lateral wall of the nasopharynx. A ridge of pharyngeal mucosa posterior to this opening constitutes the **tubal tonsil** (Figure 22.3a), whose location provides the middle ear some protection against infections that may spread from the pharynx.

The Oropharynx

The **oropharynx** lies posterior to the oral cavity (mouth); its archlike entranceway, directly behind the mouth, is the **fauces** (faw′sēz; "throat") (Figure 22.3b). The oropharynx extends inferiorly from the level of the soft palate to the level of the epiglottis (a flap posterior to the tongue). Both swallowed food and inhaled air pass through the oropharynx.

As the nasopharynx blends into the oropharynx, the lining epithelium changes from pseudostratified columnar to a thick, protective *stratified squamous epithelium.* This structural adaptation reflects the increased friction and greater chemical trauma accompanying the passage of swallowed food through the oropharynx.

Two kinds of tonsils are embedded in the mucosa of the oropharynx: The paired **palatine tonsils** lie in the lateral walls of the fauces, and the **lingual tonsil** covers the posterior surface of the tongue.

The Laryngopharynx

Like the oropharynx superior to it, the **laryngopharynx** serves as a common passageway for food and air and is lined with a stratified squamous epithelium. The laryngopharynx lies directly posterior to the larynx and is continuous with both the esophagus, which conducts food and fluids to the stomach, and the larynx, which conducts air to the respiratory tract.

The Larynx

➤ Describe the structure and functions of the larynx.

The **larynx** (lar′ingks), or voice box, extends from the level of the fourth to the sixth cervical vertebra. Superiorly, it attaches to the hyoid bone and opens into the laryngopharynx (see Figure 22.3c); inferiorly, it is continuous with the trachea (windpipe). The larynx has three functions: (1) producing vocalizations, (2) providing an open airway, and (3) acting as a switching mechanism to route air and food into the proper channels. For the latter purposes, the inlet (superior opening) to the larynx is closed during swallowing and open during breathing.

The framework of the larynx is an intricate arrangement of nine cartilages connected by membranes and ligaments **(Figure 22.5)**. The large, shield-shaped **thyroid cartilage,** which is formed by two cartilage plates, resembles an upright open book, with the book's "spine" lying in the anterior midline of the neck. This "book spine" is the ridgelike **laryngeal** (lah-rin′je-al) **prominence,** which is obvious externally as the **Adam's apple.** The thyroid cartilage is larger in men than in women because male sex hormones stimulate its growth during puberty. Inferior to the thyroid cartilage is the **cricoid cartilage** (kri′koid; "circle"), the only laryngeal cartilage that forms a complete ring. The shape of the cricoid cartilage is likened to a signet ring, a ring historically used to imprint wax seals on letters and official documents. Today, rings of this style are commonly referred to as initial rings. The comparison does accurately describe the cricoid; it is wide posteriorly and narrow anteriorly and is located on top of the trachea.

As shown in Figure 22.5c and d, three pairs of small cartilages lie just superior to the cricoid cartilage in the posterior part of the larynx: the **arytenoid cartilages** (ar″ĭ-te′noid; "ladle-like"), the **corniculate cartilages** (kor-nik′u-lāt; "little horn"), and the **cuneiform cartilages** (ku-ne′ĭ-form; "wedge-shaped"). The most important of these cartilages are the pyramid-shaped arytenoids, which anchor the vocal cords.

The ninth cartilage of the larynx, the leaf-shaped **epiglottis** (ep″ĭ-glot′is), is composed of elastic cartilage and is almost entirely covered by a mucosa. Its stalk attaches

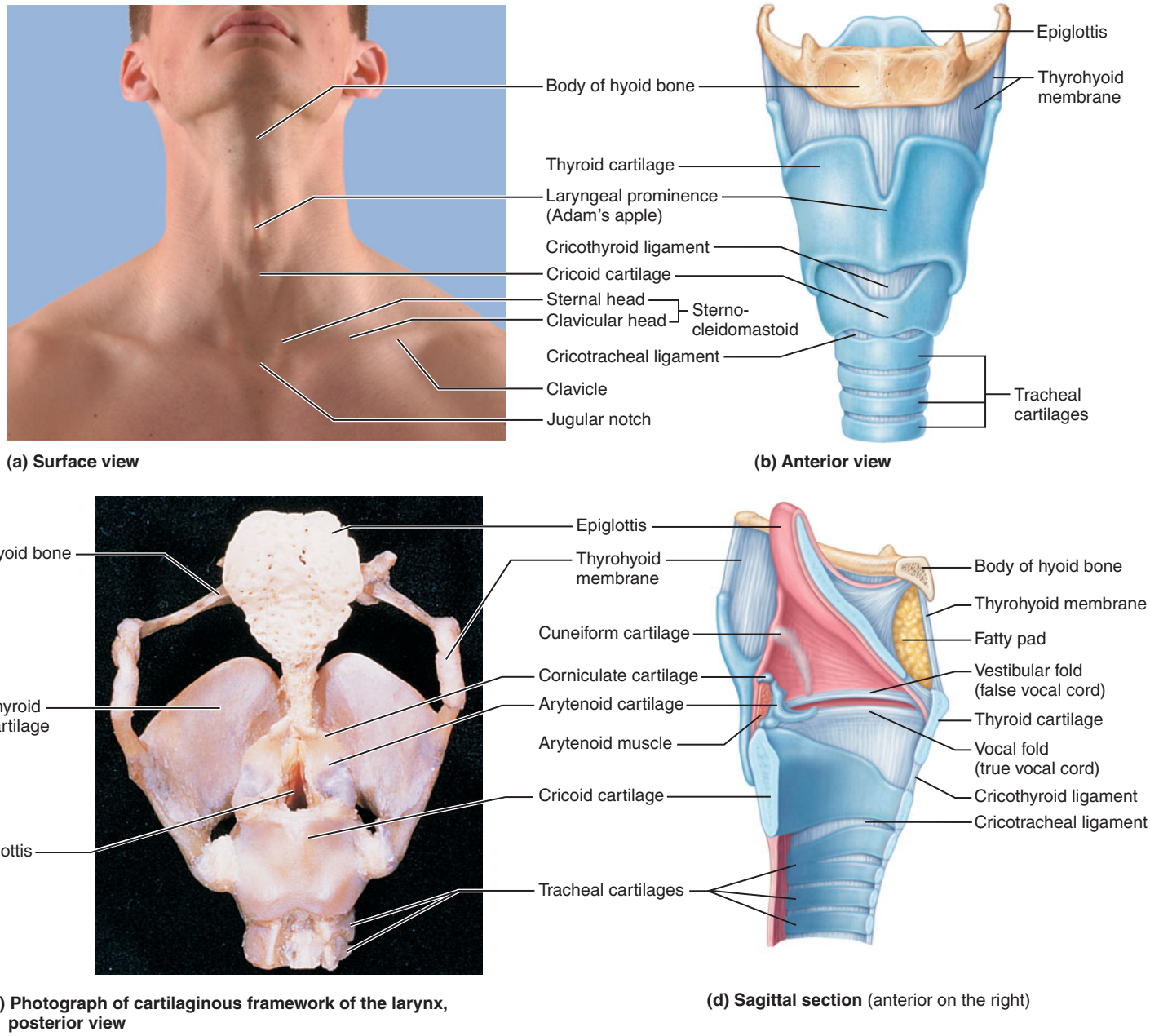


FIGURE 22.5 Anatomy of the larynx.

anteriorly to the internal aspect of the angle of the thyroid cartilage. From there, the epiglottis projects superoposteriorly and attaches to the posterior aspect of the tongue (*epiglottis* means "upon the tongue"). During swallowing, the entire larynx is pulled superiorly, and the epiglottis tips inferiorly to cover and seal the laryngeal inlet. This action keeps food out of the lower respiratory tubes. The entry into the larynx of anything other than air initiates the cough reflex, which expels the substance and prevents it from continuing into the lungs.

Because the larynx lies inferiorly in the neck and must ascend so far during swallowing, it sometimes cannot reach the protective cover of the epiglottal lid before food enters the laryngeal inlet. Although the low position of the larynx makes us more susceptible to choking, it is essential to humans' ability to talk. Its inferior location allows for greater movement of the tongue in shaping sounds and for an exceptionally long pharynx, which acts as a resonating chamber for speech. This latter feature greatly improves the quality of the vowel sounds we produce.

Within the larynx, paired *vocal ligaments* run anteriorly from the arytenoid cartilages to the thyroid cartilage. These ligaments, composed largely of elastic fibers, form the core of a pair of mucosal folds called the **vocal folds** or **(true) vocal cords** (see Figure 22.5d and Figure 22.6). Because the mucosa covering them is avascular, the vocal folds appear pearly white. Air exhaled from the lungs causes these folds to vibrate in a wave motion and to clap together, producing the

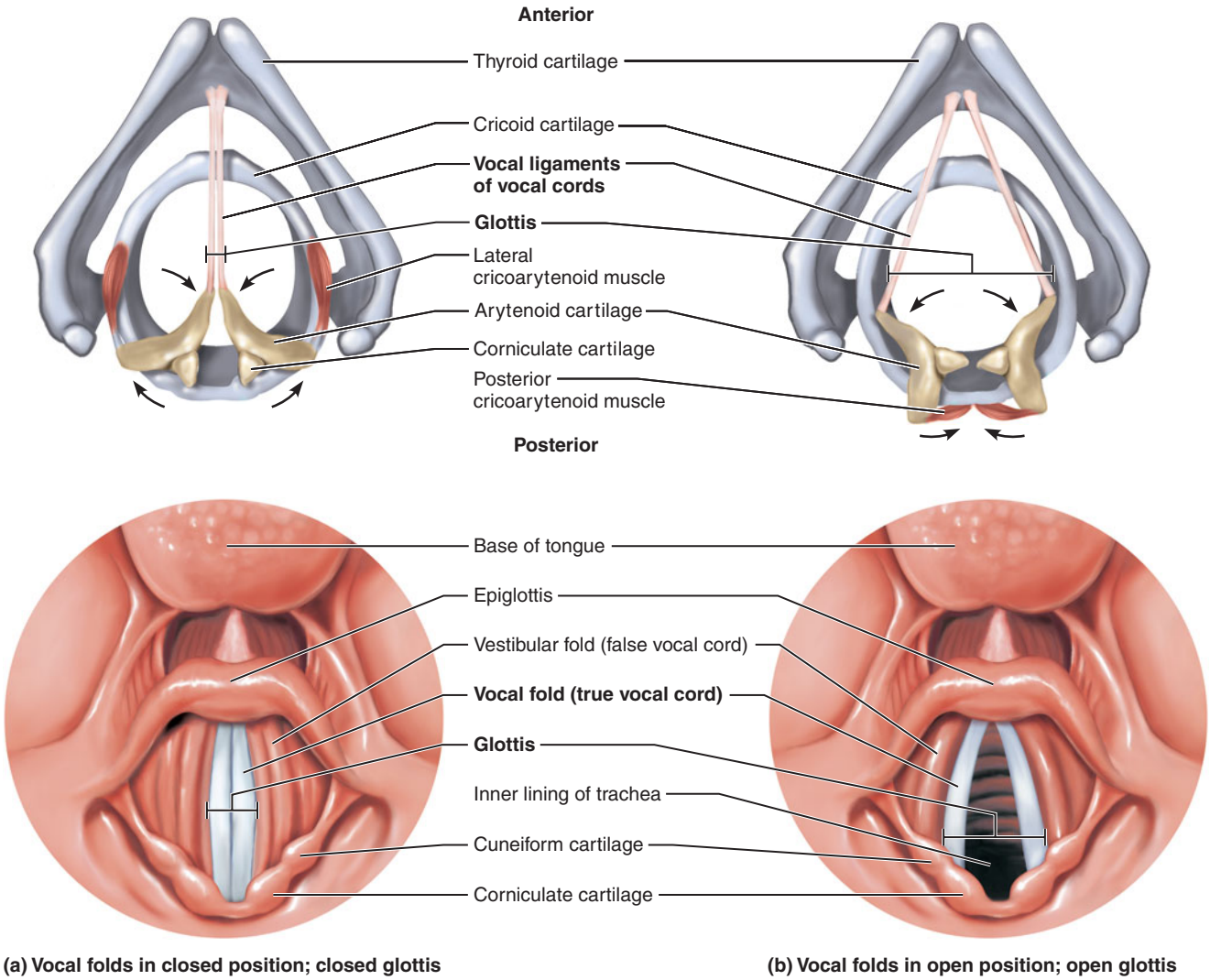


FIGURE 22.6 Movement of the vocal cords. Superior views of the larynx and vocal cords. The top illustrations show the skeletal elements and two of the many pairs of laryngeal muscles that rotate the arytenoid cartilages. The epiglottis has been left out so that the other laryngeal cartilages can be seen. Lower illustrations show the view of the larynx and vocal folds as seen through a laryngoscope.

basic sounds of speech. The medial opening between the vocal folds through which air passes is called the **rima glottidis** (*rima* = fissure), and the vocal folds together with this rima compose the **glottis.** Another pair of horizontal mucosal folds that lies directly superior to the vocal folds, the **vestibular folds,** or **false vocal cords,** play no part in sound production. However, they define a slitlike cavity between themselves and the true vocal cords (see Figure 22.5d) that enhances high-frequency sounds, functioning like the tweeter on stereo speakers.

The epithelium lining the superior part of the larynx, an area subject to food contact, is stratified squamous. Inferior to the vocal folds, the epithelium is pseudostratified ciliated columnar that entraps dust. In this epithelium, the power stroke of the cilia is directed upward, toward the pharynx, so that dust-trapping mucus is continuously moved superiorly from the lungs. "Clearing the throat" helps move this mucus up and out of the larynx.

Voice Production

Speech involves the intermittent release of exhaled air and the opening and closing of the glottis. In this process, the length of the vocal folds and the size of the rima glottidis are varied by intrinsic laryngeal muscles, most of which move the arytenoid cartilages (Figure 22.6). As the length and tension of the vocal folds change, the pitch of the produced sound changes. Generally, the tenser the vocal folds, the faster the exhaled air causes them to vibrate, and the higher the pitch.

As a boy's larynx enlarges during puberty, his laryngeal prominence grows anteriorly into a large Adam's apple, lengthening the vocal folds. Because longer vocal folds vibrate more slowly than short folds do, the voice becomes

deeper, just as a cello has a lower tone than a violin. For this reason, most men have lower voices than females or young boys. The voices of adolescent boys frequently "crack," alternating between high-pitched and low-pitched sounds, because the boys have not yet learned to control the action of their longer vocal folds.

Loudness of the voice depends on the force with which air rushes across the vocal folds. The greater the force, the stronger the vibrations and the louder the sound. The vocal folds do not move at all when we whisper, but they vibrate vigorously when we yell.

Although the vocal folds produce the basic speech sounds, the entire length of the pharynx acts as a resonating chamber to amplify the quality of sound. The oral cavity, nasal cavity, and paranasal sinuses also contribute to vocal resonance. In addition, normal speech and good enunciation depend on the "shaping" of sounds into recognizable consonants and vowels by the pharynx, tongue, soft palate, and lips.

LARYNGITIS Infection from a bad cold stimulates inflammation of the larynx, or **laryngitis,** causing the vocal folds to swell. The swelling interferes with their ability to vibrate, producing hoarseness. Hoarseness is also caused by overuse of the voice, growths on the vocal cords, inhalation of irritating chemicals (as in tobacco smoking), paralysis of some laryngeal muscles, or compression of a recurrent laryngeal nerve in the lung apex.

Sphincter Functions of the Larynx

Under certain conditions, the vocal folds act as a sphincter that prevents the passage of air. During abdominal straining, such as occurs when one strains to defecate, the abdominal muscles contract and the glottis closes to prevent exhalation, raising intrathoracic and intra-abdominal pressure. These events, collectively called **Valsalva's maneuver,** help to evacuate the rectum; they also stabilize the trunk of the body when one lifts a heavy load.

Innervation of the Larynx

The larynx receives its sensory and motor innervation through a superior laryngeal branch of each vagus nerve and from the *recurrent laryngeal nerves,* which branch off the vagus in the superior thorax and loop superiorly to reascend through the neck. The left recurrent laryngeal nerve loops under the aortic arch, whereas the right recurrent laryngeal nerve loops under the right subclavian artery. The backtracking course of these nerves is so unusual that the ancient Greeks mistook them for slings supporting the great arteries. Because these nerves supply innervation to most of the laryngeal muscles, damage to them, as can occur during surgery in this region, disrupts speech. Transection of one recurrent laryngeal nerve immobilizes one vocal fold, producing a degree of hoarseness. In such cases the other vocal fold can compensate, and speech remains almost normal. However, if both recurrent laryngeal nerves are transected, speech (except for whispering) is lost entirely.

check your understanding

3. Differentiate the pharynx from the larynx.
4. What structure forms the inferior boundary of each region of the pharynx (nasopharynx, oropharynx, and laryngopharynx)?
5. Name the laryngeal cartilages to which the vocal folds attach.

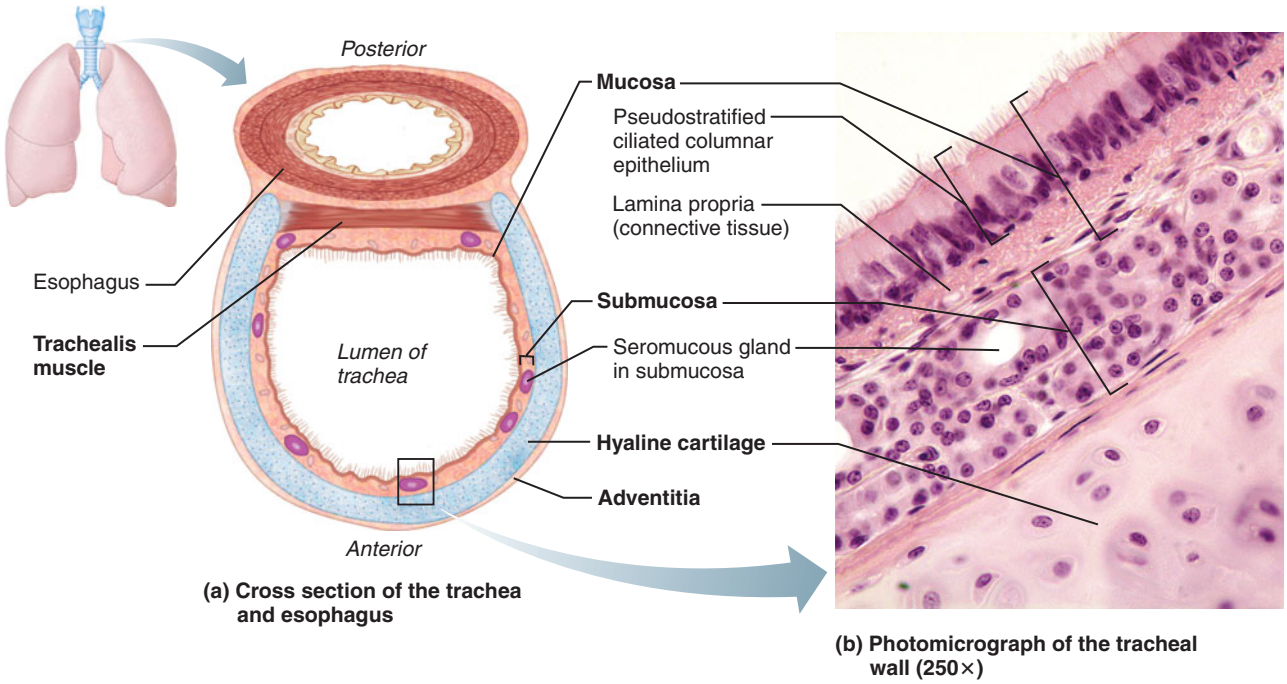
For answers, see Appendix B.

The Trachea

The flexible **trachea** (tra′ke-ah), or windpipe, descends from the larynx through the neck and into the mediastinum; it ends by dividing into the two main bronchi (primary bronchi) in the midthorax (see Figure 22.1). Early anatomists mistook the trachea for a rough-walled artery (*trachea* = rough).

The tracheal wall contains 16 to 20 C-shaped rings of hyaline cartilage **(Figure 22.7a)** joined to one another by intervening membranes of fibroelastic connective tissue. Consequently, the trachea is flexible enough to permit bending and elongation, but the cartilage rings prevent it from collapsing and keep the airway open despite the pressure changes that occur during breathing. The open posterior parts of the cartilage rings, which abut the esophagus, contain smooth muscle fibers of the **trachealis muscle** and soft connective tissue. Because the posterior wall of the trachea is not rigid, the esophagus can expand anteriorly as swallowed food passes through it. Contraction of the trachealis muscle decreases the diameter of the trachea: During coughing and sneezing, this action helps to expel irritants from the trachea by accelerating the exhaled air to a speed of 165 km/h (100 mph). A ridge on the internal aspect of the last tracheal cartilage, called the **carina** (kah-ri′nah; "keel"), marks the point where the trachea branches into the *two main (primary) bronchi* (see Figure 22.1). The mucosa that lines the carina is highly sensitive to irritants, and the cough reflex often originates here.

TRACHEOTOMY (TRACHEOSTOMY) When the upper respiratory tract is obstructed, a **tracheotomy,** or **tracheostomy,** is performed to open the airway. In a tracheotomy, the surgeon makes a vertical incision between the second and third tracheal rings in the anterior neck; a tube is then inserted to keep the airway open. In the emergency procedure called cricothyroid tracheotomy, a breathing tube is inserted through an incision made through the cricothyroid ligament of the larynx (see Figure 22.5b).



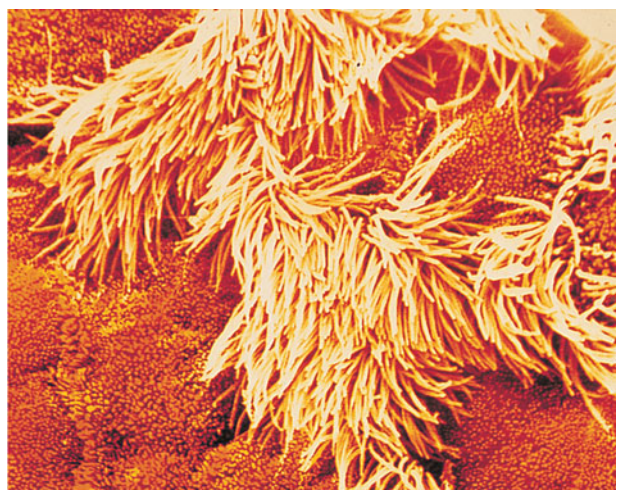


FIGURE 22.7 Tissue composition of the tracheal wall. In the scanning electron micrograph in (c), the cilia appear as yellow, grasslike projections. Mucus-secreting goblet cells (orange) with short microvilli are interspersed between the ciliated cells.

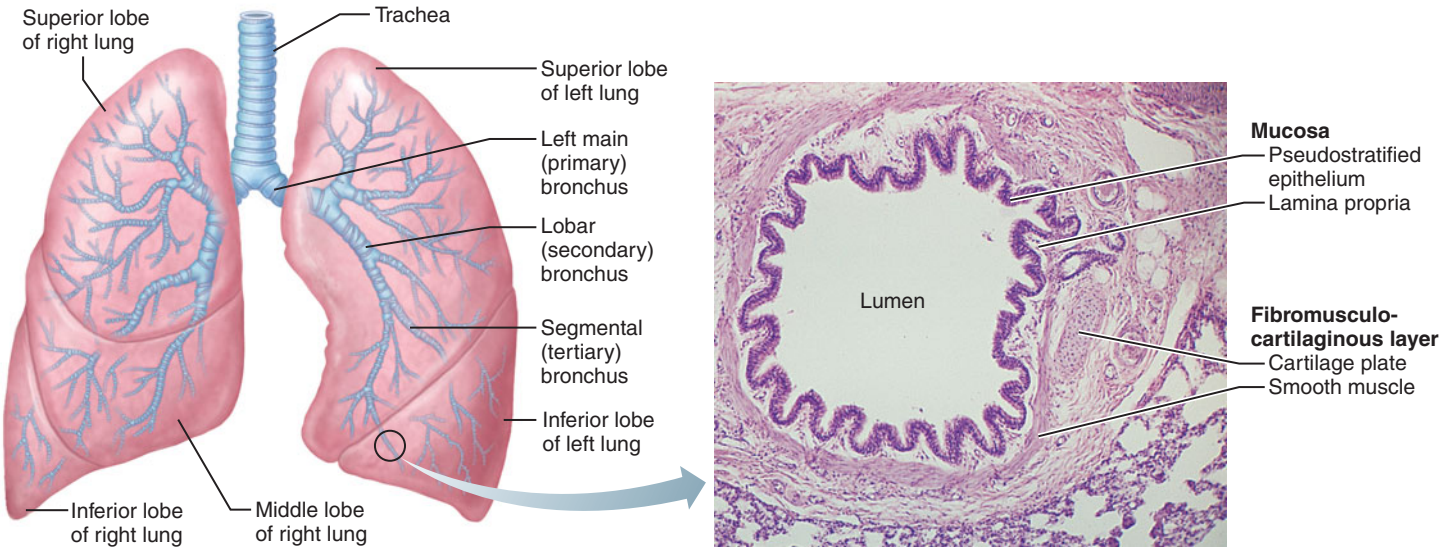
The microscopic structure of the wall of the trachea (Figure 22.7b) consists of several layers common to many tubular organs of the body: the *mucosa, submucosa,* and *adventitia.* The **mucosa,** a mucous membrane, as usual, consists of an inner epithelium and a lamina propria. The epithelium is the same air-filtering pseudostratified epithelium that occurs throughout most of the respiratory tract; its cilia continuously propel dust-laden sheets of mucus superiorly toward the pharynx (Figure 22.7c). The lamina propria contains many elastic fibers and is separated from the submucosa by a distinct sheet of elastin (not illustrated). This elastin, which occurs in all smaller air tubes as well, enables the trachea to stretch during inhalation and recoil during exhalation. The **submucosa** ("below the mucosa"), another layer of connective tissue, contains glands with both serous and mucous cells, called *seromucous glands,* which help produce the sheets of mucus within the trachea. The cartilaginous rings, the fibroelastic connective tissue connecting adjacent rings, and the trachealis muscle lie external to the submucosa and form the **fibromusculocartilaginous layer** of the trachea. The external layer of connective tissue is the **adventitia** (ad″ven-tish′e-ah).

The Bronchial Tree

- Explain how the walls of the upper respiratory passages differ histologically from those in the lower parts of the respiratory tree.
- Describe the structure of a lung alveolus and of the respiratory membrane.

Bronchi in the Conducting Zone

The right and left **main bronchi** (brong′ki), also called *primary bronchi,* are the largest conduits in the *bronchial*



(a) The branching of the bronchial tree

(b) Photomicrograph of a bronchus (13×)

FIGURE 22.8 The bronchi in the conducting zone.

tree, a system of respiratory passages that branches extensively within the lungs (Figure 22.8). The two main bronchi are branches of the trachea in the mediastinum. This bifurcation occurs at the level of the sternal angle (T_4) in the cadavers studied in the anatomy laboratory, but in living, standing individuals, it typically occurs more inferiorly, approximately at T_7. Each main bronchus runs obliquely through the mediastinum before plunging into the medial depression (hilum) of a lung. The main bronchi lie directly posterior to the large pulmonary vessels that supply the lungs. Because the right main bronchus is wider, shorter, and more vertical than the left, an accidentally inhaled object, such as a button or marble, is more likely to lodge in the right main bronchus.

As they approach and enter the lungs, the main bronchi divide into secondary or **lobar bronchi**—three on the right and two on the left—each of which supplies one lung lobe. The lobar bronchi branch into tertiary or **segmental bronchi,** which in turn divide repeatedly into smaller bronchi: fourth-order, fifth-order, and so on. Overall, there are about 23 orders of air tubes in the lungs, the tiniest almost too small to be seen without a microscope. The tubes smaller than 1 mm in diameter are called **bronchioles** ("little bronchi"), and the smallest of these, the **terminal bronchioles,** are less than 0.5 mm in diameter.

The tissue composition of the wall of each main bronchus mimics that of the trachea, but as the conducting tubes become smaller, the following changes occur:

1. **The supportive connective tissues change.** The cartilage rings are replaced by irregular *plates* of cartilage as the main bronchi enter the lungs (see Figure 22.8b). By the level of the bronchioles, supportive cartilage is no longer present in the tube walls. By contrast, elastin, which occurs in the walls throughout the bronchial tree, does not diminish.
2. **The epithelium changes.** The mucosal epithelium thins as it changes from pseudostratified columnar to simple columnar and then to simple cuboidal epithelium in the terminal and respiratory bronchioles. Neither cilia nor mucus-producing cells are present in these small bronchioles, where the sheets of air-filtering mucus end. Any inhaled dust particles that travel beyond the bronchioles are not trapped in mucus but instead are removed by macrophages in the alveoli (discussed shortly).
3. **Smooth muscle becomes important.** A layer of smooth muscle first appears in the posterior wall of the trachea, the trachealis muscle, and continues into the large bronchi. This layer forms helical bands that wrap around the smaller bronchi and bronchioles and regulate the amount of air entering the alveoli. The musculature relaxes to widen the air tubes during sympathetic stimulation, thus increasing airflow when respiratory needs are great, and it constricts the air tubes under parasympathetic direction when respiratory needs are low. Strong contractions of the bronchial smooth muscles narrow the air tubes during asthma attacks, as discussed in "Disorders of the Respiratory System" section of this chapter. The smooth muscle thins as it reaches the terminal end of the bronchiole tree and is absent around the alveoli.

The Respiratory Zone

The respiratory zone is the end part of the respiratory tree in the lungs. The respiratory zone consists of structures that contain air-exchange chambers called **alveoli** (Figure 22.9). The first respiratory zone structures, which branch from the terminal bronchioles of the conducting zone, are **respiratory bronchioles.** These can be recognized by the scattered alveoli protruding from their walls. The respiratory bronchioles lead into **alveolar ducts,** straight ducts whose walls consist almost entirely of alveoli. The alveolar ducts then lead into terminal clusters of alveoli called **alveolar sacs.** Note that alveoli and alveolar sacs are not the same things: An alveolar

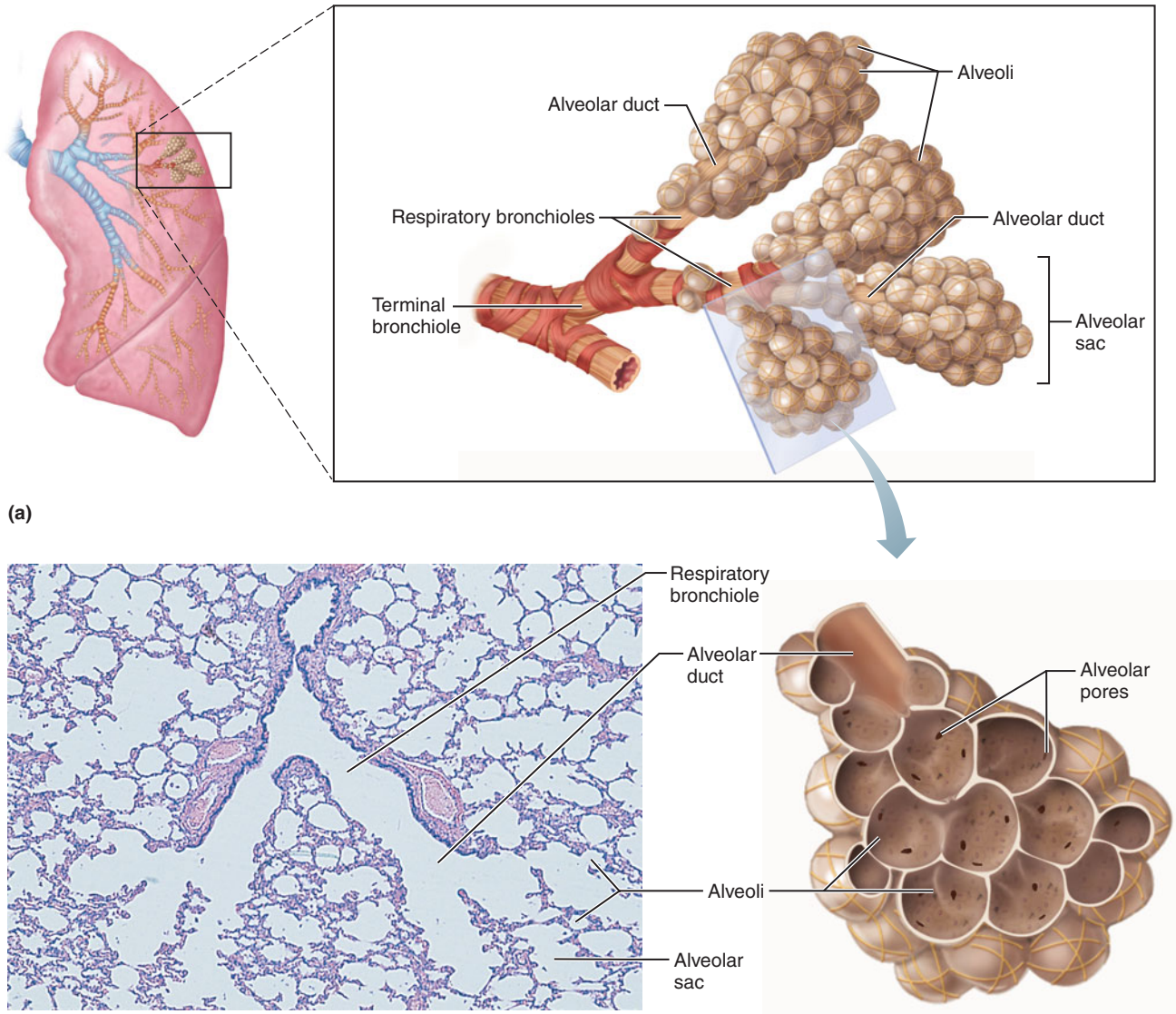


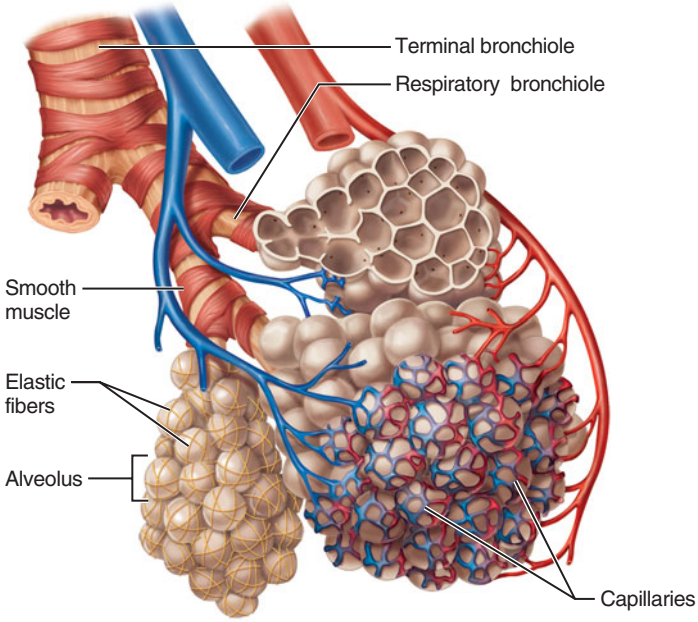
FIGURE 22.9 Structures of the respiratory zone. (a) Diagram of respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli. **(b)** Photomicrograph of a part of the lung (13×). (See *A Brief Atlas of the Human Body,* Second Edition, Plate 41.)

sac is analogous to a bunch of grapes, in which the individual grapes are the alveoli.

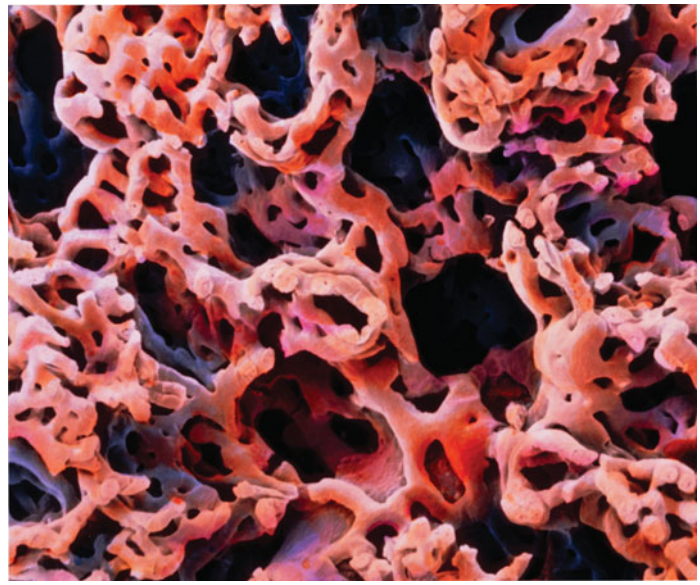
About 300 million air-filled alveoli crowd together within the lungs, accounting for most of the lung volume and providing a tremendous surface area for gas exchange. The total area of all alveoli in an average pair of lungs is 140 square meters, or 1500 square feet, which is 40 times greater than the surface area of the skin!

The wall of each alveolus consists of a single layer of squamous epithelial cells called **type I cells** surrounded by a delicate basal lamina **(Figure 22.10)**. The extreme thinness of this wall—0.5 μm—is hard to imagine. It is 15 times thinner than a sheet of tissue paper. The external surfaces of the alveoli are densely covered with a "cobweb" of pulmonary capillaries (Figure 22.10a and b), each of which is surrounded by a thin sleeve of the finest areolar connective tissue. Together, the alveolar and capillary walls and their fused basal laminae form the **respiratory membrane,** where oxygen and carbon dioxide are exchanged between the alveolus and the blood (Figure 22.10c). Air is present on the alveolar side of the membrane, and blood flows past on the capillary side. Gases pass easily through this thin membrane: Oxygen diffuses from the alveolus into the blood, and carbon dioxide diffuses from the blood to enter the air-filled alveolus.

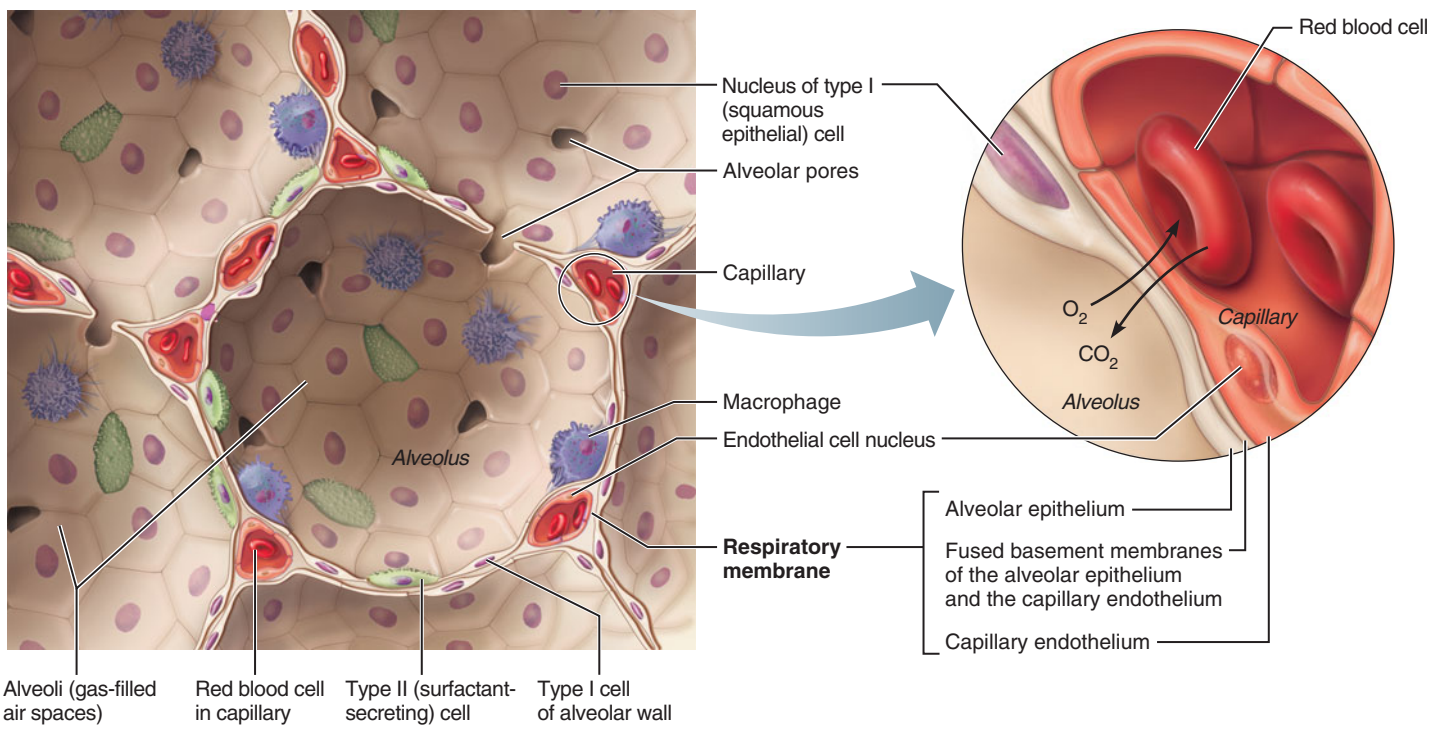
Scattered among the type I squamous cells in the alveolar walls are cuboidal epithelial cells called **type II cells** (Figure 22.10c), which secrete a fluid that coats the internal alveolar surfaces. This fluid contains a detergent-like substance called **surfactant** (ser-fak′tant) that reduces surface tension within the alveoli. Without this surfactant, the inner walls of an alveolus would stick together during exhalation.



(a) Diagrammatic view of capillary-alveoli relationships



(b) Scanning electron micrograph of pulmonary capillary casts. Tissue forming the alveoli has been removed, leaving only the capillary network (24×).



(c) Detailed anatomy of the respiratory membrane

FIGURE 22.10 Alveoli and the respiratory membrane. Elastic fibers and capillaries surround all alveoli, but for clarity they are shown only on some alveoli in (a).

Lung alveoli also have the following significant features:

1. Alveoli are surrounded by fine *elastic fibers* of the same type that surround structures along the entire respiratory tree (Figure 22.10a).
2. Adjacent alveoli interconnect via **alveolar pores** (Figure 22.10c), which allow air pressure to be equalized throughout the lung and provide alternative routes for air to reach alveoli whose bronchi have collapsed because of disease.
3. Internal alveolar surfaces provide a site for the free movement of **alveolar macrophages,** which actually live in the air space and remove the tiniest inhaled particles that were not trapped by mucus. Dust-filled macrophages

migrate superiorly from the "dead-end" alveoli into the bronchi, where ciliary action carries them into the pharynx to be swallowed. This mechanism removes over 2 million debris-laden macrophages each hour.

check your understanding

6. What features of the trachea and larger bronchi trap and remove foreign particles from inhaled air? What performs this function in the alveoli?
7. At what level of the bronchial tree are cartilage plates no longer found? What is the extent of elastin in the bronchial tree?
8. Describe the structure of the respiratory membrane.

For answers, see Appendix B.

The Lungs and Pleurae

➤ Describe the gross structure of the lungs and the pleurae.

Gross Anatomy of the Lungs

The paired **lungs** and their pleural sacs occupy all the thoracic cavity lateral to the mediastinum **(Figure 22.11)**. Each lung is roughly cone-shaped. The anterior, lateral, and posterior surfaces of a lung contact the ribs and form a continuously curving *costal surface.* Just deep to the clavicle is the **apex,** the rounded, superior tip of the lung. The concave inferior surface that rests on the diaphragm is the **base.** On the *medial (mediastinal) surface* of each lung is an indentation, the **hilum,** through which blood vessels, bronchi, lymphatic vessels, and nerves enter and exit the lung. Collectively, these structures attach the lung to the mediastinum and are called the **root** of the lung. The largest components of this root are the pulmonary artery and veins and the main (primary) bronchus. Figure 22.11b shows the arrangement of the root structures at the hilum of the lung.

Because the heart is tilted slightly to the left of the median plane of the thorax, the left and right lungs differ slightly in shape and size. The left lung is somewhat smaller than the right and has a **cardiac notch,** a deviation in its anterior border that accommodates the heart (see Figure 22.11a). Several deep fissures divide the two lungs into different patterns of **lobes.** The left lung is divided into two lobes, the **superior lobe** and the **inferior lobe,** by the **oblique fissure.** The right lung is partitioned into three lobes, the **superior, middle,** and **inferior lobes,** by the **oblique** and **horizontal fissures.** As previously mentioned, each lung lobe is served by a lobar (secondary) bronchus and its branches.

Each of the lobes, in turn, contains a number of **bronchopulmonary segments (Figure 22.12)** separated from one another by thin partitions of dense connective tissue. Each segment receives air from an individual segmental (tertiary) bronchus. There are approximately ten bronchopulmonary segments arranged in similar, but not identical, patterns in each of the two lungs.

The bronchopulmonary segments have clinical significance in that they limit the spread of some diseases within the lung, because infections do not easily cross the connective-tissue partitions between them. Furthermore, because only small veins span these partitions, surgeons can neatly remove segments without cutting any major blood vessels.

The smallest subdivision of the lung that can be seen with the naked eye is the **lobule.** Appearing on the lung surface as hexagons ranging from the size of a pencil eraser to the size of a penny (see Figure 22.11b), each lobule is served by a bronchiole and its branches. In most city dwellers and in smokers, the connective tissue that separates the individual lobules is blackened with carbon.

As previously mentioned, the lungs consist largely of air tubes and spaces. The balance of the lung tissue, its *stroma* (stro′mah; "mattress"), is a framework of connective tissue containing many elastic fibers. As a result, the lungs are light, soft, spongy, elastic organs that each weigh only about 0.6 kg (1.25 pounds). The elasticity of healthy lungs helps to reduce the effort of breathing, as described shortly.

Blood Supply and Innervation of the Lungs

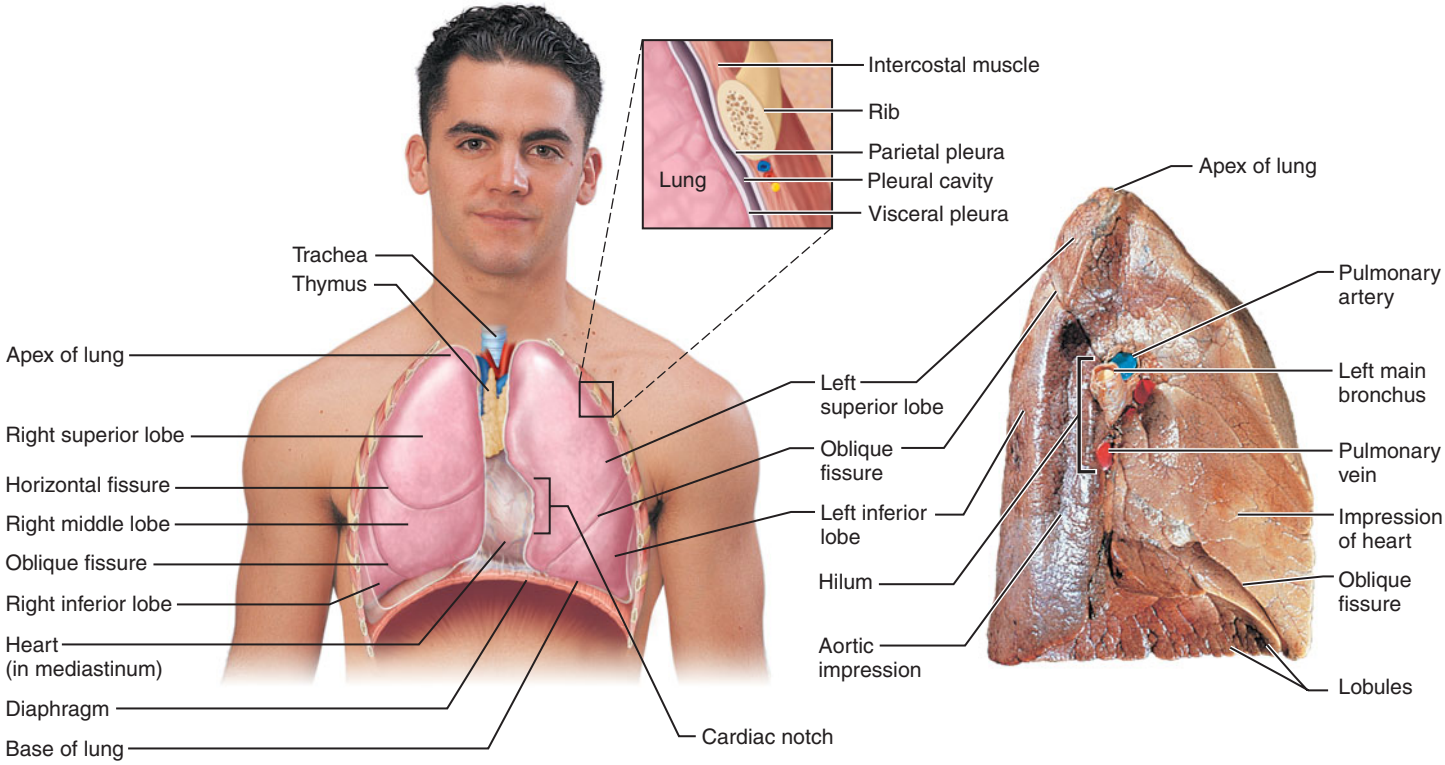
The **pulmonary arteries** (see p. 589) deliver oxygen-poor blood to the lungs for oxygenation (see Figure 22.11d and e). In the lung, these arteries branch along with the bronchial tree, generally lying *posterior* to the corresponding bronchi. The smallest arteries feed into the **pulmonary capillary networks** around the alveoli (Figure 22.10a). Oxygenated blood is carried from the alveoli of the lungs to the heart by the **pulmonary veins,** whose tributaries generally lie *anterior* to the corresponding bronchi within the lungs. However, some venous tributaries run in the connective tissue partitions between the lung lobules and between the bronchopulmonary segments.

As explained in Chapter 20 (pp. 594, 603), the *bronchial arteries* and *veins* provide and drain systemic blood to and from the lung tissues. These small vessels enter and exit the lungs at the hilum, and within the lung they lie on the branching bronchi.

The lungs are innervated by sympathetic, parasympathetic, and visceral sensory fibers that enter each lung through the *pulmonary plexus* on the lung root. From there, these nerve fibers lie along the bronchial tubes and blood vessels within the lungs. As described in Chapter 15, parasympathetic fibers constrict the air tubes, whereas sympathetic fibers dilate them.

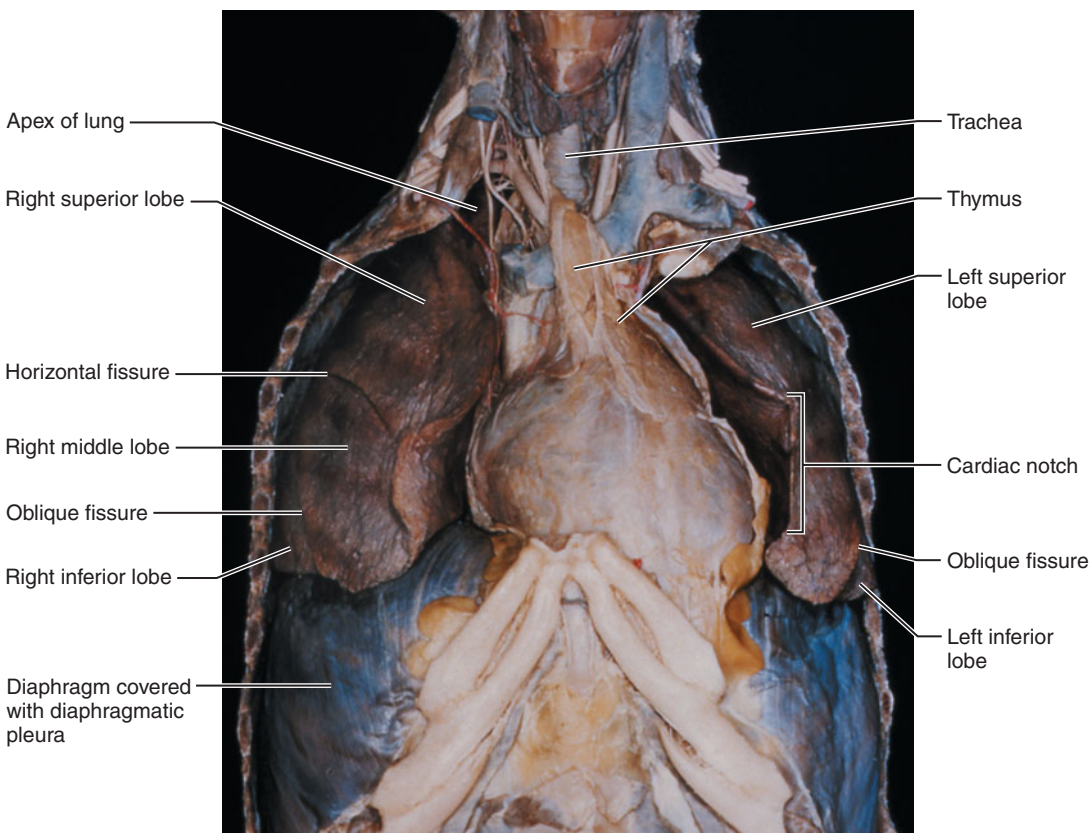
The Pleurae

Around each lung is a flattened sac whose walls consist of a serous membrane called **pleura** (ploo′rah; "the side"). As shown in Figure 22.11a, enlargement, the outer layer of this sac is the *parietal pleura,* whereas the inner layer, directly on the lung, is the *visceral pleura.* The **parietal pleura** covers the internal surface of the thoracic wall, the superior surface of the diaphragm, and the lateral surfaces of the mediastinum. From the mediastinum, it reflects laterally to enclose the great vessels running to the lung (lung root, Figure 22.11d). In the area where these vessels enter the lung, the parietal pleura is continuous with the **visceral pleura,** which covers the external lung surface.



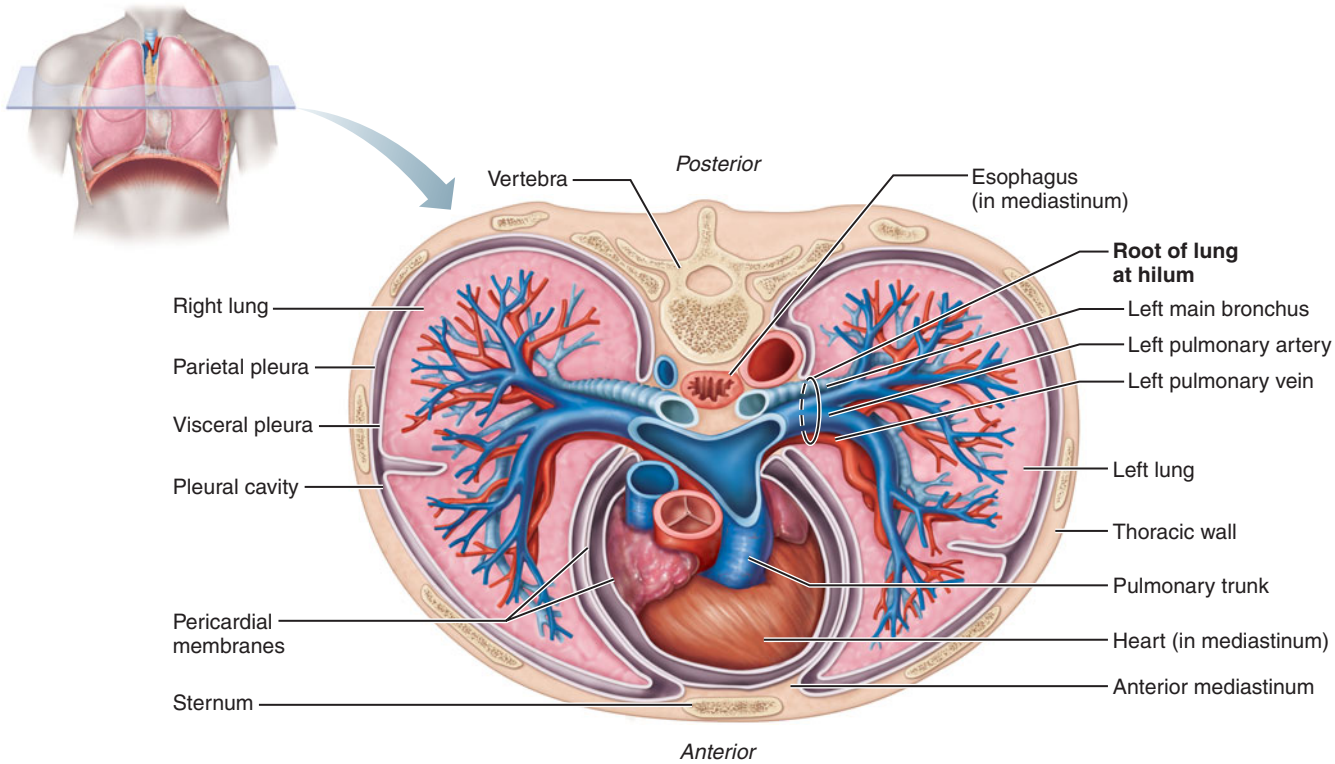
(a) Anterior view. The lungs flank mediastinal structures laterally.

(b) Photograph of medial view of the left lung

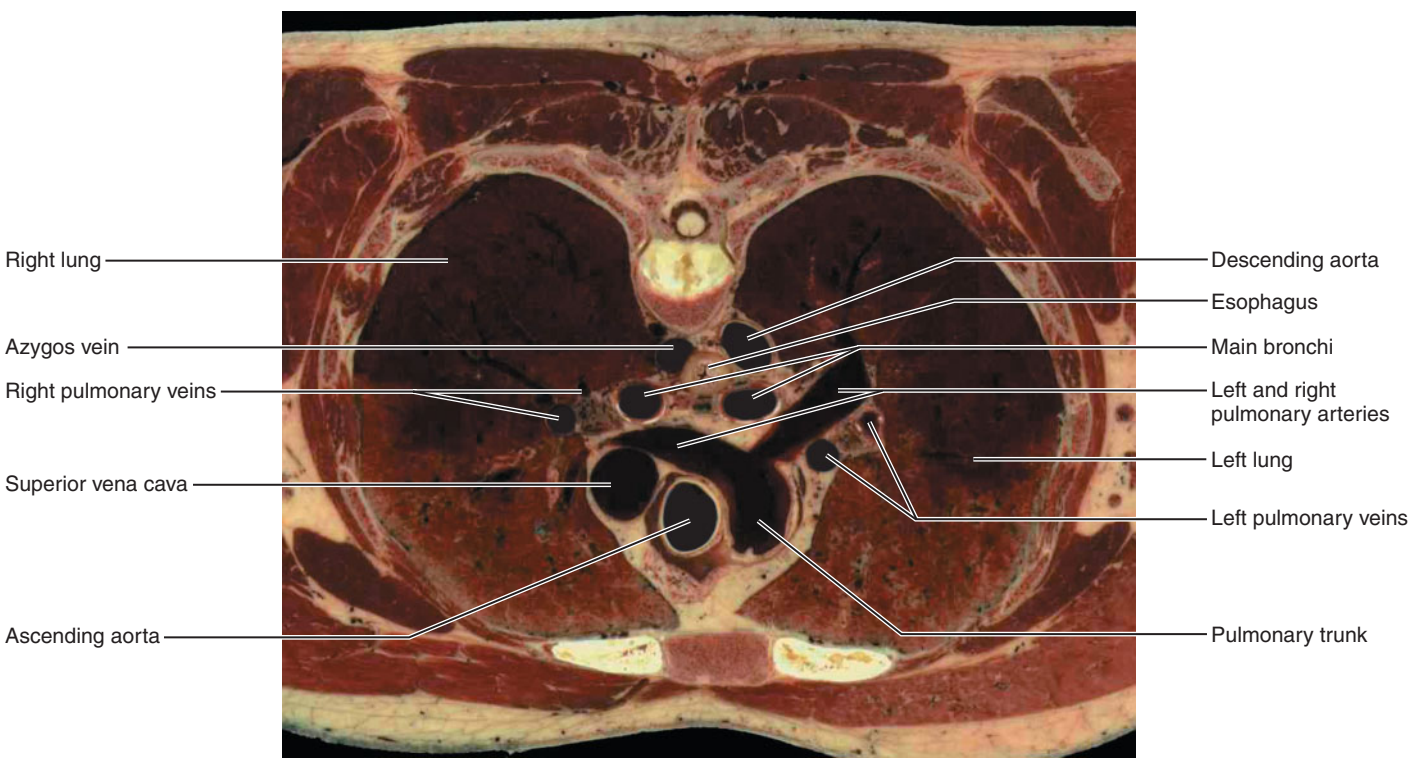


(c) Dissection of the thoracic viscera, anterior view

FIGURE 22.11 Anatomical relationships of organs in the thoracic cavity. The inset in (a) shows details of the pleurae and pleural cavity. (See *A Brief Atlas of the Human Body*, Second Edition, Figure 56.)



(d) Transverse section through the thorax, viewed from above. Lungs, pleural membranes, and major organs in the mediastinum are shown.



(e) Cross section through the thorax, view as in (d)

FIGURE 22.11 Anatomical relationships of organs in the thoracic cavity, *continued.*
In (d), the size of the pleural cavity is exaggerated for clarity.

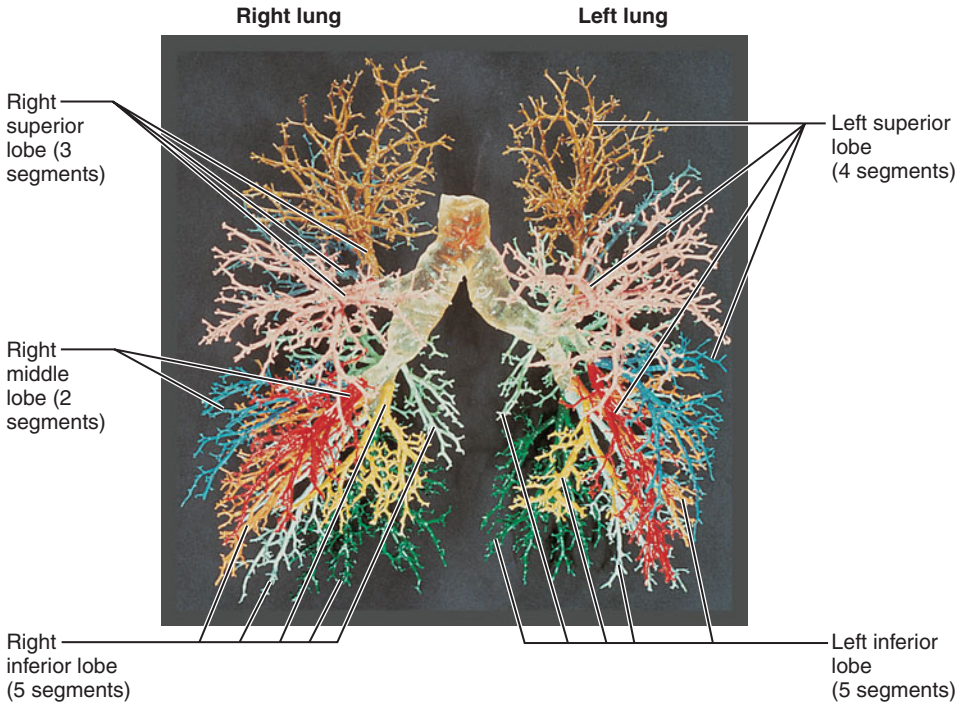


FIGURE 22.12 Bronchopulmonary segments. Resin cast of the bronchial tree; individual bronchopulmonary segments have been painted different colors.

The space between the parietal and visceral pleurae is the **pleural cavity** (Figure 22.11d). The pleural cavity is filled with a thin film of *pleural fluid.* Secreted by the pleurae, this lubricating fluid allows the lungs to glide without friction over the thoracic wall during breathing movements. The fluid also holds the parietal and visceral pleurae together, just as a film of oil or water would hold two glass plates together. The two pleurae can easily slide from side to side across each other, but their separation is strongly resisted. Consequently, the lungs cling tightly to the thoracic wall and are forced to expand and recoil as the volume of the thoracic cavity increases and decreases during breathing.

The pleurae also divide the thoracic cavity into three separate compartments—the central mediastinum and two lateral pleural compartments, each containing a lung (Figure 22.11d). This compartmentalization helps to prevent the moving lungs or heart from interfering with one another. Compartmentalization also limits the spread of local infections and the extent of traumatic injury.

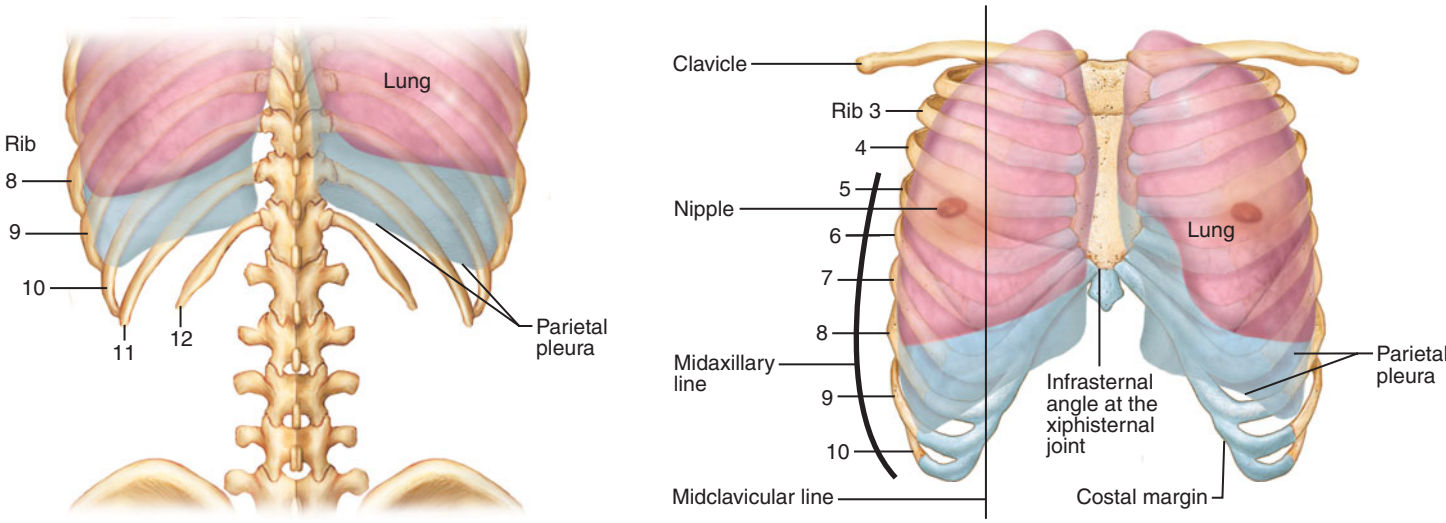
Around all surfaces of the lung, with the exception of the inferior surface, the pleural cavity is a slitlike potential space. Inferiorly, the pleural cavity extends below the inferior border of the lungs as illustrated in **Figure 22.13**. Posteriorly, the inferior margin of the parietal pleura lies adjacent to vertebra T_{12} near the posterior midline (Figure 22.13a) and runs horizontally across the back to reach rib 10 at the midaxillary line. Anteriorly, the parietal pleura ascends to rib 8 in the midclavicular line (Figure 22.13b) and to the level of the xiphisternal joint near the anterior midline. The inferior borders of the lungs are located two ribs superior to the pleural margin. Anteriorly, the lungs meet the pleural margin near the xiphisternal joint. Surgeons and other clinicians must be aware of the inferior margin of the pleural cavities because cutting into a pleural cavity causes the lung to collapse.

PLEURISY AND PLEURAL EFFUSION Lung infections such as pneumonia produce inflammation of the pleura, called **pleurisy (pleuritis).** The rubbing together (friction) of the two inflamed pleural membranes produces a stabbing chest pain with each breath. Because the visceral pleura is relatively insensitive (see the discussion of visceral pain on p. 475), the pain of pleurisy actually originates from the parietal pleura only. If pleurisy persists, the inflamed pleurae may secrete excess pleural fluid, which then overfills the pleural cavity and exerts pressure on the lungs, thus hindering breathing movements.

Pleural effusion is a general term for the accumulation of one of various kinds of fluid in the pleural cavity. Although it characterizes many cases of pleurisy, it can also result from (1) hemorrhage of a damaged lung or lung vessel or (2) leakage of a serous fluid from the lung capillaries when either right ventricular failure or heart failure causes blood to back up in the pulmonary circuit.

check your understanding

9. Name the fissure that separates the superior and middle lobes of the right lung.
10. In what order, from superior to inferior, do the pulmonary vessels and main bronchus enter the left lung at the hilum?



(a) Posterior view **(b) Anterior view**

FIGURE 22.13 Position of the lungs and the pleural cavities in reference to the thoracic cage. The pleural cavity extends approximately two ribs below the inferior border of the lungs.

11. Would a stab wound in the midclavicular line just above rib 7 puncture a lung? Would it puncture the pleural cavity?

For answers, see Appendix B.

VENTILATION

- Explain the relative roles of the respiratory muscles and lung elasticity in the act of ventilation.
- Define surfactant, and explain its function in ventilation.
- Explain how the brain and peripheral chemoreceptors control the ventilation rate.

The Mechanism of Ventilation

Breathing, or **pulmonary ventilation,** consists of two phases: *inspiration* (inhalation), the period when air flows into the lungs, and *expiration* (exhalation), the period when gases exit the lungs. This section discusses the mechanical factors that promote this movement of air; for more detailed information on the *muscles* of ventilation, see Table 11.7 on pp. 289–290.

Inspiration

The process of **inspiration** is easy to understand if you think of the thoracic cavity as an expandable container with a single entrance at the top: the tubelike trachea. Enlarging all dimensions of this container increases its volume and thus decreases the pressure within it (because pressure and volume in a closed container are inversely related). This decrease in internal gas pressure causes air to enter the container from the atmosphere, because gases always flow from areas of high pressure to areas of low pressure. During normal quiet inspiration, the inspiratory muscles—the diaphragm and intercostal muscles—function to increase the volume of the thorax. This process is illustrated and explained in Figure 22.14a.

1. **Action of the diaphragm.** When the dome-shaped diaphragm contracts, it moves inferiorly and flattens (Figure 22.14a). As a result, the superior-inferior dimension of the thoracic cavity increases.
2. **Action of the intercostal muscles.** The external intercostal muscles contract to raise the ribs. Lifting the ribs enlarges both the lateral dimensions of the thoracic cavity and the anterior-posterior dimensions (Figure 22.14a).

   The external and internal intercostal muscles also function together during quiet inspiration to stiffen the thoracic wall. Without this stiffening, the contraction of the diaphragm would result in a change of shape of the thorax but not a change in volume. Think about what would happen if the expandable container described above was made with rubber sides. As the container is enlarged by movement of its inferior surface, the flexible sides would collapse toward the center. A change of shape would result, but not a change of volume.

Although simplistic, the analogy of the expandable container provides the basic framework to discuss the mechanics of breathing. The lungs are surrounded by the pleural cavity. The air in the lungs is at atmospheric pressure and the pressure in the pleural cavity is less than atmospheric pressure, causing the lungs to expand and compress the pleural cavity against the thoracic wall (Figure 22.15, ①). As the thoracic cavity enlarges by the action of the inspiratory muscles, the pleural cavity also enlarges, as does the volume of the lungs. The increased volume results in a decrease in pressure in the pleural cavity and in the lungs. Because the lungs are open to the atmosphere via the bronchial tubes and trachea, when pressure in the lungs decreases, air from the atmosphere flows in (Figure 22.15, ②). For ventilation to occur, the pleural cavity must be intact. If the integrity of the pleural cavity is disrupted as a result of trauma or a disease process and the pressure

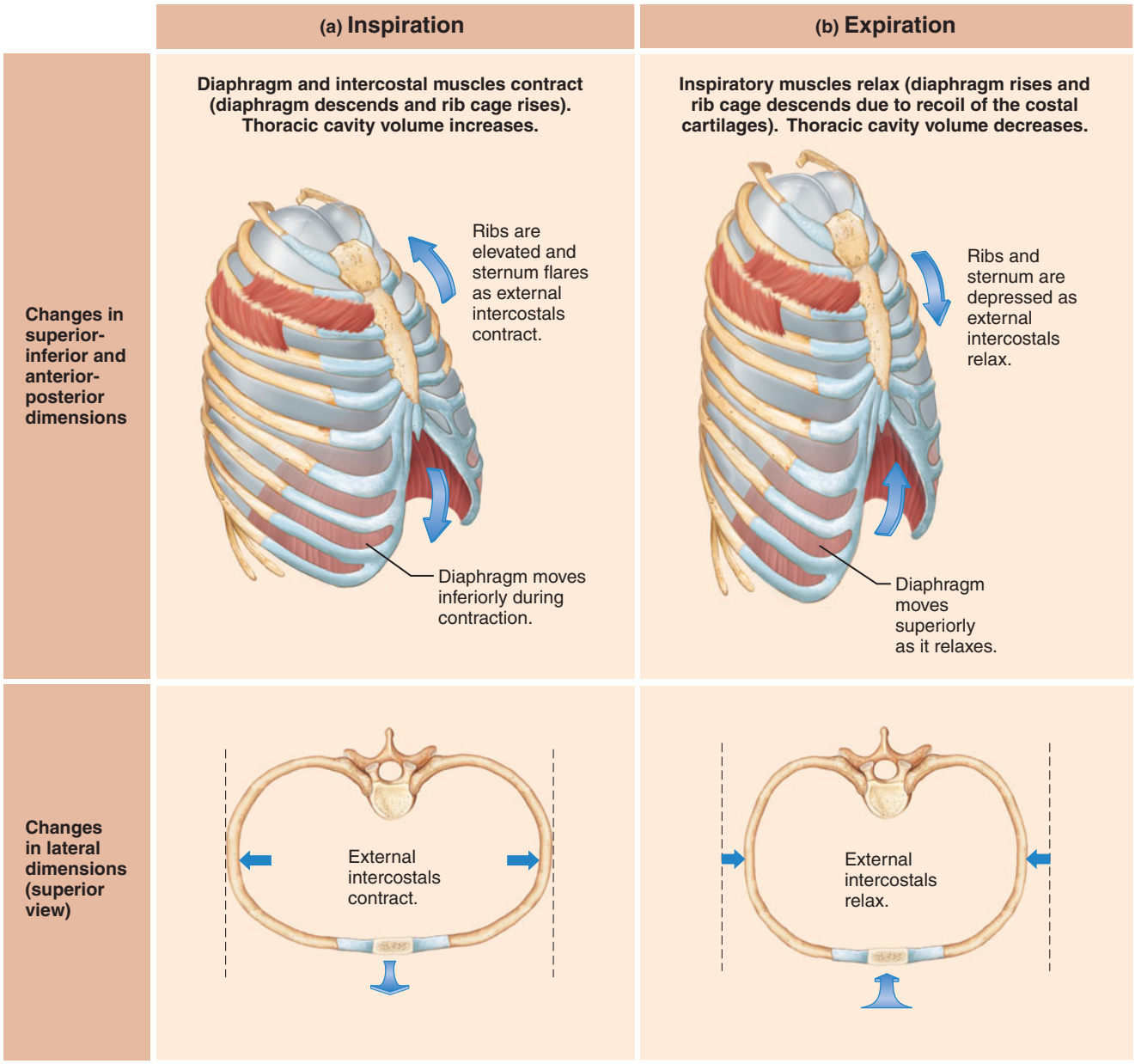


FIGURE 22.14 Actions of the respiratory muscles during normal ventilation and changes in thoracic cavity volume.

differential between the lung and the pleural cavity is lost, the lung will collapse, and normal ventilation cannot occur.

COLLAPSED LUNG A lung will collapse if air enters the pleural cavity, a condition called **pneumothorax** (nu″mo-tho′raks; "air thorax"). The air breaks the seal of pleural fluid that holds the lung to the thoracic wall, allowing the elastic lung to collapse like a deflating balloon. Pneumothorax usually results from chest trauma or overexertion that raises intrathoracic pressure such that a lung pops like one pops a blown-up paper bag; it can also result from a wound that penetrates the thoracic wall or from a disease process that erodes a hole through the external surface of the lung. Pneumothorax is reversed surgically by closing the "hole" through which air enters the pleural cavity and then gradually withdrawing the air from this cavity using chest tubes. This treatment allows the lung to reinflate and resume its normal function.

Obstruction of a bronchus by a plug of mucus, an inhaled object, a tumor, or enlarged lymph nodes may also cause a lung to collapse; the collapse occurs as the air beyond the point of blockage is gradually reabsorbed into the pulmonary capillaries. Finally, pleural effusion (see p. 652) can cause collapse as the accumulating fluid compresses the lung.

Because the lungs are in completely separate pleural cavities, one lung can collapse without affecting the function of the other.

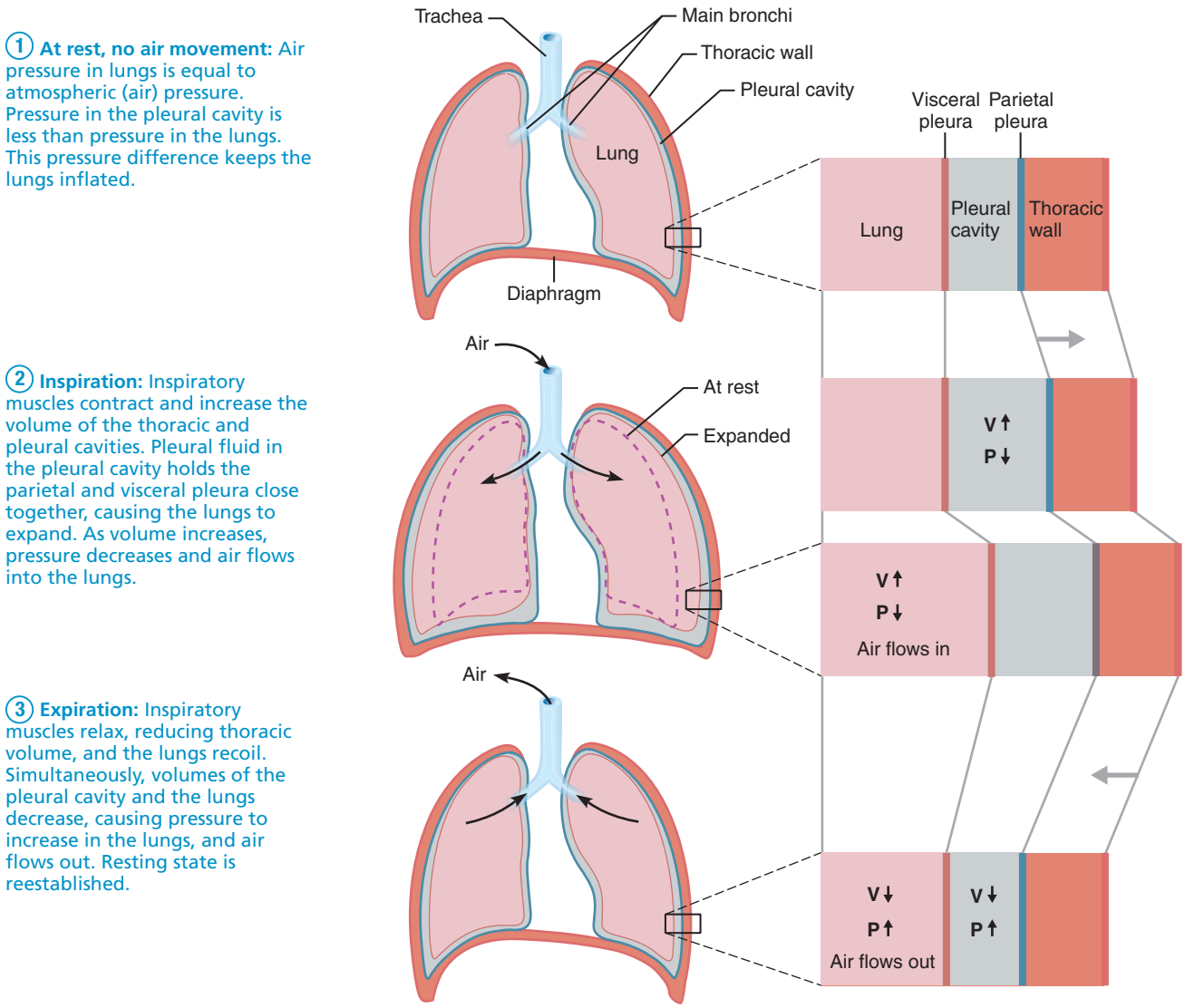


FIGURE 22.15 The volume and pressure changes that result in pulmonary ventilation.

During deep or forced inspiration, additional muscles contract and further increase thoracic volume. The rib cage is elevated by the sternocleidomastoid muscle of the neck, and by the scalenes and pectoralis minor of the chest. Additionally, the quadratus lumborum fixes the 12th rib, resulting in a more powerful downward pull of the diaphragm, and the back extends as the thoracic curvature is straightened by the erector spinae muscles.

Expiration

Quiet **expiration** in healthy people is chiefly a passive process. As the inspiratory muscles relax, the rib cage drops under the force of gravity and the relaxing diaphragm moves superiorly (Figure 22.14b). At the same time, the many elastic fibers within the lungs recoil. The result is that the volumes of the thorax and lungs decrease simultaneously, increasing the pressure within the lungs and pushing the air out (Figure 22.15, ③).

By contrast, *forced* expiration is an active process produced by contraction of muscles in the abdominal wall, primarily the internal and external obliques and the transversus abdominis. These contractions (1) increase the intra-abdominal pressure, forcing the diaphragm superiorly, and (2) sharply depress the rib cage, decreasing thoracic volume. The internal intercostal muscles and the latissimus dorsi also help to depress the rib cage.

In a healthy lung, the alveoli remain open at all times and do not collapse during exhalation. At first glance this seems to contradict the laws of physics, because a watery film coats the internal surfaces of the alveoli, and water molecules have a high attraction for one another (called *surface tension*) that should collapse the alveoli after each breath. Collapse does not occur because the alveolar film also contains surfactant secreted by the type II alveolar cells, which interferes with the attraction between water molecules, thereby reducing surface tension and enabling the alveoli to remain open.

RESPIRATORY DISTRESS SYNDROME Because pulmonary surfactant is not produced until the end of fetal life, its absence can have dire consequences for infants born prematurely. In such infants, the alveoli collapse during exhalation and must be completely reinflated during each inspiration, an effort that requires a tremendous expenditure of energy that can lead to exhaustion and respiratory failure. This condition, called **respiratory distress syndrome (RDS),** is responsible for one-third of all infant deaths. It is treated by using positive-pressure respirators to force air into the alveoli and keep them inflated between breaths and by administering natural or artificial surfactants. Even though such *surfactant therapy* has saved many lives since its introduction in 1990, many survivors still suffer from *chronic lung disease (bronchopulmonary dysplasia)* throughout childhood and beyond. This condition results from inflammatory injury to the respiratory zone, possibly caused by the high pressures the respirator exerts on the delicate lungs to distribute surfactant evenly.

Neural Control of Ventilation

The brain's most important respiratory center is in the reticular formation of the medulla oblongata. This center, called the ventral respiratory group (VRG) is a pacemaker whose neurons generate the basic ventilatory rhythm and rate with input from centers in the pons and dorsal medulla **(Figure 22.16)**. Neurons from the medullary respiratory center stimulate the somatic motor neurons to the respiratory muscles. This basic ventilatory pattern can be modified by higher centers of the brain, such as the limbic system and hypothalamus (by which emotions influence breathing—as when people gasp), and the cerebral cortex (through which people have conscious control over the rate and depth of breathing).

Although the medulla's respiratory center sets a baseline ventilatory rate, this rate is also modified by input from receptors that sense the chemistry of blood. These chemoreceptors respond to falling concentrations of oxygen, rising levels of carbon dioxide, or increased acidity of the blood by signaling the respiratory center to increase the rate and depth of breathing, which returns the blood gases to their normal concentrations. The chemoreceptors are of two types: *central chemoreceptors,* located mainly in the medulla, and *peripheral chemoreceptors,* the **aortic bodies** located on the aortic arch and the **carotid bodies** found in the fork of the common carotid artery **(Figure 22.17)**. The aortic bodies send their sensory information to the medulla through the vagus nerves, whereas the carotid bodies send theirs through the glossopharyngeal (and perhaps the vagus) nerves. In humans, it appears that the carotid bodies are the more important chemoreceptor for regulating respiration. For more information on the peripheral chemoreceptors, see Table 14.2 on pp. 437–438.

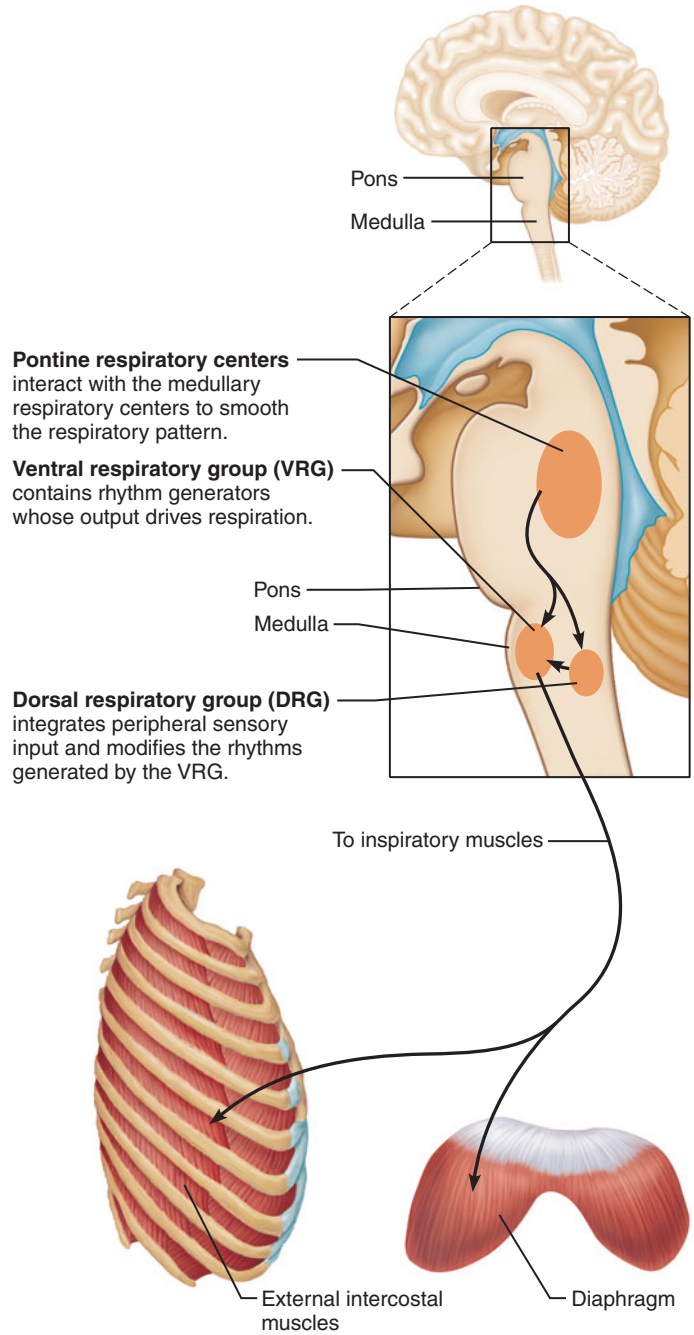


FIGURE 22.16 Respiratory centers in the brain stem. The synapses in the spinal cord with somatic motor neurons to the inspiratory muscles are not diagrammed.

check your understanding

12. How does contraction of the diaphragm affect the volume of the thoracic cavity? How does this change of volume affect the pressure in the pleural cavity?
13. What will happen to the lung in the stab wound injury described in question 11 on p. 653? Why is this type of injury called a "sucking chest wound"?
14. What accessory muscles may be used for inspiration in a person experiencing respiratory distress? What muscles are active during forced expiration?

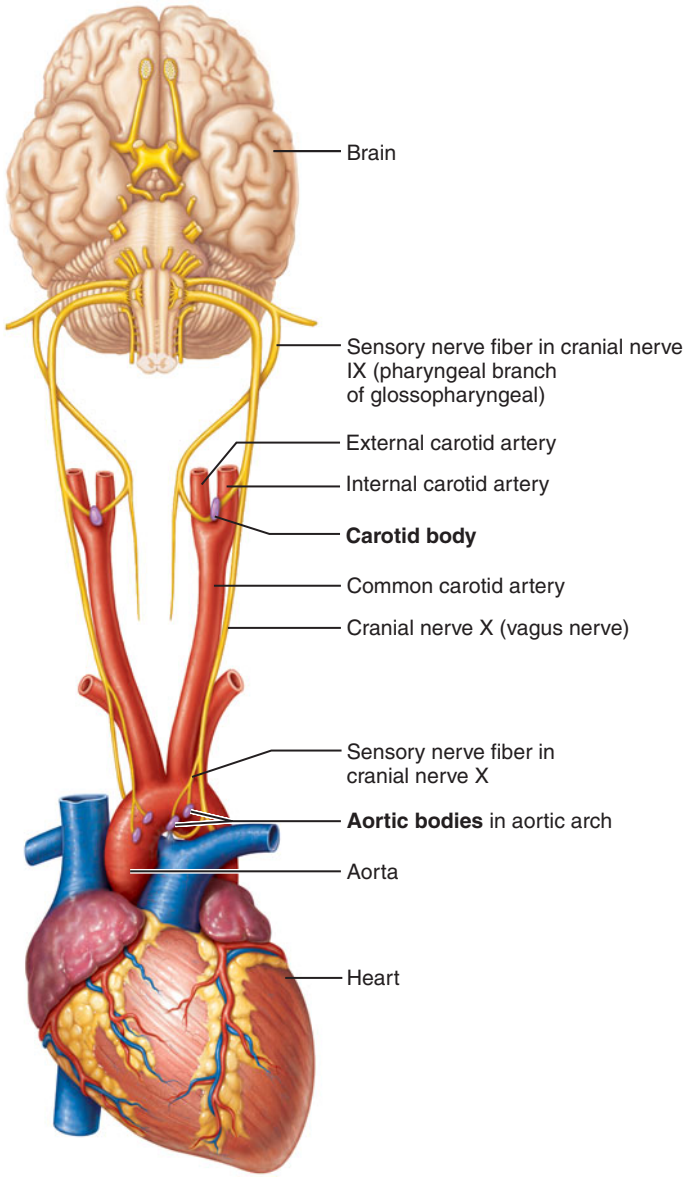


FIGURE 22.17 Location and innervation of the peripheral chemoreceptors in the carotid and aortic bodies. Also shown are the sensory pathways from these receptors through cranial nerves IX and X to the respiratory center in the medulla.

15. Although the respiratory muscles are innervated by somatic motor neurons, and thus are under voluntary control, we do not have to think about breathing. Why is this so?

For answers, see Appendix B.

DISORDERS OF THE RESPIRATORY SYSTEM

➤ Consider the causes and consequences for respiration of asthma, cystic fibrosis, chronic bronchitis, emphysema, lung cancer, and nosebleeds.

Because the respiratory system is open to the outside world and exposed to large volumes of air, it is highly susceptible to airborne microorganisms, pollutants, and irritants, leading to a remarkable number of common respiratory disorders. One of the most lethal respiratory diseases, lung cancer, is discussed in **A Closer Look** on p. 658.

Bronchial Asthma

Bronchial asthma, which affects about 7% of adults and 10% of children and is increasing in frequency, is a type of allergic inflammation in people who are hypersensitive to irritants in the air or to stress. Attacks may be triggered by inhaling substances to which the sufferer is allergic (dust mites, pollen, molds, bits of cockroach), by inhaling dust or smoke, by respiratory infections, by emotional upset, or by the mild shock of breathing cold air. Symptoms of asthma attacks include coughing, wheezing, and shortness of breath.

An asthma attack starts with an early phase, in which an allergen initiates the release of inflammation-mediating chemicals, such as histamine, from mast cells. These chemicals stimulate both contraction of the bronchial smooth musculature (bronchoconstriction) and secretion of mucus in these airways. Within several hours, a late phase develops as eosinophils, neutrophils, a certain type of helper T lymphocytes, and basophils accumulate in the bronchi and bronchioles, where these leukocytes secrete additional inflammatory chemicals that damage the bronchial mucosa and further increase bronchoconstriction and secretion of mucus.

Until about 1990, bronchoconstriction was considered the primary symptom of asthma, and quick relief is still provided by drugs that either inhibit smooth-muscle contraction (bronchodilators) or inhibit the parasympathetic stimulation of such contraction (anticholinesterases). Recently, the realization that asthma is primarily an inflammatory disease has led to new treatments using anti-inflammatory drugs (glucocorticoids and nonsteroidal anti-inflammatory agents). These drugs allow much better long-term management, leading to fewer asthma attacks and less damage to the airways.

Cystic Fibrosis

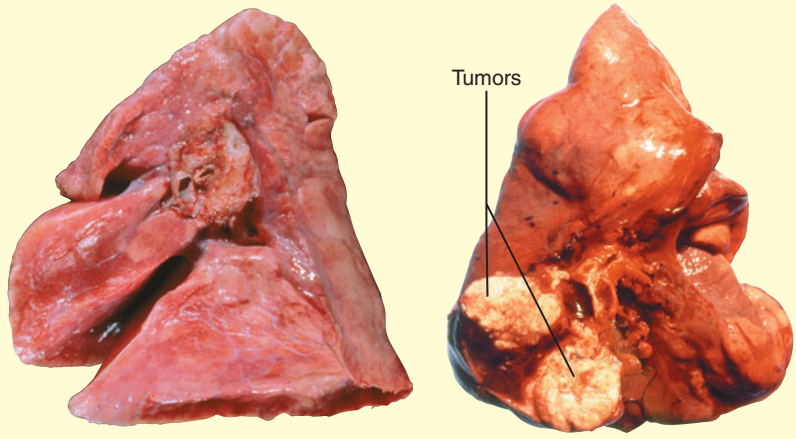
Cystic fibrosis (CF) is an inherited disease in which the functions of exocrine glands are disrupted throughout the body. Occurring mainly in people of European descent, it kills 1 of every 2400 Americans. CF affects the respiratory system by causing an oversecretion of a viscous mucus by the bronchial glands. This mucus clogs the respiratory passageways and acts as a feeding ground for bacteria, predisposing the child to early death due to respiratory infections.

In the 1980s, researchers isolated the gene responsible for cystic fibrosis (the *cftr* gene) and showed that a defect in this gene prevents the formation of a membrane channel that carries chloride ions into epithelial cells, so that the fluid covering the respiratory epithelium becomes a salty brine. A natural antibiotic produced by the epithelium is ineffective under such salty conditions. As a result, bacteria thrive, leading to inflammation and the excessive secretion of mucus. Despite

a closer look

Lung Cancer: The Facts Behind the Smoke Screen

Lung cancer accounts for fully one-third of all cancer deaths in the United States, and its incidence is increasing daily. The most common malignancy in both sexes, it is notoriously deadly. Most types of lung cancer are tremendously aggressive and metastasize rapidly and widely, and most cases cannot be diagnosed until they are far advanced. Whole-lung CT scans to detect early-stage tumors may improve treatment outcomes. This screening test is currently in clinical trial.



Medial view of lungs: healthy lung on left, cancerous lung on right.

For years, Americans were remarkably unaware of the link between lung cancer and cigarette smoking, despite the fact that over 90% of lung cancer patients are smokers. Smoking even a single cigarette increases the heart rate, constricts peripheral blood vessels throughout the body, disrupts the flow of air in the lungs, and affects brain and mood. In adolescents, that first cigarette can trigger addiction. In a study of sixth graders in Massachusetts, of those who developed physical symptoms of dependence, 10% developed symptoms after their first cigarette and 25% within 2 weeks of intermittent cigarette use!

Long-term smoking contributes to atherosclerosis and heart disease, strokes, cataracts, and osteoporosis. In addition to causing lung cancer, smoking raises the risk of cancers of the mouth, throat, esophagus, stomach, liver, pancreas, cervix, kidney, and bladder. Smoking contributes to one-fifth of all deaths in the United States, making it the single most preventable cause of death.

Ordinarily, sticky mucus and the action of cilia do a fine job of protecting the lungs from chemical and biological irritants, but smoking overwhelms these cleansing devices until they eventually become nonfunctional. Even though continuous irritation prompts the production of more mucus, smoking slows the movements of cilia that clear this mucus and depresses the activity of lung macrophages. Mucus pools in the lower respiratory tree and pulmonary illnesses, including pneumonia and COPD, become more frequent. However, it is the 15 or so carcinogens in tobacco smoke, including the highly addictive nicotine, that ultimately lead to lung cancer. These chemicals, plus the tars in tobacco, eventually cause the epithelial cells lining the bronchial tree to proliferate wildly and lose their characteristic structure. Ironically, the nicotine in tobacco smoke may be carcinogenic *because* it promotes cell survival. Normally, damaged cells either repair the damage or die, but nicotine appears to activate enzymes that disrupt the sequence of apoptosis.

The three most common types of lung cancer are (1) **squamous cell carcinoma** (25% to 30% of cases), which arises in the epithelium of the larger bronchi and tends to form masses that cavitate and bleed; (2) **adenocarcinoma** (40%), which originates in the peripheral areas of the lung as solitary nodules that develop from bronchial mucous glands and alveolar epithelial cells; and (3) **small cell carcinoma** (15% of cases, but rapidly increasing), which contains lymphocyte-like epithelial cells that originate in the main bronchi and grow aggressively in cords or small grapelike clusters within the mediastinum, a site from which metastasis is especially rapid.

The most effective treatment for lung cancer is complete removal of the diseased lung. However, removal is an option open to very few patients because metastasis has usually occurred by the time of diagnosis, and most patients' chances of survival are too poor to justify the surgery. In most cases, radiation therapy and chemotherapy are the only options, but most lung cancers are resistant to these treatments. Only small cell carcinoma responds to chemotherapy, but frequently it returns quickly and spawns brain tumors.

The good news is that quitting helps. While the incidence of lung cancer is 20 times greater in smokers than in nonsmokers, this ratio drops to 2:1 for former smokers who have not smoked in 15 years. In a British study, people who stopped smoking before age 35 lived full life spans, and quitting at any age prolonged survival. Overall, 48.8% of U.S. adults who ever smoked cigarettes have quit. Many succeeded by quitting "cold turkey," and others benefited from aids such as nicotine nasal sprays, patches, and inhalers.

this knowledge of the cause of CF, attempts to cure it by delivering nondefective copies of the *cftr* gene to the affected respiratory epithelial cells have not succeeded. Modern antibiotics allow the average CF patient to survive until age 30, an increase in life span of 16 years since 1969. For information on the nonrespiratory effects of CF, see p. 700.

Chronic Obstructive Pulmonary Disease

Chronic obstructive pulmonary disease (COPD) is a category of disorders in which the flow of air into and out of the lungs is difficult or obstructed. It mostly refers to *chronic bronchitis* or *obstructive emphysema* (or both of these occurring together), and it is a major cause of death and disability in the United States. These two diseases share certain features: The patients almost always have a history of smoking; they experience difficult or labored breathing called **dyspnea** (disp-ne′ah; "bad breathing"); coughing and pulmonary infections occur frequently; and most COPD victims ultimately develop respiratory failure **(Figure 22.18)**.

In **chronic bronchitis,** inhaled irritants lead to a prolonged secretion of excess mucus by the mucosa of the lower respiratory passages and to inflammation and fibrosis (formation of scar tissue) of this mucosa. These responses obstruct the airways, severely impairing ventilation and gas exchange. Infections frequently develop because bacteria and viruses thrive in the stagnant pools of mucus. Coughing is especially persistent and productive. Patients with chronic bronchitis are sometimes called "blue bloaters" because lowered blood oxygenation often results in cyanosis and other signs of right heart failure, including edema. However, the degree of dyspnea is usually moderate compared to that of emphysema sufferers. So significant is cigarette smoking as a causative factor of chronic bronchitis that this disease would be an insignificant health problem if cigarettes were unavailable. Air pollution is another, although minor, causative factor.

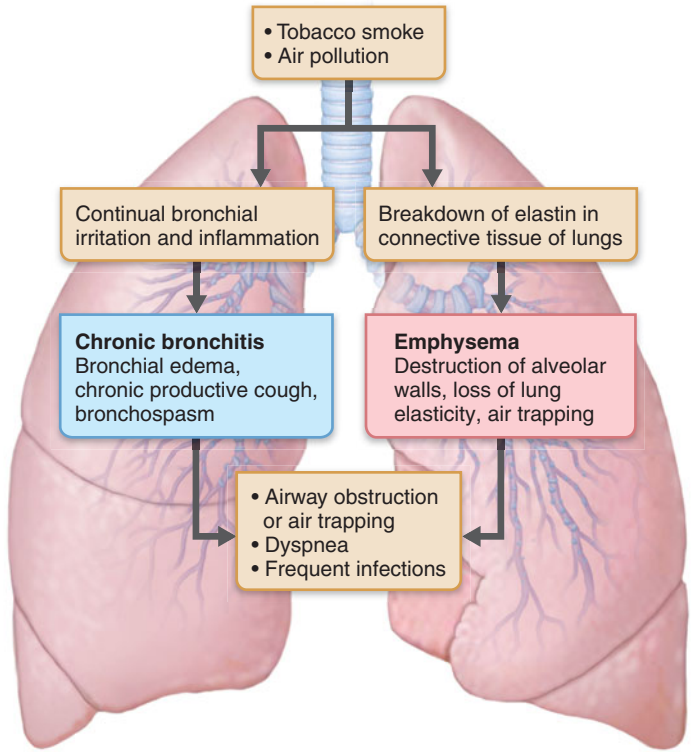
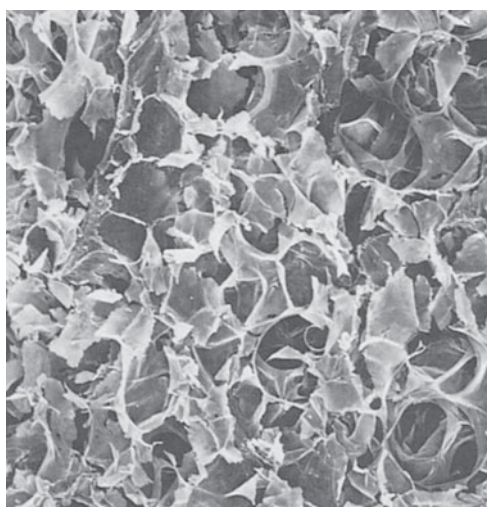
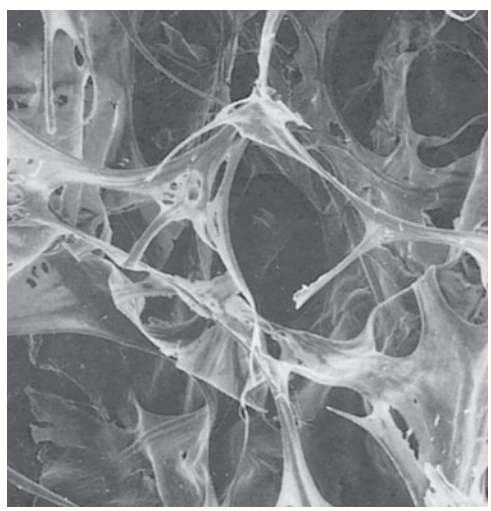


FIGURE 22.18 The pathogenesis of chronic obstructive pulmonary disease (COPD).

Obstructive emphysema (em″fĭ-se′mah; "to inflate") is characterized by a permanent enlargement of the alveoli caused by a deterioration of the alveolar walls **(Figure 22.19)**. The disease is most often associated with a smoking-related chronic inflammation of the lungs and increased activity of lung macrophages, whose lysosomal enzymes seem responsible for destroying the alveolar walls and breaking down elastin. Chronic inflammation also leads to fibrosis, and the lungs become progressively less elastic,

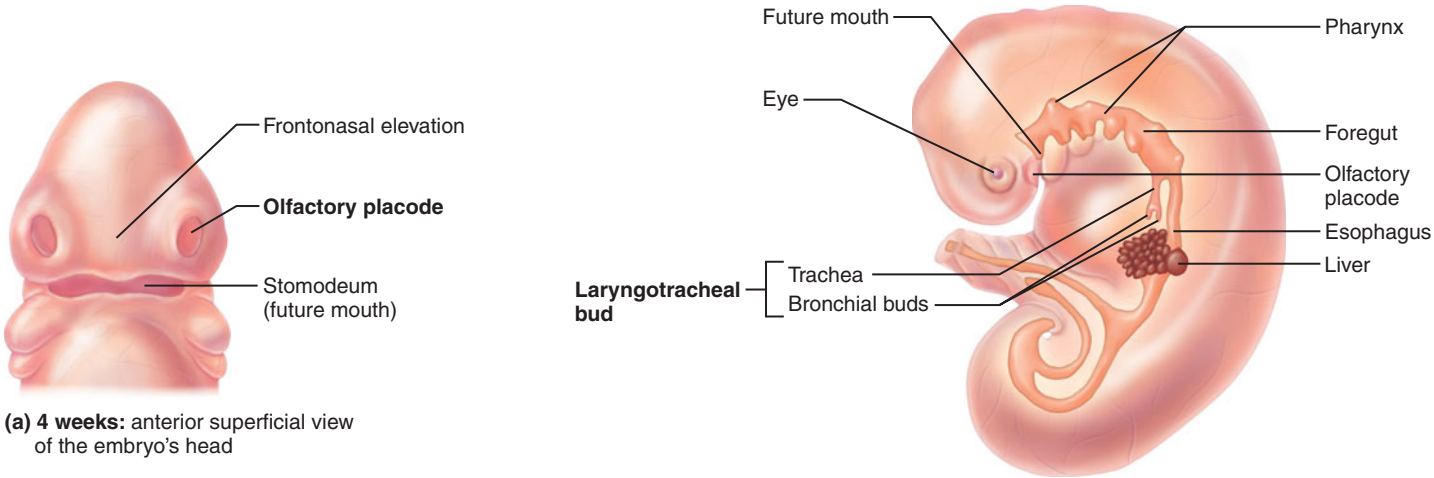


(a) Scanning electron micrograph of alveoli from a normal lung (6×)



(b) Scanning electron micrograph of alveoli from the lung of a patient with emphysema (6×)

FIGURE 22.19 Alveolar changes in emphysema.



(a) 4 weeks: anterior superficial view of the embryo's head

(b) 5 weeks: left lateral view of the developing lower respiratory passageway mucosae

FIGURE 22.20 Embryonic development of the respiratory system.

making expiration difficult and exhausting. For complex reasons, the bronchioles open during inspiration but collapse during expiration, trapping huge volumes of air in the alveoli. This enlarges the lung, leads to the development of an expanded "barrel chest," and flattens the diaphragm, which decreases ventilatory efficiency. Damage to the lung capillaries increases the resistance in the pulmonary vascular circuit, forcing the heart's right ventricle to enlarge through overwork.

COPD is routinely treated with bronchodilators and anti-inflammatory drugs (including glucocorticoids) in aerosol form. Severe dyspnea and lowered uptake of oxygen nearly mandates supplemental oxygen use. A treatment used initially in the late 1950s and recently reintroduced, called *lung volume reduction surgery,* has been performed on some emphysema patients. In this surgery, part of the grossly enlarged lung is removed to give the remaining lung tissue room to expand. This surgery has improved the breathing capacity and the quality of life in moderately ill patients, but it is less beneficial in patients with less severe emphysema.

Epistaxis

Epistaxis (ep″ĭ-stak′sis; "to bleed from the nose") is a nosebleed, or nasal hemorrhage. It commonly follows trauma to the nose, excessive nose blowing or nose picking, or drying of the mucous membrane lining the nasal cavity, as can occur in dry climates or during the winter heating months in cold climates. Most nasal bleeding is from the highly vascularized *anterior* part of the nasal septum and can be stopped by pinching the nostrils together or packing them with cotton. Bleeding from the *posterior* part of the nasal cavity, however, is less common but more serious because it usually involves more bleeding that is harder to stop. Posterior nasal bleeding usually indicates a cardiovascular disease such as high blood pressure or a clotting disorder.

check your understanding

16. Which of the described disorders results in damage to the respiratory zone?
17. What causes "smoker's cough," the persistent cough that commonly afflicts long-term or heavy smokers?

For answers, see Appendix B.

THE RESPIRATORY SYSTEM THROUGHOUT LIFE

- Trace the development of the respiratory system in the embryo and fetus.
- Describe the normal changes that occur in the respiratory system from infancy to old age.

Because embryos develop in a craniocaudal (head-to-tail) direction, the upper respiratory structures appear before the lower ones. By week 4, a thickened plate of ectoderm called the **olfactory placode** (plak′ōd; "plate") has appeared on each side of the future face **(Figure 22.20a)**. These placodes quickly invaginate to form *olfactory pits* that form the nasal cavity, including the olfactory epithelium in its roof. The nasal cavity then connects with the future pharynx of the developing foregut, which forms at the same time.

The lower respiratory organs develop from a tubular outpocketing off the pharyngeal foregut called the **laryngotracheal bud** (Figure 22.20b). The proximal part of this bud forms the trachea, and its distal part branches repeatedly to form the bronchi and their subdivisions, including (eventually) the lung alveoli. The respiratory tubes, like the gut tube from which they arise, are lined by endoderm and covered by splanchnic mesoderm. The endoderm becomes the lining epithelium (and glands) of the trachea, bronchial

tree, and alveoli. The splanchnic mesoderm gives rise to all other layers of the tracheal and bronchial walls (cartilage, smooth muscle, and all connective tissues) and to the stroma of the lungs.

The respiratory system reaches functional maturity relatively late in development. No alveoli appear until month 6 (24 weeks), and the alveolar type I cells do not attain their extreme thinness until the time of birth. Type II alveolar cells begin to produce surfactant by week 26. It is not until 26–30 weeks that a prematurely born baby can survive and breathe on its own. Infants born before this time are most severely threatened by respiratory distress syndrome resulting from inadequate production of surfactant.

During fetal life, the lungs are filled with fluid, and all respiratory exchange occurs across the placenta. At birth, the first breaths bring air into the lungs, and the alveoli inflate and begin to function in gas exchange. During the nearly 2 weeks it takes for the lungs to become fully inflated, the fluid that originally filled the lungs is absorbed into the alveolar capillaries.

At birth, only one-sixth of the final number of lung alveoli are present. The lungs continue to mature throughout childhood, and more alveoli are formed until young adulthood. Research has revealed that in individuals who begin smoking in their early teens, the lungs never completely mature, and those additional alveoli never form.

Under normal conditions, the respiratory system works so efficiently that people are not even aware of it. Most problems that occur are the result of external factors, such as viral or bacterial infections, irritants that trigger asthma in susceptible individuals, or obstruction of the trachea by a piece of food. For many years, tuberculosis and bacterial pneumonia (discussed in the "Related Clinical Terms" section at the end of this chapter) were the worst killers in the United States and Europe; even though antibiotics have decreased their threat to a large extent, they are still dangerous diseases. Influenza, a respiratory flu virus, killed at least 20 million people worldwide in 1918, and each year government health agencies keep a watchful eye on outbreaks caused by new viral strains. Despite the attention given to such dramatic illnesses, by far the most troublesome respiratory disorders are smoking-related COPD and lung cancer.

As humans age, the number of glands in the nasal mucosa declines, as does the flow of blood in this mucosa. Thus, the nose dries and crusts and produces a thickened mucus that makes one want to "clear the throat." In the elderly, the thoracic wall grows more rigid, and the lungs gradually lose their elasticity, resulting in a slowly declining ability to ventilate the lungs. The levels of oxygen in the blood may fall slightly while levels of carbon dioxide rise. Additionally, just as the overall efficiency of the immune system declines with age, many of the respiratory system's protective mechanisms become less effective—the activity of the cilia in its epithelial lining decreases, and the macrophages in the lungs become sluggish. The net result is that the elderly are at greater risk for respiratory infections, particularly pneumonia and influenza.

check your understanding

18. Why do babies born earlier than 26 weeks experience severe respiratory distress?

For the answer, see Appendix B.

RELATED CLINICAL TERMS

ATELECTASIS (at″ĕ-lek′tah-sis) Collapse of the lung, either from airway obstruction or from the compression of pleural effusion. Can also refer to a failure of the lungs to inflate, as in premature infants.

BRONCHOSCOPY (*scopy* = viewing) Use of a viewing tube to examine the internal surface of the main bronchi in the lung. The tube is inserted through the nose or mouth and guided inferiorly through the larynx and trachea. Forceps may be attached to the tip of the tube to remove trapped objects, take biopsy samples, or retrieve samples of mucus for examination.

CROUP Disease in children in which viral-induced inflammation causes the air passageways to narrow; is characterized by coughing that sounds like the bark of a dog, hoarseness, and wheezing or grunting sounds during inspiration. Most cases resolve after a few days, but severe cases may require anti-inflammatory aerosols or a tracheotomy (see p. 644) to bypass the obstructed upper respiratory tubes.

PNEUMONIA An infectious inflammation of the lungs in which fluid accumulates in the alveoli. Of the over 50 known varieties, most are caused by viruses or bacteria (however, the type of pneumonia associated with AIDS, *Pneumocystis carinii* pneumonia, is caused by a fungus). Extremely common, pneumonia is the sixth most frequent cause of death in the United States because almost any severely ill person can develop it.

SUDDEN INFANT DEATH SYNDROME (SIDS) Unexpected death of an apparently healthy infant during sleep. Commonly called *crib death,* SIDS is the most frequent cause of death in infants under 1 year old. The cause is unknown, but it may reflect immaturity of the brain's respiratory control centers. Since 1992, a campaign to have babies sleep on their backs instead of on their bellies has led to a decline of 40% or more in the incidence of SIDS in the United States.

TUBERCULOSIS (TB) A lung disease caused by the bacterium *Mycobacterium tuberculosis,* which is spread by coughing and enters the body in inhaled air. TB typically affects the lungs but can spread through lymphatic vessels to other organs. A massive inflammatory and immune response contains the primary infection within fibrous or calcified nodules in the lungs (tubercles), but the bacteria often survive, break out, and cause repeated infections. Symptoms of TB are coughing, weight loss, mild fever, and chest pain. Some strains of TB are now resistant to antibiotics, and TB cases are increasing in the United States. Current vaccines protect more than half of the children who receive them, but are not effective in adults.

CHAPTER SUMMARY

You can use the following media study tool for additional help when you review specific key topics in Chapter 22.

PAL = Practice Anatomy Lab™

1. Respiration involves the delivery of O_2 to the body cells for use in energy production and the removal of the waste product CO_2. The respiratory processes are as follows: pulmonary ventilation, external respiration, cardiovascular transport of respiratory gases, internal respiration, and cellular respiration. Both the respiratory system and the cardiovascular system participate in respiration.

Functional Anatomy of the Respiratory System (pp. 636–654)

2. The organs of the respiratory system include a conducting zone (nose to terminal bronchioles), which warms, moistens, and filters the inhaled air, and a respiratory zone (respiratory bronchioles to alveoli), where gas exchange occurs.

The Nose and the Paranasal Sinuses (pp. 637–640)

3. The nose and nasal cavity provide an airway for respiration and house the olfactory receptors.
4. The external nose is shaped by bone and by cartilage plates. The nasal cavity begins at the external nares and ends posteriorly at the posterior nasal apertures (choanae). Air-swirling conchae occupy its lateral walls. The paranasal sinuses drain into the nasal cavity.
5. The respiratory mucosa lines the nasal cavity and paranasal sinuses. Its epithelium, a pseudostratified ciliated columnar epithelium with goblet cells, is covered with a sheet of mucus that filters dust particles and moistens inhaled air. Its lamina propria contains glands that contribute to the mucus sheet and blood vessels that warm the air.

The Pharynx (pp. 640–641)

6. The pharynx has three regions: The nasopharynx (behind the nasal cavity) is an air passageway; the oropharynx (behind the mouth) and the laryngopharynx (behind the larynx) are passageways for both food and air.
7. The soft palate moves superiorly to seal off the nasopharynx during swallowing. The tubal and pharyngeal tonsils occupy the mucosa that lines the nasopharynx. The oropharynx contains the lingual and palatine tonsils. The laryngopharynx opens into the larynx anteriorly and into the esophagus inferiorly.

The Larynx (pp. 641–644)

8. The larynx, or voice box, is the entryway to the trachea and lower respiratory tubes. The larynx has a skeleton of nine cartilages: the thyroid; the cricoid; the paired arytenoid, corniculate, and cuneiform cartilages; and the epiglottis.
9. The leaf-shaped epiglottis acts as a lid that prevents food or liquids from entering the lower respiratory channels. The larynx moves superiorly under this protective flap during swallowing.
10. The larynx contains the vocal folds (vocal cords). Exhaled air causes these folds to vibrate, producing the sounds of speech. The vocal folds extend anteriorly from the arytenoids to the thyroid cartilage, and muscles that move the arytenoids change the tension on the folds to vary the pitch of the voice. The pharynx, nasal cavity, lips, and tongue also aid in vocal articulation.
11. The main sensory and motor nerves of the larynx (and of speech) are the left and right recurrent laryngeal nerves, which are branches of the vagus nerves.

The Trachea (pp. 644–645)

12. The trachea, which extends from the larynx in the neck to the main bronchi in the thorax, is a tube reinforced by C-shaped cartilage rings, which keep it open. The trachealis muscle narrows the trachea, increasing the speed of airflow during coughing and sneezing.
13. The wall of the trachea contains several layers: a mucous membrane, a submucosa, a fibromusculocartilaginous layer, and an outer adventitia. The mucous membrane consists of an air-filtering pseudostratified ciliated columnar epithelium and a lamina propria rich in elastic fibers.

The Bronchial Tree (pp. 645–649)

14. The right and left main (primary) bronchi supply the lungs. Within the lungs, they subdivide into lobar (secondary) bronchi to the lobes, segmental (tertiary) bronchi to the bronchopulmonary segments, and finally to bronchioles and terminal bronchioles.
15. As the respiratory tubes become smaller, the cartilage in their walls is reduced and finally lost (in bronchioles); the epithelium thins and loses its air-filtering function (in the terminal bronchioles); smooth muscle becomes increasingly important; and elastic fibers continue to surround all of the tubes.
16. The terminal bronchioles lead into the respiratory zone, which consists of respiratory bronchioles, alveolar ducts, and alveolar sacs, all of which contain tiny chambers called alveoli. Gas exchange occurs in the alveoli, across the thin respiratory membrane. The respiratory membrane consists of alveolar type I cells, fused basement membranes, and capillary endothelial cells.
17. Alveoli also contain alveolar type II cells, which secrete surfactant, and freely wandering alveolar macrophages that remove dust particles that reach the alveoli.

The Lungs and Pleurae (pp. 649–653)

18. The lungs flank the mediastinum in the thoracic cavity. Each lung is suspended in a pleural cavity by its root (the vessels supplying it) and has a base, an apex, and medial and costal surfaces. The root structures enter the lung hilum.
19. The right lung has three lobes (superior, middle, and inferior lobes, as defined by the oblique and horizontal fissures); the left lung has two lobes (superior and inferior lobes, as defined by the oblique fissure). The lobes are divided into bronchopulmonary segments, which are supplied by segmental bronchi.
20. The lungs consist primarily of air tubes and alveoli, but they also contain a stroma of elastic connective tissue.
21. The pulmonary arteries carry deoxygenated blood and generally lie posterior to their corresponding bronchi within the lungs. The pulmonary veins, which carry oxygenated blood, tend to lie anterior to their corresponding bronchi. The lungs are innervated by sympathetic and parasympathetic fibers through the pulmonary plexus.
22. Pleurae are serous membranes. The parietal pleura lines the thoracic wall, diaphragm, and mediastinum; the visceral pleura covers the external surfaces of the lungs. The slitlike pleural cavity between the parietal and visceral pleura is filled with a serous fluid

that holds the lungs to the thorax wall and reduces friction during breathing movements. Inferiorly, the pleural cavity extends below the inferior border of the lungs.

PAL **Human Cadaver/Respiratory System**

Ventilation (pp. 653–657)

The Mechanism of Ventilation (pp. 653–656)

23. Ventilation consists of inspiration and expiration. Inspiration occurs when the diaphragm and intercostal muscles contract, increasing the volume of the thorax. As the intrathoracic pressure drops, pressure in the lungs also drops and air rushes into the lungs.

24. Expiration is largely passive, occurring as the inspiratory muscles relax. The volume of the thorax decreases and the lungs recoil elastically, raising pressure and expelling air from the lungs.

25. Surface tension of the alveolar fluid threatens to collapse the alveoli after each breath. This tendency is resisted by surfactant secreted by alveolar type II cells.

Neural Control of Ventilation (pp. 656–657)

26. The main respiratory control center is in the reticular formation of the medulla oblongata, although this center is influenced by input from the pons, limbic system, hypothalamus, and cerebral cortex.

27. Chemoreceptors monitor concentrations of respiratory gases and acid in the blood. The central chemoreceptors are neurons in the medulla, and the peripheral chemoreceptors are the carotid and aortic bodies.

Disorders of the Respiratory System (pp. 657–660)

28. Bronchial asthma is a chronic, allergic inflammatory condition.

29. Cystic fibrosis is an inherited disease in which the functions of the body's exocrine glands are disrupted. It involves an oversecretion of a viscous mucus in the bronchi, in which bacteria grow, leading to early death through respiratory infection.

30. Chronic bronchitis, a COPD, is characterized by an excessive production of mucus in the lower respiratory passages, which impairs ventilation and leads to infection. Patients may become cyanotic as a result of low oxygen levels in the blood.

31. Obstructive emphysema, also a COPD, is characterized by permanent enlargement and destruction of alveoli. The lungs lose elasticity, and expiration becomes an active, exhaustive process.

32. Most lung cancers are caused by smoking. They are extremely aggressive, metastasize rapidly, and are most often fatal.

33. Epistaxis (nosebleed) results from trauma to the nose or drying of the nasal mucosa.

The Respiratory System Throughout Life (pp. 660–661)

34. The nasal cavity develops from the olfactory placodes; the pharynx forms as part of the foregut; and the lower respiratory tubes grow from an outpocketing of the embryonic pharynx (laryngotracheal bud). The epithelium lining the pharynx and the lower respiratory tubes derives from endoderm. Mesoderm forms all other parts of these tubes and the lung stroma as well.

35. The respiratory system completes its development very late in the prenatal period: No alveoli or surfactant appear until month 6, and only one-sixth of the final number of alveoli are present at birth. Respiratory immaturity, especially a lack of surfactant, is a serious complication of premature birth.

36. With age, the nose dries, the thorax becomes more rigid, the lungs become less elastic, and ventilation capacity declines. Respiratory infections become more common in old age.

REVIEW QUESTIONS

Multiple Choice/Matching Questions

For answers, see Appendix B.

1. When the inspiratory muscles contract, (a) only the lateral dimension of the thoracic cavity increases, (b) only the anteroposterior dimension of the thoracic cavity increases, (c) the volume of the thoracic cavity decreases, (d) both the lateral and the anteroposterior dimensions of the thoracic cavity increase, (e) the diaphragm bulges superiorly.

2. The part of the respiratory mucosa that warms the inhaled air is the (a) pseudostratified epithelium, (b) vessels in the lamina propria, (c) alveolar type I cells, (d) cartilage and bone.

3. Which of the following statements about the vocal cords is *false*? (a) They are the same as the vocal folds. (b) They attach to the arytenoid cartilages. (c) Exhaled air flowing through the glottis vibrates them to produce sound. (d) They are also called the vestibular folds.

4. In both lungs, the surface that is the largest is the (a) costal, (b) mediastinal, (c) inferior (base), (d) superior (apex).

5. Match the proper type of lining epithelium from the key with each of the following respiratory structures.

Key: a. stratified squamous c. simple squamous
b. pseudostratified columnar d. stratified columnar

____ **(1)** nasal cavity

____ **(2)** nasopharynx

____ **(3)** laryngopharynx

____ **(4)** trachea and bronchi

____ **(5)** alveoli (type I cells)

6. Match the air tube in column B with the lung region in column A supplied by that air tube.

Column A	**Column B**
____ **(1)** bronchopulmonary segment	**(a)** main bronchus
____ **(2)** lobule	**(b)** lobar bronchus
____ **(3)** alveolar ducts and sacs	**(c)** segmental bronchus
____ **(4)** whole lung	**(d)** large bronchiole
____ **(5)** lobe	**(e)** respiratory bronchiole

7. The respiratory membrane consists of (a) alveolar type I cell, basement membranes, endothelial cell; (b) air, connective tissue, lung; (c) type II cell, macrophage, type I cell; (d) pseudostratified epithelium, lamina propria, capillaries.

8. The trachealis muscle and the smooth muscle around the bronchi develop from which of the following embryonic layers? (a) ectoderm, (b) endoderm, (c) mesoderm, (d) neural crest.

9. A serous cell of a gland secretes (a) the slippery serous fluid in the body cavities, (b) mucus, (c) a watery lubricating fluid, (d) tissue fluid.
10. The function of alveolar type I cells is to (a) produce surfactant, (b) propel sheets of mucus, (c) remove dust particles through phagocytosis, (d) allow rapid diffusion of respiratory gases.

Short Answer Essay Questions

11. Trace the route of exhaled air from an alveolus to the external nares, naming all the structures through which the air passes. Then indicate which of these structures are in the respiratory zone and which are in the conducting zone.
12. The cilia lining the upper respiratory passages (superior to the larynx) beat inferiorly, whereas the cilia lining the lower respiratory passages (larynx and inferior) beat superiorly. What is the functional reason for this difference?
13. What is the function of the abundant elastin fibers that occur in the stroma of the lung and around all respiratory tubes from the trachea through the respiratory tree?
14. (a) Which tonsils lie in the fauces of the oropharynx? (b) Does the lingual tonsil lie near the superior part of the epiglottis? Explain.
15. (a) It is easy to confuse the hilum of the lung with the root of the lung. Define both structures, and contrast hilum and root. (b) Define the parietal pleura and the visceral pleura.
16. The three terms choanae, conchae, and carina are easily confused. Define each of them, clarifying the differences.
17. Briefly explain the anatomical reason why most men have deeper voices than boys or women.
18. Sketch a picture of the right and left lungs in anterior view, showing all the fissures and lung lobes, as well as the cardiac notch.
19. Explain the functions of the pseudostratified epithelium of the respiratory mucosa.
20. What is the function of the alveolar macrophages in the lung alveoli?

CRITICAL REASONING & CLINICAL APPLICATION QUESTIONS

1. (a) Two girls in a high school cafeteria were giggling over lunch, and milk accidentally sprayed out of the nostrils of one of them. Explain in anatomical terms why swallowed fluids can sometimes come out the nose. (b) A boy in the same cafeteria then stood on his head to show he could drink milk upside down without any of it entering his nasal cavity or nose. What prevented the milk from flowing downward into his nose?
2. A surgeon had to remove three adjacent bronchopulmonary segments from the left lung of a patient with tuberculosis. Even though almost half of the lung was removed, there was no severe bleeding, and relatively few blood vessels had to be cauterized (closed off). Why was this surgery so easy to perform?
3. While getting his 1-year-old daughter (who puts almost everything in her mouth) ready for bed, Mr. Gregoire could not find one of the small barrettes he had just removed from her hair. Two days later, she developed a cough and became feverish. What probably had happened to the barrette, and where (anatomically) would you expect to find it?
4. A taxi driver was carried into an emergency room after being knifed once in the left side of the thorax. The diagnosis was pneumothorax and a collapsed lung. (a) Explain exactly why the lung collapsed. (b) Explain why only one lung (not both) collapsed.
5. A man was driving to work when his car was hit broadside by a car that ran a red light. When freed from the wreckage, the man was deeply cyanotic, and his breathing had stopped. His heart was still beating, but his pulse was fast and thready. His head was cocked at a peculiar angle, and the emergency personnel said he seemed to have a fracture at the level of vertebra C_2. (a) How might these findings account for the cessation of breathing? (b) Define cyanosis (see Chapter 5), and indicate why the man was cyanotic.
6. After a 4-year-old child had surgery that successfully closed off a patent ductus arteriosus (see p. 612), the child's voice was hoarse, and an examination revealed that many muscles in the left half of the larynx were paralyzed. What had gone wrong during surgery to cause the paralysis?