Mechanics of Breathing

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Background Basics
- Ciliated and exchange epithelia
- Pressure, volume, flow, and resistance
- Pulmonary circulation
- Surface tension
- Autonomic and somatic motor neurons
- Velocity of flow

This being of mine, whatever it really is, consists of a little flesh, a little breath, and the part which governs.
— Marcus Aurelius Antoninus (C.E. 121–180)
Imagine covering the playing surface of a racquetball court (about 75 m²) with thin plastic wrap, then crumpling up the wrap and stuffing it into a 3-liter soft drink bottle. Impossible? Maybe so, if you use plastic wrap and a drink bottle. But the lungs of a 70-kg man have a gas exchange surface the size of that plastic wrap, compressed into a volume that is less than that of the bottle. This tremendous surface area for gas exchange is needed to supply the trillions of cells in the body with adequate amounts of oxygen.

Aerobic metabolism in cells depends on a steady supply of oxygen and nutrients from the environment, coupled with the removal of carbon dioxide. In very small aquatic animals, simple diffusion across the body surface meets these needs. Distance limits diffusion rate, however, so most multicelled animals require specialized respiratory organs associated with a circulatory system. Respiratory organs take a variety of forms, but all possess a large surface area compressed into a small space.

Besides needing a large exchange surface, humans and other terrestrial animals face an additional physiological challenge: dehydration. The exchange surface must be thin and moist to allow gases to pass from air into solution, and yet at the same time it must be protected from drying out as a result of exposure to air. Some terrestrial animals, such as the slug (a shell-less snail), meet the challenge of dehydration with behavioral adaptations that restrict them to humid environments and nighttime activities.

A more common solution is anatomical: an internalized respiratory epithelium. Human lungs are enclosed in the chest cavity to control their contact with the outside air. Internalization creates a humid environment for the exchange of gases with the blood and protects the delicate exchange surface from damage.

Internalized lungs create another challenge, however: how to move air between the atmosphere and an exchange surface deep within the body. Air flow requires a muscular pump to create pressure gradients. More complex respiratory systems therefore consist of two separate components: a muscle-driven pump and a thin, moist exchange surface. In humans, the pump is the musculoskeletal structure of the thorax. The lungs themselves consist of the exchange epithelium and associated blood vessels.

The four primary functions of the respiratory system are:

1. **Exchange of gases between the atmosphere and the blood.** The body brings in O₂ for distribution to the tissues and eliminates CO₂ waste produced by metabolism.
2. **Homeostatic regulation of body pH.** The lungs can alter body pH by selectively retaining or excreting CO₂.
3. **Protection from inhaled pathogens and irritating substances.** Like all other epithelia that contact the external environment, the respiratory epithelium is well supplied with defense mechanisms to trap and destroy potentially harmful substances before they can enter the body.
4. **Vocalization.** Air moving across the vocal cords creates vibrations used for speech, singing, and other forms of communication.

In addition to serving these functions, the respiratory system is also a significant source of water loss and heat loss from the body. These losses must be balanced using homeostatic compensations.

In this chapter you will learn how the respiratory system carries out these functions by exchanging air between the environment and the interior air spaces of the lungs. This exchange is the bulk flow of air, and it follows many of the same principles that govern the bulk flow of blood through the cardiovascular system:

1. **Flow takes place from regions of higher pressure to regions of lower pressure.**
2. A muscular pump creates pressure gradients.
3. Resistance to air flow is influenced primarily by the diameter of the tubes through which the air is flowing.

Air and blood are both fluids. The primary difference between air flow in the respiratory system and blood flow in the circulatory system is that air is a less viscous, compressible mixture of gases while blood is a noncompressible liquid.

### The Respiratory System

The word respiration has several meanings in physiology (Fig. 17.1). **Cellular respiration** refers to the intracellular reaction of oxygen with organic molecules to produce carbon...
Mechanics of Breathing consists of structures involved in ventilation and gas exchange (Fig. 17.2):

1. **The conducting system** of passages, or airways, that lead from the external environment to the exchange surface of the lungs.
2. The **alveoli** (singular alveolus) {alveus, a concave vessel}, a series of interconnected sacs and their associated pulmonary capillaries. These structures form the exchange surface, where oxygen moves from inhaled air to the blood, and carbon dioxide moves from the blood to air that is about to be exhaled.
3. The bones and muscles of the thorax (chest cavity) and abdomen that assist in ventilation.

The respiratory system can be divided into two parts. The **upper respiratory tract** consists of the mouth, nasal cavity, pharynx, and larynx. The **lower respiratory tract** consists of the trachea, two primary bronchi {bronchos, windpipe; singular—bronchus}, their branches, and the lungs. The lower tract is also known as the thoracic portion of the respiratory system because it is enclosed in the thorax.

### Bones and Muscles of the Thorax

**Surround the Lungs**

The thorax is bounded by the bones of the spine and rib cage and their associated muscles. Together the bones and muscles are called the thoracic cage. The ribs and spine (the chest wall) form the sides and top of the cage. A dome-shaped sheet of skeletal muscle, the **diaphragm**, forms the floor (Fig. 17.2b).

Two sets of **intercostal muscles**, internal and external, connect the 12 pairs of ribs (Fig. 17.2a). Additional muscles, the **sternocleidomastoids** and the **scalenes**, run from the head and neck to the sternum and first two ribs.

Functionally, the thorax is a sealed container filled with three membranous bags, or sacs. One, the **pericardial sac**, contains the heart. The other two bags, the **pleural sacs**, each surround a lung {pleura, rib or side}. The esophagus and thoracic blood vessels and nerves pass between the pleural sacs (Fig. 17.2c).

### Pleural Sacs Enclose the Lungs

The **lungs** (Fig. 17.2b, d) consist of light, spongy tissue whose volume is occupied mostly by air-filled spaces. These irregular cone-shaped organs nearly fill the thoracic cavity, with their bases resting on the curved diaphragm. Semi-rigid conducting airways—the bronchi—connect the lungs to the main airway, the trachea.

Each lung is surrounded by a double-walled pleural sac whose membranes line the inside of the thorax and cover the outer surface of the lungs (Fig. 17.3). Each pleural membrane, or **pleura**, contains several layers of elastic...
connective tissue and numerous capillaries. The opposing layers of pleural membrane are held together by a thin film of pleural fluid whose total volume is only about 25–30 mL in a 70-kg man. The result is similar to an air-filled balloon (the lung) surrounded by a water-filled balloon (the pleural sac). Most illustrations exaggerate the volume of the pleural fluid, but you can appreciate its thinness if you imagine spreading 25 mL of water evenly over the surface of a 3-liter soft drink bottle.

Pleural fluid serves several purposes. First, it creates a moist, slippery surface so that the opposing membranes can slide across one another as the lungs move within the thorax. Second, it holds the lungs tight against the thoracic wall. To visualize this arrangement, think of two panes of glass stuck together by a thin film of water. You can slide the panes back and forth across each other, but you cannot pull them apart because of the cohesiveness of the water. A similar fluid bond between the two pleural membranes makes the lungs “stick” to the thoracic cage and holds them stretched in a partially inflated state, even at rest.

**Airways Connect Lungs to the External Environment**

Air enters the upper respiratory tract through the mouth and nose and passes into the pharynx, a common passageway for food, liquids, and air (pharynx, throat). From the pharynx, air flows through the larynx into the trachea, or windpipe (Fig. 17.2b). The larynx contains the vocal cords, connective tissue bands that vibrate and tighten to create sound when air flows through them.

The trachea is a semiflexible tube held open by 15 to 20 C-shaped cartilage rings. It extends down into the thorax, where it branches (division 1) into a pair of primary bronchi, one bronchus to each lung (Fig. 17.2b). Within the lungs, the bronchi branch repeatedly (divisions 2–11) into progressively smaller bronchi (Fig. 17.2e). Like the trachea, the bronchi are semi-rigid tubes supported by cartilage.

Within the lungs, the smallest bronchi branch to become bronchioles, small collapsible passageways with walls of smooth muscle. The bronchioles continue branching (divisions 12–23) until the respiratory bronchioles form a transition between the airways and the exchange epithelium of the lung.

The diameter of the airways becomes progressively smaller from the trachea to the bronchioles, but as the individual airways get narrower, their numbers increase geometrically (Fig. 17.4). As a result, the total cross-sectional area increases with each division of the airways. Total cross-sectional area is lowest in the upper respiratory tract and greatest in the bronchioles, analogous to the increase in cross-sectional area that occurs from the aorta to the capillaries in the circulatory system.

**Concept Check**

1. What is the difference between cellular respiration and external respiration?
2. Name the components of the upper respiratory tract and those of the lower respiratory tract.
3. Based on the total cross-sectional area of different airways, where is the velocity of airflow highest and lowest?
4. Give two functions of pleural fluid.
5. Name the components (including muscles) of the thoracic cage. List the contents of the thorax.
6. Which air passages of the respiratory system are collapsible?

**The Airways Warm, Humidify, and Filter Inspired Air**

During breathing, the upper airways and the bronchi do more than simply serve as passageways for air. They play an important role in conditioning air before it reaches the alveoli. Conditioning has three components:

1. **Warming** air to body temperature (37°C), so that core body temperature does not change and alveoli are not damaged by cold air;
2. **Adding water vapor** until the air reaches 100% humidity, so that the moist exchange epithelium does not dry out; and
3. **Filtering out foreign material**, so that viruses, bacteria, and inorganic particles do not reach the alveoli.

Inhaled air is warmed by the body’s heat and moistened by water evaporating from the mucosal lining of the airways. Under normal circumstances, by the time air reaches the trachea, it has been conditioned to 100% humidity and 37°C. Breathing through the mouth is not nearly as effective at warming and moistening air as breathing through the nose. If you exercise outdoors in very cold weather, you may be familiar with the ache in your chest that results from breathing cold air through your mouth.

Air is filtered both in the trachea and in the bronchi. These airways are lined with ciliated epithelium whose cilia are bathed in a watery saline layer (Fig. 17.5). The saline is produced by epithelial cells when Cl− secreted into the lumen by apical anion channels draws Na+ into the lumen through the paracellular pathway (Fig. 17.5c). Movement of solute from the ECF to the lumen creates an osmotic gradient, and water follows the ions into the airways. The CFT channel, whose malfunction causes cystic fibrosis, is one of the anion channels found on the apical surface of this epithelium.

A sticky layer of mucus floats over the cilia to trap most inhaled particles larger than 2 μm. The mucus layer is secreted...
The Lungs and Thoracic Cavity

(a) Muscles of the thorax, neck, and abdomen create the force to move air during breathing.

(b) The respiratory system is divided into upper and lower regions.

(c) Sectional view of chest. Each lung is enclosed in two pleural membranes. The esophagus and aorta pass through the thorax between the pleural sacs.

(d) On external view, the right lung is divided into three lobes, and the left lung is divided into two lobes.
The Bronchi and Alveoli

(e) Branching of airways creates about 80 million bronchioles.

(f) Structure of lung lobule. Each cluster of alveoli is surrounded by elastic fibers and a network of capillaries.

(g) Alveolar structure

(h) Exchange surface of alveoli

- Type I alveolar cell for gas exchange
- Endothelial cell of capillary
- Type II alveolar cell (surfactant cell) synthesizes surfactant.
- Limited interstitial fluid
- Alveolar macrophage ingests foreign material.

Blue arrow represents gas exchange between alveolar air space and the plasma.
Alveoli Are the Site of Gas Exchange

The alveoli, clustered at the ends of terminal bronchioles, make up the bulk of lung tissue (Fig. 17.2f, g). Their primary function is the exchange of gases between themselves and the blood.

Each tiny alveolus is composed of a single layer of epithelium (Fig. 17.2g). Two types of epithelial cells are found in the alveoli. The smaller but thicker type II alveolar cells synthesize and secrete a chemical known as surfactant. Surfactant mixes with the thin fluid lining of the alveoli to aid lungs as they expand during breathing, as you will see later in this chapter. Type II cells also help minimize the amount of fluid present in the alveoli by transporting solutes, followed by water, out of the alveolar air space.

The larger type I alveolar cells occupy about 95% of the alveolar surface area and are very thin so that gases can diffuse rapidly through them (Fig. 17.2h). In much of the exchange area, a layer of basement membrane fuses the alveolar epithelium to the capillary endothelium. In the remaining area only a small amount of interstitial fluid is present.

The thin walls of the alveoli do not contain muscle because muscle fibers would block rapid gas exchange. As a result,
Mechanics of Breathing

Proximity of capillary blood to alveolar air is essential for the rapid exchange of gases.

Pulmonary Circulation Is High-Flow, Low-Pressure

The pulmonary circulation begins with the pulmonary trunk, which receives low-oxygen blood from the right ventricle. The pulmonary trunk divides into two pulmonary arteries, one to

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**AIRWAY EPITHELIUM**

(a) Epithelial cells lining the airways and submucosal glands secrete saline and mucus.

(b) Cilia move the mucus layer toward the pharynx, removing trapped pathogens and particulate matter.

(c) One model of saline secretion by airway epithelial cells

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**Fig. 17.5**

Lung tissue itself cannot contract. However, connective tissue between the alveolar epithelial cells contains many elastin and collagen fibers that create elastic recoil when lung tissue is stretched.

The close association of the alveoli with an extensive network of capillaries demonstrates the intimate link between the respiratory and cardiovascular systems. Blood vessels fill 80–90% of the space between alveoli, forming an almost continuous “sheet” of blood in close contact with the air-filled alveoli. The proximity of capillary blood to alveolar air is essential for the rapid exchange of gases.

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**Pulmonary Circulation Is High-Flow, Low-Pressure**

The pulmonary circulation begins with the pulmonary trunk, which receives low-oxygen blood from the right ventricle. The pulmonary trunk divides into two pulmonary arteries, one to
each lung. Oxygenated blood from the lungs returns to the left atrium via the pulmonary veins.

At any given moment, the pulmonary circulation contains about 0.5 liter of blood, or 10% of total blood volume. About 75 mL of this amount is found in the capillaries, where gas exchange takes place, with the remainder in pulmonary arteries and veins. The rate of blood flow through the lungs is much higher than the rate in other tissues because the lungs receive the entire cardiac output of the right ventricle: 5 L/min. This means that as much blood flows through the lungs in one minute as flows through the entire rest of the body in the same amount of time!

Despite the high flow rate, pulmonary blood pressure is low. Pulmonary arterial pressure averages 25/8 mm Hg, much lower than the average systemic pressure of 120/80 mm Hg. The right ventricle does not have to pump as forcefully to create blood flow through the lungs because resistance of the pulmonary circulation is low. This low resistance can be attributed to the shorter total length of pulmonary blood vessels and to the distensibility and large total cross-sectional area of pulmonary arterioles.

Normally, the net hydrostatic pressure filtering fluid out of a pulmonary capillary into the interstitial space is low because of low mean blood pressure. The lymphatic system efficiently removes filtered fluid, and lung interstitial fluid volume is usually minimal. As a result, the distance between the alveolar air space and the capillary endothelium is short, and gases diffuse rapidly between them.

Gas Laws

Respiratory air flow is very similar in many respects to blood flow in the cardiovascular system because both air and blood are fluids. Their primary difference is that blood is a non-compressible liquid but air is a compressible mixture of gases.

Figure 17.6 summarizes the laws that govern the behavior of gases in air and provide the basis for the exchange of air between the external environment and the alveoli.

In this course, blood pressure and environmental air pressure (atmospheric pressure) are both reported in millimeters of mercury (mm Hg). Respiratory physiologists sometimes report gas pressures in centimeters of water instead, where 1 mm Hg = 1.36 cm H$_2$O, or in kiloPascals (kPa), where 760 mm Hg = 101.325 kPa.

At sea level, normal atmospheric pressure is 760 mm Hg. However, in this course we follow the convention of designating atmospheric pressure as 0 mm Hg. Because atmospheric pressure varies with altitude and because very few people live exactly at sea level, this convention allows us to compare pressure differences that occur during ventilation without correcting for altitude.

RUNNING PROBLEM

Edna has not been able to stop smoking, and her COPD is a combination of emphysema and bronchitis. Patients with chronic bronchitis have excessive mucus production and exhibit general inflammation of the entire respiratory tract. The mucus narrows the airways and makes breathing difficult.

Q1: What does narrowing of the airways do to airway resistance?
Gas Laws

This figure summarizes the rules that govern the behavior of gases in air. These rules provide the basis for the exchange of air between the external environment and the alveoli.

(a) The ideal gas equation

\[ PV = nRT \]

Where \( P \) is pressure, \( V \) is volume, \( n \) is the moles of gas, \( T \) is absolute temperature, and \( R \) is the universal gas constant, 8.3145 \( \text{J/mol} \times \text{K} \).

In the human body we can assume that the number of moles and temperature are constant. Removing the constants leaves the following equation:

\[ V = \frac{1}{P} \]

This relationship says that if the volume of gas increases, the pressure decreases, and vice versa.

(b) Boyle’s Law

Boyle’s law also expresses this inverse relationship between pressure and volume.

\[ P_1V_1 = P_2V_2 \]

For example, the container on the left is 1 L (\( V_1 \)) and has a pressure of 100 mm Hg (\( P_1 \)).

What happens to the pressure when the volume decreases to 0.5 L?

\[ \begin{align*}
100 \text{ mm Hg} \times 1 \text{ L} &= P_2 \times 0.5 \text{ L} \\
200 \text{ mm Hg} &= P_2
\end{align*} \]

The pressure has increased \( \times 2 \).

The Ideal Gas law and Boyle’s law apply to all gases or mixtures of gases.

(c) Dalton’s Law

Dalton’s law says that the total pressure of a mixture of gases is the sum of the pressures of the individual gases. The pressure of an individual gas in a mixture is known as the partial pressure of the gas (\( P_{\text{gas}} \)).

For example, at sea level, atmospheric pressure (\( P_{\text{atm}} \)) is 760 mm Hg, and oxygen is 21% of the atmosphere. What is the partial pressure of oxygen (\( P_{O_2} \))?

To find the partial pressure of any one gas in a sample of dry air, multiply the atmospheric pressure (\( P_{\text{atm}} \)) by the gas’s relative contribution (%) to \( P_{\text{atm}} \):

\[ P_{\text{gas}} = P_{\text{atm}} \times \% \text{ of gas in atmosphere} \]

\[ P_{O_2} = 760 \text{ mm Hg} \times 21\% = 160 \text{ mm Hg} \]

The partial pressure of oxygen (\( P_{O_2} \)) in dry air at sea level is 160 mm Hg.

In humid air, water vapor “dilutes” the contribution of other gases to the total pressure.

To calculate the partial pressure of a gas in humid air, you must first subtract the water vapor pressure from the total pressure. At 100% humidity and 25\(^\circ\) C, water vapor pressure (\( P_{H_2O} \)) is 24 mm Hg.

\[ P_{\text{gas in humid air}} = (P_{\text{atm}} - P_{H_2O}) \times \% \text{ of gas} \]

\[ P_{O_2} = (760 - 24) \times 21\% = 155 \text{ mm Hg} \]
Air Is a Mixture of Gases

The atmosphere surrounding the earth is a mixture of gases and water vapor. Dalton’s law states that the total pressure exerted by a mixture of gases is the sum of the pressures exerted by the individual gases (Fig. 17.6c). For example, in dry air at an atmospheric pressure of 760 mm Hg, 78% of the total pressure is due to N₂, 21% to O₂, and so on.

In respiratory physiology, we are concerned not only with total atmospheric pressure but also with the individual pressures of oxygen and carbon dioxide. The pressure of a single gas in a mixture is known as its partial pressure (Pᵢ). The pressure exerted by an individual gas is determined only by its relative abundance in the mixture and is independent of the molecular size or mass of the gas.

The partial pressures of gases in air vary slightly depending on how much water vapor is in the air because the pressure of water vapor “dilutes” the contribution of other gases to the total pressure. The table in Figure 17.6c compares the partial pressures of some gases in dry air and at 100% humidity.

Boyle’s Law Describes Pressure-Volume Relationships

The pressure exerted by a gas or mixture of gases in a sealed container is created by the collisions of moving gas molecules with the walls of the container and with each other. If the size of the container is reduced, the collisions between the gas molecules and the walls become more frequent, and the pressure rises (Fig. 17.6b). This relationship between pressure and volume was first noted by Robert Boyle in the 1600s and can be expressed by the equation of Boyle’s law of gases:

\[ P_1V_1 = P_2V_2 \]

where \( P \) represents pressure and \( V \) represents volume.

Boyle’s law states that if the volume of a gas is reduced, the pressure increases. If the volume increases, the pressure decreases.

In the respiratory system, changes in the volume of the chest cavity during ventilation cause pressure gradients that create air flow. When chest volume increases, alveolar pressure falls, and air flows into the respiratory system. When the chest volume decreases, alveolar pressure increases, and air flows out into the atmosphere. This movement of air is bulk flow because the entire gas mixture is moving rather than merely one or two of the gases in the air.

Ventilation

This bulk flow exchange of air between the atmosphere and the alveoli is ventilation, or breathing (Fig. 17.1). A single respiratory cycle consists of an inspiration followed by an expiration.

Lung Volumes Change During Ventilation

Physiologists and clinicians assess a person’s pulmonary function by measuring how much air the person moves during quiet breathing, then with maximum effort. These pulmonary function tests use a spirometer, an instrument that measures the volume of air moved with each breath (Fig. 17.7a). (Most spirometers in clinical use today are small computerized machines rather than the traditional wet spirometer illustrated here.) When a subject is attached to the traditional spirometer through a mouthpiece and the subject’s nose is clipped closed, the subject’s respiratory tract and the spirometer form a closed system. When the subject breathes in, air moves from the spirometer into the lungs, and the recording pen, which traces a graph on a rotating cylinder, moves up. When the subject exhales, air moves from the lungs back into the spirometer, and the pen moves down.

Lung Volumes The air moved during breathing can be divided into four lung volumes: (1) tidal volume, (2) inspiratory reserve

Gases Move Down Pressure Gradients

Air flow occurs whenever there is a pressure gradient. Bulk flow of air, like blood flow, is directed from areas of higher pressure to areas of lower pressure. Meteorologists predict the weather by knowing that areas of high atmospheric pressure move in to replace areas of low pressure. In ventilation, bulk flow of air down pressure gradients explains how air is exchanged between the external environment and the lungs. Movement of the thorax during breathing creates alternating conditions of high and low pressure in the lungs.

Diffusion of gases down concentration (partial pressure) gradients applies to single gases. For example, oxygen moves from areas of higher oxygen partial pressure (Pₐₒ₂) to areas of lower oxygen partial pressure. Diffusion of individual gases is important in the exchange of oxygen and carbon dioxide between alveoli and blood and from blood to cells.
PULMONARY FUNCTION TESTS

(a) The Spirometer

This figure shows a traditional wet spirometer. The subject inserts a mouthpiece that is attached to an inverted bell filled with air or oxygen. The volume of the bell and the volume of the subject’s respiratory tract create a closed system because the bell is suspended in water.

(b) Lung Volumes and Capacities

The four lung volumes

- RV = Residual volume
- ERV = Expiratory reserve volume
- VT = Tidal volume
- IRV = Inspiratory reserve volume

Capacities are sums of 2 or more volumes.

- Inspiratory capacity = VT + IRV
- Vital capacity = VT + IRV + ERV
- Total lung capacity = VT + IRV + ERV + RV
- Functional residual capacity = ERV + RV

Pulmonary Volumes and Capacities*

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRV</td>
<td>3000 mL</td>
<td>1900 mL</td>
</tr>
<tr>
<td>VT</td>
<td>500 mL</td>
<td>500 mL</td>
</tr>
<tr>
<td>ERV</td>
<td>1100 mL</td>
<td>700 mL</td>
</tr>
<tr>
<td>Residual volume</td>
<td>1200 mL</td>
<td>1100 mL</td>
</tr>
<tr>
<td>Total lung capacity</td>
<td>5800 mL</td>
<td>4200 mL</td>
</tr>
</tbody>
</table>

*Pulmonary volumes are given for a normal 70-kg man or a 50-kg woman, 28 years old.
volume, (3) expiratory reserve volume, and (4) residual volume. The numerical values used on the graph in Figure 17.7b represent average volumes for a 70-kg man. The volumes for women are typically less, as shown in Figure 17.7b. Lung volumes vary considerably with age, sex, height, and weight, so clinicians use algorithms based on those parameters to predict lung volumes. (An algorithm is an equation or a series of steps used to solve a problem.)

Each of the following paragraphs begins with the instructions you would be given if you were being tested for these volumes.

“Breathe quietly.” The volume of air that moves during a single inspiration or expiration is known as the tidal volume \( V_t \). Average tidal volume during quiet breathing is about 500 mL. (It is hard for subjects to breathe normally when they are thinking about their breathing, so the clinician may not give this instruction.)

“Now, at the end of a quiet inspiration, take in as much additional air as you possibly can.” The additional volume you inspire above the tidal volume represents your inspiratory reserve volume (IRV). In a 70-kg man, this volume is about 3000 mL, a sixfold increase over the normal tidal volume.

“Now stop at the end of a normal exhalation, then exhale as much air as you possibly can.” The amount of air forcefully exhaled after the end of a normal expiration is the expiratory reserve volume (ERV), which averages about 1100 mL.

The fourth volume cannot be measured directly. Even if you blow out as much air as you can, air still remains in the lungs and the airways. The volume of air in the respiratory system after maximal exhalation—about 1200 mL—is called the residual volume (RV). Most of this residual volume exists because the lungs are held stretched against the ribs by the pleural fluid.

**Lung Capacities** The sum of two or more lung volumes is called a capacity. The vital capacity (VC) is the sum of the inspiratory reserve volume, expiratory reserve volume, and tidal volume. Vital capacity represents the maximum amount of air that can be voluntarily moved into or out of the respiratory system with one breath. To measure vital capacity, you would instruct the person to take in as much air as possible, then blow it all out. Vital capacity decreases with age as muscles weaken and the lungs become less elastic.

Vital capacity plus the residual volume yields the total lung capacity (TLC). Other capacities of importance in pulmonary medicine include the inspiratory capacity (tidal volume + inspiratory reserve volume) and the functional residual capacity (expiratory reserve volume + residual volume).

**Inspiration Occurs When Alveolar Pressure Decreases**

For air to move into the alveoli, pressure inside the lungs must become lower than atmospheric pressure. According to Boyle's law, an increase in volume will create a decrease in pressure. During inspiration, thoracic volume increases when certain skeletal muscles of the rib cage and diaphragm contract.

When the diaphragm contracts, it drops down toward the abdomen. In quiet breathing, the diaphragm moves about 1.5 cm, increasing thoracic volume (Fig. 17.8b). Contraction of the diaphragm causes between 60% and 75% of the inspiratory volume change during normal quiet breathing.

Movement of the rib cage creates the remaining 25–40% of the volume change. During inhalation, the external intercostal
and scalene muscles (see Fig. 17.2a) contract and pull the ribs upward and out (Fig. 17.8b). Rib movement during inspiration has been likened to a pump handle lifting up and away from the pump (the ribs moving up and away from the spine) and to the movement of a bucket handle as it lifts away from the side of a bucket (ribs moving outward in a lateral direction). The combination of these two movements broadens the rib cage in all directions. As thoracic volume increases, pressure decreases, and air flows into the lungs.

For many years, quiet breathing was attributed solely to the action of the diaphragm and the external intercostal muscles. It was thought that the scalenes and sternocleidomastoid muscles were active only during deep breathing. In recent years, however, studies have changed our understanding of how these accessory muscles contribute to quiet breathing.

If an individual’s scalenes are paralyzed, inspiration is achieved primarily by contraction of the diaphragm. Observation of patients with neuromuscular disorders has revealed that although the contracting diaphragm increases thoracic volume by moving toward the abdominal cavity, it also tends to pull the lower ribs inward, working against inspiration. In normal individuals, we know that the lower ribs move up and out during inspiration rather than inward. The fact that there is no up-and-out rib motion in patients with paralyzed scalenes tells us that normally the scalenes must be contributing to inspiration by lifting the sternum and upper ribs.
New evidence also downplays the role of the external intercostal muscles during quiet breathing. However, the external intercostals play an increasingly important role as respiratory activity increases. Because the exact contribution of external intercostals and scalenes varies depending on the type of breathing, we group these muscles together and simply call them the inspiratory muscles.

Now let’s see how alveolar pressure ($P_a$) changes during a single inspiration. Follow the graphs in Figure 17.9 as you read through the process. Remember that atmospheric pressure is assigned a value of 0 mm Hg. Negative numbers designate subatmospheric pressures, and positive numbers denote higher-than-atmospheric pressures.

**Time 0.** In the brief pause between breaths, alveolar pressure is equal to atmospheric pressure (0 mm Hg at point $A_1$). When pressures are equal, there is no air flow.

**GRAPH QUESTIONS**

1. At what point in the cycle is alveolar pressure greatest? Least? Equal to atmospheric pressure?
   - When lung volume is at its minimum, alveolar pressure is ________ and external intercostal muscle contraction is ________.
     - (a) maximum
     - (b) minimum
     - (c) moving from maximum to minimum
     - (d) moving from minimum to maximum

2. Why do people with chronic bronchitis have a higher-than-normal rate of respiratory infections?

3. What is this person’s ventilation rate?
Time 0–2 sec: Inspiration. As inspiration begins, inspiratory muscles contract, and thoracic volume increases. With the increase in volume, alveolar pressure falls about 1 mm Hg below atmospheric pressure (−1 mm Hg, point A₁), and air moves into the alveoli (point C₁ to point C₂). Because the thoracic volume changes faster than air can flow, alveolar pressure reaches its lowest value about halfway through inspiration (point A₂).

As air continues to flow into the alveoli, pressure increases until the thoracic cage stops expanding, just before the end of inspiration. Air movement continues for a fraction of a second longer, until pressure inside the lungs equals atmospheric pressure (point A₂). At the end of inspiration, lung volume is at its maximum for the respiratory cycle (point C₂), and alveolar pressure is equal to atmospheric pressure.

You can demonstrate this phenomenon by taking a deep breath and stopping the movement of your chest at the end of inspiration. (Do not “hold your breath” because doing so closes the opening of the pharynx and prevents air flow.) If you do this correctly, you notice that air flow stops after you freeze the inspiratory movement. This exercise shows that at the end of inspiration, alveolar pressure is equal to atmospheric pressure.

Expiration Occurs When Alveolar Pressure Increases

At the end of inspiration, impulses from somatic motor neurons to the inspiratory muscles cease, and the muscles relax. Elastic recoil of the lungs and thoracic cage returns the diaphragm and rib cage to their original relaxed positions, just as a stretched elastic waistband recoils when released. Because expiration during quiet breathing involves passive elastic recoil rather than active muscle contraction, it is called passive expiration.

Time 2–4 sec: expiration. As lung and thoracic volumes decrease during expiration, air pressure in the lungs increases, reaching a maximum of about 1 mm Hg above atmospheric pressure (Fig. 17.9, point A₂). Alveolar pressure is now higher than atmospheric pressure, so air flow reverses and air moves out of the lungs.

Time 4 sec. At the end of expiration, air movement ceases when alveolar pressure is again equal to atmospheric pressure (point A₂). Lung volume reaches its minimum for the respiratory cycle (point C₂). At this point, the respiratory cycle has ended and is ready to begin again with the next breath.

The pressure differences shown in Figure 17.9 apply to quiet breathing. During exercise or forced heavy breathing, these values become proportionately larger. Active expiration occurs during voluntary exhalations and when ventilation exceeds 30–40 breaths per minute. (Normal resting ventilation rate is 12–20 breaths per minute for an adult.) Active expiration uses the internal intercostal muscles and the abdominal muscles (see Fig. 17.2a), which are not used during inspiration. These muscles are collectively called the expiratory muscles.

The internal intercostal muscles line the inside of the rib cage. When they contract, they pull the ribs inward, reducing the volume of the thoracic cavity. To feel this action, place your hands on your rib cage. Forcefully blow as much air out of your lungs as you can, noting the movement of your hands as you do so.

The internal and external intercostals function as antagonistic muscle groups to alter the position and volume of the rib cage during ventilation. The diaphragm, however, has no antagonistic muscles. Instead, abdominal muscles contract during active expiration to supplement the activity of the internal intercostals. Abdominal contraction pulls the lower rib cage inward and decreases abdominal volume, actions that displace the intestines and liver upward. The displaced viscera push the diaphragm up into the thoracic cavity and passively decrease chest volume even more. The action of abdominal muscles during forced expiration is why aerobics instructors tell you to blow air out as you lift your head and shoulders during abdominal “crunches.” The active process of blowing air out helps contract the abdominals, the muscles you are trying to strengthen.

Any neuromuscular disease that weakens skeletal muscles or damages their motor neurons can adversely affect ventilation. With decreased ventilation, less fresh air enters the lungs. In addition, loss of the ability to cough increases the risk of pneumonia and other infections. Examples of diseases that affect the motor control of ventilation include myasthenia gravis, an illness in which acetylcholine receptors of the motor end plates of skeletal muscles are destroyed, and polio (poliomyelitis), a viral illness that paralyzes skeletal muscles.

Concept Check

19. Scarlett O’Hara is trying to squeeze herself into a corset with an 18-inch waist. Will she be more successful by taking a deep breath and holding it or by blowing all the air out of her lungs? Why?

20. Why would loss of the ability to cough increase the risk of respiratory infections? (Hint: What does coughing do to mucus in the airways?)

Intrapleural Pressure Changes During Ventilation

Ventilation requires that the lungs, which are unable to expand and contract on their own, move in association with the expansion and relaxation of the thorax. As we noted earlier in this chapter, the lungs are enclosed in the fluid-filled pleural sac. The surface of the lungs is covered by the visceral pleura, and the portion of the sac that lines the thoracic cavity is called the parietal pleura (paries, wall). Cohesive forces exerted by the fluid between the two pleural membranes cause the stretchable lung to adhere to the thoracic cage. When the thoracic cage moves during breathing, the lungs move with it.
The intrapleural pressure in the fluid between the pleural membranes is normally subatmospheric. This subatmospheric pressure arises during fetal development, when the thoracic cage with its associated pleural membrane grows more rapidly than the lung with its associated pleural membrane. The two pleural membranes are held together by the pleural fluid bond, so the elastic lungs are forced to stretch to conform to the larger volume of the thoracic cavity. At the same time, however, elastic recoil of the lungs creates an inwardly directed force that tries to pull the lungs away from the chest wall (Fig. 17.10a). The combination of the outward pull of the thoracic cage and inward recoil of the elastic lungs creates a subatmospheric intrapleural pressure of about $-3$ mm Hg.

You can create a similar situation by half-filling a syringe with water and capping it with a plugged-up needle. At this point, the pressure inside the barrel is equal to atmospheric pressure. Now hold the syringe barrel (the chest wall) in one hand while you try to withdraw the plunger (the elastic lung pulling away from the chest wall). As you pull on the plunger, the volume inside the barrel increases slightly, but the cohesive forces between the water molecules cause the water to resist expansion. The pressure in the barrel, which was initially equal to atmospheric pressure, decreases slightly as you pull on the plunger. If you release the plunger, it snaps back to its resting position, restoring atmospheric pressure inside the syringe.

What happens to subatmospheric intrapleural pressure if an opening is made between the sealed pleural cavity and the atmosphere? A knife thrust between the ribs, a broken rib that punctures the pleural membrane, or any other event that opens

---

**RUNNING PROBLEM**

Emphysema is characterized by a loss of elastin, the elastic fibers that help the alveoli recoil during expiration. Elastin is destroyed by elastase, an enzyme released by alveolar macrophages, which must work overtime in smokers to rid the lungs of irritants. People with emphysema have more difficulty exhaling than inhaling. Their alveoli have lost elastic recoil, which makes expiration—normally a passive process—require conscious effort.

**Q3:** Name the muscles that patients with emphysema use to exhale actively.

---

**SUBATMOSPHERIC PRESSURE IN THE PLEURAL CAVITY HELPS KEEP THE LUNGS INFLATED**

(a) In the normal lung at rest, pleural fluid keeps the lung adhered to the chest wall.

(b) Pneumothorax. If the sealed pleural cavity is opened to the atmosphere, air flows in. The bond holding the lung to the chest wall is broken, and the lung collapses, creating a pneumothorax (air in the thorax).
the pleural cavity to the atmosphere allows air to flow down its pressure gradient into the cavity, just as air enters when you break the seal on a vacuum-packed can.

Air in the pleural cavity breaks the fluid bond holding the lung to the chest wall. The chest wall expands outward while the elastic lung collapses to an unstretched state, like a deflated balloon (Fig. 17.10b). This condition, called pneumothorax (pneuma, air + thorax, chest), results in a collapsed lung that is unable to function normally. Pneumothorax can also occur spontaneously if a congenital bleb, a weakened section of lung tissue, ruptures, allowing air from inside the lung to enter the pleural cavity.

Correction of a pneumothorax has two components: removing as much air from the pleural cavity as possible with a suction pump, and sealing the hole to prevent more air from entering. Any air remaining in the cavity is gradually absorbed into the blood, restoring the pleural fluid bond and reinflating the lung.

Pressures in the pleural fluid vary during a respiratory cycle. At the beginning of inspiration, intrapleural pressure is about −3 mm Hg (Fig. 17.9, point B1). As inspiration proceeds, the pleural membranes and lungs resist the expanding thoracic cage because of the pleural fluid bond, but the elastic lung tissue resists being stretched. The lungs attempt to pull farther away from the chest wall, causing the intrapleural pressure to become even more negative (Fig. 17.9, point B2).

Because this process is difficult to visualize, let’s return to the analogy of the water-filled syringe with the plugged-up needle. You can pull the plunger out a small distance without much effort, but the cohesiveness of the water makes it difficult to pull the plunger out any farther. The increased amount of work you do trying to pull the plunger out is paralleled by the work your inspiratory muscles must do when they contract during inspiration. The bigger the breath, the more work is required to stretch the elastic lung.

By the end of a quiet inspiration, when the lungs are fully expanded, intrapleural pressure falls to around −6 mm Hg (Fig. 17.9, point B2). During exercise or other powerful inspirations, intrapleural pressure may reach −8 mm Hg or lower.

During expiration, the thoracic cage returns to its resting position. The lungs are released from their stretched position, and the intrapleural pressure returns to its normal value of about −3 mm Hg (point B3). Notice that intrapleural pressure never equilibrates with atmospheric pressure because the pleural cavity is a closed compartment.

Pressure gradients required for air flow are created by the work of skeletal muscle contraction. Normally, about 3–5% of the body’s energy expenditure is used for quiet breathing. During exercise, the energy required for breathing increases substantially. The two factors that have the greatest influence on the amount of work needed for breathing are the stretchability of the lungs and the resistance of the airways to air flow.

Lung Compliance and Elastance May Change in Disease States

Adequate ventilation depends on the ability of the lungs to expand normally. Most of the work of breathing goes into overcoming the resistance of the elastic lungs and the thoracic cage to stretching. Clinically, the ability of the lung to stretch is called compliance.

Compliance refers to the amount of force that must be exerted in a body to deform it. In the lung, we can express compliance as the change of volume (V) that results from a given force or pressure (P) exerted on the lung: \( \frac{\Delta V}{\Delta P} \). A high-compliance lung stretches easily, just as a compliant person is easy to persuade. A low-compliance lung requires more force from the inspiratory muscles to stretch it.

Compliance is the reciprocal of elastance (elastic recoil), the ability to resist being deformed. Elastance also refers to the ability of a body to return to its original shape when a deforming force is removed. A lung that stretches easily (high compliance) has probably lost its elastic tissue and will not return to its resting volume when the stretching force is released (low elastance). You may have experienced something like this with old gym shorts. After many washings the elastic waistband is easy to stretch (high compliance) but lacking in elastance, making it impossible for the shorts to stay up around your waist.

Analogous problems occur in the respiratory system. For example, as noted in the Running Problem, emphysema is a disease in which elastin fibers normally found in lung tissue are destroyed. Destruction of elastin results in lungs that exhibit high compliance and stretch easily during inspiration. However, these lungs also have decreased elastance, so they do not recoil to their resting position during expiration.

To understand the importance of elastic recoil to expiration, think of an inflated balloon and an inflated plastic bag. The balloon is similar to the normal lung. Its elastic walls squeeze on the air inside the balloon, thereby increasing the internal air pressure. When the neck of the balloon is opened to the atmosphere, elastic recoil causes air to flow out of the balloon. The inflated plastic bag, on the other hand, is like the lung of an individual with emphysema. It has high compliance and is easily infl ated, but it has little elastic recoil. If the inflated...
Mechanics of Breathing

A decrease in lung compliance affects ventilation because more work must be expended to stretch a stiff lung. Pathological conditions in which compliance is reduced are called restrictive lung diseases. In these conditions, the energy expenditure required to stretch less-compliant lungs can far exceed the normal work of breathing. Two common causes of decreased compliance are inelastic scar tissue formed in fibrotic lung diseases, and inadequate alveolar production of surfactant, a chemical that facilitates lung expansion.

Pulmonary fibrosis is characterized by the development of stiff, fibrous scar tissue that restricts lung inflation. In idiopathic pulmonary fibrosis (idios, one’s own), the cause is unknown. Other forms of fibrotic lung disease result from chronic inhalation of fine particulate matter, such as asbestos and silicon, that escapes the mucus lining the airways and reaches the alveoli. Wandering alveolar macrophages (see Fig. 17.2g) then ingest the inhaled particulate matter. If the particles are organic, the macrophages can digest them with lysosomal enzymes. However, if the particles cannot be digested or if they accumulate in large numbers, an inflammatory process ensues. The macrophages then secrete growth factors that stimulate fibroblasts in the lungs’ connective tissue to produce inelastic collagen. Pulmonary fibrosis cannot be reversed.

Surfactant Decreases the Work of Breathing

For years, physiologists assumed that elastin and other elastic fibers were the primary source of resistance to stretch in the lung. However, studies comparing the work required to expand air-filled and saline-filled lungs showed that air-filled lungs are much harder to inflate. From this result, researchers concluded that lung tissue itself contributes less to resistance than once thought. Some other property of the normal air-filled lung, a property not present in the saline-filled lung, must create most of the resistance to stretch.

This property is the surface tension created by the thin fluid layer between the alveolar cells and the air. At any air-fluid interface, the surface of the fluid is under tension, like a thin membrane being stretched. When the fluid is water, surface tension arises because of the hydrogen bonds between water molecules. The water molecules on the fluid’s surface are attracted to other water molecules beside and beneath them but are not attracted to gases in the air at the air-fluid interface.

Alveolar surface tension is similar to the surface tension that exists in a spherical bubble, even though alveoli are not perfect spheres. The surface tension created by the thin film of fluid is directed toward the center of the bubble and creates pressure in the interior of the bubble. The law of Laplace is an expression of this pressure. It states that the pressure (P) inside a bubble formed by a fluid film is a function of two factors: the surface tension of the fluid (T) and the radius of the bubble (r). This relationship is expressed by the equation

\[ P = \frac{2T}{r} \]

Notice in Figure 17.11a that if two bubbles have different diameters but are formed by fluids with the same surface tension, the pressure inside the smaller bubble is greater than that inside the larger bubble.

How does this apply to the lung? In physiology, we can equate the bubble to a fluid-lined alveolus (although alveoli are not perfect spheres). The fluid lining all the alveoli creates surface tension. If the surface tension (T) of the fluid were the

![LAW OF LAPLACE](image-url)

**LAW OF LAPLACE**

<table>
<thead>
<tr>
<th>(a) The two bubbles shown have the same surface tension (T). According to the Law of Laplace, pressure is greater in the smaller bubble.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larger bubble</td>
</tr>
<tr>
<td>r = 2</td>
</tr>
<tr>
<td>T = 3</td>
</tr>
<tr>
<td>( P = \frac{2 \times 3}{2} )</td>
</tr>
<tr>
<td>( P = 3 )</td>
</tr>
<tr>
<td>Smaller bubble</td>
</tr>
<tr>
<td>r = 1</td>
</tr>
<tr>
<td>T = 3</td>
</tr>
<tr>
<td>( P = \frac{2 \times 3}{1} )</td>
</tr>
<tr>
<td>( P = 6 )</td>
</tr>
</tbody>
</table>

**Law of Laplace**

\[ P = \frac{2T}{r} \]

| P = pressure  |
| T = surface tension  |
| r = radius  |

According to the law of Laplace, if two bubbles have the same surface tension, the smaller bubble will have higher pressure.

**Fig. 17.11**
same in small and large alveoli, small alveoli would have higher inwardly directed pressure than larger alveoli, and increased resistance to stretch. As a result, more work would be needed to expand smaller alveoli.

Normally, however, our lungs secrete a surfactant that reduces surface tension. Surfactants ("surface active agents") are molecules that disrupt cohesive forces between water molecules by substituting themselves for water at the surface. For example, that product you add to your dishwasher to aid in the rinse cycle is a surfactant that keeps the rinse water from beading up on the dishes (and forming spots when the water beads dry). In the lungs, surfactant decreases surface tension of the alveolar fluid and thereby decreases resistance of the lung to stretch.

Surfactant is more concentrated in smaller alveoli, making their surface tension less than that in larger alveoli (Fig. 17.11b). Lower surface tension helps equalize the pressure among alveoli of different sizes and makes it easier to inflate the smaller alveoli. With lower surface tension, the work needed to expand the alveoli with each breath is greatly reduced. Human surfactant is a mixture containing proteins and phospholipids, such as dipalmitoylphosphatidylcholine, which are secreted into the alveolar air space by type II alveolar cells (see Fig. 17.2g).

Normally, surfactant synthesis begins about the twenty-fifth week of fetal development under the influence of various hormones. Production usually reaches adequate levels by the thirty-fourth week (about six weeks before normal delivery). Babies who are born prematurely without adequate concentrations of surfactant in their alveoli develop newborn respiratory distress syndrome (NRDS). In addition to having "stiff" (low-compliance) lungs, NRDS babies also have alveoli that collapse each time they exhale. These infants must use a tremendous amount of energy to expand their collapsed lungs with each breath. Unless treatment is initiated rapidly, about 50% of these infants die. In the past, all physicians could do for NRDS babies was administer oxygen. Today, however, the prognosis for NRDS babies is much better. Amniotic fluid can be sampled to assess whether or not the fetal lungs are producing adequate amounts of surfactant. If they are not, and if delivery cannot be delayed, NRDS babies can be treated with aerosol administration of artificial surfactant until the lungs mature enough to produce their own. The current treatment also includes artificial ventilation that forces air into the lungs (positive-pressure ventilation) and keeps the alveoli open.

**RUNNING PROBLEM**

Edna has been experiencing shortness of breath while exercising, so her physician runs some tests, including measuring Edna’s lung volumes with spirometry. Part of the test is a forced expiratory volume. With her lungs filled to their maximum with air, Edna is told to blow out as fast and as forcefully as she can. The volume of air that Edna expels in the first second of the test (the forced expiratory volume in one second, or FEV1) is lower than normal because in COPD, airway resistance is increased. Another test the physician orders is a complete blood count (CBC). The results of this test show that Edna has higher-than-normal red blood cell count and hematocrit.

**Q4:** When Edna fills her lungs maximally, the volume of air in her lungs is known as the ________ capacity. When she exhales all the air she can, the volume of air left in her lungs is the ________.

**Q5:** Why are Edna’s RBC count and hematocrit increased? (*Hint: Because of Edna’s COPD, her arterial P02 is low.*)
Mechanics of Breathing

increase resistance. If you have ever tried breathing through your nose when you have a cold, you can appreciate how the narrowing of an upper airway limits air flow!

The bronchioles normally do not contribute significantly to airway resistance because their total cross-sectional area is about 2000 times that of the trachea. Because the bronchioles are collapsible tubes, however, a decrease in their diameter can suddenly turn them into a significant source of airway resistance. Bronchoconstriction increases resistance to air flow and decreases the amount of fresh air that reaches the alveoli.

Bronchioles, like arterioles, are subject to reflex control by the nervous system and by hormones. However, most minute-to-minute changes in bronchiolar diameter occur in response to paracines. Carbon dioxide in the airways is the primary paracrine that affects bronchiolar diameter. Increased CO₂ in expired air relaxes bronchiolar smooth muscle and causes bronchodilation.

Histamine is a paracrine that acts as a powerful bronchoconstrictor. This chemical is released by mast cells in response to either tissue damage or allergic reactions. In severe allergic reactions, large amounts of histamine may lead to widespread bronchoconstriction and difficult breathing. Immediate medical treatment in these patients is imperative.

The primary neural control of bronchioles comes from parasympathetic neurons that cause bronchoconstriction, a reflex designed to protect the lower respiratory tract from inhaled irritants. There is no significant sympathetic innervation of the bronchioles in humans. However, smooth muscle in the bronchioles is well supplied with β₂-receptors that respond to epinephrine. Stimulation of β₂-receptors relaxes airway smooth muscle and results in bronchodilation. This reflex is used therapeutically in the treatment of asthma and various allergic reactions characterized by histamine release and bronchoconstriction. Table 17.1 summarizes the factors that alter airway resistance.

<table>
<thead>
<tr>
<th>Factor</th>
<th>Affected by</th>
<th>Mediated by</th>
</tr>
</thead>
<tbody>
<tr>
<td>Length of the system</td>
<td>Constant; not a factor</td>
<td></td>
</tr>
<tr>
<td>Viscosity of air</td>
<td>Usually constant; humidity and altitude may alter slightly</td>
<td></td>
</tr>
<tr>
<td>Diameter of airways</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper airways</td>
<td>Physical obstruction</td>
<td>Mucus and other factors</td>
</tr>
<tr>
<td>Bronchioles</td>
<td>Bronchoconstriction</td>
<td>Parasympathetic neurons (muscarinic receptors), histamine, leukotrienes</td>
</tr>
<tr>
<td></td>
<td>Bronchodilation</td>
<td>Carbon dioxide, epinephrine (β₂-receptors)</td>
</tr>
</tbody>
</table>

Table 17.1

Rate and Depth of Breathing Determine the Efficiency of Breathing

The efficiency of the heart is measured by the cardiac output, which is calculated by multiplying heart rate by stroke volume. Likewise, we can estimate the effectiveness of ventilation by calculating total pulmonary ventilation, the volume of air moved into and out of the lungs each minute (Fig. 17.12a). Total pulmonary ventilation, also known as the minute volume, is calculated as follows:

Total pulmonary ventilation = ventilation rate × tidal volume
Ventilation

(a) Total pulmonary ventilation is greater than alveolar ventilation because of dead space.

**Total pulmonary ventilation**

\[ \text{Total pulmonary ventilation} = \text{ventilation rate} \times \text{tidal volume} (V_T) \]

For example: 12 breaths/min \( \times \) 500 mL breath = 6000 mL/min

(b) Because the conducting airways do not exchange gases with the blood, they are known as anatomic dead space.

**Alveolar ventilation**

Alveolar ventilation is a better indication of how much fresh air reaches the alveoli. Fresh air remaining in the dead space does not get to the alveoli.

\[ \text{Alveolar ventilation} = \text{ventilation rate} \times (V_T - \text{dead space volume} V_D) \]

If dead space is 150 mL: 12 breaths/min \( \times \) (500 – 150 mL) = 4200 mL/min

---

**KEY**

- P_{O_2} = 150 mm Hg (fresh air)
- P_{O_2} = 100 mm Hg (stale air)

---

**FIGURE QUESTION**

Complete this table showing the effects of breathing pattern on alveolar ventilation. Assume dead space volume is 150 mL. Which pattern is the most efficient?

<table>
<thead>
<tr>
<th>Tidal volume (mL)</th>
<th>Ventilation rate (breaths/min)</th>
<th>Total pulmonary ventilation (mL/min)</th>
<th>Fresh air to alveoli (mL)</th>
<th>Alveolar ventilation (mL/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>500 (normal)</td>
<td>12 (normal)</td>
<td>6000</td>
<td>350</td>
<td>4200</td>
</tr>
<tr>
<td>300 (shallow)</td>
<td>20 (rapid)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>750 (deep)</td>
<td>8 (slow)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The normal ventilation rate for an adult is 12–20 breaths (br) per minute. Using the average tidal volume (500 mL) and the slowest ventilation rate, we get:

\[
\text{Total pulmonary ventilation} = 12 \text{ br/} \text{min} \times 500 \text{ mL/br} = 6000 \text{ mL/min} = 6 \text{ L/min}
\]

Total pulmonary ventilation represents the physical movement of air into and out of the respiratory tract, but is it a good indicator of how much fresh air reaches the alveolar exchange surface? Not necessarily.

Some air that enters the respiratory system does not reach the alveoli because part of every breath remains in the conducting airways, such as the trachea and bronchi. Because the conducting airways do not exchange gases with the blood, they are known as the anatomic dead space. Anatomic dead space averages about 150 mL.

To illustrate the difference between the total volume of air that enters the airways and the volume of fresh air that reaches the alveoli, let’s consider a typical breath that moves 500 mL of air during a respiratory cycle (Fig. 17.12b).

1. At the end of an inspiration, lung volume is maximal, and fresh air from the atmosphere fills the dead space.
2. The tidal volume of 500 mL is exhaled. However, the first portion of this 500 mL to exit the airways is the 150 mL of fresh air that had been in the dead space, followed by 350 mL of “stale” air from the alveoli. Even though 500 mL of air exited the alveoli, only 350 mL of that volume left the body. The remaining 150 mL of “stale” alveolar air stays in the dead space.
3. At the end of expiration, lung volume is at its minimum, and stale air from the most recent expiration fills the anatomic dead space.
4. With the next inspiration, 500 mL of fresh air enters the airways. The first air to enter the alveoli is the 150 mL of stale air that was in the anatomic dead space. The remaining 350 mL of air to go into the alveoli is fresh air. The last 150 mL of inspired fresh air again remains in the dead space and never reaches the alveoli.

Thus, although 500 mL of air entered the alveoli, only 350 mL of that volume was fresh air. The fresh air entering the alveoli equals the tidal volume minus the dead space volume.

Because a significant portion of inspired air never reaches an exchange surface, a more accurate indicator of ventilation efficiency is alveolar ventilation, the amount of fresh air that reaches the alveoli each minute. Alveolar ventilation is calculated by multiplying ventilation rate by the volume of fresh air that reaches the alveoli:

\[
\text{Alveolar ventilation} = \text{ventilation rate} \times (\text{tidal volume} - \text{dead space})
\]

Using the same ventilation rate and tidal volume as before, and a dead space of 150 mL, then

\[
\text{Alveolar ventilation} = 12 \text{ br/} \text{min} \times (500 - 150 \text{ mL/br}) = 4200 \text{ mL/min}
\]

Thus, at 12 breaths per minute, the alveolar ventilation is 4.2 L/min. Although 6 L/min of fresh air enters the respiratory system, only 4.2 L reaches the alveoli.

Alveolar ventilation can be drastically affected by changes in the rate or depth of breathing, as you can calculate using the figure question in Figure 17.12. Maximum voluntary ventilation, which involves breathing as deeply and quickly as possible, may increase total pulmonary ventilation to as much as 170 L/min. Table 17.2 describes various patterns of ventilation, and Table 17.3 gives normal ventilation values.

### Gas Composition in the Alveoli Varies Little During Normal Breathing

How much can a change in alveolar ventilation affect the amount of fresh air and oxygen that reach the alveoli? Figure 17.13 shows how the partial pressures \( P_{O_2} \) and \( P_{CO_2} \) in the alveoli vary with hyper- and hypoventilation. As alveolar ventilation increases above normal levels during hyperventilation, alveolar \( P_{O_2} \) increases, and alveolar \( P_{CO_2} \) falls. During hypoventilation, when less fresh air enters the alveoli, alveolar \( P_{O_2} \) decreases and alveolar \( P_{CO_2} \) increases.

A dramatic change in alveolar ventilation pattern can affect gas partial pressures in the alveoli, but the \( P_{O_2} \) and \( P_{CO_2} \) in the alveoli change surprisingly little during normal quiet breathing. Alveolar \( P_{O_2} \) is fairly constant at 100 mm Hg, and alveolar \( P_{CO_2} \) stays close to 40 mm Hg.

Intuitively, you might think that \( P_{O_2} \) would increase when fresh air first enters the alveoli, then decrease steadily as oxygen leaves to enter the blood. Instead, we find only very small swings in \( P_{O_2} \). Why? The reasons are that (1) the amount of oxygen that enters the alveoli with each breath is roughly equal to the amount of oxygen that enters the blood, and (2) the amount of fresh air that enters the lungs with each breath is only a little more than 10% of the total lung volume at the end of inspiration.

### Ventilation and Alveolar Blood Flow Are Matched

Moving oxygen from the atmosphere to the alveolar exchange surface is only the first step in external respiration. Next, gas exchange must occur across the alveolar-capillary interface. Finally, blood flow (perfusion) past the alveoli must be high enough to
to recruit additional capillary beds during exercise is an example of the reserve capacity of the body.

At the local level, the body attempts to match air flow and blood flow in each section of the lung by regulating the diameters of the arterioles and bronchioles. Bronchiolar diameter is mediated primarily by $\text{CO}_2$ levels in exhaled air passing through them (Fig. 17.14). An increase in the $P_{\text{CO}_2}$ of expired air causes bronchioles to dilate. A decrease in the $P_{\text{CO}_2}$ of expired air causes bronchioles to constrict.

Although there is some autonomic innervation of pulmonary arterioles, there is apparently little neural control of pulmonary blood flow. The resistance of pulmonary arterioles to blood flow is regulated primarily by the oxygen content of the interstitial fluid around the arteriole. If ventilation of alveoli in one area of the lung is diminished, as shown in Figure 17.14b, the $P_{\text{O}_2}$ in that area decreases, and the arterioles respond by constricting, as shown in Figure 17.14c. This local vasoconstriction is adaptive because it diverts blood away from the under-ventilated region to better-ventilated parts of the lung.

Another important point must be noted here. Local control mechanisms are not effective regulators of air and blood flow under all circumstances. If blood flow is blocked in one pulmonary artery, or if air flow is blocked at the level of the larger capillary beds in which blood pressure is higher.

In a person at rest, some capillary beds in the apex (top) of the lung are closed off because of low hydrostatic pressure. Capillary beds at the base of the lung have higher hydrostatic pressure because of gravity and thus remain open. Consequently, blood flow is diverted toward the base of the lung. During exercise, when blood pressure rises, the closed apical capillary beds open, ensuring that the increased cardiac output can be fully oxygenated as it passes through the lungs. The ability of the lungs to recruit additional capillary beds during exercise is an example of the reserve capacity of the body.

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Mechanics of Breathing

**ALVEOLAR GASES**

As alveolar ventilation increases, alveolar \( \text{PO}_2 \) increases and \( \text{PCO}_2 \) decreases. The opposite occurs as alveolar ventilation decreases.

![Graph showing alveolar gases](image)

**Concept Check**

30. If a lung tumor decreases blood flow in one small section of the lung to a minimum, what happens to \( \text{PO}_2 \) in the alveoli in that section and in the surrounding interstitial fluid? What happens to \( \text{PO}_2 \) in that section? What is the compensatory response of the bronchioles in the affected section? Will the compensation bring ventilation in the affected section of the lung back to normal? Explain.

**Auscultation and Spirometry Assess Pulmonary Function**

Most pulmonary function tests are relatively simple to perform. Auscultation of breath sounds is an important diagnostic technique in pulmonary medicine, just as auscultation of heart sounds is an important technique in cardiovascular diagnosis. Breath sounds are more complicated to interpret than heart sounds, however, because breath sounds have a wider range of normal variation.

Normally, breath sounds are distributed evenly over the lungs and resemble a quiet “whoosh” made by flowing air. When air flow is reduced, such as in pneumothorax, breath sounds may be either diminished or absent. Abnormal sounds include various squeaks, pops, wheezes, and bubbling sounds caused by fluid and secretions in the airways or alveoli. Inflammation of the pleural membrane results in a crackling or grating sound known as a friction rub. It is caused by swollen, inflamed pleural membranes rubbing against each other, and it disappears when fluid again separates them.

Diseases in which air flow is diminished because of increased airway resistance are known as **obstructive lung diseases**. When patients with obstructive lower airway diseases are asked to exhale forcefully, air whistling through the narrowed airways creates a wheezing sound that can be heard even without a stethoscope. Depending on the severity of the disease, the bronchioles may even collapse and close off before a forced expiration is completed, reducing both the amount and rate of air flow as measured by a spirometer.

Obstructive lung diseases include asthma, obstructive sleep apnea, emphysema, and chronic bronchitis. The latter two are sometimes called **chronic obstructive pulmonary disease** (COPD) because of their ongoing, or chronic, nature. **Obstructive sleep apnea** (apnoea, breathless) results from obstruction of the upper airway, often due to abnormal relaxation of the muscles of the pharynx and tongue that increases airway resistance during inspiration.

**Asthma** is an inflammatory condition, often associated with allergies, that is characterized by bronchoconstriction and airway edema. Asthma can be triggered by exercise (exercise-induced asthma) or by rapid changes in the temperature or humidity of inspired air. Asthmatic patients complain of “air hunger” and difficulty breathing, or **dyspnea**. The severity of asthma attacks ranges from mild to life threatening. Studies of asthma at the cellular level show that a variety of chemical signals may be responsible for inducing asthmatic bronchoconstriction. Among these are acetylcholine, histamine, substance P (a neuropeptide), and leukotrienes secreted by mast cells, macrophages, and eosinophils. **Leukotrienes** are lipid-like bronchoconstrictors that are released during the inflammatory response. Asthma is treated with inhaled and oral medications that include \( \beta_2 \)-adrenergic agonists, anti-inflammatory drugs, and leukotriene antagonists.

**Concept Check**

31. Restrictive lung diseases decrease lung compliance. How will inspiratory reserve volume change in patients with a restrictive lung disease?

32. Chronic obstructive lung disease causes patients to lose the ability to exhale fully. How does residual volume change in these patients?

This completes our discussion of the mechanics of ventilation.
Local control mechanisms attempt to match ventilation and perfusion.

(a) Normally perfusion of blood past alveoli is matched to alveolar ventilation to maximize gas exchange.

(b) Ventilation-perfusion mismatch caused by under-ventilated alveoli.

If ventilation decreases in a group of alveoli, \( P_{CO_2} \) increases and \( P_{O_2} \) decreases. Blood flowing past those alveoli does not get oxygenated.

(c) Local control mechanisms try to keep ventilation and perfusion matched.

(d) Bronchiole diameter is mediated primarily by \( CO_2 \) levels in exhaled air passing through them.

Local Control of Arterioles and Bronchioles by Oxygen and Carbon Dioxide

<table>
<thead>
<tr>
<th>Gas composition</th>
<th>Bronchioles</th>
<th>Pulmonary arteries</th>
<th>Systemic arteries</th>
</tr>
</thead>
<tbody>
<tr>
<td>( P_{CO_2} ) increases</td>
<td>Dilate</td>
<td>(Constrict)*</td>
<td>Dilate</td>
</tr>
<tr>
<td>( P_{CO_2} ) decreases</td>
<td>Constrict</td>
<td>(Dilate)</td>
<td>Constrict</td>
</tr>
<tr>
<td>( P_{O_2} ) increases</td>
<td>(Constrict)</td>
<td>(Dilate)</td>
<td>Constrict</td>
</tr>
<tr>
<td>( P_{O_2} ) decreases</td>
<td>(Dilate)</td>
<td>Constrict</td>
<td>Dilate</td>
</tr>
</tbody>
</table>

* Parentheses indicate weak responses.

FIGURE QUESTIONS
A blood clot prevents gas exchange in a group of alveoli.

1. What happens to tissue and alveolar gases?
2. What do bronchioles and arterioles do in response?

Blood clots prevent gas exchange.
Mechanics of Breathing

RUNNING PROBLEM CONCLUSION

Emphysema
Edna leaves the office with prescriptions for a mucus-thinning drug, a bronchodilator, and anti-inflammatory drugs to keep her airways as open as possible. She has agreed to try to stop smoking once more and also has a prescription and brochures for that. Unfortunately, the lung changes that take place with COPD are not reversible, and Edna will require treatment for the rest of her life. According to the American Lung Association (www.lungusa.org), COPD is the fourth leading cause of death in the United States and costs more than $30 billion per year in direct medical costs and indirect costs such as lost wages.

In this running problem you learned about chronic obstructive pulmonary disease. Now check your understanding of the physiology in the problem by comparing your answers with those in the following table.

<table>
<thead>
<tr>
<th>Question</th>
<th>Facts</th>
<th>Integration and Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. What does narrowing of the airways do to the resistance airways offer to air flow?</td>
<td>The relationship between tube radius and resistance is the same for air flow as for blood flow: as radius decreases, resistance increases.</td>
<td>When resistance increases, the body must use more energy to create air flow.</td>
</tr>
<tr>
<td>2. Why do people with chronic bronchitis have a higher-than-normal rate of respiratory infections?</td>
<td>Cigarette smoke paralyzes the cilia that sweep debris and mucus out of the airways. Without the action of cilia, mucus and trapped particles pool in the airways.</td>
<td>Bacteria trapped in the mucus can multiply and cause respiratory infections.</td>
</tr>
<tr>
<td>3. Name the muscles that patients with emphysema use to exhale actively.</td>
<td>Normal passive expiration depends on elastic recoil of muscles and elastic tissue in the lungs.</td>
<td>Forceful expiration involves the internal intercostal muscles and the abdominal muscles.</td>
</tr>
<tr>
<td>4. When Edna fills her lungs maximally, the volume of air in her lungs is known as the __________ capacity. When she exhales all the air she can, the volume of air left in her lungs is the __________.</td>
<td>The maximum volume of air in the lungs is the <em>total lung capacity</em>. Air left in the lungs after maximal exhalation is the <em>residual volume</em>.</td>
<td>N/A</td>
</tr>
<tr>
<td>5. Why are Edna’s RBC count and hematocrit increased?</td>
<td>Because of Edna’s COPD, her arterial $P_{O_2}$ is low. The major stimulus for red blood cell synthesis is hypoxia.</td>
<td>Low arterial oxygen levels trigger EPO release, which increases the synthesis of red blood cells. More RBCs provide more binding sites for oxygen transport.</td>
</tr>
</tbody>
</table>

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- Interactive Physiology Animations

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<thead>
<tr>
<th>Page</th>
<th>Content</th>
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</thead>
</table>
| 2 | **Mechanics of Breathing**

Air flow into and out of the lungs is another example of the principle of mass flow. Like blood flow, air flow is bulk flow that requires a pump to create a pressure gradient and that encounters resistance, primarily from changes in the diameter of the tubes through which it flows. The mechanical properties of the pleural sacs and elastic recoil in the chest wall and lung tissue are essential for normal ventilation.

1. Aerobic metabolism in living cells consumes oxygen and produces carbon dioxide.)
2. Gas exchange requires a large, thin, moist exchange surface; a pump to move air; and a circulatory system to transport gases to the cells.
3. Respiratory system functions include gas exchange, pH regulation, vocalization, and protection from foreign substances.

**The Respiratory System**

4. Cellular respiration refers to cellular metabolism that consumes oxygen. External respiration is the exchange of gases between the atmosphere and cells of the body. It includes ventilation, gas exchange at the lung and cells, and transport of gases in the blood. Ventilation is the movement of air in and out of the lungs. (Fig. 17.1)

5. The respiratory system consists of anatomical structures involved in ventilation and gas exchange.

6. The upper respiratory tract includes the mouth, nasal cavity, pharynx, and larynx. The lower respiratory tract includes the trachea, bronchi, bronchioles, and exchange surfaces of the alveoli. (Fig. 17.2b)

7. The thoracic cage is bounded by the ribs, spine, and diaphragm. Two sets of intercostal muscles connect the ribs. (Fig. 17.2a)

8. Each lung is contained within a double-walled pleural sac that contains a small quantity of pleural fluid. (Figs. 17.2c, 17.3)

9. The two primary bronchi enter the lungs. Each primary bronchus divides into progressively smaller bronchi and finally into collapsible bronchioles. (Figs. 17.2e, 17.4)

10. The upper respiratory system filters, warms, and humidifies inhaled air.

11. The alveoli consist mostly of thin-walled type I alveolar cells for gas exchange. Type II alveolar cells produce surfactant. A network of capillaries surrounds each alveolus. (Fig. 17.2f, g)

12. Blood flow through the lungs equals cardiac output. Resistance to blood flow in the pulmonary circulation is low. Pulmonary arterial pressure averages 25/8 mm Hg.

**Gas Laws**

13. The total pressure of a mixture of gases is the sum of the pressures of the individual gases in the mixture (Dalton’s law). Partial pressure is the pressure contributed by a single gas in a mixture. (Fig. 17.6)

14. Bulk flow of air occurs down pressure gradients, as does the movement of any individual gas making up the air.

15. Boyle’s law states that as the volume available to a gas increases, the gas pressure decreases. The body creates pressure gradients by changing thoracic volume. (Fig. 17.6b)

**Ventilation**

16. A single respiratory cycle consists of an inspiration and an expiration.

17. Tidal volume is the amount of air taken in during a single normal inspiration. Vital capacity is tidal volume plus expiratory and inspiratory reserve volumes. Air volume in the lungs at the end of maximal expiration is the residual volume. (Fig. 17.7b)

18. Air flow in the respiratory system is directly proportional to the pressure gradient, and inversely related to the resistance to air flow offered by the airways.

19. During inspiration, alveolar pressure decreases, and air flows into the lungs. Inspiration requires contraction of the inspiratory muscles and the diaphragm. (Fig. 17.9)

20. Expiration is usually passive, resulting from elastic recoil of the lungs.

21. Active expiration requires contraction of the internal intercostal and abdominal muscles.

22. Intrapleural pressures are subatmospheric because the pleural cavity is a sealed compartment. (Figs. 17.9, 17.10)

23. Compliance is a measure of the ease with which the chest wall and lungs expand. Loss of compliance increases the work of breathing. Elastance is the ability of a stretched lung to return to its normal volume.

24. Surfactant decreases surface tension in the fluid lining the alveoli. Reduced surface tension prevents smaller alveoli from collapsing and also makes it easier to inflate the lungs. (Fig. 17.11)

25. The diameter of the bronchioles determines how much resistance they offer to air flow.

26. Increased CO₂ in expired air dilates bronchioles. Parasympathetic neurons cause bronchoconstriction in response to irritant stimuli. There is no significant sympathetic innervation of bronchioles, but epinephrine causes bronchodilation. (Tbl. 1.7)

27. Total pulmonary ventilation = tidal volume x ventilation rate. Alveolar ventilation = ventilation rate x (tidal volume – dead space volume). (Fig. 17.12a)

28. Alveolar gas composition changes very little during a normal respiratory cycle. Hyperventilation increases alveolar P O₂ and decreases alveolar P CO₂. Hypoventilation has the opposite effect. (Fig. 17.13)

29. Local mechanisms match airflow and blood flow around the alveoli. Increased levels of CO₂ dilate bronchioles, and decreased O₂ constricts pulmonary arterioles. (Fig. 17.14)
Mechanics of Breathing

Questions

Level One Reviewing Facts and Terms

1. List four functions of the respiratory system.
2. Give two definitions for the word **respiration**.
3. Which sets of muscles are used for normal quiet inspiration? For normal quiet expiration? For active expiration? What kind(s) of muscles are the different respiratory muscles (skeletal, cardiac, or smooth)?
4. What is the function of pleural fluid?
5. Name the anatomical structures that an oxygen molecule passes on its way from the atmosphere to the blood.
6. Diagram the structure of an alveolus, and state the function of each part. How are capillaries associated with an alveolus?
7. Trace the path of the pulmonary circulation. About how much blood is found here at any given moment? What is a typical arterial blood pressure for the pulmonary circuit, and how does this pressure compare with that of the systemic circulation?
8. What happens to inspired air as it is conditioned during its passage through the airways?
9. During inspiration, most of the thoracic volume change is the result of movement of the ________.
10. Describe the changes in alveolar and intrapleural pressure during one respiratory cycle.
11. What is the function of surfactants in general? In the respiratory system?
12. Of the three factors that contribute to the resistance of air flow through a tube, which plays the largest role in changing resistance in the human respiratory system?
13. Match the following items with their correct effect on the bronchioles:
   - **(a)** histamine
   - **(b)** epinephrine
   - **(c)** acetylcholine
   - **(d)** increased \( P_{CO_2} \)
   - 1. bronchoconstriction
   - 2. bronchodilation
   - 3. no effect
14. Refer to the spirogram in the figure below:

![Spirogram](image)

(a) Label tidal volume \( (V_T) \), inspiratory and expiratory reserve volumes \( (IRV \text{ and } ERV) \), residual volume \( (RV) \), vital capacity \( (VC) \), total lung capacity \( (TLC) \).
(b) What is the value of each of the volumes and capacities you labeled?
(c) What is this person’s ventilation rate?

Level Two Reviewing Concepts

15. Compare and contrast the terms in each of the following sets:
   - (a) compliance and elastance
   - (b) inspiration, expiration, and ventilation
   - (c) intrapleural pressure and alveolar pressure
   - (d) total pulmonary ventilation and alveolar ventilation
   - (e) type I and type II alveolar cells
   - (f) pulmonary circulation and systemic circulation
16. List the major paracrines and neurotransmitters that cause bronchoconstriction and bronchodilation. What receptors do they act through? (muscarinic, nicotinic, \( \alpha, \beta_1, \beta_2 \))
17. Compile the following terms into a map of ventilation. Use up arrows, down arrows, greater than symbols (\( > \)), and less than symbols (\( < \)) as modifiers. You may add other terms.

<table>
<thead>
<tr>
<th>Term</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>abdominal muscles</td>
<td></td>
</tr>
<tr>
<td>air flow</td>
<td></td>
</tr>
<tr>
<td>contract</td>
<td></td>
</tr>
<tr>
<td>diaphragm</td>
<td></td>
</tr>
<tr>
<td>expiratory muscles</td>
<td></td>
</tr>
<tr>
<td>external intercostals</td>
<td></td>
</tr>
<tr>
<td>forced breathing</td>
<td></td>
</tr>
<tr>
<td>in, out, from, to</td>
<td></td>
</tr>
<tr>
<td>intercostals</td>
<td></td>
</tr>
<tr>
<td>pleural fluid</td>
<td></td>
</tr>
<tr>
<td>relax</td>
<td></td>
</tr>
<tr>
<td>scalenes</td>
<td></td>
</tr>
</tbody>
</table>

18. Decide whether each of the following parameters will increase, decrease, or not change in the situations given.

   - (a) airway resistance with bronchodilation
   - (b) intrapleural pressure during inspiration
   - (c) air flow with bronchoconstriction
   - (d) bronchiolar diameter with increased \( P_{CO_2} \)
   - (e) tidal volume with decreased compliance
   - (f) alveolar pressure during expiration
19. Define the following terms: pneumothorax, spirometer, auscultation, hypoventilation, bronchoconstriction, minute volume, partial pressure of a gas.
20. The cartoon coyote is blowing up a balloon in another attempt to catch the roadrunner. He first breathes in as much air as he can, then blows out all he can into the balloon.
   - (a) The volume of air in the balloon is equal to the ________ of the coyote’s lungs. This volume can be measured directly by measuring the balloon volume or by adding which respiratory volumes together?
   - (b) In 10 years, when the coyote is still chasing the roadrunner, will he still be able to put as much air into the balloon in one breath? Explain.
21. Match the descriptions to the appropriate phase(s) of ventilation:

   - (a) usually depend(s) on elastic recoil
   - (b) is/are easier when lung compliance decreases
   - (c) is/are driven mainly by positive intrapleural pressure generated by muscular contraction
   - (d) is usually an active process requiring smooth muscle contraction

<table>
<thead>
<tr>
<th>Phase</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. inspiration</td>
<td>(a) usually depend(s) on elastic recoil</td>
</tr>
<tr>
<td>2. expiration</td>
<td>(b) is/are easier when lung compliance decreases</td>
</tr>
<tr>
<td>3. both inspiration and expiration</td>
<td>(c) is/are driven mainly by positive intrapleural pressure generated by muscular contraction</td>
</tr>
<tr>
<td>4. neither</td>
<td>(d) is usually an active process requiring smooth muscle contraction</td>
</tr>
</tbody>
</table>
Mechanics of Breathing

22. Draw and label a graph showing the $P_{O_2}$ of air in the primary bronchi during one respiratory cycle. (Hint: What parameter goes on each axis?)

23. Lung compliance increases but chest wall compliance decreases as we age. In the absence of other changes, would the following parameters increase, decrease, or not change as compliance decreases?
(a) work required for breathing
(b) ease with which lungs inflate
(c) lung elastance
(d) airway resistance during inspiration

24. Will pulmonary surfactant increase, decrease, or not change the following?
(a) work required for breathing
(b) lung compliance
(c) surface tension in the alveoli

Level Three Problem Solving

25. Assume a normal female has a resting tidal volume of 400 mL, a respiratory rate of 15 breaths/min, and an anatomic dead space of 125 mL. When she exercises, which of the following scenarios would be most efficient for increasing her oxygen delivery to the lungs?
(a) increase respiratory rate to 20 breaths/min but have no change in tidal volume
(b) increase tidal volume to 550 mL but have no change in respiratory rate
(c) increase tidal volume to 500 mL and respiratory rate to 15 breaths/min

Which of these scenarios is most likely to occur during exercise in real life?

26. A 30-year-old computer programmer has had asthma for 15 years. When she lies down at night, she has spells of wheezing and coughing. Over the years, she has found that she can breathe better if she sleeps sitting nearly upright. Upon examination, her doctor finds that she has an enlarged thorax. Her lungs are overinflated on X-ray. Here are the results of her examination and pulmonary function tests. Use the normal values and abbreviations in Figure 17.8 to help answer the questions.
Ventilation rate: 16 breaths/min
Tidal volume: 600 mL
ERV: 1000 mL
RV: 3500 mL
Inspiratory capacity: 1800 mL
Vital capacity: 2800 mL
Functional residual capacity: 4500 mL
TLC: 6300 mL

After she is given a bronchodilator, her vital capacity increased to 3650 mL.
(a) What is her minute volume?
(b) Explain the change in vital capacity with bronchodilators.
(c) Which other values are abnormal? Can you explain why they might be, given her history and findings?

Level Four Quantitative Problems

27. A container of gas with a movable piston has a volume of 500 mL and a pressure of 60 mm Hg. The piston is moved, and the new pressure is 150 mm Hg. What is the new volume of the container?

28. You have a mixture of gases in dry air, with an atmospheric pressure of 760 mm Hg. Calculate the partial pressure of each gas if the composition of the air is:
(a) 21% oxygen, 78% nitrogen, 0.3% carbon dioxide
(b) 40% oxygen, 13% nitrogen, 45% carbon dioxide, 2% hydrogen
(c) 10% oxygen, 15% nitrogen, 1% argon, 25% carbon dioxide

29. Li is a tiny woman, with a tidal volume of 400 mL and a respiratory rate of 12 breaths per minute at rest. What is her total pulmonary ventilation? Just before a physiology exam, her ventilation increases to 18 breaths per minute from nervousness. Now what is her total pulmonary ventilation? Assuming her anatomic dead space is 120 mL, what is her alveolar ventilation in each case?

30. You collected the following data on your classmate Neelesh:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minute volume</td>
<td>5004 mL/min</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>3 breaths/15 sec</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>4800 mL</td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>1000 mL</td>
</tr>
</tbody>
</table>

What are Neelesh’s tidal volume and inspiratory reserve volume?

31. Use the figure below to help solve this problem. A spirometer with a volume of 1 liter ($V_1$) is filled with a mixture of oxygen and helium, with the helium concentration being 4 g/L ($C_1$). Helium does not move from the lungs into the blood or from the blood into the lungs. A subject is told to blow out all the air he possibly can. Once he finishes that exhalation, his lung volume is $V_2$. He then puts the spirometer tube in his mouth and breathes quietly for several breaths. At the end of that time, the helium is evenly dispersed in the spirometer and the subject’s lungs. A measurement shows the new concentration of helium is 1.9 g/L. What was the subject’s lung volume at the start of the experiment? (Hint: $C_1V_1 = C_2V_2$)

32. The graph shows one lung under two different conditions, A and B. What does this graph show? (a) the effect of lung volume on pressure, or (b) the effect of pressure on lung volume? In which condition does the lung have higher compliance, or is compliance the same in the two situations?
Mechanics of Breathing

Answers to Concept Check Questions

1. Cellular respiration is intracellular and uses O₂ and organic substrates to produce ATP. External respiration is exchange and transport of gases between the atmosphere and cells.

2. The upper respiratory tract includes the mouth, nasal cavity, pharynx, and larynx. The lower respiratory tract includes the trachea, bronchi, bronchioles, and exchange surface of lungs.

3. Velocity is highest in the trachea and lowest in the bronchioles.

4. Pleural fluid reduces friction and holds the lungs tight against the chest wall.

5. The thoracic cage consists of the rib cage with intercostal muscles, spinal (vertebral) column, and diaphragm. The thorax contains two lungs in pleural sacs, the heart and pericardial sac, esophagus, and major blood vessels.

6. The bronchioles are collapsible.

7. If cilia cannot move mucus, the mucus collecting in the airways triggers a cough reflex to clear out the mucus.

8. Blood flow is approximately equal in the pulmonary trunk and aorta. (Normally some venous blood leaving the bronchi, pleura, and part of the heart bypasses the pulmonary circulation and drains directly into the left side of the heart. This is called an anatomic shunt.)

9. Increased hydrostatic pressure causes greater net filtration out of capillaries and may result in pulmonary edema.

10. Mean pressure = 8 mm Hg + 1/3(25 – 8) mm Hg = 8 + 17/3 mm Hg = 13.7 mm Hg.

11. 720 mm Hg × 0.78 = 562 mm Hg

12. 700 mm Hg – 47 mm Hg = 653 mm Hg × 21% = 137.1 mm Hg PₐO₂

13. Lung capacities are the sum of two or more lung volumes.

14. Residual volume cannot be measured directly.

15. If aging individuals have reduced vital capacity while total lung capacity does not change, then residual volume must increase.

16. As air becomes humidified, the PₐO₂ decreases.

17. Air flow reverses direction during a respiratory cycle, but blood flows in a loop and never reverses direction.

18. See Figures 17.2c and 17.3. The lungs are enclosed in a pleural sac. One pleural membrane attaches to the lung, and the other lines the thoracic cage. Pleural fluid fills the pleural sac.

19. Scarlett will be more successful if she exhales deeply, as this will decrease her thoracic volume and will pull her lower rib cage inward.

20. Inability to cough decreases the ability to expel the potentially harmful material trapped in airway mucus.

21. A hiccup causes a rapid decrease in both intrapleural pressure and alveolar pressure.

22. The knife wound would collapse the left lung if the knife punctured the pleural membrane. Loss of adhesion between the lung and chest wall would release the inward pressure exerted on the chest wall, and the rib cage would expand outward. The right side would be unaffected as the right lung is contained in its own pleural sac.

23. Normally, lung and chest wall elastance contribute more to the work of breathing.

24. Scar tissue reduces lung compliance.

25. Without surfactant, the work of breathing increases.

26. When bronchiolar diameter decreases, resistance increases.

27. Neurotransmitter is acetylcholine, and receptor is muscarinic.

28. Increased tidal volume increases alveolar PₐO₂.

29. Increased breathing rate increases alveolar PₐO₂. Increasing breathing rate or tidal volume increases alveolar ventilation.

30. PₐO₂ in alveoli in the affected section will increase because O₂ is not leaving the alveoli. PₐCO₂ will decrease because new CO₂ is not entering the alveoli from the blood. Bronchioles constrict when PₐCO₂ decreases (see Fig. 17.14), shunting air to areas of the lung with better blood flow. This compensation cannot restore normal ventilation in this section of lung, and local control is insufficient to maintain homeostasis.

31. Inspiratory reserve volume decreases.

32. Residual volume increases in patients who cannot fully exhale.

Answers to Figure and Graph Questions

Figure 17.9: 1. Alveolar pressure is greatest in the middle of expiration and least in the middle of inspiration. It is equal to atmospheric pressure at the beginning and end of inspiration and expiration. 2. When lung volume is at its minimum, alveolar pressure is (c) moving from maximum to minimum and external intercostal muscle contraction is (b) minimal. 3. 2 breaths/8 sec = 7 breaths/60 sec = 15 breaths/min.

Figure 17.12: Shallow and rapid: total pulmonary ventilation = 6000 mL/min, 150 mL fresh air, alveolar ventilation = 3000 mL/min. Slow and deep: total pulmonary ventilation = 6000 mL/min, 600 mL fresh air, alveolar ventilation = 4800 mL/min. Slow and deep is the most efficient.

Figure 17.13: Alveolar PₐO₂ goes to 120 mm Hg and PₐCO₂ falls to about 19 mm Hg.

Figure 17.14: 1. Alveolar PₐO₂ increases and PₐCO₂ decreases in the affected alveoli. Local tissue PₐO₂ increases. 2. This constricts local arterioles, which then shunts blood to better-perfused sections of lung. Bronchioles constrict to divert air to better-perfused alveoli.
Answers to Review Questions

Level One  Reviewing Facts and Terms
1. gas exchange, vocalization, pH regulation, and protection
2. Cellular respiration—oxygen and nutrients are used for energy production. External respiration—gas exchange between atmosphere and cells.
3. Quiet inspiration—external intercostals, scalenes, and diaphragm. Quiet expiration—no significant muscle contraction. Active expiration—internal intercostals and abdominal muscles. These are all skeletal muscles.
4. Lubrication between lungs and internal thoracic surface
5. Nose and mouth, pharynx, larynx, trachea, main bronchi, secondary bronchi, bronchioles, epithelium of the alveoli, interstitial fluid, and capillary endothelium
6. See Figure 17.2a and b. Type I—gas exchange; type II—surfactant. Macrophages ingest foreign material. Capillary endothelium is almost fused to the alveolar epithelium, and the space between alveoli is almost filled with capillaries.
7. Right ventricle to pulmonary trunk, to left and right pulmonary arteries, smaller arteries, arterioles, capillaries, venules, small veins, pulmonary veins, left atrium. Contains about 0.5 L of blood. Arterial pressure is 25/8, compared with 120/80 for systemic.
8. Warmed, humidified, and cleaned (filtered)
9. diaphragm
10. See Figure 17.9.
11. Surfactant decreases surface tension of water and makes it easier for lungs to inflate and stay inflated.
12. radius of the airways
13. (a) 1 (b) 2 (c) 1 (d) 2
14. (a) See Figure 17.7. (b) $V_t = 0.5 \text{ L}$, IRV = 1.25 L, ERV = 1.0 L (c) 3 breaths/15 sec $\times$ 60 sec/min = 12 br/min

Level Two  Reviewing Concepts
15. (a) Compliance—ability to deform in response to force; elastance—ability to resume original shape after deforming force has been removed. (b) Ventilation—air exchange between atmosphere and lungs. Inspiration—air movement into lungs.Expiration—air movement out of lungs. (c) Intrapleural pressure—always subatmospheric (except during forced expiration, when it may become positive); alveolar pressures vary from subatmospheric to above atmospheric. (d) Total pulmonary ventilation—volume of air entering or leaving alveoli in a given period of time. Alveolar ventilation—volume of air entering or leaving alveoli in a given period of time. (e) Type I—thin cells for gas exchange; Type II—synthesize and secrete surfactant. (f) Pulmonary—from right heart to lung and back to left atrium. Systemic—from left heart to most tissues and back to right atrium.
16. Bronchoconstrictors: histamine, leukotrienes, acetylcholine (muscarinic); bronchodilators: carbon dioxide, epinephrine ($\beta_2$)
17. See Figs. 17.8 and 17.9.
18. (a) decrease (b) decrease (c) decrease (d) increase (e) decrease (f) increase
20. (a) tidal capacity Sum of tidal volume and expiratory and inspiratory reserve volumes. (b) No, because lung function decreases with age as elastance and compliance diminish.
21. (a) 2, (b) 2, (c) 4, (d) 4
22. X-axis—time; Y-axis—$P_{O_2}$. During inspiration, the $P_{O_2}$ of the primary bronchi will increase, as fresh air ($P_{O_2} = 160 \text{ mm Hg}$) replaces the stale air ($P_{O_2} = 100 \text{ mm Hg}$). During expiration, the $P_{O_2}$ will decrease, as the oxygen-depleted air exits the alveoli. The curve will vary from 100 mm Hg to 160 mm Hg.
23. (a) Work increases. (b) Lungs inflate more easily. (c) Elastance decreases. (d) Airway resistance is not affected.
24. (a) decrease (b) increase (c) decrease

Level Three  Problem Solving
25. Resting alveolar ventilation = 3575 mL/min. Exercising: (a) 5500 mL/min (b) 5525 mL/min (c) 5625 mL/min. Increasing both rate and depth has the largest effect and is what would happen in real life.
26. (a) 9600 mL/min. (b) Dilating bronchioles reduces airway resistance. The patient is able to force more air out of the lungs on expiration, which increases her ERV and decreases her RV. (c) Respiratory rate is normal, but lung volumes are abnormal. Her high RV is confirmed by the X-ray. In obstructive lung diseases such as asthma, the bronchioles collapse on expiration, trapping air in the lungs and resulting in hyperinflation. Her low IRV accounts for most of the low vital capacity and is to be expected in someone with asthma, where the lungs are already overinflated at the beginning of inspiration. Her higher tidal volume may be the result of the effort she must exert to breathe.

Level Four  Quantitative Problems
27. $P_1V_1 = P_2V_2$. New volume = 200 mL.
28. (a) $O_2 = 160 \text{ mm Hg}$, nitrogen = 593 mm Hg, $CO_2 = 2.3 \text{ mm Hg}$. (b) $O_2 = 304 \text{ mm Hg}$, nitrogen = 99 mm Hg, $CO_2 = 342 \text{ mm Hg}$, $H_2 = 15 \text{ mm Hg}$. (c) $O_2 = 76 \text{ mm Hg}$, nitrogen = 114 mm Hg, argon = 8 mm Hg, $CO_2 = 190 \text{ mm Hg}$.
29. Total pulmonary ventilation = 4800 mL/min. Before an exam, ventilation is 7200 mL/min. Alveolar ventilation is 3360 mL/min (at rest) and 5040 mL/min (before exam).
30. Tidal volume = 417 mL/breath. IRV = 3383 mL.
31. Lung volume is 1.1 L. (Did you forget to subtract the volume of the spirometer?)
32. (b) The lung in A has the highest compliance.