

The Cardiovascular System

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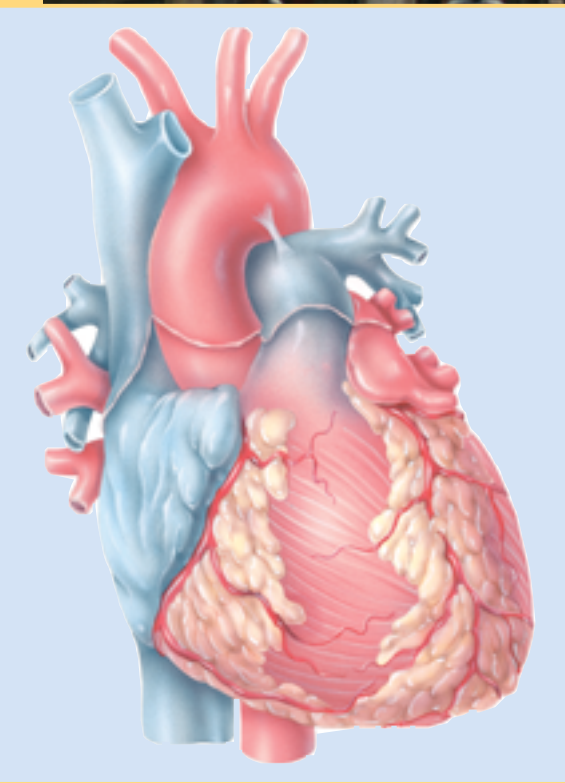
LANCE ARMSTRONG AND THE INCREDIBLE HEART

Lance Armstrong, seven-time winner of the Tour de France, is not like the rest of us, and one of his most unusual features is his heart. At rest, for example, Armstrong's ticker is said to beat 32 times a minute, less than half of the average for people his age. That slow rhythm provides a big clue to the efficiency of his heart (and musculature) because it suggests how much power he can produce when his heart speeds up.

Armstrong obviously has terrific genetics for an endurance athlete, and he has also perfected the arduous training needed to beat all contenders in the 3,200+ kilometer Tour de France. But most healthy people can get some benefits from "athletic heart syndrome," which is the series of changes in the heart's anatomy and physiology that result from regular, strenuous exercise. This training allows the heart's four chambers to dilate more, so that they accept and discharge more blood on each stroke, thereby delivering more oxygen and nutrients to the body. These changes, combined with many others that emerge from a combination of genetics and training, explain why Armstrong had the fastest times on the Tour, seven years running.

In this chapter, we will introduce the structure and function of the heart as well as the rest of the cardiovascular system, and then look at some things that can go awry with the body's essential delivery and trash-removal system. The cardiovascular system, or CV, is a triumph of sophisticated design, but cardiovascular problems are also the predominant cause of death in many developed countries.

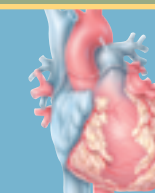
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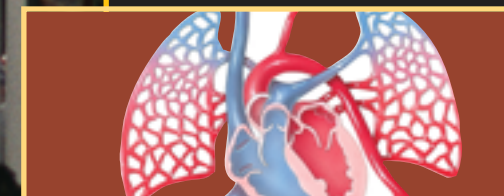
CHAPTER OUTLINE



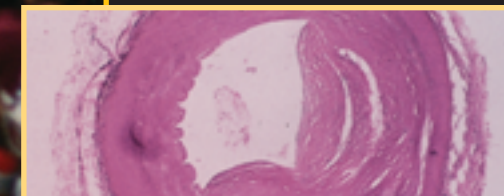
The Heart Ensures Continual, 24/7 Nutrient Delivery p. 000



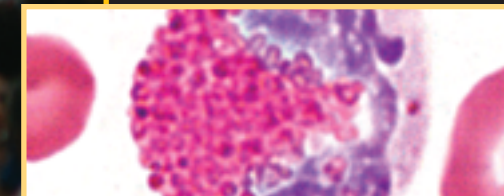
Blood Vessels and Capillary Transport Involve Miles of Sophisticated Plumbing p. 000



Different Circulatory Pathways Have Specific Purposes p. 000



Cardiovascular Disorders Have Life-threatening Consequences p. 000



Blood Consists of Plasma, Cells, and Other Formed Elements p. 000



Blood Can Suffer Many Disorders p. 000



The Heart Ensures Continual, 24/7 Nutrient Delivery

LEARNING OBJECTIVES

Describe the structure of the heart.

Trace the flow of blood through the heart.

Explore the electrical signaling that produces contraction.

Describe how the tracings on an ECG reflect the heart's beat.

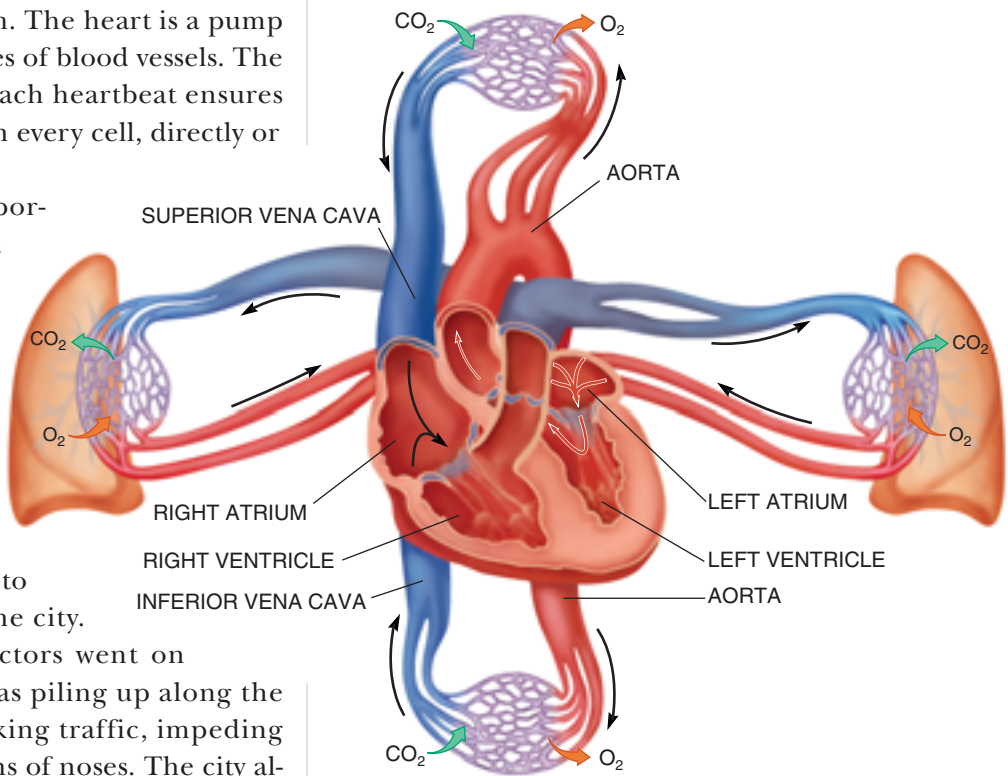
On the day Dr. Seuss's Grinch discovered the true meaning of Christmas his heart grew three sizes. The tin man in the **Wizard of Oz** wanted a heart so he could have emotions. We've all heard the heart described as our emotional center, but the heart is literally the center of the cardiovascular system. The heart is a pump that pushes blood through miles of blood vessels. The blood pressure generated by each heartbeat ensures that nutrients and oxygen reach every cell, directly or indirectly.

To understand the importance of the CV system, look at any large city. Vehicles transport food, goods, and raw materials into the city and deliver them to residents and institutions. After the goods are consumed, waste that is left over must be recycled, burned, reused, or shipped away. Any obstruction to this flow is likely to damage the city.

Within days after trash collectors went on strike in the 1980s, garbage was piling up along the streets of New York City, blocking traffic, impeding business, and offending millions of noses. The city almost ground to a halt until a new contract was signed and trash removal resumed. Similarly, if the human body cannot move water, nutrients, and oxygen into the tissues, and remove wastes from them, tissues will die, and eventually the organism will die as well.

In delivering oxygen and removing carbon dioxide, the cardiovascular and respiratory systems work to-

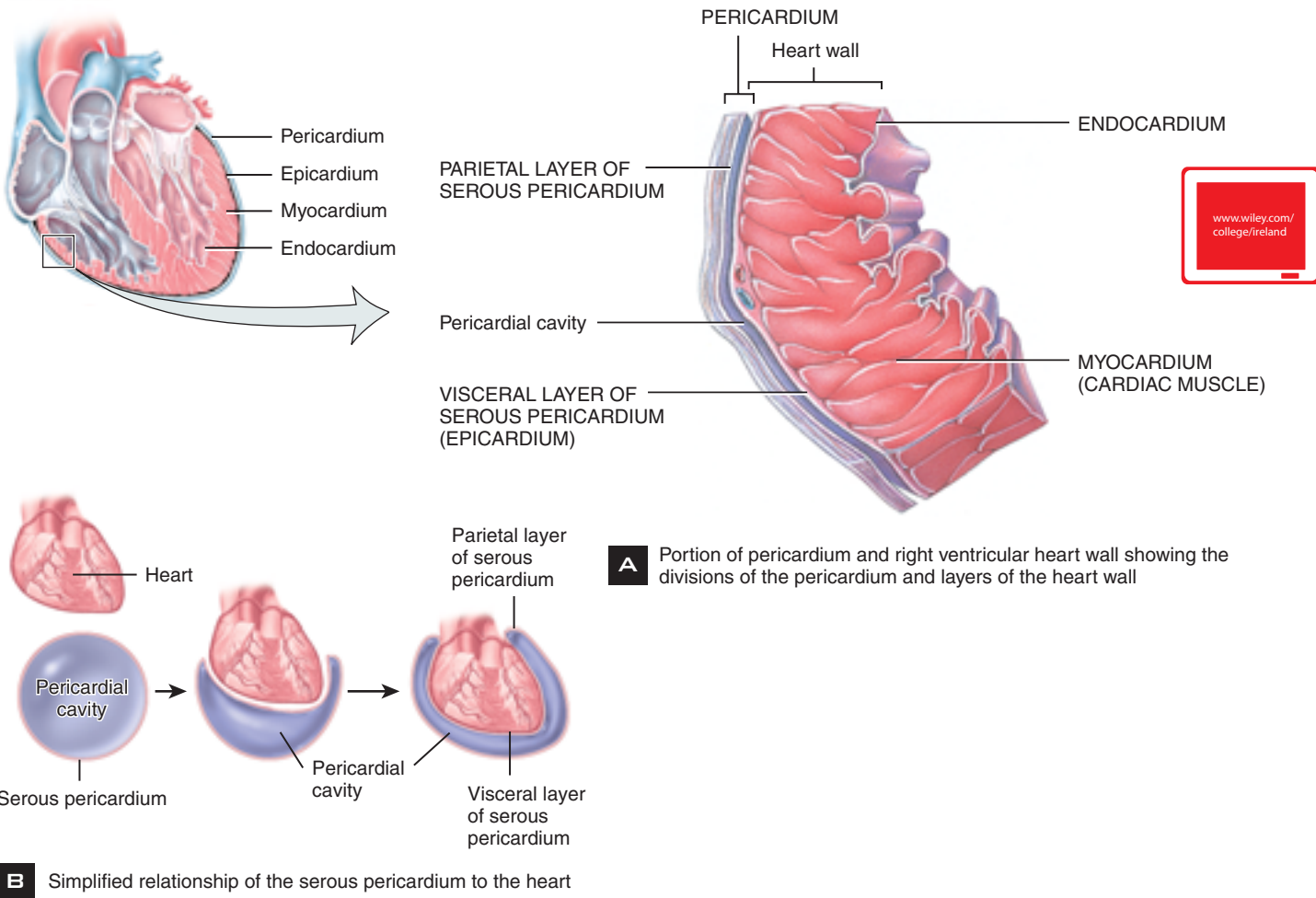
gether. The **respiratory** system (Chapter 12) brings oxygen to the blood and removes carbon dioxide from it. The **cardiovascular** system transports that blood, carry-



Basic schematic of the CV system

FIGURE 11.1

The heart and blood vessels form a *closed circuit* that transports blood from the heart to various parts of the body and back to the heart.



A Portion of pericardium and right ventricular heart wall showing the divisions of the pericardium and layers of the heart wall

Pericardium FIGURE 11.2

The pericardium includes two layers, one lining the walls of the thoracic cavity and the other attached to the cardiac muscle of the heart. Between these two membranes is a thin slippery

layer of serous fluid, allowing the heart to move within the cavity without damaging itself or the thoracic area.

ing nutrients, wastes, and dissolved gases to and from the tissues. The cardiovascular system includes the **heart**, **blood vessels**, and **blood**. We will look first at the heart and the blood vessels, and then at the blood that flows through that closed circuit (**FIGURE 11.1**).

The heart resides in the center of the thoracic cavity, literally hanging by the great blood vessels that deliver and remove blood. The **pericardium** (**FIGURE 11.2**) is a serous membrane that surrounds the heart and allows it to beat without causing

damage to itself—beating causes the heart to jump around in the **mediastinum**.

The heart is composed of four chambers—two **ventricles** and two **atria**. The atria are smaller, thin-walled chambers sitting atop the thick-walled, muscular ventricles. The atria receive blood from large veins

Mediastinum

The area between the lungs, containing the heart, lymphatics, and vessels of the thoracic region.

and direct it into the ventricles, which expel the blood under great pressure toward the lungs or body.

During development, the heart forms from two adjacent vessels. By the third week of development, these two vessels fuse to form one large chamber (a ventricle) with two smaller vessels delivering blood to it. The heart is upside down at this point, with the ventricle above the two incoming vessels. Around week 5, the heart curves back on itself in an “S” turn, creating the familiar anatomy, with the atria on top (**FIGURE 11.3**).

The adult heart is shown in **FIGURE 11.4**. Note the thick ventricular walls, especially in the left ventricle. It is the left ventricle that must generate enough force to push blood throughout the body. The

less muscular right ventricle pushes blood only to the nearby lungs. The walls of the atria are even less muscular because these chambers are essentially holding tanks for blood after it returns from the body or lungs, rather than pumping chambers.

Each heart chamber contains one valve that opens to allow blood to enter and then closes when the chamber contracts to pump. Because these valves are found between the atria and the ventricles, they are referred to as atrioventricular valves. The atrioventricular valves are the **tricuspid**

Tricuspid

The valve between the right atrium and right ventricle, composed of three points (cusps) of connective tissue.

Bicuspid

The valve between the left atrium and left ventricle, composed of two opposing cusps or flaps of connective tissue.

Prolapse

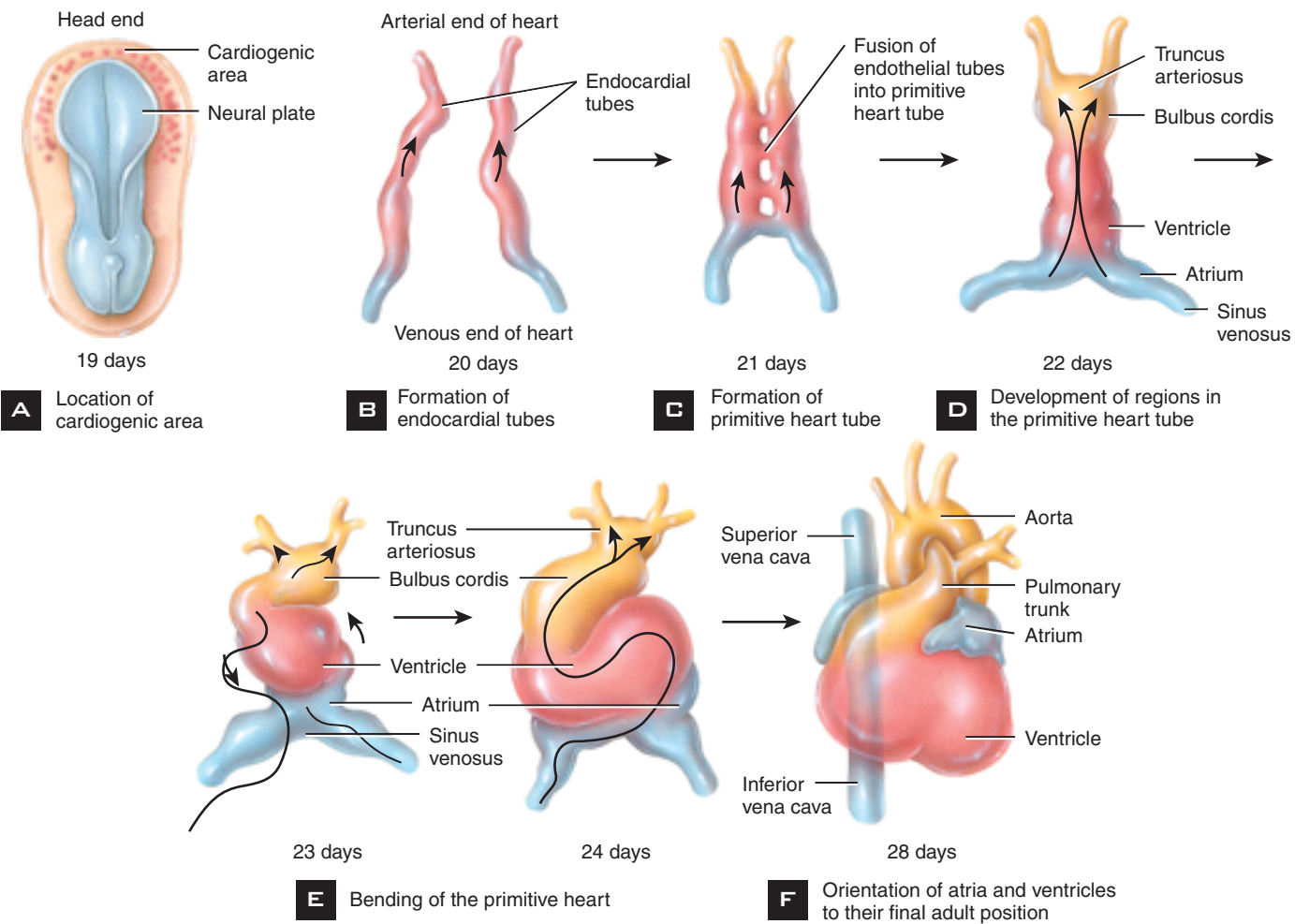
Movement of an organ from its original position in the body, usually because of gravity or other pressure.

valve in the right ventricle and the **bicuspid**, or **mitral** valve, in the left ventricle. Valves are composed of dense, irregular connective tissue and are held in place by the tuft-like **papillary muscles**. The **chordae tendineae** (literally chords of tendons) are the “heart strings” that anchor the cusps of the valves to the papillary muscles. When we listen to a heart beat, even without a stethoscope, part of what we are hearing is the thrumming of the heartstrings as they are pulled tight and pressurized blood flows past them.

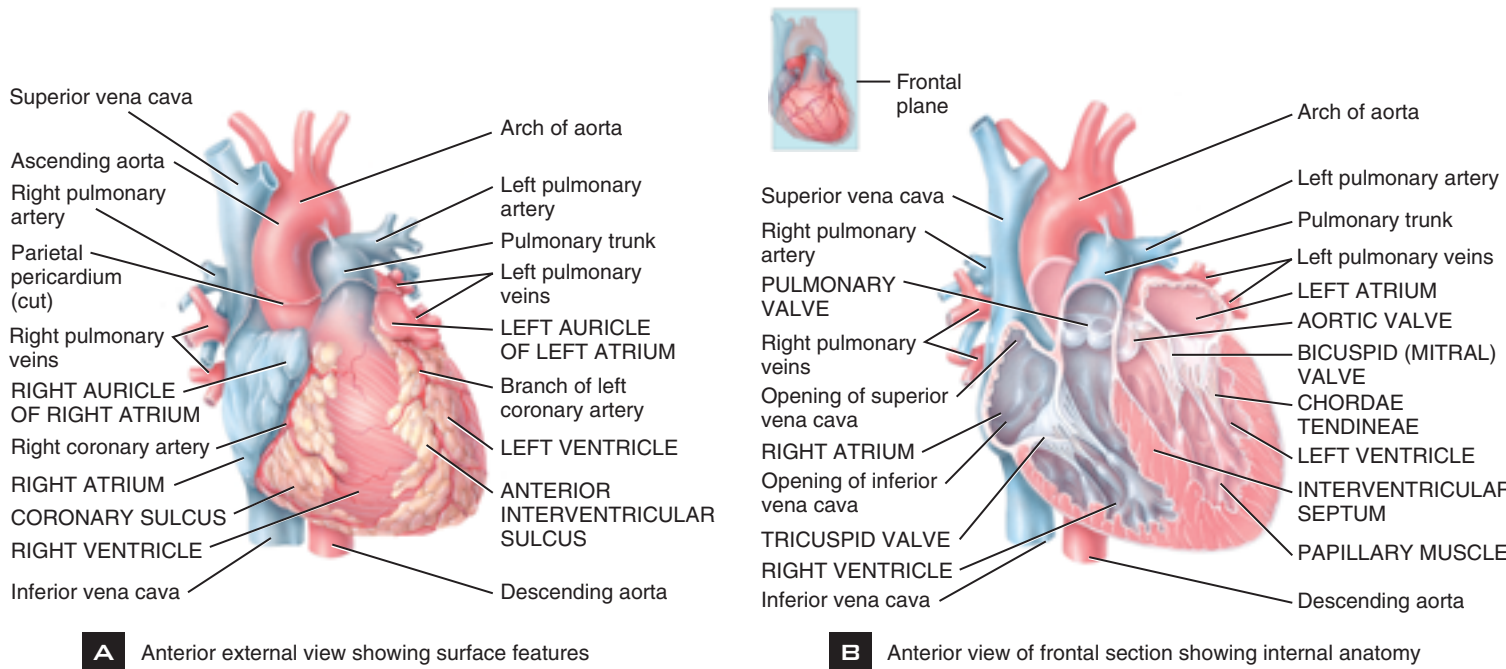
If the mating surfaces of a valve fit poorly, blood can slip past, causing the valve to flutter. This fluttering creates a murmur (an audible change in heart sound) and can possibly lead to valve **prolapse**. The most leak-

prone valve is the mitral valve in the left ventricle. When the mitral valve fits poorly, a condition called mitral valve prolapse (MVP) results. MVP runs in families and affects more women than men. Although MVP saps heart efficiency, patients rarely report symptoms and require no medical treatment.

At the base of the great arteries leaving the heart are the **pulmonary** and **aortic valves**. These valves are shaped like three flexible bowls, anchored to the walls of the great vessel (**FIGURE 11.5**). When the heart pushes blood into the pulmonary or aortic artery, the bowls flatten against the artery walls so that the blood can flow freely. When pressure drops inside the heart, blood in the arteries pushes back, ballooning the three bowls so that they open and contact each another, closing the arterial opening leading back to the heart. Because these valves lack the chordae tendineae, these valves make no humming sound when they close. Instead, they produce a tapping noise as they fill and knock against one another. This sharper noise can be heard when listening to the heart beat.



Development of the heart **FIGURE 11.3**

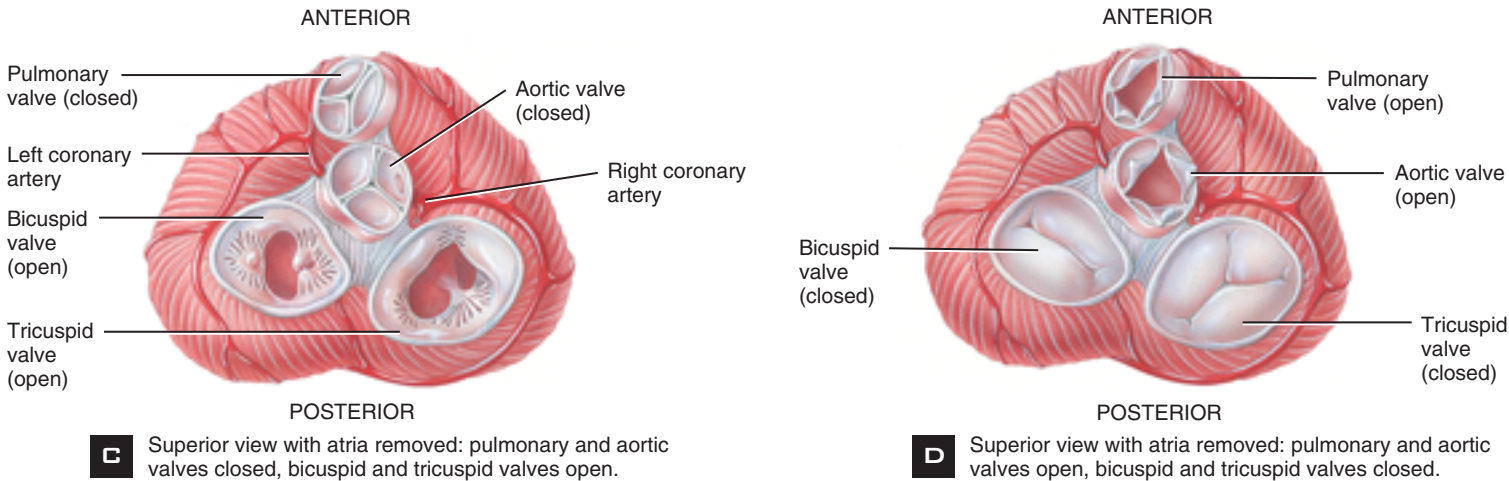
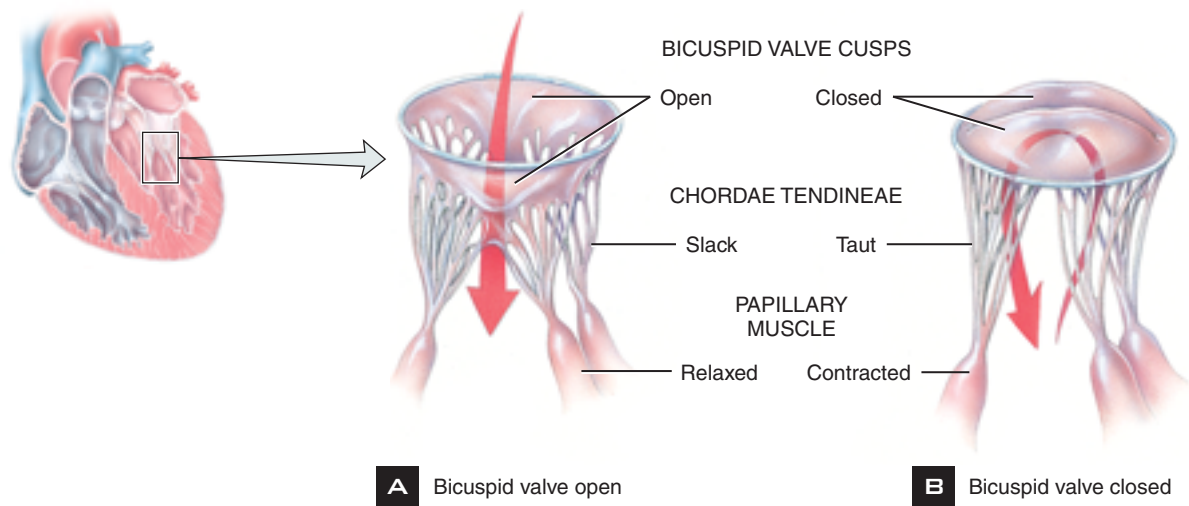


The adult heart **FIGURE 11.4**

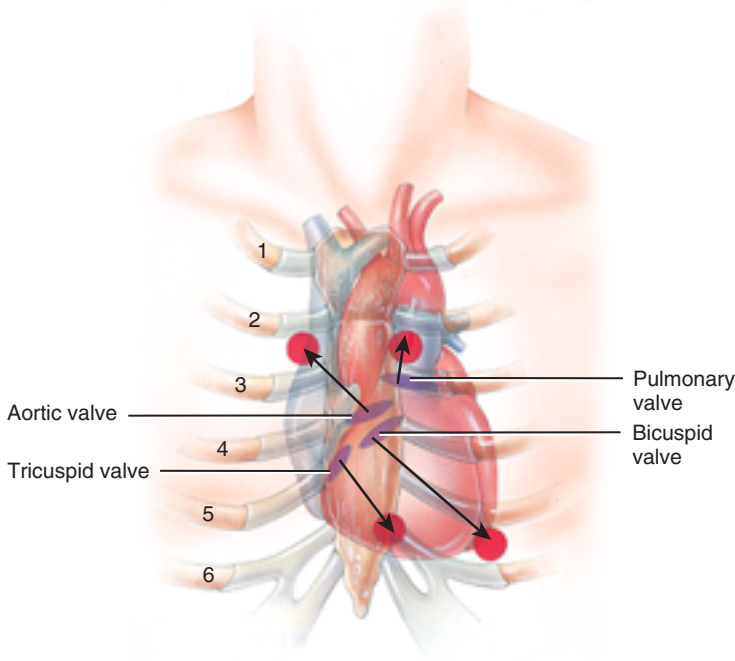
Thinking about the heartbeat, we immediately imagine the characteristic “lubb-dupp” sound (**FIGURE 11.6**). This sound is generated by the heart valves, and it can have clinical significance. Normal heart sounds are called S1 (“lubb”) and S2 (“dupp”). S1 is a loud, resonating sound caused by blood pressure against the atrioventricular valves. This pressure closes the bicuspid and tricuspid valves, pulling the chordae tendinae and the entire supporting framework of cardiac muscle. The second sound forms when the ventricles relax and blood in the pulmonary artery and the aorta flows back toward the ventricles. The arterial valves catch the backflow and snap against one another—“dupp.” If the two ventricles are slightly out

of sequence, so that one closes first, S2 may “stutter” or “split.” An occasional split S2 is normal, but a constant split may indicate **hypertrophy** of one ventricle, a serious cardiac disorder. Listening to these heart sounds, or any internal body sounds for that matter, is termed **auscultation**.
A heart “murmur” can indicate valve malfunction. This whooshing, blowing, or rasping noise occurs when blood passes the valves in a turbulent flow. Murmurs may signal serious valve trouble, but not all murmurs are cause for alarm. Children often develop a

Hypertrophy
Enlargement of an organ owing to enlarged cells rather than an increasing number of cells.



Heart valves **FIGURE 11.5**



Anterior view of heart valve locations and auscultation sites

Heart sound auscultation spots **FIGURE 11.6**

When listening to the valve functioning in the heart, it is best to position the stethoscope bell immediately over those valves.

murmur because the cardiac muscle grows much faster than the valves, which are made of connective tissue. For a while, the valves are simply too small for the heart! Many women develop a murmur during pregnancy, as a result of the dramatic increase in blood volume. Pumping the extra volume exaggerates any small murmurs that are present.

Blood flows twice through the heart Blood enters the heart twice during one complete circuit of the cardiovascular system—once through the right side and then again through the left. Blood enters the right atrium from the **superior** and **inferior vena cavae** and the **cardiac sinus**, and then drops through the tricuspid atrioventricular valve into the right ventricle, which pumps blood to the lungs.

Cardiac sinus
Large vein on the dorsal surface of the right atrium that collects blood from the cardiac veins and returns it to the chambers of the heart.

Blood that returns from the lungs enters the left atrium and drops through the mitral valve into the left ventricle, which pumps the blood throughout the body, with the exception of the respiratory membranes of the lungs. This cyclic movement of blood through the heart and body is referred to as the cardiac cycle.

Diastole
Relaxation of the heart.
Systole
Contraction of the heart.

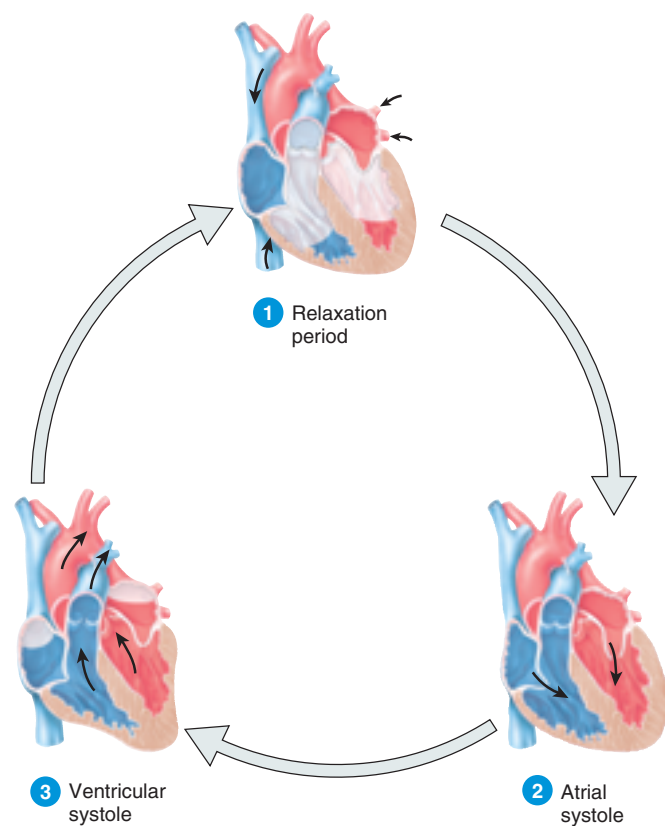
At the beginning of the cardiac cycle, the heart is in **diastole**. The ventricles have relaxed after their recent contraction, and their volume increases. This increase in volume quickly decreases the pressure in the ventricles below that of the atria, drawing in blood through the atrioventricular (AV) valves. The majority of ventricular filling occurs as these AV valves open. Then the two atria undergo **systole** and contract, forcing the remaining atrial blood into the ventricles. This step takes approximately 0.15 second.

After atrial systole is complete, the ventricles contract, taking another 0.30 second. The rapid pressure increase inside the ventricles forces the atrioventricular valves closed, and the semilunar valves open. The blood trapped in the ventricles cannot escape back to the atria through the atrioventricular valves, and so it is forced through the semilunar valves, into the great arteries. Blood leaves the right side of the heart via the pulmonary valve and enters the **pulmonary trunk**, which takes it to the lungs. The blood exiting the left ventricle passes through the aortic valve and reaches the rest of the body.

As the ventricles contract, the atria relax. After a brief ventricular contraction, the entire heart relaxes. Most of the cardiac cycle (an average of 0.40 second) is spent in diastole (**FIGURE 11.7**).

The meaning of blood pressure The heart-beat propels blood through the closed cardiovascular system. As the ventricles undergo systole, they exert pressure on the blood in the entire cardiovascular system. The volume of the blood in the system does not change, but its pressure does. The force the left ventricle creates generates the pulse we can feel and the blood pressure that is measured at the doctor’s office.

The cardiac cycle FIGURE 11.7



The majority of the cardiac cycle is spent in diastole.

- 1 At the beginning of the cycle, the heart is completely relaxed, with blood entering both the left and right atria.
- 2 As the heartbeat begins, the atria contract. This forces blood from the atria into the ventricles.
- 3 Soon after atrial systole, ventricular systole occurs. The atria relax during ventricular systole. The ventricles remain contracted for a measurable period of time, and then the entire heart returns to diastole.

You may be able to recite your blood pressure, which is presented in standard form as systolic pressure over diastolic pressure, such as 110/60 or 193/85.

These numbers have physiological meaning. Systolic pressure measures the force of left ventricle contraction, which pushes blood through the circulatory system. This number is low in children and creeps up with age, as the blood vessels become less elastic. Diastolic pressure is the force your blood exerts on the walls of your closed circulatory system while the heart is

in complete diastole (relaxation). Contrary to popular belief, the diastolic number cannot be zero unless all the blood has been drained from the organism. High blood pressure is loosely defined as a blood pressure reading of 140/90 or above (see the section, “Cardiovascular Disorders Have Life-threatening Consequences,” later in this chapter).

The heartbeat is under intrinsic and extrinsic control Your heart began beating during your third week of development, and it must continue beating to supply your body’s oxygen and nutrient demands until the last minutes of your life. The rate of heartbeat is under two types of control: **Intrinsic controls** establish the usual, day-in, day-out pace of heart beats; **extrinsic controls** modulate the baseline rate to meet the body’s immediate demands.

Unlike other muscle cells, cardiac muscle cells undergo rhythmic contractions without receiving nerve impulses (FIGURE 11.8). The particular rhythm of each cell is based on the rate of calcium ion leakage from the sarcoplasmic reticulum. Recall that the trigger for skeletal muscle contractions is the calcium ion. Cardiac muscle cells are constantly leaking this important ion, and when the intercellular calcium concentration reaches threshold, the cell spontaneously contracts. When two or more cardiac muscle cells touch one another, they begin to beat in unison, following the pace of the faster cell.

A group of cells in the upper wall of the right atrium has the fastest intrinsic beat, and it serves as the heart’s pacemaker. Because these pacemaker cells are near the entrance of the **coronary sinus**, they are called the SA (sinoatrial) node. When the SA node initiates the heartbeat, the signal to contract passes in wave-like fashion from cell to cell through the right and then the left atrium causing them both to contract.

At the base of the left atrium, near the ventricle, lies a group of cells called the **AV node**. AV stands for atrioventricular, due to the placement of this node. These cells serve as a relay station that delays the contraction impulse before sending it on. (Like the SA node, these cells cannot be distinguished visually.) The delay allows the atria to complete their contraction before the ventricles are stimulated to begin contracting. After the delay, the impulse passes through a series of



An individual cardiac cell in a culture

FIGURE 11.8

conductive tissues before reaching the cells of the ventricles.

From their relative size, it’s obvious that ventricles have far more cells than atria. Although the impulse to contract could spread from cell to cell in the ventricles just as in the atria, the contraction would be ineffective because closer cells would have finished contracting before the contraction impulse reached more

distant cells. Instead of producing the forceful contraction needed to build up pressure and open the semilunar valves, blood would just slosh around in the ventricle. If you try to pop a water balloon by grabbing the top, the middle, and bottom in order, the water will simply move away from your hands without breaking the balloon.

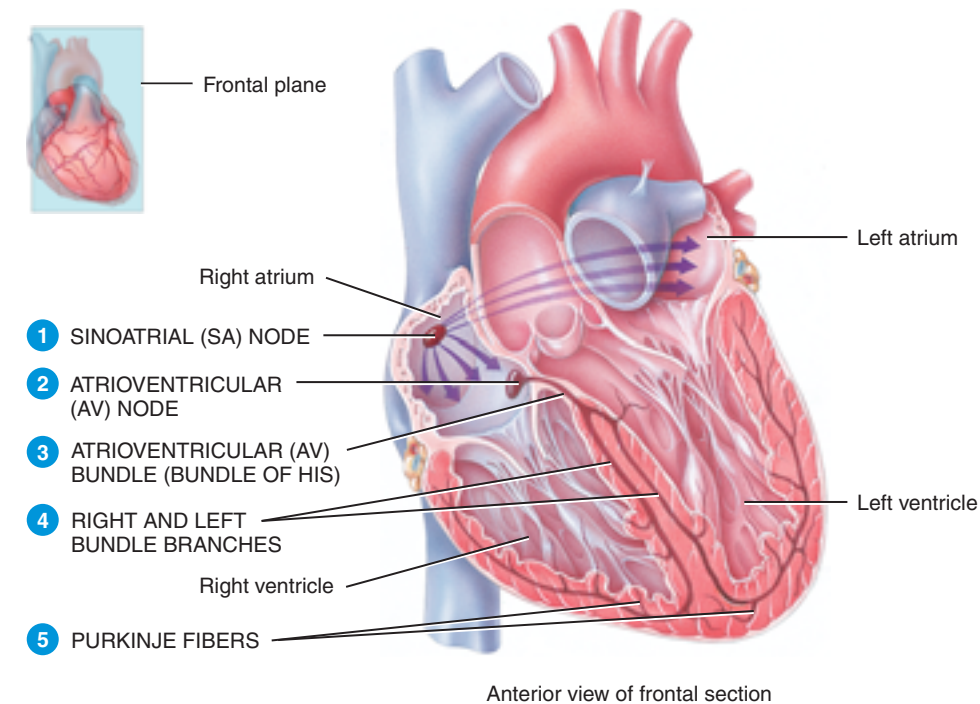
But if you can grab the balloon everywhere at once, the dramatic rise in internal pressure will pop it.

To obtain simultaneous contraction, the ventricles require a conduction system for the contraction impulse. This system starts at the AV node and goes to the **AV bundle** at the center of the heart, near the inter-ventricular septum. Here the system splits into the **left** and **right bundle branches**, which carry the impulse to the apex of the heart, and then up the outer walls. From the bundle branches, the impulse travels on smaller **Purkinje fibers** (FIGURE 11.9), which end at

Purkinje fibers

Conduction myofibers that reach individual cells of the ventricles, named for Johannes Purkinje, Czech physiologist (1787–1869).

Conduction system of the heart FIGURE 11.9



The contraction impulse begins in the SA node 1. The contraction passes in a wave-like fashion through the atria and is collected at the AV node 2 before being passed on to the ventricles. From the AV node, the impulse is sent down the AV bundle 3 to the left and right bundle branches 4, through the Purkinje fibers 5, and then on to the cells of the ventricles.



clusters of ventricular cells. Using this system, all ventricular cardiac muscle cells contacted by Purkinje fibers get the impulse simultaneously, resulting in synchronous contraction of the entire ventricle.

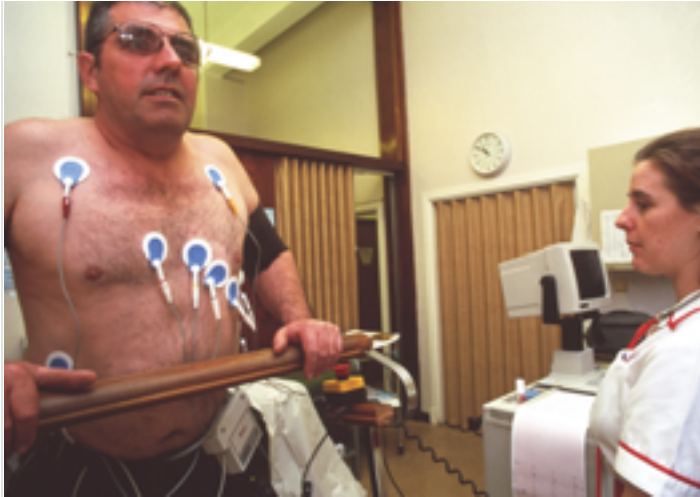
The SA node and the conduction system govern the baseline, or resting, heart rate. But if the body needs more blood than this heart rate can deliver, several **extrinsic heart rate controls** may enter the picture. One extrinsic control resides in the cardiac control center in the medulla oblongata. This center can override the intrinsic heartbeat, increasing or decreasing the rate as necessary. When the sympathetic division of the autonomic nervous system is active, heart rate increases significantly. Similarly, heart rate immediately rises in cardiac cells that are exposed to norepinephrine, the sympathetic division neurotransmitter.

The heart itself can also affect contraction rate and strength. **Starling's law** states that when the ventricles are stretched by increased blood volume, they recoil with matching force. Thus increased blood flow to the heart, which occurs when we start hard physical work or exercise, causes the heart to respond with more forceful pumping—just what we need to move oxygenated blood to the active muscles.

The electrocardiogram records electrical activity Regardless of what is controlling the heart rate, the cardiac muscle cells generate a pattern of recognizable electrical signals as they go through the cardiac cycle. The cells of the myocardium depolarize immediately before they contract and re-polarize as they relax. Because so many cells are involved in this cycle, the electrical signals are strong enough to be detected on the skin, where they can be recorded on an **electrocardiogram**, or ECG (**FIGURE 11.10**).

The ECG tracing has a defined series of peaks and valleys (**FIGURE 11.11**). As the SA node fires, the atrial cells depolarize, causing a hill-shaped upward deflection called the **P wave**. Within 100 milliseconds, atrial systole follows the P wave. The ECG tracing briefly flattens, then starts a large upward deflection. This **QRS complex** is created by the simultaneous depolarization of the many

Electrocardiogram A graphic representation of the electrical changes during a heartbeat.



Person hooked up to an ECG machine **FIGURE 11.10**

The electrical changes associated with cardiac systole and diastole are strong enough to be picked up on the surface of the body with strategically placed electrodes. These leads are attached to the skin, with a conductive gel between the surface of the lead and the skin to enhance the sensitivity of the lead. The tracing that results from this is used to diagnose cardiac function.

ventricular cells. As the ventricles briefly remain in systole, the ECG is momentarily flat. As the ventricles relax, the cells re-polarize, creating the hill-shaped deflection called the **T wave**, which marks the return of cardiac diastole.

These deflections can help clinicians evaluate cardiac function. During the **P-R interval** (from atrial depolarization to ventricular depolarization), the contraction impulse is transmitted from the SA node, through the atria, to the AV node, and finally through the conduction system. An interval longer than 0.2 second may indicate damage to the conduction system or the AV node. A long **Q-T interval** (the total time of ventricular contraction and relaxation) may indicate **congenital** heart defects, conduction problems, coronary **ischemia**, or even cardiac tissue damage from a previous heart attack. If the problems seen in the ECG are severe, the heart muscle may

Congenital
Present at birth.

Ischemia
Lack of oxygen to a tissue because of constriction or blockage of the blood vessels.



Typical ECG tracing **FIGURE 11.11**

stop functioning properly or it may be too weak to be effective. We have the technology and medical skill to replace the heart, but the operation raises complex issues. The Ethics and Issues box, “The ethics of heart replacement” further explores the controversy surrounding this complex procedure.

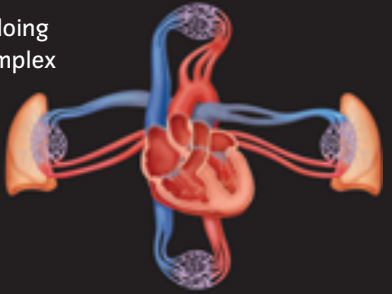
CONCEPT CHECK

Trace blood flow through the heart, including all chambers, valves, major arteries, and veins.

Define diastole and distinguish it from systole.

Trace the electrical pathway through the heart, beginning at the SA node and ending with the conduction systems that cause ventricular systole.

What is the heart doing during the QRS complex of an ECG?



Blood Vessels and Capillary Transport Involve Miles of Sophisticated Plumbing

LEARNING OBJECTIVES

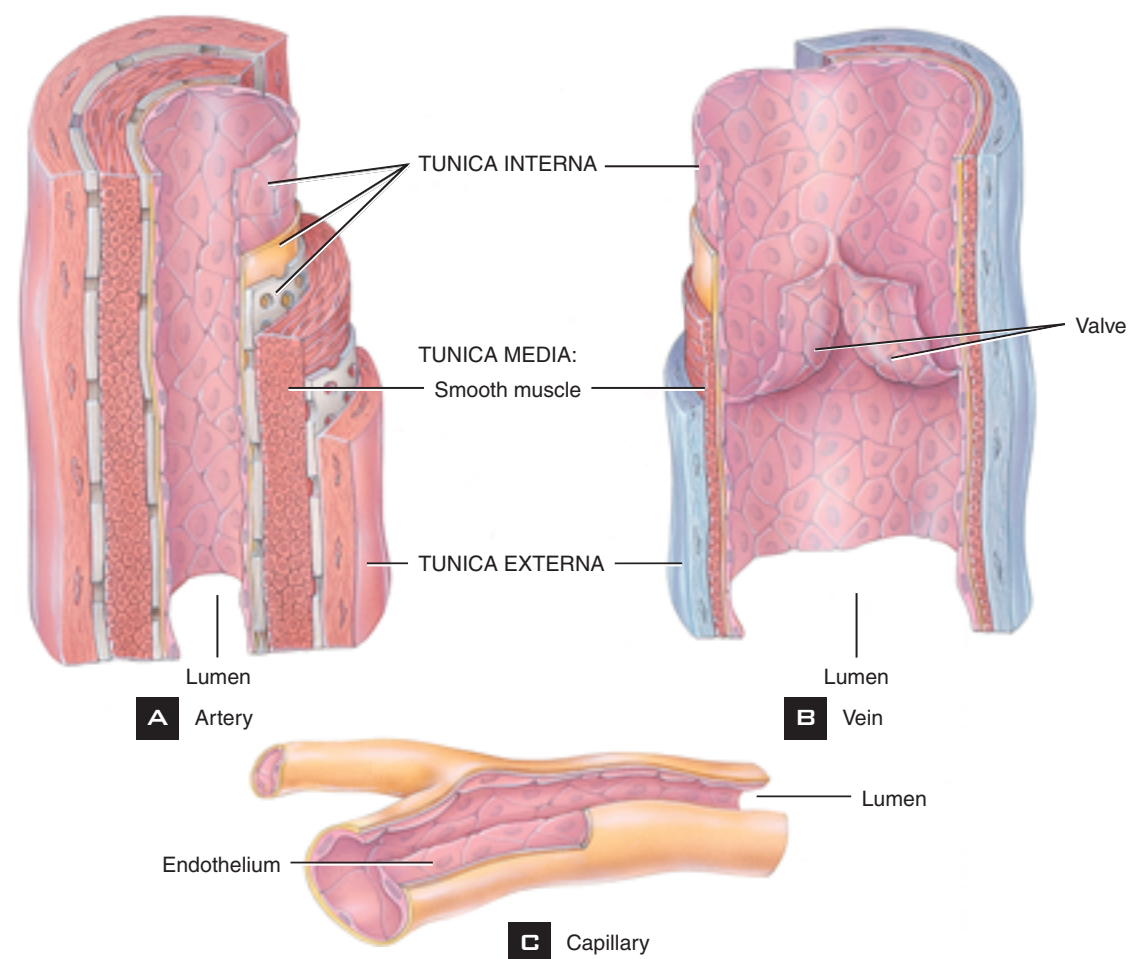
Compare the structure and function of the three types of blood vessels.

Discuss the function of capillary beds and venous valves.

The cardiovascular system has three categories of vessels that are strung together in a large web that begins at the heart, reaches the tissues, and returns to the heart. The vessels in this continuous circuit are the **arteries**, **capillaries**, and **veins** (**FIGURE 11.12**). Each type of vessel has a different function.

Arteries are blood vessels on the output (ventricular) side of the heart. Arteries closest to the heart

have large diameters and thick walls because the heart's pumping causes them to stretch and recoil with each beat. Further from the heart, diameter and wall thickness both decrease because this distance reduces the pressure the heart is able to exert on these vessels. **Arterioles** are small vessels that branch from larger arteries and are structurally similar. In the arterioles, the total cross-sectional area of the blood vessels increases, even though each vessel is smaller in diameter. This



Artery, capillary, and vein structure FIGURE 11.12

Note that the artery is the thickest of the vessels. Arteries take blood from the heart to the tissues of the body and are subjected to the largest pressures. They have a layer of resilient muscle in their walls that allows for the bouncing pulse we can feel through the skin. Capillaries are extremely thin-walled,

usually only one cell thick. They are the diffusion vessels of this system. Veins are thinner than arteries but have more substance than the capillaries. Valves prevent backflow of blood in these weak-walled vessels.

Lumen

The inner, hollow portion of a tubular structure; the center of the blood vessel.

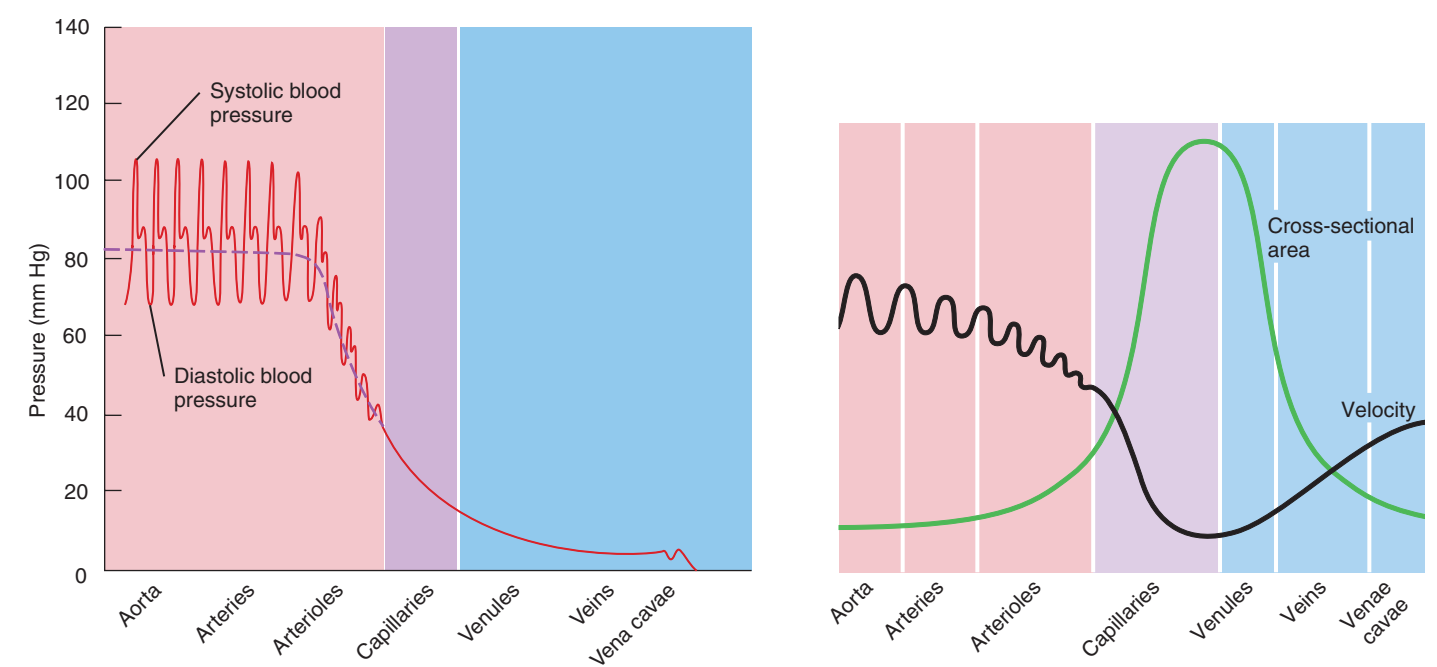
larger cross section allows the blood to slow. The smaller **lumen** exposes the blood to more surface area, which creates friction, further causing the flow to slow (FIGURE 11.13).

Arterioles lead to **capillaries**, the smallest blood vessels. The wall of a capillary is one cell layer thick, and the lumen is barely big enough for one blood cell. Capillaries are exchange vessels that reach to almost every cell, and they are the only vessels that permit the vital

exchange of gases, nutrients, and waste across the blood vessel wall. The slow blood flow and high cross-sectional area provide enough time and surface area for exchange to occur. Capillaries form large **capillary beds** within the tissues, where blood flow is regulated by **pre-capillary sphincters**. These small, ring-like muscles can close or open parts of a capillary bed, depending on the oxygen and nutrient demands of the tissue (FIGURE 11.14).

Capillary bed

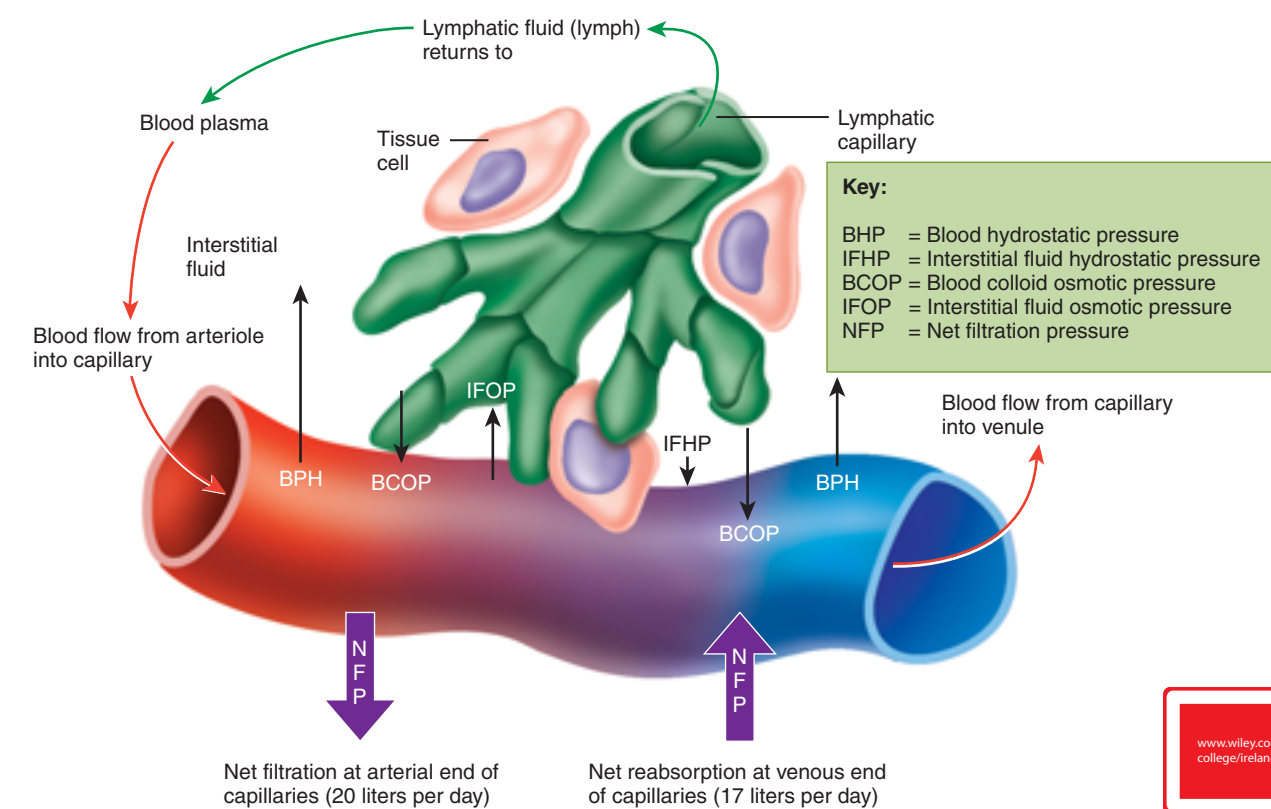
Interwoven mat of capillaries threading through a tissue.



Relative blood pressure and blood flow in arteries, capillaries and veins. FIGURE 11.13

Capillary bed and exchange flow

FIGURE 11.14



Process Diagram



Ethics and Issues: Ethics of Heart Replacement

Replacing diseased hearts has long been a dream of medical science. The heart may look like a simple pump, but the difficulty of replacing it with metal and plastic emerged in 1983, when dentist Barney Clark received an artificial heart, then died a slow and painful death in the glare of massive publicity due to small blood clots created by the action of the replacement heart.

Clark's death raised a slew of thorny ethical questions. Were the doctors justified in performing a transplant with the prototype heart? Were they and Clark medical pioneers who were selflessly working to perfect and test a technology that would later benefit thousands of others with failing hearts? Or were the heart developers self-promoters who took advantage of a dying man in their search for fame and fortune?

The Clark experience put a damper on the quest for an artificial heart—although some machines are now used as “bridge” to sustain patients until a heart transplant becomes available. A second design of an artificial heart was tested—with much less publicity—in 2001; that machine also had problems.

It's now clear that the best replacement for a human heart is another human heart. Many early heart transplants were rejected by the body's immune system, but the use of immune-suppressing drugs after 1980 vastly improved success rates. According to the American Heart Association, about 2,000 heart transplants are performed each year in the United States.

Transplant organs must still be tissue matched to the recipient to reduce the chance of rejection, and many heart transplant recipients enjoy 10 or more fairly normal years after the surgery. Sadly, despite our ability to successfully transplant organs patients are still dying of diseases a transplant would “cure.” With too few donated organs available, patients with kidney, liver, or heart disease are dying on waiting lists, and transplants raise their own set of ethical problems:

- Should people wait their turn on the list and receive transplants when they are near death? Or should they get transplants sooner, when they are more likely to benefit from the procedure?



- Should states that actively solicit organ donors (through check-offs on drivers' licenses, for example) be permitted to keep organs, or should they be shared regionally or nationally?
- Is the immense cost of a transplant always worth it, or only when a certain life span is expected?

Here's a final question. Some argue that modern medicine, with its focus on the individual patient, has become too conservative. In 1900, Dr. Walter Reed and colleagues proved that mosquitoes carry the dreaded yellow fever virus by exposing volunteers to mosquito bites. One of those volunteers died from the experiment, but the results were used to support mosquito eradication campaigns that brought yellow fever to its knees. Would a similar experiment be permitted today, given the potential for massive social benefits?

The possible use of replacement tissues derived from stem cells raises a related set of ethical questions that you've seen in Chapter 3's Ethics and Issues box on stem cell research. But all cases of organ replacement or transplant raise certain key ethical issues: Who will pay for these elaborate procedures? Can desperately sick patients make informed decisions about their own treatment? And can doctors and scientists who are enthusiastically working on potential “medical miracles” make wise decisions about the risks and benefits of untried procedures?

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Skeletal muscle squeezing vein, assisting in return FIGURE 11.15



At rest, skeletal muscles do not impede the flow of blood back to the heart through veins. When the muscles of the leg are contracted however, they push against and flatten portions of the veins, helping to move the blood to the heart. This rhythmic flattening and releasing “milks” the veins, moving the blood more efficiently.

Blood leaving capillaries collects in larger vessels called **venules** and veins heading back toward the heart. At this point, circulation resembles the flow of water from rivulets into creeks into rivers and eventually to the sea. As the veins get bigger, the walls thicken slightly.

Veins never reach the thickness of arterial walls because there is no need for so much resilient muscle in these vessels. The blood in the veins is moving with barely any pressure, so the veins themselves do not need to be terribly strong.

Because the veins are beyond the capillaries, the heart's pumping cannot put much pressure on ve-

nous blood. However, blood continues to flow toward the heart. Part of the reason is fluid dynamics: Fluids flow easily from a smaller vessel to a larger one, where there is less friction from the vessel walls. Returning the blood from the legs to the heart poses a special challenge because the flow must counteract gravity, with little or no help from the heart. Blood does not pool or flow backwards in the legs because a series of valves in the large veins prevent reverse flow. Also, the contraction of skeletal muscle squeezes the veins and creates a pumping action, pushing blood up toward the heart (**FIGURE 11.15**). Exercise is often prescribed to move blood and prevent **edema** of the lower extremities.

Venules Small veins that drain blood from capillaries to larger veins.

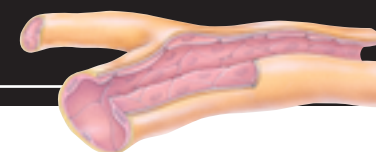
Edema Abnormal swelling in tissues.

CONCEPT CHECK

What are the three types of cardiovascular vessels?

Why do veins have valves? Are valves necessary in arteries?

How does the large surface area of the arteriole affect blood pressure in the arterioles?



Different Circulatory Pathways Have Specific Purposes

LEARNING OBJECTIVES

Define a closed circuit. Compare the function and flow of the systemic, pulmonary, and hepatic portal pathways in humans.

Blood can take one of two pathways from the heart: the **pulmonary circuit** toward the lungs or the **systemic circuit** toward the tissues. The purpose of the pulmonary circuit is to exchange carbon dioxide in the blood for oxygen from the environment. The systemic circuit brings this oxygen (and nutrients) to the tissues and removes carbon dioxide.

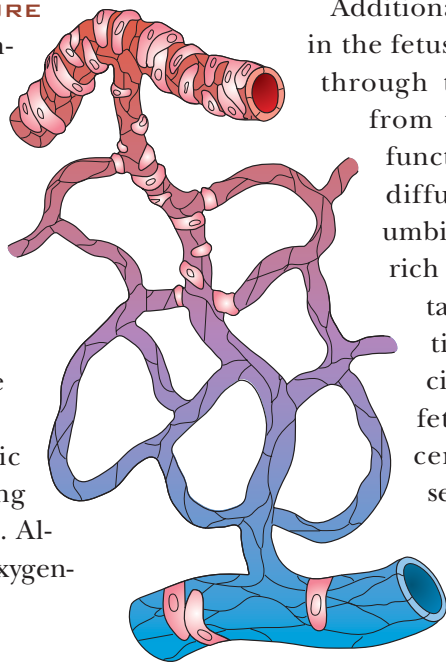
The **pulmonary circuit** extends from the right side of the heart to the capillary beds of the lungs and on to the left atrium. Blood entering the right atrium is low in oxygen, having just returned from the body. This deoxygenated, carbon-dioxide-rich blood drops to the right ventricle, which propels it to the lungs, where it picks up oxygen and releases carbon dioxide, and returns to the left atrium.

The **systemic circuit** begins when oxygen-rich blood enters the left atrium (**FIGURE 11.16**). This oxygen-rich blood then enters the left ventricle, and, during ventricular systole, is pumped through the aortic arch to the body. After passing through the capillaries, venous blood makes its way back to the superior and inferior vena cavae. These large veins drain into the right atrium, where blood reenters the pulmonary circuit. The systemic circuit includes most of the blood vessels in the body.

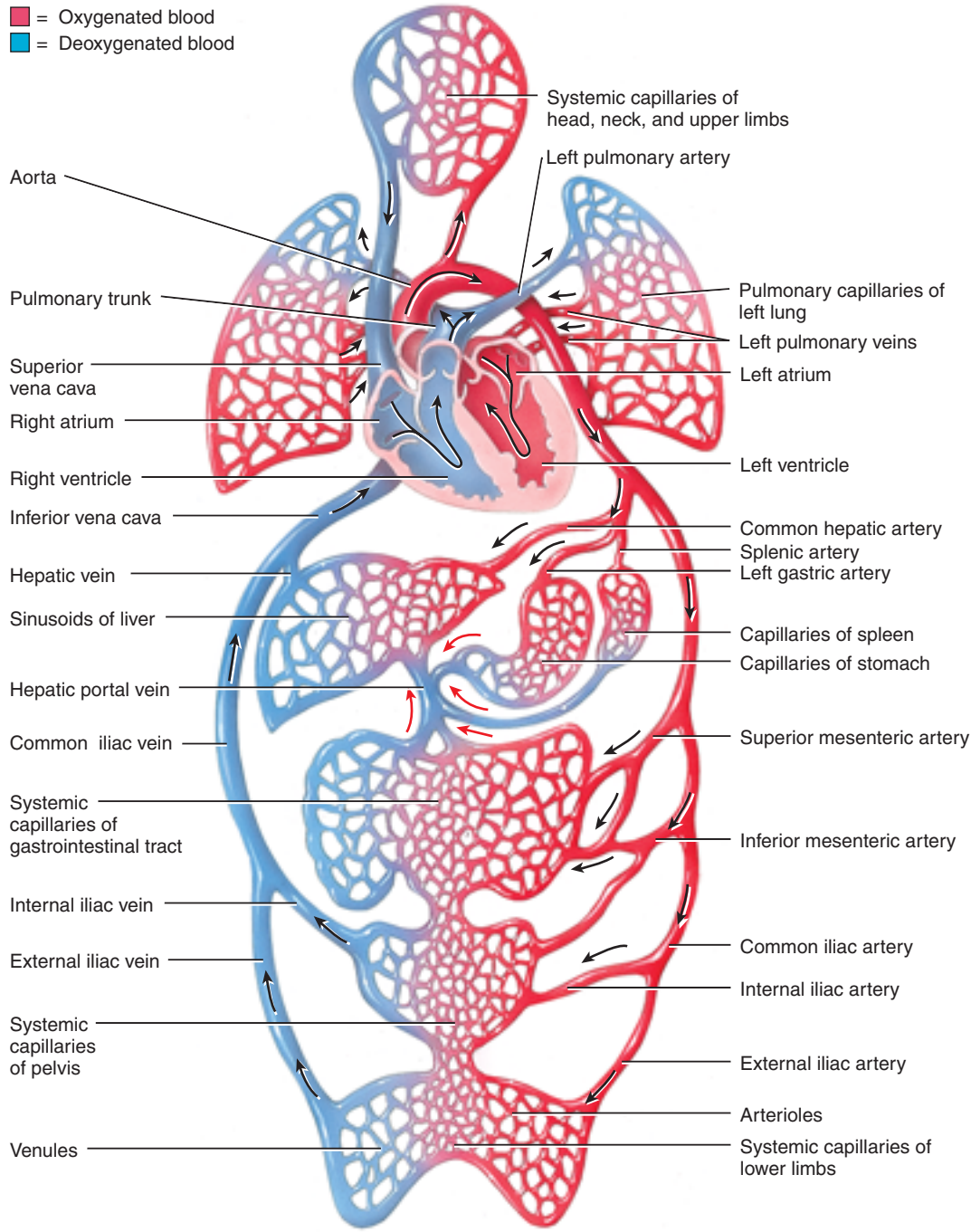
The first branches from the aortic arch are the **coronary arteries**, delivering oxygen-rich blood to the cardiac muscle. Although the left side of the heart is full of oxygen-

rich blood, that blood and its oxygen are not available to the heart tissue because the inner lining of the heart, the **endocardium**, is not a diffusion membrane. Therefore, cardiac tissue must obtain oxygen through a capillary bed, just like every other tissue. These coronary arteries are narrow and prone to clogging. If they are blocked, less oxygen-rich blood is delivered to the heart, causing a heart attack. Heart attacks are discussed later in this chapter.

Although blood usually flows from arteries to capillaries to veins, this pattern is modified in a few places. In **portal systems**, blood flows from arteries to capillaries to veins, as usual (**FIGURE 11.17**). The veins, however, break up into another set of capillary beds before the blood returns to the heart. This allows the blood to slow down in the organ before it is pushed back to the heart.



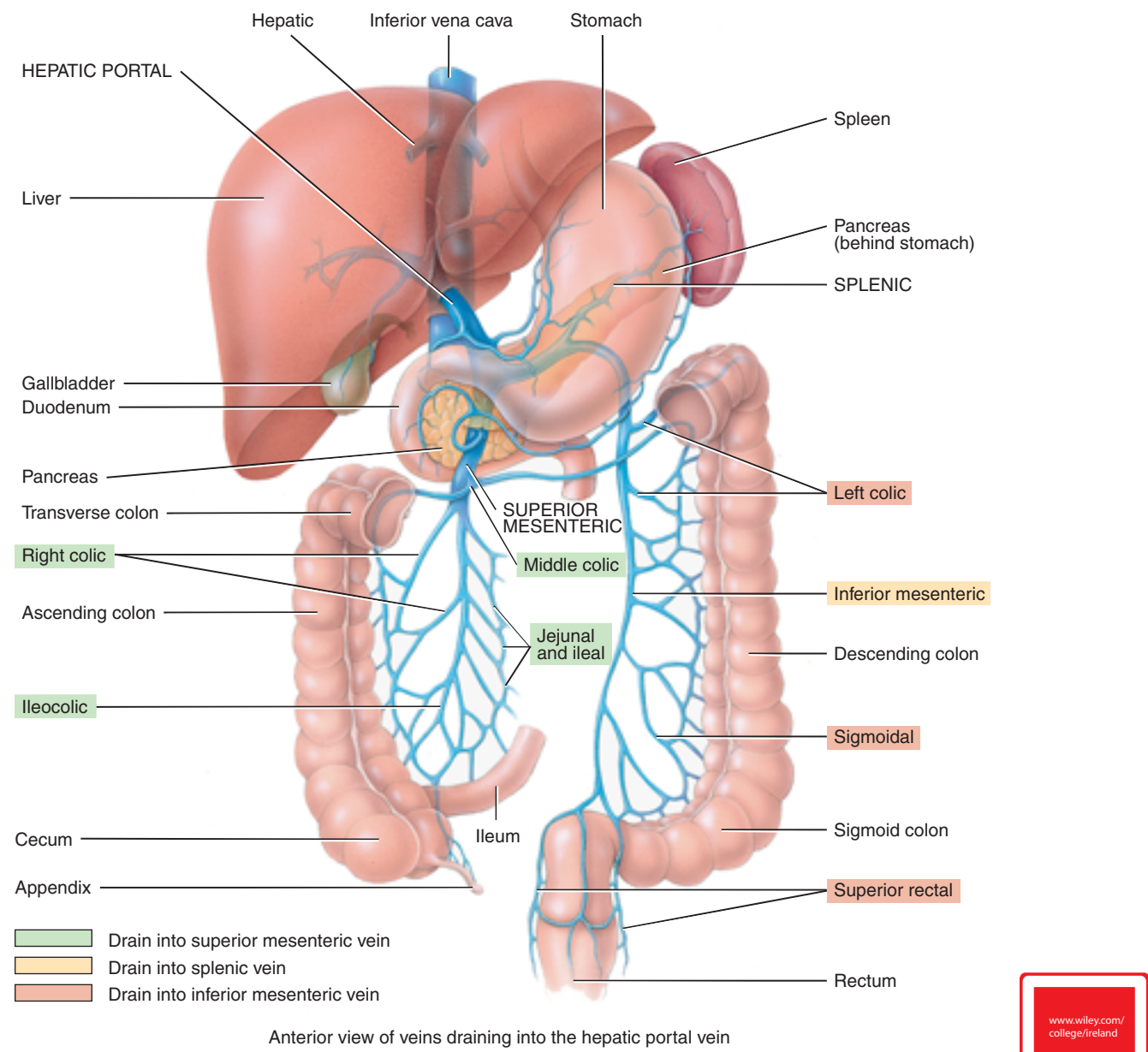
Additionally, there is an altered blood flow in the fetus, which gets its oxygenated blood through the placenta, or umbilicus, not from the lungs. The lungs are not yet functioning, and will not be needed to diffuse oxygen until after birth. The umbilicus carries oxygenated, nutrient-rich blood from the placenta to the fetal liver, where the blood then continues through the fetal systemic circuit. The umbilical arteries carry fetal blood from the fetus to the placenta to be cleansed. The complete set of fetal circulatory modifications is discussed in more detail in Chapter 17.



Pulmonary and systemic circulatory routes **FIGURE 11.16**

The two main circulatory routes in the body can be seen here. The pulmonary circulatory route takes blood from the heart to the respiratory surface of the lungs and back to the heart (short black arrows). The much more complicated systemic circuit

delivers blood to the organs of the body, and then back to the heart (longer black arrows). The red arrows represent the hepatic portal circulation (see Figure 11.17).



Anterior view of veins draining into the hepatic portal vein

Hepatic portal system **FIGURE 11.17**

In the *hepatic portal system*, blood flow begins and ends with capillaries. Before blood enters the liver, it absorbs nutrients in capillary beds in the small intestine. This blood collects in the hepatic portal vein, which drains from the small intestine to the liver, and then passes through another capillary bed.

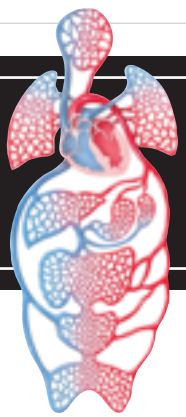
The blood slows in the capillary bed of the liver so that *hepatocytes* (liver cells) can remove detrimental ions and compounds that were picked up by the digestive tract. The cleansed blood collects in the hepatic vein and drains to the inferior vena cava.

CONCEPT CHECK

How does the pulmonary circuit differ from the systemic circuit?

What is meant by a “portal system?”

Why does the fetus need an altered blood flow?



Cardiovascular Disorders Have Life-Threatening Consequences

LEARNING OBJECTIVES

List the most common disorders of the heart and vessels.

Probe the risk factors for heart attack.

Explore the role of lifestyle in cardiovascular disease.

Many cultures equate great emotional pain with a “broken heart,” but in reality love gone awry does not interfere with cardiac function. However, **cardiovascular disease** (CVD) does and in fact is the leading cause of death in adults in Western countries. CVD takes many forms, each with its own symptoms and treatments. In every case, however, the common thread is a decrease in effective cardiac function.

The most common cardiovascular diseases include hypertension, atherosclerosis, heart attack, heart failure, embolism, stroke, and varicose veins. You probably know someone who has suffered from one of these common conditions. The risk factors for cardiovascular disease can be genetic or environmental. Genetic risk factors include family history, gender, and ethnic background. A family history of heart attack prior to age 55 indicates a genetic predisposition to heart disease. Males suffer from CVD more frequently than females, although this gap is closing. The reasons for this change are uncertain but could include the advance of women into the higher stress jobs once dominated by men, women’s longer life spans, and the post-menopausal reduction in estrogen levels. The incidence of CVD is higher in African Americans than in Americans of European descent, indicating a genetic predisposition.

Even if you have genetic risk factors, all is not lost. Many risk factors, such as smoking, overeating, and spending too much time on the sofa rather than exercising, are fairly easy to control. Monitoring your diet

and getting a modicum of exercise will decrease your chance of CVD. Some studies indicate that 40 minutes a day of low-impact exercise reduces the chance of heart disease by one-third. And the exercise need not be terribly strenuous—gardening, yoga, or ballroom dancing can all improve cardiovascular performance. In many cases, preventing or controlling CVD is not terribly difficult; it just requires some dedication and understanding.

HIGH BLOOD PRESSURE STRESSES THE ENTIRE BODY

One of the most prevalent CVDs is **hypertension**, or high blood pressure. Hypertension is often called the “silent killer” because it may produce no symptoms before disaster strikes. As mentioned, hypertension is diagnosed when systolic blood pressure is above 140, or diastolic is above 90. A high diastolic number indicates a decline in blood-vessel elasticity that increases the chance that the force of systolic contraction will exceed the capacity of the circulatory system. In chronic hypertension, capillary beds leak blood into the surrounding tissues, or break entirely, causing internal bruising. Although hypertension is harmful to many organs, the key risk is stroke. Dietary restrictions, moderate exercise, reducing smoking and drinking, and medications can all control hypertension.

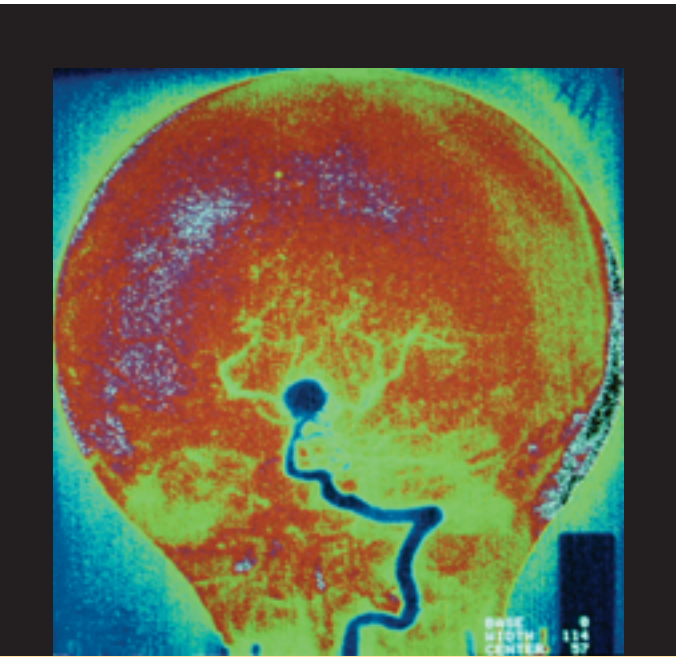
Recent research is discovering a genetic link to some forms of high blood pressure. In particular, two genes are involved in the conversion and activation of the protein angiotensinogen. This enzyme converts angiotensinogen in the plasma into angiotensin, which constricts the blood vessels. If this pathway is hyperactive, constricted vessels will reduce the blood-flow capacity, and the noncompressible blood will push harder against the vessel walls, increasing blood pressure.

ARTERY DAMAGE IS A MAJOR CAUSE OF MORTALITY AND DISABILITY

Atherosclerosis (literally “hardened vessels”) is another disease of the blood vessels. When **plaques**, fatty deposits of cholesterol, accumulate inside the vessel walls, they **occlude** (slow or block) the lumen, reducing blood flow (**FIGURE 11.18**). More serious complications can arise if the plaque causes a clot to form within the vessel. A clot that is attached to the vessel wall is called a thrombus, but if it loosens and floats in the bloodstream, it is called an **embolism**. This floating clot can lodge in a smaller vessel, completely blocking blood flow and causing tissue death.

An **aneurysm** occurs when a vessel wall balloons under pressure, forming a weak spot that can be burst by the increased blood pressure generated with each heartbeat (**FIGURE 11.19**). Burst aneurysms are usually fatal, because they tend to develop in large, high-volume arteries in the abdomen or brain. Because arteries are not exchange vessels, aneurysms can sometimes be repaired before they burst by replacing the ballooned area with plastic tubing, but only if they are detected as they develop.

An embolism or aneurysm in the brain causes stroke. Whether the problem is a blockage or excess bleeding, stroke starves the tissues fed by the blocked or broken artery of oxygen and nutrients. Although quick removal of an embolism can control the amount of damage, brain tissue usually dies. Initial symptoms of stroke include sudden difficulty speaking, blindness in



Aneurysm **FIGURE 11.19**

one eye, or numbness and/or weakness, usually on one side of the body. Stroke can also cause **aphasia** (loss of speech), loss of fine motor control, paralysis, or even death. New emphasis on quick treatment of strokes has reduced the resulting disability, but many of the 700,000 Americans who suffer a stroke each year suffer widespread brain damage.

THE CAUSES AND CONSEQUENCES OF HEART ATTACK

The most fearsome cardiovascular disorder is **heart attack**, the death of a portion of the heart muscle due to a lack of oxygen. Heart attack, or **myocardial infarction**

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(MI), causes one in five deaths in the United States. Each year, the population of the United States suffers more than 1.2 million nonfatal heart attacks, and of those patients, an incredible 40 percent will die within one year. (See the “I Wonder . . .” box on page 347.)

Dead cardiac tissue ceases to conduct electricity, so the contraction impulse cannot pass. A ventricle that cannot contract completely is unable to move blood efficiently, and the result is reduced cardiac output.

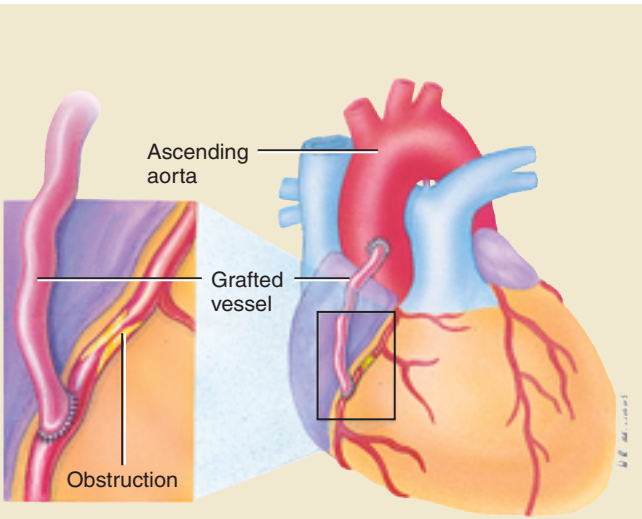
MI is usually due to plaque in a coronary artery that occludes the blood flow. While the plaque is forming and blood flow is diminishing, the heart tissue may act like a cramped muscle. The pain from this temporary loss of oxygen is usually described as a crushing feeling in the chest, pain that radiates through the chest and left arm, or a numbness in the left arm. This condition is called **angina pectoris**, often abbreviated as simply angina.

Angina can arise when the heart is working hard, such as during strenuous exercise, or when it is stressed by, for example, smoking cigarettes. This “**stable angina**” can be treated by reducing activity and/or quitting smoking. **Unstable angina**, in contrast, appears with no apparent stimulus and is often an early warning of impending heart attack. Fortunately, people with unstable angina often think they are having a heart attack and seek immediate medical attention.

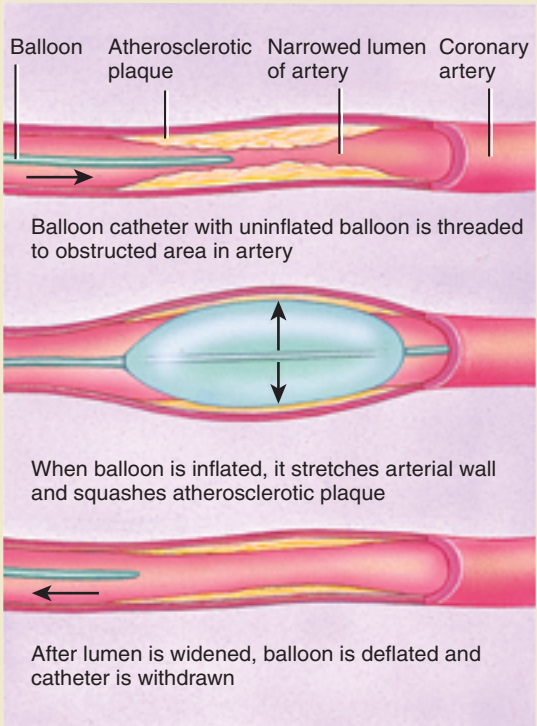
Angina can often be controlled with nitrate drugs, such as nitroglycerine and isosorbide, which relax cardiovascular smooth muscle. As the smooth muscle in the walls of the coronary arteries relax, blood pressure decreases and blood can flow more smoothly past the obstructive plaque.

If medication does not restore a normal lifestyle, surgical procedures may be recommended, including **balloon angioplasty**, placement of a **stent**, or **bypass surgery** (**FIGURE 11.20**). Balloon angioplasty pushes soft, fatty plaque against the vessel wall, reopening the lumen. The physician inserts a catheter with a deflated balloon at the end into the femoral artery and

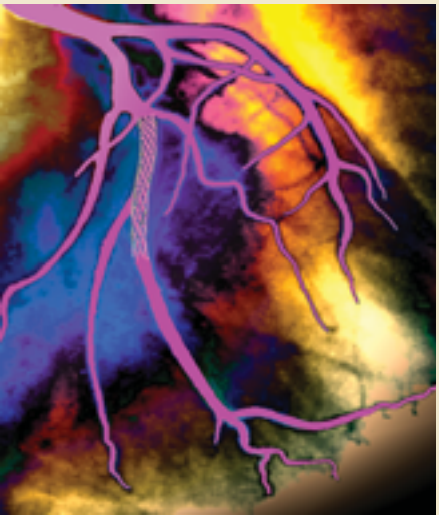
Surgical procedures for reestablishing blood flow in occluded coronary arteries **FIGURE 11.20**



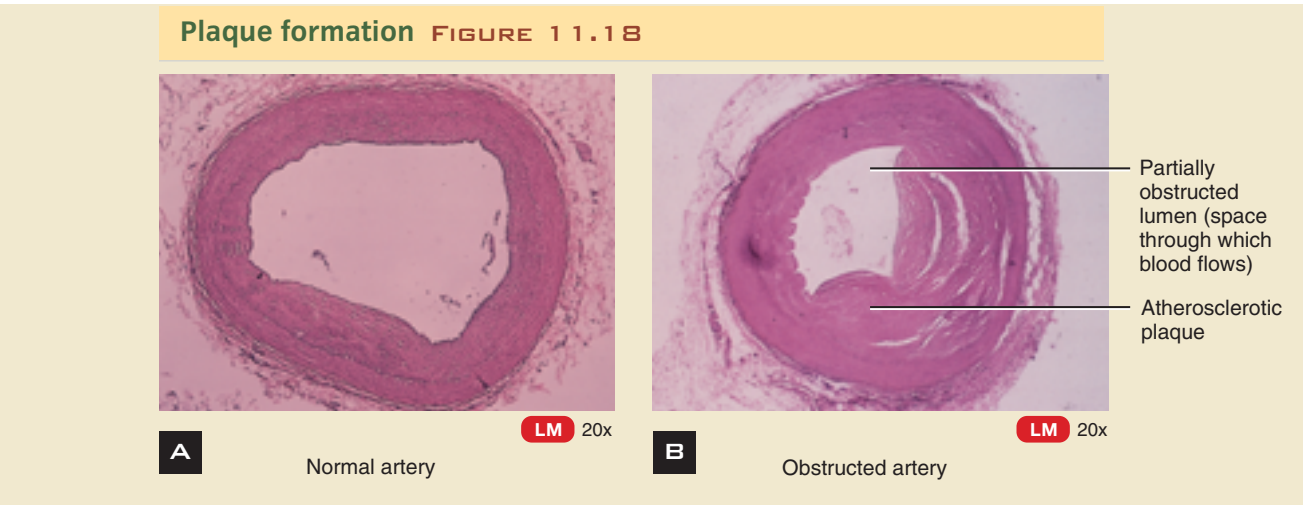
A Bypass surgery



B Balloon angioplasty



C Angiogram showing a stent in the circumflex artery



threads the catheter to the occluded coronary artery. When the balloon is inflated, the lumen expands.

Balloon angioplasty can fail if the plaque does not stick to the vessel wall. Stents, which look like a tiny roll of chicken wire, are designed to overcome this difficulty. A stent supports the arterial walls, permanently opening the vessel to improve blood flow. A stent may be coated with medicine to block plaque buildup; as the medicine leaches from the stent, it supplies a constant dose exactly where it is needed.

If the coronary artery is physically damaged, or the plaque buildup is severe, bypass surgery may be required. This is open-heart surgery and is obviously much more invasive than angioplasty. Surgeons

break the breastbone to reach the heart, and periodically stop the heart to perform delicate suturing. A section of blood vessel, usually from the femoral vein, is removed to serve as the bypass vessel. (Blood return from

the legs is not hindered because the venous system includes many **anastomoses** and alternate pathways for blood return.) The surgeons suture a small length of femoral vein around the blockage in the coronary artery, creating something that works like a highway detour: Blood bypasses the congestion and returns to normal circulation beyond the blockage. Each detour counts as one bypass, so a triple bypass surgery involves three detours. Bypass surgery is usually highly effective, but recovery takes much longer than it does for balloon angioplasty or stent placement, owing to the major thoracic surgery and the healing required in the leg.

Congestive heart failure is due to a weak heart With age, many hearts simply weaken and fail to push enough blood through the circulatory system. Such a condition, called **congestive heart failure**, is growing more common now that more people are living deep into old age. A weakened left ventricle fails to move the blood, allowing fluid to back up and leak into the lungs, causing **pulmonary edema**. As the blood flow slows, fluid also builds up elsewhere. Congestive heart failure is named for the resulting congestion in the tho-

racic cavity. When this fluid presses against the heart, beating becomes even more difficult.

Heart failure, unlike angina or MI, is a gradual disease. As the heart weakens, the body attempts to compensate. The heart itself expands, resulting in a larger volume in each ventricle. Enlarged volume requires more muscle mass to push the blood, causing a further expansion in the heart. As these two remedies fail, **tachycardia** pushes more blood through the body, until the heart cannot maintain the rapid pulse. Symptoms of congestive heart failure include fatigue, difficulty breathing, tachycardia, and possibly even death as fluid builds up in the lungs, effectively drowning the respiratory membranes.

Tachycardia
Resting heart rate
above 100 beats
per minute.

When veins become visible they function less effectively Not all cardiovascular diseases are fatal. **Varicose veins** and **spider veins** are unsightly and can be painful (**FIGURE 11.21**). Varicose veins are distensions of the venous walls near valves. As the blood



Varicose veins and spider veins **FIGURE 11.21**

Why do trained athletes have heart attacks ?

It's a paradox. People exercise to improve their overall health and their CV systems. Yet once in a while, trained athletes keel over from heart attacks. Why? Is exercise dangerous?

A series of studies over the past 20 years found that extreme athletic effort does seem to raise the risk of heart attack, at least for about 24 hours after the event. But they also show that most of the stricken athletes already had heart disease to begin with.

Among youths, sudden cardiac death during exercise is quite rare. The key danger seems to be hypertrophic cardiomyopathy—a syndrome of heart “overgrowth” that usually can be detected with ultrasound examination.

A wide variety of preexisting conditions can cause athletic heart failure among people over 40. Most common is atherosclerosis—hardened arteries that suffer an embolism or aneurysm during the event. But spasms of the coronary arteries, deformed valves, and other common causes of MI are also to blame. Arrhythmias—erratic heart rhythms—can also cause athletic heart failure because extreme exertion can sometimes trigger a fatal arrhythmia.

The number of athletic deaths is low. In 1998, a South African scientist reported that “6 in 100,000 middle-aged men die during exertion per year. . . the evidence suggests that exercise seems to trigger or cause sudden cardiac death in athletes with underlying heart disease.” (*Encyclopedia of Sports Medicine and Science*, 1998, Dr Timothy D. Noakes.)

BJ Maron, LC Poliac and WO Roberts of the Minneapolis Heart Institute found that of 215,413 competitive marathon runners studied over a 30-year period only four suffered sudden cardiac deaths. Three resulted from coronary artery disease, and the other from unusual anatomy of a coronary artery. This study indicated that adults who were fit enough

to run 26-mile marathon races faced a 1 in 50,000 risk of sudden cardiac death during the event, or immediately afterward.

The stricken athletes may be unwittingly close to a heart attack. After a severe snowfall in the United States, one study found a temporary spike in heart-attack deaths during the storm but no change in the monthly toll. The researchers concluded that the people who had heart attacks while shoveling snow would probably have died within a few days anyway.

Most experts say exercise helps the heart and overall CV system by strengthening cardiac muscle and the vasculature, and by improving the efficiency of circulation and respiration. Although athletes can get MI from many underlying causes, “none of these conditions is caused by exercise, however vigorous,” wrote Timothy Noakes, of the University of Cape Town's physiology department. “Rather, the evidence is clear that regular exercise acts against the development especially of coronary atherosclerosis.”

But there is one line of evidence showing that exercise can damage the heart: A few recent studies have found the enzyme troponin I in athletes' blood after endurance events. Troponin I in the blood stream is considered an indication of death of heart muscle cells. Nevertheless, a 2005 study of data from a long research project in Massachusetts associated working out almost every day with an extended life span. Among about 5,000 Americans, middle-aged or older, a moderate or high level of activity added 1.3 to 3.7 years to the life span, compared to those with little exercise. The major benefit was a reduction in heart disease.

What is the best advice for an adult embarking on an exercise program? See the doctor first, and be sure to report any cardiac symptoms. Although there is some dispute on the matter, athletes live longer and healthier lives than non-athletes, and for most people, exercise helps control weight, improves mood and the immune system, and keeps the cardiovascular system in tip-top condition.



The Great North Run in London attracts many athletes, both well trained and amateur.

I WONDER. . .

moves into the veins, it pools against the valves. If the walls of the vein are weak, owing to disease or genetics, the vessel will expand. More blood will move into the distended area with each heartbeat without moving up toward the heart, pushing the walls out even further. The vein eventually pops out of the musculature and is visible as a bluish wrinkled cord directly beneath the skin. The varicose portion of the vein can be removed surgically if it becomes too painful or unsightly.

Spider veins are less visible because they involve venules, not veins. These surface venules fill with blood

but do not empty. They are visible through the skin as pale purple or blue tracings and usually occur in small to large patches on the face or thighs. Treatment for spider veins is purely cosmetic, and involves injecting the blocked venule with sterile saline solution to displace the pooled blood.

The exact cause of these two venous disorders is not known, but they do run in families. More women suffer varicose veins than men, and hormonal changes are often implicated. Many women develop varicose veins or spider veins during pregnancy.

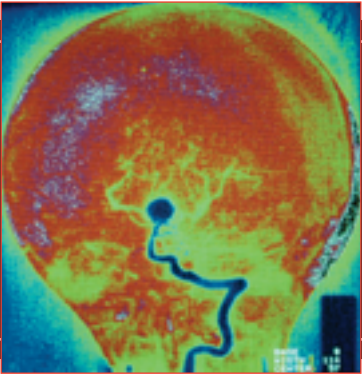
CONCEPT CHECK

List four risk factors for CVD.

How is atherosclerosis related to myocardial infarction?

Describe an aneurysm.
What can an aneurysm cause in the brain?

What is meant by “double bypass”?



Blood Consists of Plasma, Cells, and Other Formed Elements

LEARNING OBJECTIVES

Explore the role of plasma in blood.

Explain how red blood cells carry oxygen to the tissues.

List the various formed elements in blood.

Understand the physiological basis of blood typing.

Describe the functions of red and white blood cells.

Describe how clots form.

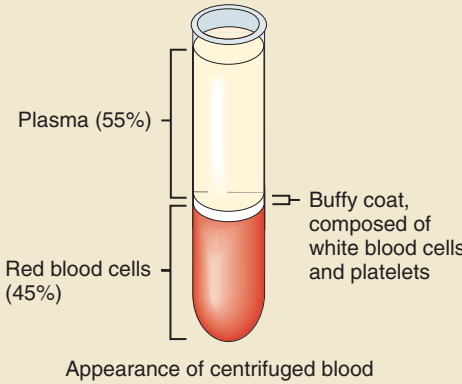
Hormones

Compounds secreted in one area of the body that are active in another, usually carried by the blood.

Many people are squeamish about blood. They do not like to see it outside the circulatory system, and the mere thought of it can make their knees weak. This is unfortunate because blood is a unique and essential connective tissue. It is composed of a liquid portion, the **plasma**, and a solid portion, the **formed elements**, which are mainly cells (**FIGURE 11.22**).

The functions of blood are all critical to maintaining homeostasis. Blood regulates the internal envi-

ronment of the body by diffusing ions and other materials into the interstitial fluid. It forms clots to prevent fluid loss at injuries. Blood also transports heat between the body core and the skin. Dissolved in the plasma are **hormones**, nutrients and gases that are needed in other areas, so blood serves as a mode of transport for these compounds. In addition, the formed elements in the blood deliver oxy-



Centrifuge tube of spun blood **FIGURE 11.22**

When blood is spun in a centrifuge, the formed elements settle to the bottom of the tube, leaving the plasma on the top. The red blood cells (RBC) are heavier than the white blood cells, so the white blood cells are found in a small “buffy coat” on top of the packed red blood cells.

gen and patrol the body to destroy pathogens. Both specific and non-specific immunity occur within the blood. None of these functions is reason to fear blood; instead they indicate just how remarkable this tissue is.

The formed elements in the blood are the cells and cellular fragments—99.9 percent of the formed elements are red blood cells, giving blood its red color; the other 0.1 percent are white blood cells and platelets.

Plasma, the fluid portion of blood, comprises approximately 46 to 63 percent of total blood volume. Plasma is 92 percent water, 7 percent dissolved proteins, and 1 percent **electrolytes**, nutrients, and wastes. The proteins help maintain blood’s osmotic pressure, so water remains inside the vessels instead of diffusing into the tissues. The protein albumin is particularly important in maintaining osmotic pressure. If the albumin level drops, osmotic pressure of the blood shifts, forcing water from the blood into the tissues, causing edema. Edema can also be caused by many other factors that alter the osmotic balance between blood and tissue.

Electrolytes

Compounds that dissolve into ions capable of conducting electricity.

Colony stimulating factors

Blood-borne compounds that cause cells in the bone marrow to produce new blood cells

The formed elements of the blood are cells or bits of cells that originate in the red bone marrow (**FIGURE 11.23**). In adults, red marrow is located within the epiphyses of the long bones, in the hip and sternum. Under the direction of hormones and **colony stimulating factors**, blood

stem cells differentiate into **erythrocytes**, platelets, or **leukocytes**. Leukocytes further differentiate into five types of white blood cells.

Erythrocytes

Red blood cells.

Leukocytes

White blood cells.

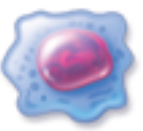
LEUKOCYTES ARE DEFENSIVE CELLS

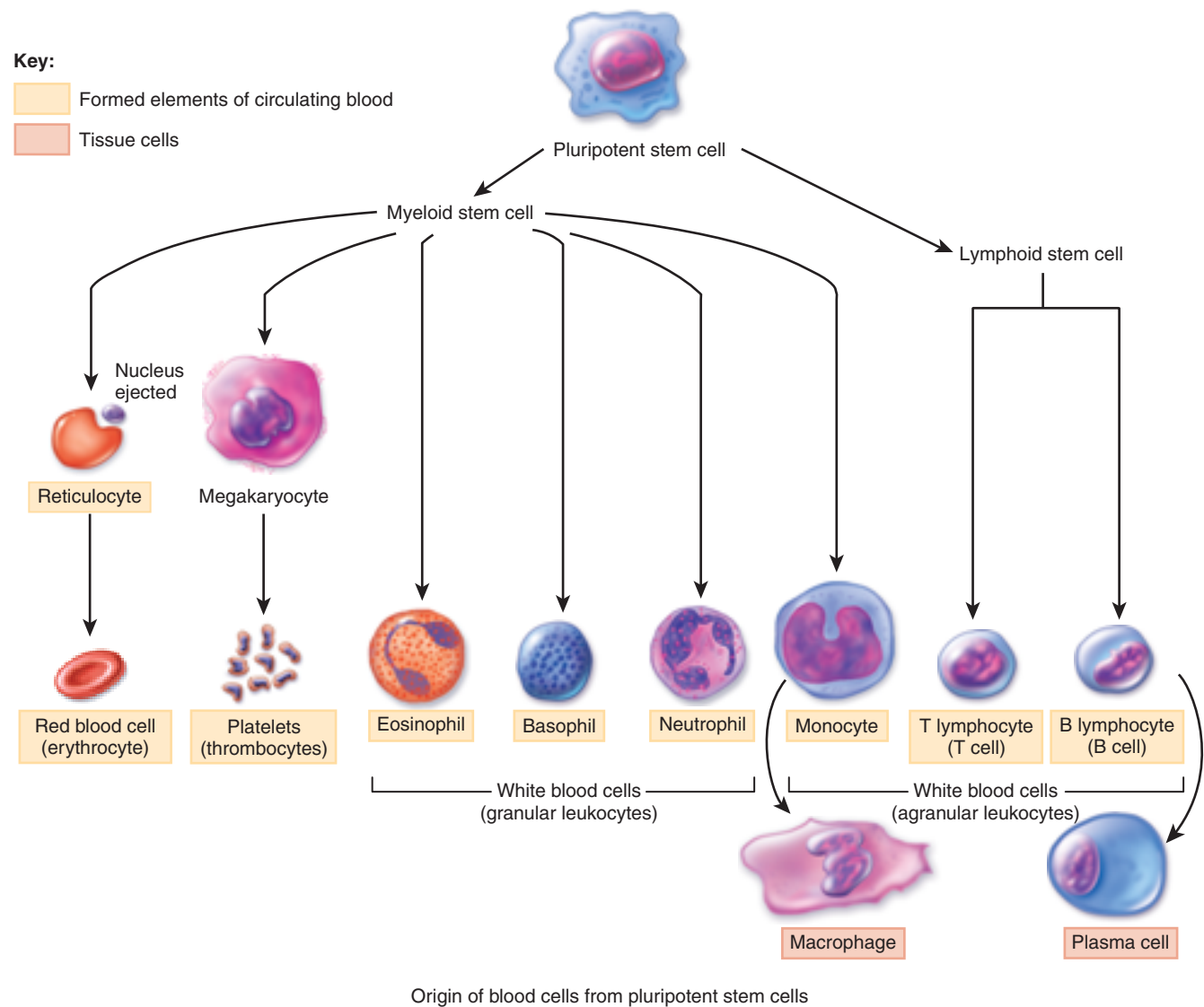
Leukocytes are specialized for defense, and though not abundant, they are critical to the immune system. There are approximately 5,000 to 11,000 white blood cells per mm³ of blood, compared to the 4 to 6 million red blood cells per mm³.

The five types of white blood cell (WBC) include three granular cells: **neutrophils**, **eosinophils**, and **basophils**, and two agranular cells: **lymphocytes** and **monocytes**. “Granular” means that when the cells are stained, dark granules appear in the cytoplasm under a microscope.

The odd names of the granulocytes (neutrophil, eosinophil, and basophil) reflect what happens when they are placed in Wright’s stain, a mixture of stains used to identify white blood cells. Neutrophil granules become stained with the neutral stain (their granules “like” neutral stain, which is the literal translation of “neutrophil”). Eosinophil granules stain a bright orange-pink, the color of the eosin portion of the stain. Basophil granules take on the basic (pH 11) stain color, nearly black.

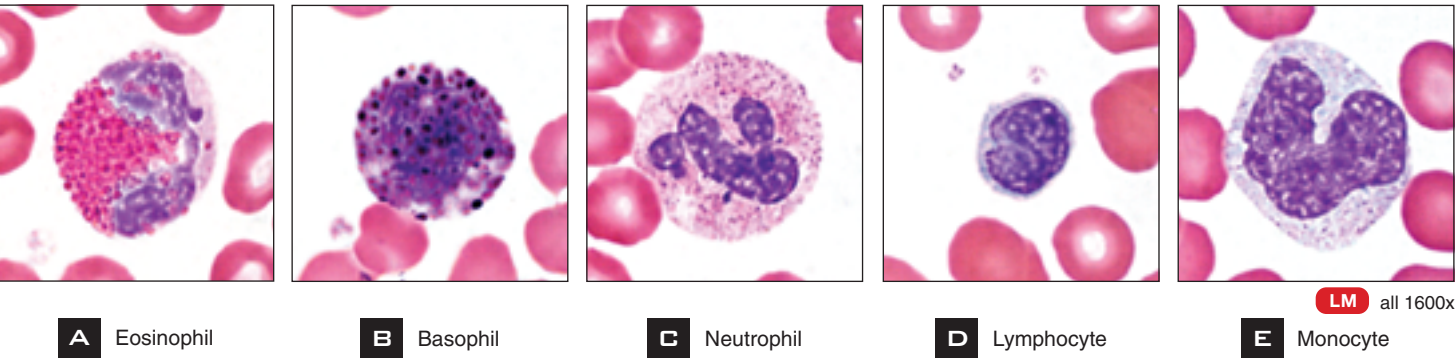
Agranulocytes contain no granules in their cytoplasm. Lymphocytes are small, round cells with little visible cytoplasm, whereas the monocytes are the largest of the white blood cells, with quite a bit





Blood cell formation FIGURE 11.23

Blood cell production, called **hematopoiesis**, occurs in bone marrow after birth. All the different types of blood cell arise from one type of pluripotent stem cell.










Leukocyte comparison FIGURE 11.24

of cytoplasm surrounding a large, kidney-shaped nucleus (FIGURE 11.24).

The proportions of leukocytes remain fairly constant in a healthy individual. Neutrophils make up the majority of circulating WBCs, with lymphocytes a close second. Monocytes are the third most common WBC, followed by eosinophils and lastly basophils. You can remember this order with this catchphrase: “Never let monkeys eat bananas.” (N = neutrophils,

l = lymphocytes, m = monocytes, e = eosinophils, and b = basophils.)

Each cell has a specific function in warding off pathogens. When necessary, specific populations of WBCs increase, altering the overall proportions. The proportion of white blood cells gives an indication of what type of pathogen is present. See TABLE 11.1 for a physical description and the main function of each type of WBC.

Summary of formed elements in blood TABLE 11.1			
Name and Appearance	Number	Characteristics*	Functions
Red Blood Cells (RBC) or Erythrocytes 	4.8 million/ μL in females; 5.4 million/ μL in males	7–8 μm diameter, biconcave discs, without nuclei; live for about 120 days.	Hemoglobin within RBCs transports most of the oxygen and part of the carbon dioxide in the blood.
White Blood Cells (WBCs) or Leukocytes	5000–10,000/ μL	Most live for a few hours to a few days. [†]	Combat pathogens and other foreign substances that enter the body.
Granular leukocytes			
Neutrophils 	60–70% of all WBCs	10–12 μm diameter; nucleus has 2–5 lobes connected by thin strands of chromatin; cytoplasm has very fine, pale lilac granules.	Phagocytosis. Destruction of bacteria with lysozyme, defensins, and strong oxidants, such as superoxide anion, hydrogen peroxide, and hypochlorite anion.
Eosinophils 	2–4% of all WBCs	10–12 μm diameter; nucleus usually has 2 lobes connected by a thick strand of chromatin; large, red–orange granules fill the cytoplasm.	Combat the effects of histamine in allergic reactions, phagocytize antigen–antibody complexes, and destroy certain parasitic worms.
Basophils 	0.5–1% of all WBCs	8–10 μm diameter; nucleus has 2 lobes; large cytoplasmic granules appear deep blue–purple.	Liberate heparin, histamine, and serotonin in allergic reactions that intensify the overall inflammatory response.
Agranular leukocytes			
Lymphocytes (T cells, B cells, and natural killer cells) 	20–25% of all WBCs	Small lymphocytes are 6–9 μm in diameter; large lymphocytes are 10–14 μm in diameter; nucleus is round or slightly indented; cytoplasm forms a rim around the nucleus that looks sky blue; the larger the cell, the more cytoplasm is visible.	Mediate immune responses, including antigen–antibody reactions. B cells develop into plasma cells, which secrete antibodies. T cells attack invading viruses, cancer cells, and transplanted tissue cells. Natural killer cells attack a wide variety of infectious microbes and certain spontaneously arising tumor cells.
Monocytes 	3–8% of all WBCs	12–20 μm diameter; nucleus is kidney shaped or horseshoe shaped; cytoplasm is blue–gray and has foamy appearance.	Phagocytosis (after transforming into fixed or wandering macrophages).
Platelets (Thrombocytes) 	50,000–400,000/ μL	2–4 μm diameter cell fragments that live for 5–9 days; contain many vesicles but no nucleus.	Form platelet plug in hemostasis; release chemicals that promote vascular spasm and blood clotting.

*Colors are those seen when using Wright’s stain.

[†]Some lymphocytes, called T and B memory cells, can live for many years once they are established.

ERYTHROCYTES CARRY OXYGEN

Erythrocytes, or red blood cells, transport oxygen to the tissues, and are by far the most common blood cells. Red blood cells (RBCs) are little more than a membrane-bound sac of **hemoglobin**, a protein that contains the pigment heme (**FIGURE 1 1.25**). Each RBC carries approximately 200 million hemoglobin molecules. Each of these molecules has at its center an atom of iron. This iron picks up oxygen (it rusts, in essence) in an environment where the oxygen content is high, and releases oxygen where oxygen is scarce. The concentration of oxygen in these areas is measured in bars, and is written as the partial pressure of the gas. Often the total gas pressure is due to more than one gaseous element. Each individual gas exerts a portion of the total, or a partial pressure. The partial pressure of oxygen is annotated as P_{O_2} , and the partial pressure of carbon dioxide is indicated P_{CO_2} . Hemoglobin responds to the P_{O_2} in tissues and blood. This is a perfect setup because the body needs to transport oxygen from the lungs (where oxygen concentration is high) to the tissues (where the concentration is low). Hemoglobin is so perfect, in fact, that it is the only respiratory protein found

Invertebrate

Organism without a vertebral column, such as an earthworm, crab, or starfish.

Hemolymph

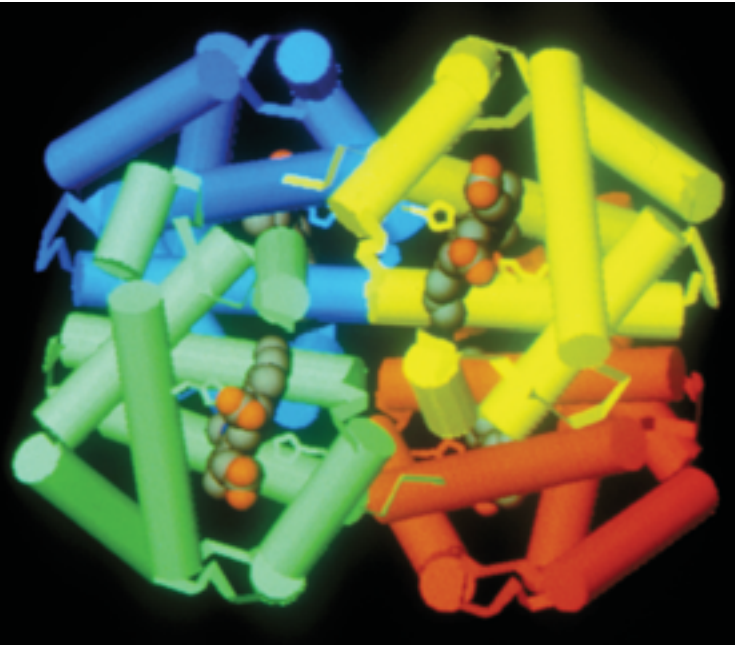
An oxygen-carrying fluid that circulates through the tissues of many invertebrates with open circulatory systems.

in vertebrates; the same protein that carries oxygen in your arteries also carries oxygen for fish, whales, and frogs. Hemoglobin also appears throughout the **invertebrates**, where it floats in the blood or **hemolymph** of some insects, clams, and worms.

Hemoglobin also responds to changes in pH and temperature. In low pH or high temperature, both of which occur in active muscle, hemoglobin drops its oxygen more readily, so that the RBC delivers the oxygen exactly where it is needed (**FIGURE 1 1.26**). No wonder this respiratory protein

is almost ubiquitous.

Erythrocytes are unique in several ways. As the immature red blood cell develops, it kicks out the nu-



Hemoglobin molecule **FIGURE 1 1.25**

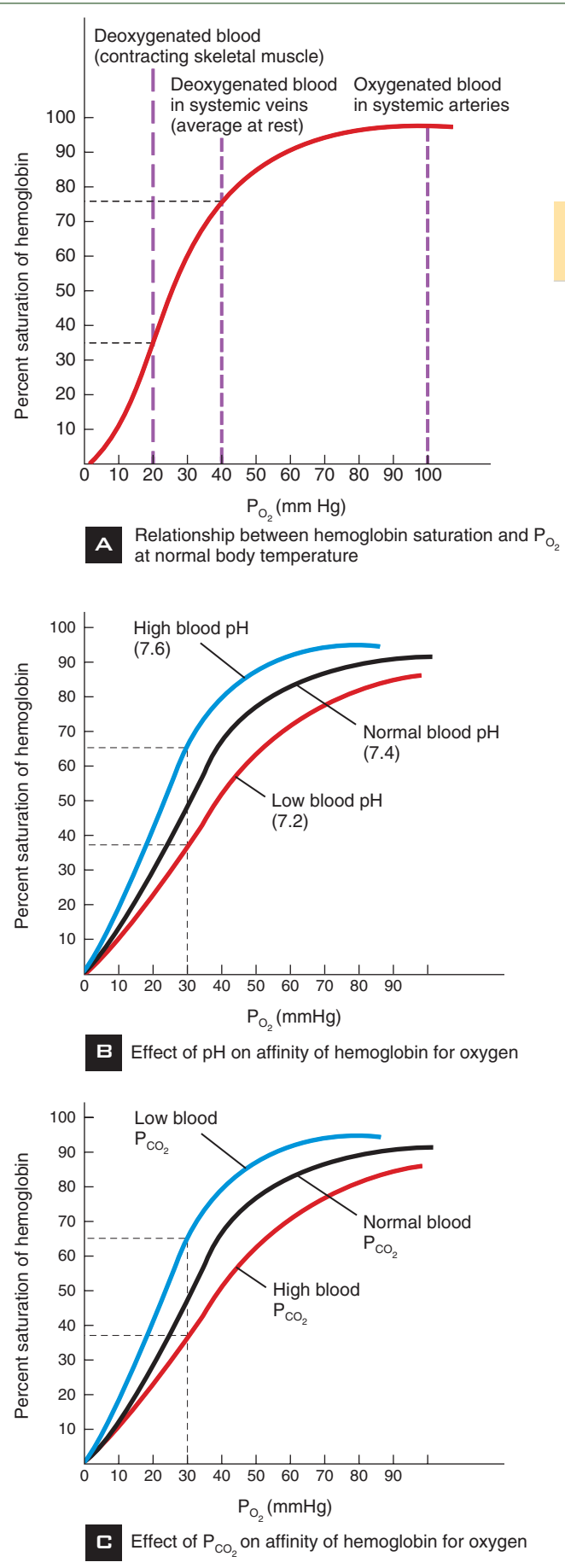
cleus to make room for more hemoglobin. Without a nucleus, the cell cannot repair itself, nor can it direct cellular activities, including such basics as cellular respiration. Red blood cells do not survive for long in the circulatory system. All the pressure from the left ventricle of the heart races the RBCs through the vessels and squishes them, one cell at a time, through the capillary beds (**FIGURE 1 1.27**). While passing through these beds, RBCs not only drop their oxygen, but they also suffer physical damage, which cannot be repaired in the absence of a nucleus.

RBCs circulate for approximately 120 days before they are damaged enough to need removal from the circulatory system. The spleen and liver are responsible for removing these cells, breaking them down, and recycling their constituent minerals and proteins. An estimated 2 million RBCs are broken down per second. Because we do not run out of RBCs, they must be produced at the same rate: an incredible 2 million cells created per second.

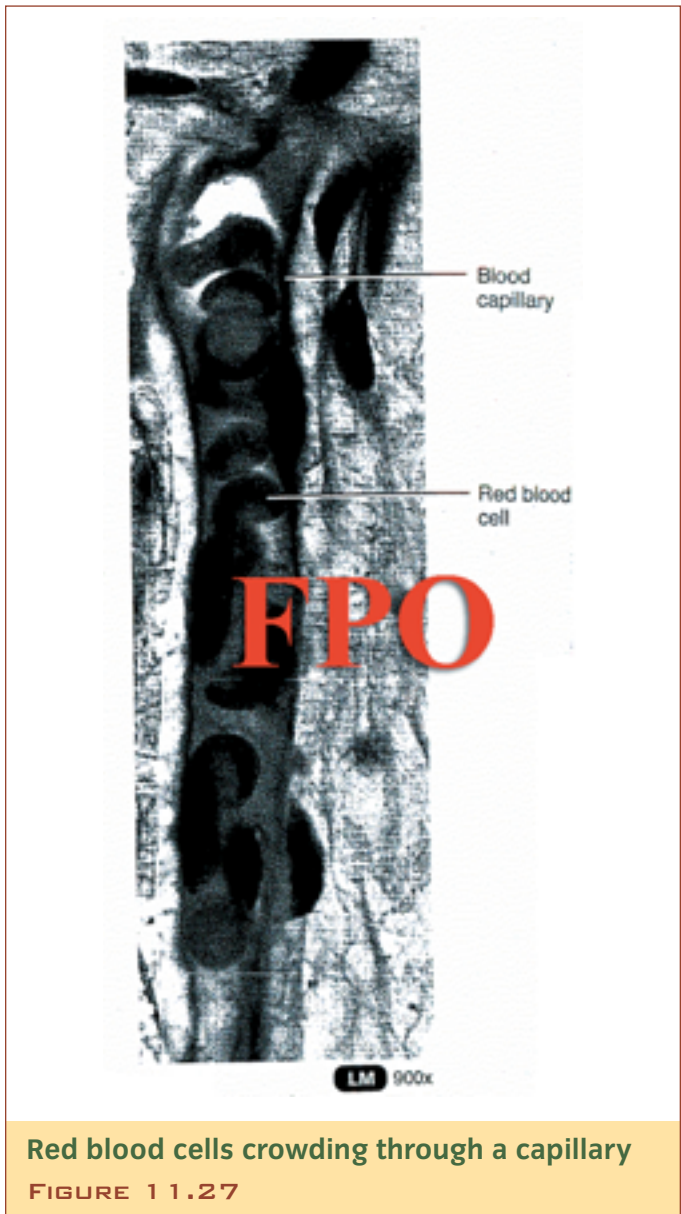
The rate of **erythropoiesis** is affected by hormones and environmental need. When blood oxygen drops, the kidneys are stimulated to produce erythropoietin, a hormone that stimulates RBC production. Because the presence of more red blood cells translates into more oxygen-carrying capacity, athletes can use this physiological fact to

Erythropoiesis

The formation of red blood cells (*poiesis* = to form, *erythro* = red).



Graphs of hemoglobin uptake and release in varying conditions **FIGURE 1 1.26**



Red blood cells crowding through a capillary **FIGURE 1 1.27**

improve their training. Because oxygen gets scarce at higher altitude, many athletes train at elevation just to stimulate RBC production. Some athletes have also used commercial erythropoietin, or EPO, to do the same thing. Although this hormone does increase RBC production, the performance advantage is unproven, and EPO is banned in many sports.

RED BLOOD CELL SURFACE PROTEINS DETERMINE BLOOD TYPE

Red blood cells, like other somatic cells, have many marker proteins on their surfaces, but the most important set is the markers that determine blood type. Blood type is described as A, B, AB, or O. Although you probably know your blood type, you may have no idea what those letters mean.

Co-dominant

Neither form of a gene will overshadow the other; when both forms are present, the individual will express both equally.

A, B, and O were arbitrarily chosen to identify the protein markers on the surface of your red blood cells. People with the “A” marker have type A blood; people with the “B” marker have type B. Because these traits are **co-dominant**, some people have

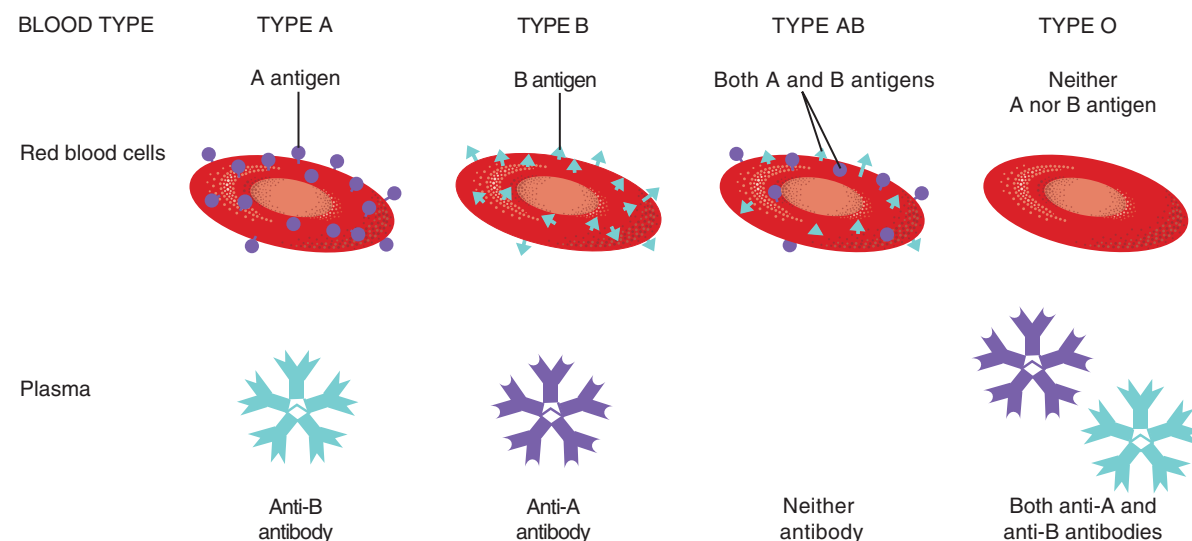
both A and B markers, which we call type AB blood. Those with neither A nor B markers have type O blood, which represents the condition described as “no markers.”

Recent findings, however, indicate that type O blood has the precursor to the A and B markers on its surface. This precursor, called H substance, is modified to form the A and B antigens on the surface of types A, B, and AB blood (**FIGURE 1 1.28**). Apparently, people with type O do not modify the H substance, leaving it as it appears in original form, able to trigger antibodies to both A and B antigens.

Despite certain dieting fads, the A, B, and O blood markers are important only when we must receive blood. The plasma of people with type A blood contains an anti-B **agglutinin** that will clump B blood. Similarly, those with type B blood have plasma that contains an anti-A agglutinin which clumps type A blood. Type O blood carries both anti-A and anti-B agglutinins. This does not harm the individual because their RBCs have neither marker. It stands to reason that those with type AB blood have neither anti-A nor anti-B agglutinins because either would agglutinate their blood, with fatal results. If the blood type is not

Agglutinin

Agent that causes cells to clump together or agglutinate.



The antigens and antibodies involved in blood typing **FIGURE 1 1.28**

matched before a blood transfusion, the recipients' agglutinins will clump the introduced blood, negating any benefits of the additional blood volume and possibly causing life-threatening problems.

These agglutinins are also used to “type blood” (determine its type). Technicians mix small samples of blood with each type of agglutinin and observe any reaction. If your blood clumps up when mixed with anti-A agglutinin, your RBCs have the surface marker A, and your blood is type A. If no clumping occurs when your blood is mixed with anti-A, you have type B or type O. Samples that clump when mixed with anti-B agglutinins have the B marker, and are type B. A sample that reacts with both anti-A and anti-B must have both A and B markers, and is thus type AB. Conversely, if no reaction appears with either agglutinin, the sample is type O.

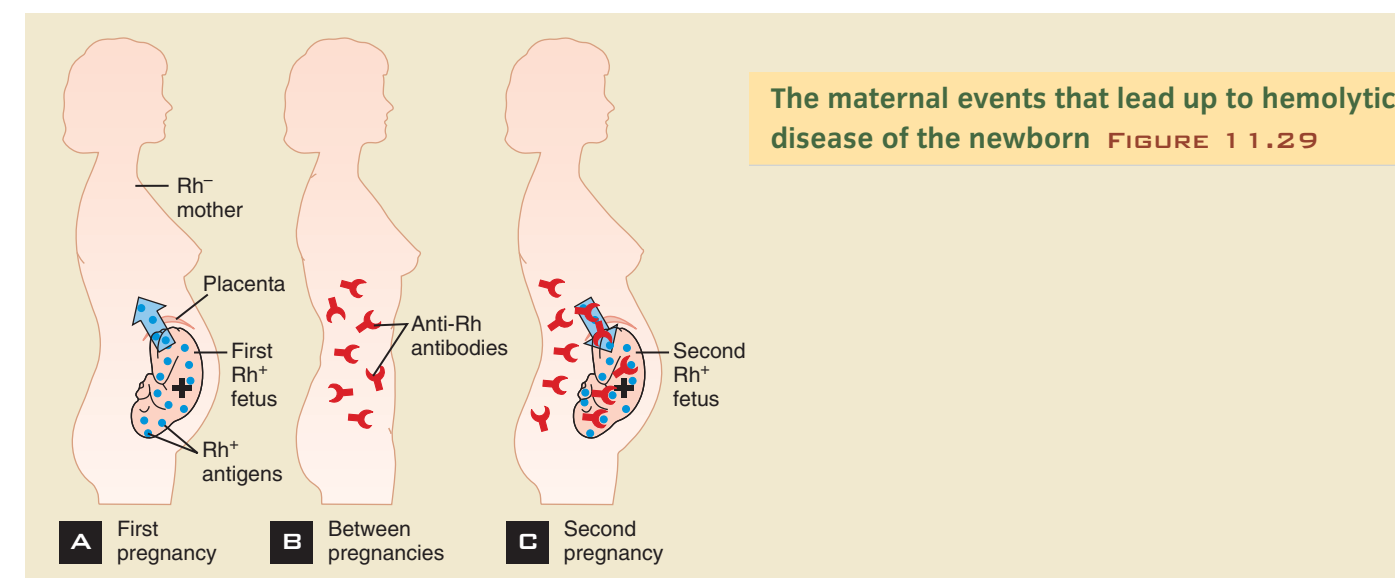
In the United States, type O blood is more common than type A, however the proportions of each type differ among ethnic groups. Slightly more than 46 percent of the Caucasian U.S. population are type O, whereas 38.8 percent are type A. Types B and AB are much rarer, comprising 11.1 and 3.9 percent of the population, respectively. Among African Americans, A and B are more evenly distributed: 49 percent are type O, 27 percent are type A, 20 percent are type B, and 4 percent are type AB. As a comparison, the Native American population is almost exclusively type O (79 percent). Very few Native Americans have type A blood (16

percent), and even fewer have type B (4 percent) or type AB (1 percent).

Because blood types are genetically based, they can be used to identify fathers in paternity suits, to eliminate or incriminate suspects in criminal investigations, and even to study ancient population migrations. A total of 26 blood groups other than ABO have been identified for these pursuits, including the MNS's, P, Lutheran (LU), and Kell (KEL).

Another blood-cell antigen, called **Rh factor**, is either present (Rh+) or absent (Rh-) on RBCs. On a blood-type card, Rh factor is the plus or minus sign after the A-B-O designation: “A+” or “O-.” Rh factor works differently than ABO because people who are Rh- do not ordinarily have anti-Rh agglutinin. But once they are exposed to Rh+ blood, their immune system starts to produce anti-Rh. To prevent complications from anti-Rh-, the Rh factor must be matched in an Rh- person who has previously received an unmatched transfusion.

Eighty-five percent of the U.S. population is Rh+. Rh- mothers are at risk of **hemolytic disease of the newborn**, or HDN (**FIGURE 1 1.29**), if the father is Rh+. As an Rh+ child develops in the uterus, fetal blood and maternal blood do not mix, so the mother will not make anti-Rh agglutinin. But during birth, these two blood supplies may contact each other, in which case the mother will begin to produce anti-Rh.



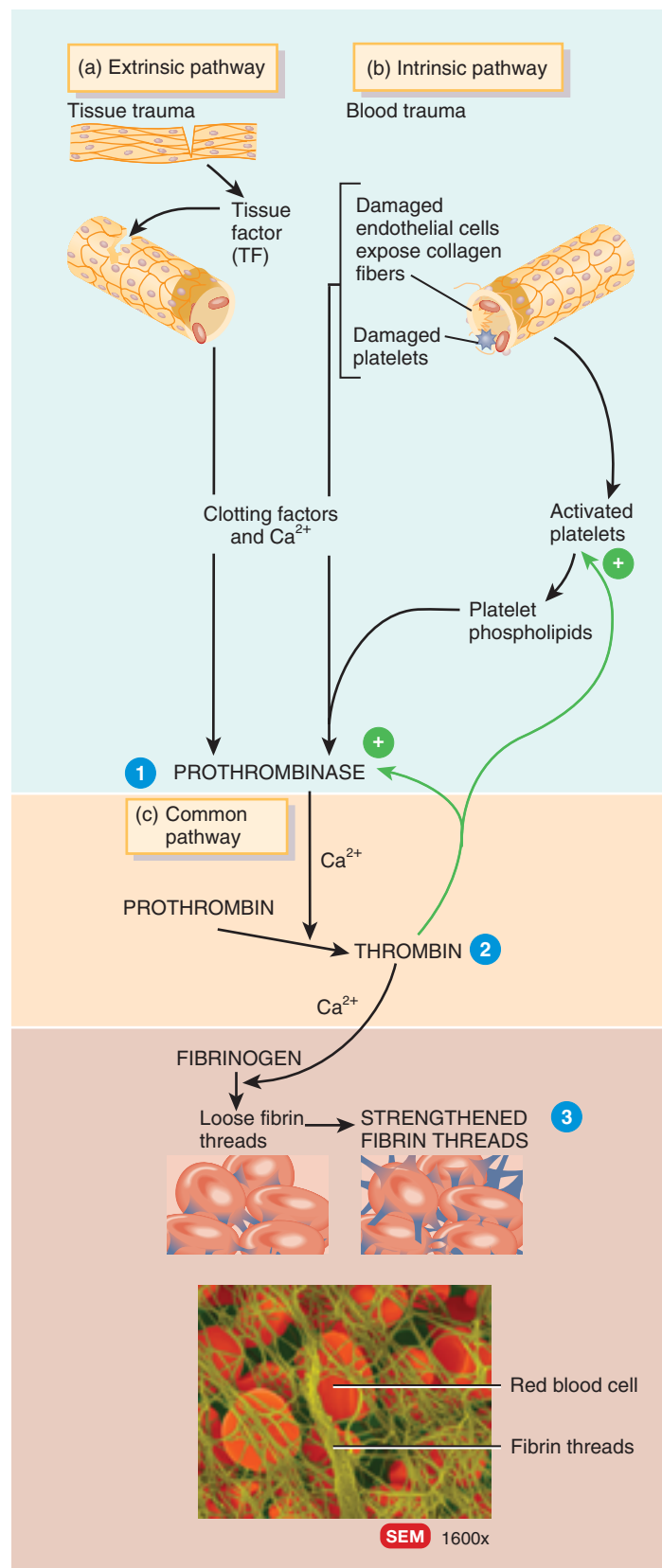
The maternal events that lead up to hemolytic disease of the newborn **FIGURE 1 1.29**



Process Diagram

Clot formation **FIGURE 11.33**

- 1 Prothrombin is formed by one of two pathways, the extrinsic pathway or the intrinsic pathway.
- 2 Prothrombinase converts prothrombin into the enzyme thrombin.
- 3 Thrombin converts soluble fibrinogen into insoluble fibrin. Fibrin forms the threads of the clot.



These antibodies will not affect her, or her first child, but if she becomes pregnant with a second Rh+ child, her Rh+ antibodies will cross the placenta and cause agglutination and destruction of this second baby's blood. Rh- mothers can be prevented from producing these antibodies **during the second birth** by inoculation with a dose of anti-Rh+ antibodies **immediately after the first birth**. These antibodies clump the Rh+ blood and remove it from the mother's blood supply before her immune response is launched.

PLATELETS GOVERN BLOOD CLOTTING

Platelets, the final type of formed element, are not even complete cells, but rather fragments of large cells called **megakaryocytes** that remain in the bone marrow. These huge cells bud pieces from their cytoplasm and release them into the bloodstream, forming more than 200 billion platelets per day. The fragments lack organelles and energy stores, but they do contain packets of physiologically active compounds. Once these compounds are released from the platelet into the surrounding plasma, they begin a series of events leading to the formation of a blood clot, in a process called hemostasis.

Clotting is necessary for maintaining fluid homeostasis; as we know, severe bleeding is a life-threatening emergency. Clotting is a complicated process in

which a series of plasma proteins interact with clotting factors released by the platelets (**FIGURE 11.30**). Clotting begins when a blood vessel is damaged, turning its normally smooth interior rough. These rough edges catch platelets flowing past, and a **platelet plug** may form and seal the wound without forming a true clot. A platelet plug is what prevents bleeding from a paper cut.

If the rip is too large for a platelet plug, a clot will form as the stuck platelets rupture with the pressure of the passing blood and release compounds that react with plasma components. These interactions begin a series of events that will continue until blood flow ceases. The damaged tissue and trapped platelets release **prothrombinase activator**, which converts the plasma protein **prothrombin** into its active form, **thrombin**. Thrombin, in turn, activates the plasma protein **fibrinogen**, forming long thin fibers of **fibrin**. The fibrin threads get caught in the rough edges of the torn vessel, creating a net. As blood flows through the fibrin net, red blood cells get trapped. More fibers are delivered by fresh plasma that reaches the wound. The new fibrinogen interacts with fresh thrombin, and the clotting cascade continues until the plasma stops flowing and ceases bringing more protein. When plasma stops flowing, clotting has succeeded at stopping the bleeding. Clotting is a rare example of positive feedback in the body (see the green arrows in Figure 11.30).

CONCEPT CHECK

What homeostatic functions does plasma provide?

physiologically, from the monocyte?

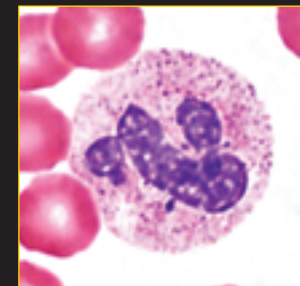
What is the main physiological difference between the Rh factor and the ABO blood group?

What is the function of the neutrophil? How is this cell different, anatomically and

Why do red blood cells live only 120 days?

How are blood types determined?

How do platelets initiate blood clotting?



Blood Can Suffer Many Disorders

LEARNING OBJECTIVES

Describe the general pathology of leukemia.

Define anemia and list its symptoms.

List common blood-borne pathogens

Because blood is so vital, when something goes wrong with it, our quality of life is severely diminished. Disorders of the blood can be life-threatening, as in leukemia, or they can cause acute disabilities, as in anemia. **Infectious mononucleosis**, commonly called mono, is a common blood disease on college campuses. Mono is caused by the **Epstein-Barr virus** and is transmitted through saliva—hence the popular name of “kissing disease.” (We discussed mono in detail in Chapter 10.)

BLOOD CANCER: LEUKEMIA

Perhaps the most frightening blood disorder is **leukemia**, literally: “white blood.” Leukemia is a general term for several cancers of the bone marrow. In most leukemias, the white blood cells are shaped abnormally and do not function properly. More than 2,000 children and 27,000 adults in the United States are diagnosed with leukemia every year.

Many symptoms of leukemia are flu-like, and all are related to those nonfunctional white blood cells. Infections take hold more readily and are more persistent. Lymph nodes and the spleen swell in an effort to rid the body of these defective leukocytes. To add to the difficulty, when the bone marrow is pushing out too many white blood cells, it often reduces its output of red blood cells, which reduces the blood’s oxygen-carrying capacity, causing fatigue and weakness.

The causes of leukemia are unknown, and although some risk factors have been identified, having these factors does not mean you will necessarily develop leukemia any more than having the risk factors associ-

ated with cardiovascular disease means you will have a heart attack. The risk factors include exposure to ionizing radiation from nuclear weapons and nuclear waste, exposure to carcinogens such as benzene, or a family history of the disease.

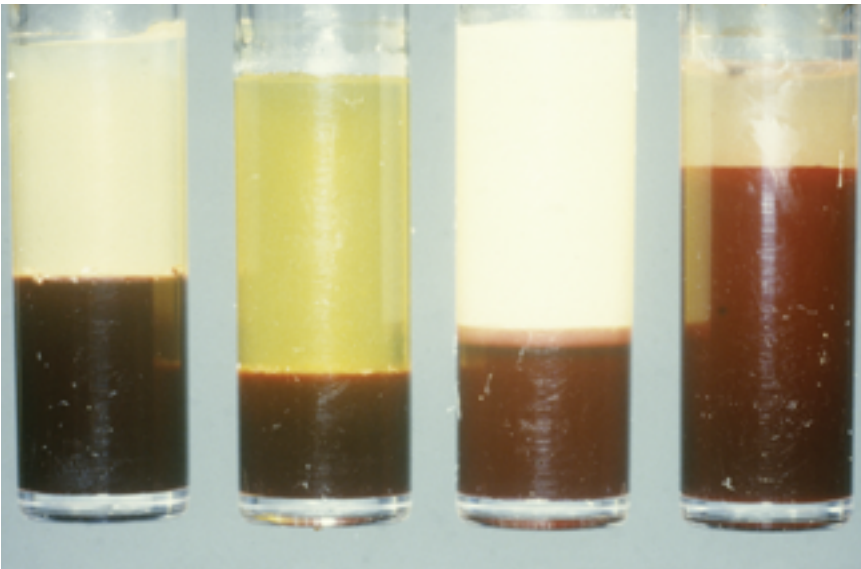
Leukemia can be classified by its pattern of onset or by the specific cells affected. **Acute** leukemia appears quickly, filling the blood with extremely immature white blood cells called **blasts**. **Chronic** leukemia appears far more slowly, with blood cells that are more developed, but still immature. Both acute and chronic leukemia can affect either **myeloid** or **lymphoid** cells. Both cells mature in the bone marrow, but myeloid cells become the granulocyte form of white blood cells, whereas lymphoid cells mature in the lymph glands and become lymphocytes (see Figure 11.23).

Treatments for leukemia vary depending on the stage of disease and the type of affected leukocytes, but the goal is to move the patient into **remission**—where the disease may remain in the patient’s bone marrow, but the leukocytes are functionally normal. Treatment—chemotherapy, radiation therapy, bone marrow transplant, or biological therapy—is often successful for a period. Because many times the disease reappears, leukemia patients need constant medical monitoring.

ANEMIA MEANS A SHORTAGE OF ERYTHROCYTES

Anemia is a reduction in the red blood cell population and thus in the blood’s oxygen-carrying capacity. The symptoms of anemia include fatigue, weakness, shortness of breath, and sometimes chest pains like angina. Anemia is easily diagnosed via **hematocrit** (FIGURE 11.31). A packed cell level below 42 percent in adult males, or 38 percent in adult females, often indicates some form of anemia.

The many types of anemia are based on the cause of the red blood cell deficiency. A shortage of iron, vitamin B12, intrinsic factor (a hormone that allows for iron absorption), or other essential proteins all inhibit RBC production. Excessive bleeding will reduce



Hematocrit tubes

FIGURE 11.31

The level of packed cells in the hematocrit tube after **spining** in a **centrifuge** indicates the amount of oxygen-carrying capacity of that sample of blood. Adults should have a packed cell volume above 38 percent. If the packed cell volume is lower than that, anemia is suspected.

RBC counts. Improper formation of red blood cells and nutritional deficits can also cause anemia. Sickle cell anemia (see the accompanying Health, Wellness, and Disease box) is a special case wherein the hemoglobin molecule is not properly formed.

CARBON MONOXIDE PREVENTS THE BLOOD FROM CARRYING OXYGEN

Carbon monoxide is an odorless environmental poison that prevents the blood from carrying oxygen and can cause death or disability. Carbon monoxide (CO) molecules establish an irreversible bond to hemoglobin, thereby causing the hemoglobin molecule to lose the ability to carry oxygen. Red blood cells contaminated with CO float uselessly through the blood until they wear out and are destroyed. Normally, air contains almost no CO, so this irreversible binding is irrelevant. But the CO concentration increases dramatically in some environments, primarily when fossil fuels are burned and the exhaust fumes are returned to the combustion zone. This can happen if a car runs in a closed garage or a malfunctioning furnace recycles fumes into a residence. Because severe CO poisoning can starve the tissues of oxygen, causing brain damage, myocardial infarction, or death, carbon monoxide detectors

(much like smoke detectors), are an affordable and sensible precaution. The first symptoms of CO poisoning are drowsiness and headache. If you suspect carbon monoxide poisoning, move to fresh air and seek medical help. Blood transfusions may be needed to replace carbon monoxide-polluted red blood cells with functional erythrocytes.

PATHOGENS CAN LIVE IN THE BLOOD

Though not necessarily a disorder of the blood itself, many pathogens travel in the blood, including **hepatitis**, **HIV**, and other sexually transmitted diseases. The best defense against blood-borne pathogens is to prevent your blood from contacting another person’s blood. A key source of infection is unprotected sex, which can tear **mucus** membranes, causing unintentional contact between the two bloodstreams. Healthcare workers are constantly reminded to take precaution around all “sharps,” because an inadvertent “stick” with a used needle can spread blood-borne pathogens. To prevent infection through transfusion of tainted blood, blood banks routinely test their stocks of blood for viral contamination. Because blood-borne pathogens include such a variety of diseases, each case is treated with an eye to the ultimate goal: removing the pathogen from the blood.

Sickle cell anemia

About 72,000 Americans have sickle cell anemia. Sickle cell disease is a genetic defect that deforms red blood cells and impairs their ability to circulate and transport oxygen. The sickle cell mutation changes the oxygen-transport protein, hemoglobin. Normally, red blood cells (RBCs) are shaped like donuts. In sickle cell anemia, they are hard and crescent or “sickle” shaped in environments with a low PO_2 . The deformed cells get caught in capillaries, forming a plug that prevents other RBCs from delivering oxygen to these tissues. And because these abnormal RBCs live only 10 to 20 days, in contrast to the normal 120-day life span, patients with sickle-cell disease also have anemia, a shortage of RBCs.

Sickle cell disease is an inherited defect in the gene that forms hemoglobin. A person who inherits one “sickle” gene and one normal gene becomes a carrier for sickle cell disease; they have “sickle cell trait.” These carriers do not have symptoms of sickle cell disease, but their children are at risk. If both parents have sickle cell trait, each child has a 1 in 4 chance of having the disease and a 2 in 4 chance of being carriers (having the sickle cell trait), leaving only a 1 in 4 chance of being free of the sickle cell trait.

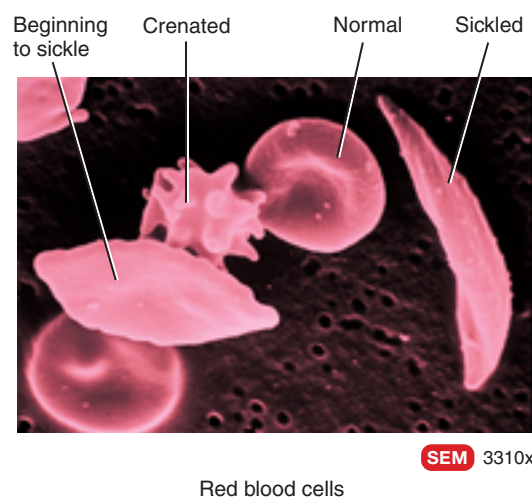
Why does the sickle cell trait persist among humans? Evolution, after all, tends to remove defective genes from the population, and they should eventually disappear. The answer is that the sickle cell trait protects people from the deadly consequences of malaria, a parasite which at-

tacks red blood cells. This resistance to malaria explains why the sickle cell gene is common in Africa, South and Central America, some Mediterranean **countries** and India. In these places, protection against malaria is an adaptive trait, even if it does reduce the fitness of the individual by placing their offspring at risk of inheriting sickle cell anemia.

Although sickle cell disease is inherited and present at birth, symptoms usually don't occur until after 4 months of age. Beyond the problem with oxygen transport, the deformed cells also cause small blood clots and recurrent painful episodes called “sickle cell crises.” Other crises include potentially life-threatening *hemolytic crises* when damaged red blood cells break down, *splenic sequestration crises* as the spleen enlarges and traps the damaged blood cells, and *aplastic crises* if a certain type of infection causes the bone marrow to stop producing red blood cells.

Repeated crises can damage the kidneys, lungs, bones, eyes, and central nervous system. Blocked blood vessels and damaged organs cause acute painful episodes, which most patients suffer at some point, that can last hours to days. These acute painful episodes affect the bones of the back, the long bones, and the chest. The crises may require hospitalization for pain control, oxygen, and intravenous fluids.

Sickle cell disease can cause death by organ failure and infection. Some patients experience minor, brief, and infrequent episodes, while others endure severe, prolonged, and frequent episodes with many complications. People with sickle cell disease need treatment to prevent and reduce symptoms. Blood transfusions can treat the anemia portion of the disease. In the past, death from organ failure usually occurred between ages 20 and 40. More recently, because of better understanding and management, affected people live into their 40s and 50s.



Again and again in this discussion, we have returned to one of the primary roles of the blood: to distribute oxygen and remove carbon dioxide. To do its work, the cardiovascular system must interact closely with the respiratory system, which is the point of entry for oxygen and the point of departure for carbon dioxide. If you need more evidence of the tight interaction

between the CV and respiratory systems, pay attention to your own body. Take your pulse while resting, and simultaneously count your breaths. Then run up some stairs and repeat. Notice that both your pulse and your breathing have accelerated. To understand what is happening during this interaction of heartbeats and breaths, we must move on to the respiratory system.

CONCEPT CHECK

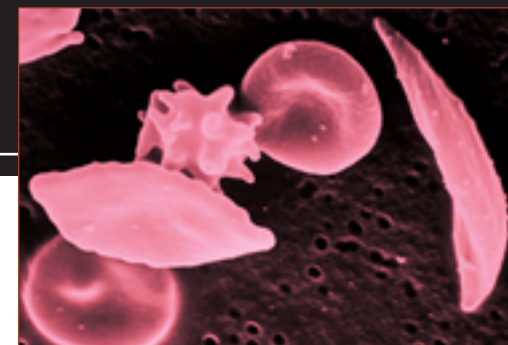
Differentiate

between chronic and acute leukemia.

Why is sickle cell anemia still present in the human gene pool?

How does carbon monoxide reduce oxygen-carrying capacity of blood?

List two blood-borne pathogens.

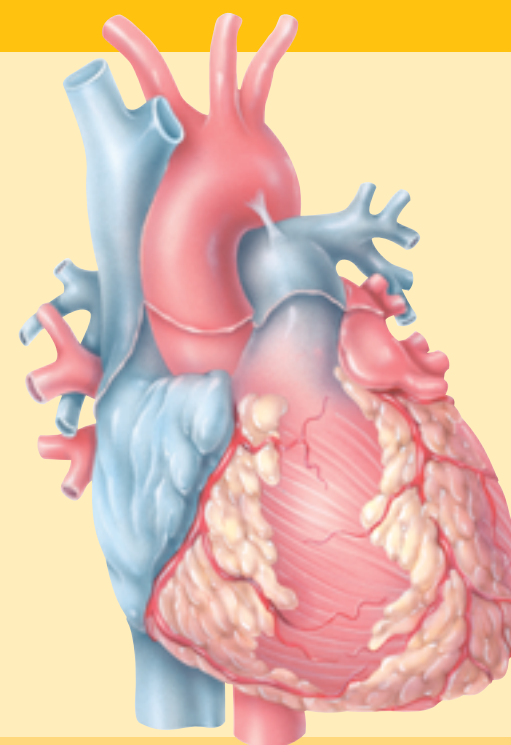


CHAPTER SUMMARY

1 The Heart Ensures Continual, 24/7 Nutrient Delivery

The cardiovascular system is responsible for the transport of nutrients, gases, and waste products in the body. It is a closed system, consisting of the heart, arteries, veins, and capillaries. The heart serves as the pump for the cardiovascular system, pushing blood in one direction through the body. The heart has four chambers: two atria and two ventricles. The ventricles generate the force needed to move the blood. The bicuspid, tricuspid, aortic, and pulmonary valves in the heart prevent backflow. Cardiac output is a measure of the amount of blood pumped by the heart in one minute.

The conduction system of the heart consists of the SA node, AV node, AV bundle, bundle branches, and the Purkinje fibers. The impulse to contract follows this pathway, ensuring the heart will contract effectively. The ECG records the changes in electrical charge as the cardiac cells contract. The P wave is the depolarization of the atria, the QRS complex is the depolarization of the ventricles, and the T wave is the repolarization of the ventricles. The time between events measures the speed of transfer of the contraction impulse.



CHAPTER SUMMARY

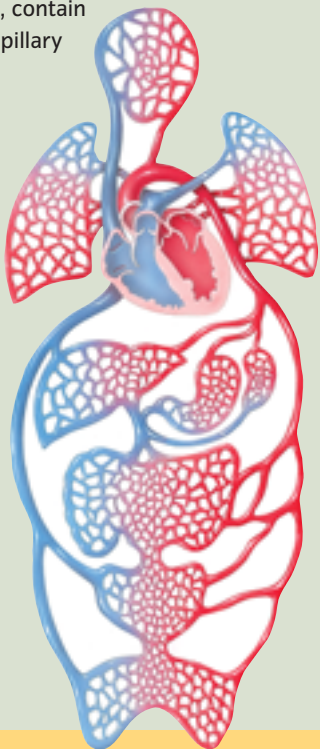
2 Blood Vessels and Capillary Transport Involve Miles of Sophisticated Plumbing

Arteries carry blood from the heart, capillaries are the exchange vessels, and veins return the blood to the heart. The walls of these vessels differ according to the differing pressures they each carry. Veins, with extremely low-pressure flow, require valves in order to prevent backflow.



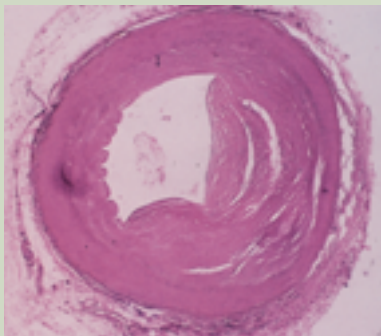
3 Different Circulatory Pathways Have Specific Purposes

Vessels that lead from the heart to the lungs and back to the heart comprise the pulmonary system. The systemic system includes vessels that leave the heart, travel through the tissues, and return to the heart. Portal systems, like the hepatic portal system, contain two capillary beds.



4 Cardiovascular Disorders Have Life-Threatening Consequences

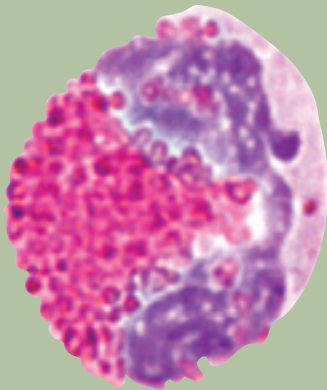
Cardiovascular disease is the leading cause of death in the United States. It includes many different problems, such as hypertension, atherosclerosis, heart attack, heart failure, embolism, and stroke. Genetic factors play a role in hypertension, atherosclerosis, heart **attack** and heart failure. High blood pressure affects the tissues of the body by damaging or destroying capillary beds. Vessels become clogged with fatty deposits in atherosclerosis. Heart attack, or myocardial infarction, is due to a lack of blood flow to a region of the heart. Angioplasty, stent **placement** or bypass surgery are used to correct for cardiac atherosclerosis, in order to prevent heart attack. Heart failure is an inability of the heart to pump blood from the left ventricle through the body.



5 Blood Consists of Plasma, Cells, and Other Formed Elements

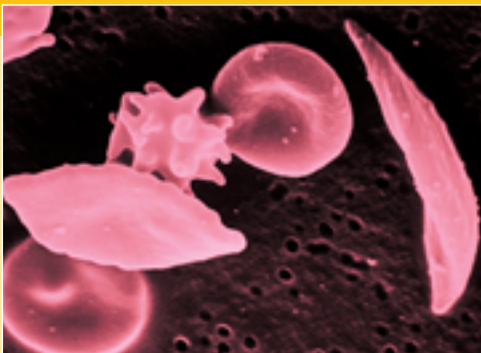
Blood is a liquid connective tissue composed of plasma, red blood cells, white blood cells, and platelets. The plasma serves to hydrate the body and dissolve nutrients. The red blood cells transport oxygen, using hemoglobin, which drops oxygen in areas of low oxygen concentration, and picks it up in areas of high concentration. RBCs carry marker substances on their surface, designating them as type A, B, AB, or O. In addition, there is an Rh factor on most people's RBCs. The ABO blood groups are genetically determined and can be used to trace lineage. Type A blood has anti-B agglutinins; Type B blood has anti-A agglutinins, and Type O blood has both agglutinins. Type AB blood has neither agglutinin because that would be detrimental to the individual. There are many other blood groups based on proteins and glycoproteins on the surface of the RBCs.

White blood cells provide immunity and nonspecific defense. There are five types of white blood cells: neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Each has a specific job and occurs in a specific percentage in a healthy individual. Platelets maintain fluid hemostasis. They either form a platelet plug blocking the loss of blood in small tears, or they release factors that initiate clotting. The clot formation is a positive feedback loop, continuing until blood no longer flows past the injured area.



6 Blood Can Suffer Many Disorders

Anemia is the most common blood disorder. In this disorder, RBC numbers decline and oxygen-carrying capacity of the blood drops. Causes of this range from lack of iron in the diet, to inadequate protein formation to bleeding and loss of blood volume. Sickle cell anemia is a special type of anemia in which the hemoglobin is incorrectly formed, causing a drop in RBC levels. Leukemia is another blood disorder,



this time affecting the white blood cells. Causes of leukemia may include exposure to benzenes or nuclear fallout.

Leukemia is treated with chemotherapy, or bone marrow transplant surgery. Blood can carry a wide range of pathogens. Many are spread by contact with contaminated blood, which is one reason for using caution in sexual activity. Blood banks must test blood for viruses before distributing blood for transfusions. HIV, herpes and other STDs are examples of blood-borne pathogens.

KEY TERMS

- agglutinin p. 000
- anastomoses p. 000
- bicuspid p. 000
- capillary bed p. 000
- cardiac sinus p. 000
- co-dominant p. 000
- congenital p. 000
- coronary sinus p. 000
- diastole p. 000
- edema p. 000

- electrocardiogram p. 000
- electrolyte p. 000
- erythrocytes p. 000
- erythropoiesis p. 000
- hemolymph p. 000
- hormones p. 000
- hypertrophy p. 000
- invertebrate p. 000
- ischemia p. 000
- leukocytes p. 000

- lumen p. 000
- mediastinum p. 000
- prolapse p. 000
- Purkinje fibers p. 000
- systole p. 000
- tachycardia p. 000
- tricuspid p. 000
- venules p. 000

CRITICAL THINKING QUESTIONS

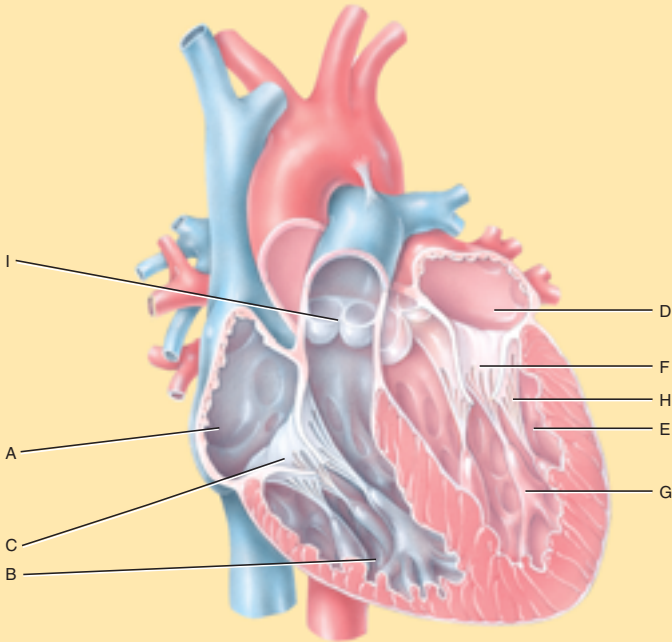
1. Reptiles and amphibians have a three-chambered heart, with only one ventricle. Blood flows from the lungs and body into this single pumping chamber, which pushes it to the body or the lungs. How does this compare with the functioning of the four-chambered heart of mammals? Explain the physiological advantage of separate left and right ventricles.
2. Artificial pacemakers can override the natural heartbeat set by the SA node. These electronic devices set a constant heartbeat that is not sensitive to the body's demands. List some activities that would be challenging for a patient with an artificial pacemaker. What innovation could improve pacemaker technology?
3. Most capillaries are diffusion vessels, meaning that nutrients, oxygen, waste material, and hormones can pass through their walls and into surrounding cells (or vice versa). What features

- of the structure of a capillary wall raise diffusion capacity—how does structure relate to function in this case? What special modifications would you expect to see in areas where diffusion is prevented, as in capillaries of the brain?
4. Marie was born and raised in Denver, Colorado, the “mile high” city. She has been a cross-country runner since grade school. When Marie went to college in Florida, her running times were much improved over her Denver times. What might explain her sudden improvement?
 5. Hemophilia is an inherited clotting disorder attributable to the absence of one necessary blood-clotting factor. How might the inability to form blood clots affect daily life? Why is homeostasis a vital function of the blood?

SELF TEST

1. The correct pattern of blood flow through the cardiovascular system is as follows:
- a. Heart→veins→arteries→capillaries→heart
 - b. Heart→arteries→capillaries→veins→heart
 - c. Heart→veins→capillaries→arteries→heart
 - d. Heart→capillaries→veins→arteries→heart

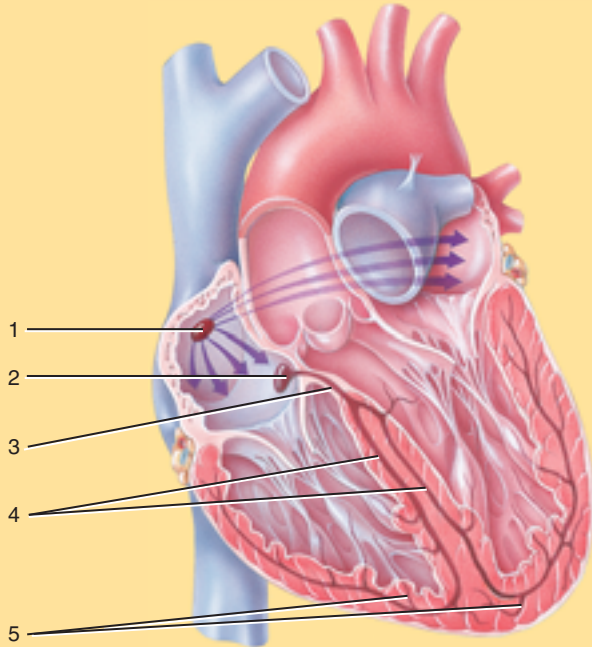
For questions 2,3 and 4, refer to the following figure.



2. The chamber of the heart that receives blood from the lungs is
- a. A
 - b. B
 - c. D
 - d. E
3. The valve that prevents backflow of blood returning from the body is
- a. A
 - b. C
 - c. F
 - d. I
4. The structure(s) responsible for supporting and stabilizing the interventricular valves is(are)
- a. C
 - b. F
 - c. G
 - d. H
 - e. Both G and H

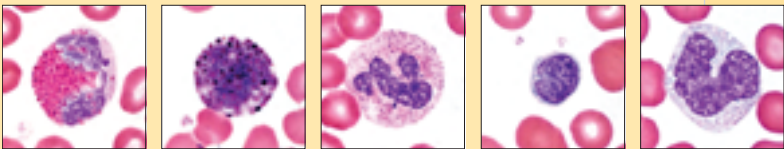
5. True or False: When the heart is relaxed, it is said to be in diastole.
6. During the cardiac cycle, the stage that immediately follows atrial systole is
- a. atrial diastole.
 - b. ventricular systole.
 - c. ventricular diastole.
 - d. whole heart diastole.

The following figure should be use to answer questions 7, 8, and 9.



7. The structure that initiates the heartbeat, indicated by the number 1, is the
- a. Purkinje fibers.
 - b. AV node.
 - c. bundle branches.
 - d. SA node.
8. Once the heartbeat begins, the function of the structure labeled 2 is to
- a. spread the impulse to contract to the cells of the atria.
 - b. slow the impulse to contract and pass it to the AV bundle and on to the ventricles.
 - c. allow the impulse to reach all the cells of the ventricles simultaneously.
 - d. send the impulse to contract on to the bundle branches.

9. The structure responsible for the P wave on an ECG is number
- a. 1
 - b. 2
 - c. 3
 - d. 4
10. The blood vessel that is thin-walled, includes valves, and carries blood under little pressure is the
- a. artery.
 - b. capillary.
 - c. vein.
 - d. All of the above fit this description.
11. The main difference between the pulmonary circuit and the systemic circuit is that in the pulmonary circuit,
- a. oxygen-rich blood leaves the heart for the lungs.
 - b. pulmonary veins carry oxygen-poor blood.
 - c. pulmonary arteries carry oxygen-poor blood.
 - d. blood in the pulmonary circuit goes to the brain only.
12. When a blood vessel of the leg becomes occluded (blocked) by a fatty deposit, the resulting condition is
- a. stroke.
 - b. aneurysm.
 - c. myocardial infarction.
 - d. atherosclerosis.
13. Congestive heart failure
- a. causes a build-up of fluid in the lungs and pericardium.
 - b. is more common in the elderly than the young.
 - c. is due to a weakened left ventricle.
 - d. All of the above are true.
14. The most common cell in the blood is the
- a. neutrophil.
 - b. leukocyte.
 - c. erythrocyte.
 - d. platelet.
15. True or False: The liquid portion of the blood, the plasma contains water, proteins, and cells.
16. Which of the cells shown in this figure is least common in the blood?

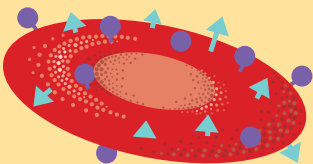


- (a) Eosinophil
 - (b) Basophil
 - (c) Neutrophil
 - (d) Lymphocyte
 - (e) Monocyte
- a. Neutrophil
b. Eosinophil
c. Basophil
d. Monocyte
e. Lymphocyte

17. Hemoglobin is specialized to _____ oxygen where pH is low, oxygen concentration is low, or temperatures are high.
- a. release
 - b. pick up

18. What is the blood type of the cell indicated?

- a. A
- b. B
- c. O
- d. AB



19. The plasma protein that is activated and forms a network of fibers across a wound to trap RBCs is
- a. prothrombin.
 - b. thrombin.
 - c. fibrinogen.
 - d. fibrin.
20. This test is used to diagnose
- a. anemia.
 - b. acute leukemia.
 - c. infectious mononucleosis.
 - d. angina.

