

CHAPTER 19

PHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM

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KEY TERMS

- | | |
|-----------------------|------------------|
| cardiac cycle | pressoreflex |
| cardiac output | stroke volume |
| chemoreceptor reflex | systole |
| diastole | vasoconstriction |
| electrocardiogram | vasodilation |
| pacemaker | venous return |
| peripheral resistance | |

The vital role of the cardiovascular system in maintaining homeostasis depends on the continuous and controlled movement of blood through the thousands of miles of capillaries that permeate every tissue and reach every cell in the body. It is in the microscopic capillaries that blood performs its ultimate transport function. Nutrients and other essential materials pass from capillary blood into fluids surrounding the cells as waste products are removed. Blood must not only be kept moving through its closed circuit of vessels by the pumping activity of the heart, but it must also be directed and delivered to those capillary beds surrounding cells that need it most. Blood flow to cells at rest is minimal. In contrast, blood is shunted to the digestive tract after a meal or to skeletal muscles during exercise. The thousands of miles of capillaries could hold far more than the body's total blood volume if it were evenly distributed. Regulation of blood pressure and flow must therefore change in response to cellular activity.

Numerous control mechanisms help to regulate and integrate the diverse functions and component parts of the cardiovascular system to supply blood to specific body areas according to need. These mechanisms ensure a constant *milieu intérieur*, that is, a constant internal environment surrounding each body cell regardless of differing demands for nutrients or production of waste products. This chapter presents information about several of the control mechanisms that regulate the pumping activity of the heart and the smooth and directed flow of blood through the complex channels of the circulation.

HEMODYNAMICS

Hemodynamics is a term used to describe a collection of mechanisms that influence the active and changing—or dynamic—circulation of blood. Circulation is, of course, a vital function. It constitutes the only means by which cells can receive materials needed for their survival and can have their wastes removed. Circulation is necessary, and circulation of different volumes of blood per minute is also essential for healthy survival. More active cells need more blood per minute than less active cells. The reason underlying this principle is obvious. The more work cells do, the more energy they use, and the more oxygen and nutrients they remove from the blood. Because blood circulates, it can continually bring in more oxygen and nutrients to replace what is consumed. The greater the activity of any part of the body, the greater the volume of blood circulating through it. This requires that circulation control mechanisms accomplish two functions: maintain circulation (keep blood flowing) and vary the volume and distribution of the blood circulated. Therefore as any structure increases its activity, an increased volume of blood must be distributed to it—must be shifted from the less active to the more active tissues.

To achieve these two ends, a great many factors must operate together as one smooth-running, although complex, machine. Incidentally, this is an important physiological principle that you have no doubt observed by now—that every body function depends on many other functions. A constellation of separate processes or mechanisms act as a single integrated mechanism. Together, they perform one large function. For example, many mechanisms together accomplish the large function we call *circulation*.

This chapter is about hemodynamics—the mechanisms that keep blood flowing properly. We begin with a discussion of the heart as a pump, then move on to the even bigger picture of blood flow through the entire cardiovascular system.

THE HEART AS A PUMP

In Chapter 18 we discussed the functional anatomy of the heart. Its four chambers and their valves make up two pumps: a left pump and a right pump. The left pump (left side of the heart) helps move blood through the systemic circulation and the right pump (right side of the heart) helps move blood through the pulmonary circulation. We will now step back from our previous discussion of the valves and chambers of the heart to look at the bigger picture and see how these two linked pumps function together as a single unit. First, we will discuss the role of the electrical conduction system of the heart in coordinating heart contractions. Then we will discuss how these coordinated contractions produce the pumping cycle of the heart.

CONDUCTION SYSTEM OF THE HEART

The anatomy of four structures that compose the conduction system of the heart—sinoatrial (SA) node, atrioven-

tricular (AV) node, AV bundle, and Purkinje system—was discussed briefly in Chapter 18. Each of these structures consists of cardiac muscle modified enough in structure to differ in function from ordinary cardiac muscle. The specialty of ordinary cardiac muscle is contraction. In this, it is like all muscle, and like all muscle, ordinary cardiac muscle can also conduct impulses. But the conduction system structures are more highly specialized, both structurally and functionally, than ordinary cardiac muscle tissue. They are not contractile. Instead, they permit only generation or rapid conduction of an action potential through the heart.

The normal cardiac impulse that initiates mechanical contraction of the heart arises in the SA node (or pacemaker), located just below the atrial epicardium at its junction with the superior vena cava (Figure 19-1). Specialized pacemaker cells in the node possess an *intrinsic rhythm*. This means that without any stimulation by nerve impulses from the brain and cord, they themselves initiate impulses at regular intervals. Even if pacemaker cells are removed from the body and placed in a nutrient solution, completely separated from all nervous and hormonal control, they will continue to beat! In an intact living heart, of course, nervous and hormonal regulation does occur and the SA node generates a pace accordingly.

Each impulse generated at the SA node travels swiftly throughout the muscle fibers of both atria. An **interatrial bundle** of conducting fibers facilitates rapid conduction to the left atrium. Thus stimulated, the atria begin to contract. As the action potential enters the AV node by way of three **internodal bundles** of conducting fibers, its conduction slows markedly, thus allowing for complete contraction of both atrial chambers before the impulse reaches the ventricles. After passing slowly through the AV node, conduction velocity increases as the impulse is relayed through the AV bundle (bundle of His) into the ventricles. Here, right and left *bundle branches* and the Purkinje fibers in which they terminate conduct the impulses throughout the muscle of both ventricles, stimulating them to contract almost simultaneously.

Thus the SA node initiates each heartbeat and sets its pace—it is the heart's own natural *pacemaker* (Box 19-1). Under the influence of autonomic and endocrine control, the SA node will normally “discharge,” or “fire,” at an intrinsic rhythmical rate of 70 to 75 beats per minute under resting conditions. However, if for any reason the SA node loses its ability to generate an impulse, pacemaker activity will shift to another excitable component of the conduction system such as the AV node or the Purkinje fibers. Pacemakers other than the SA node are called *abnormal*, or **ectopic, pacemakers**. Although ectopic pacemakers fire rhythmically, their rate of discharge is generally much slower than that of the SA node. For example, a pulse of 40 to 60 beats per minute would result if the AV node were forced to assume pacemaker activity.

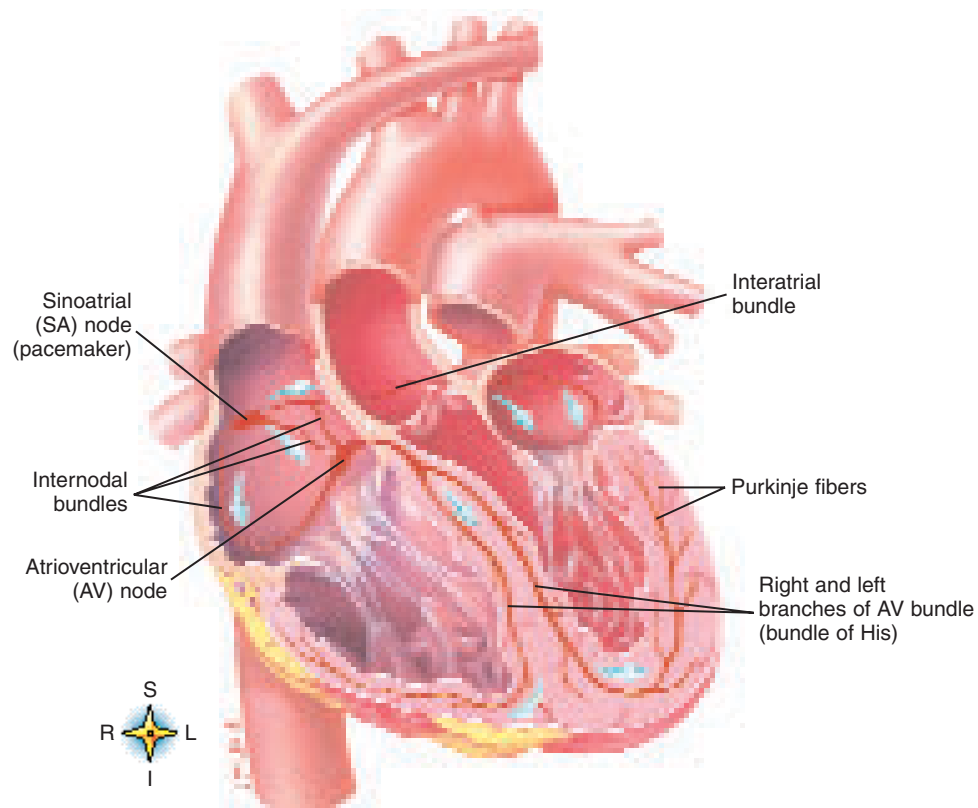


Figure 19-1 Conduction system of the heart. Specialized cardiac muscle cells in the wall of the heart rapidly initiate or conduct an electrical impulse throughout the myocardium. The signal is initiated by the SA node (pacemaker) and spreads to the rest of right atrial myocardium directly, to the left atrial myocardium by way of a bundle of interatrial conducting fibers, and to the AV node by way of three internodal bundles. The AV node then initiates a signal that is conducted through the ventricular myocardium by way of the AV (bundle of His) and Purkinje fibers.

Box 19-1

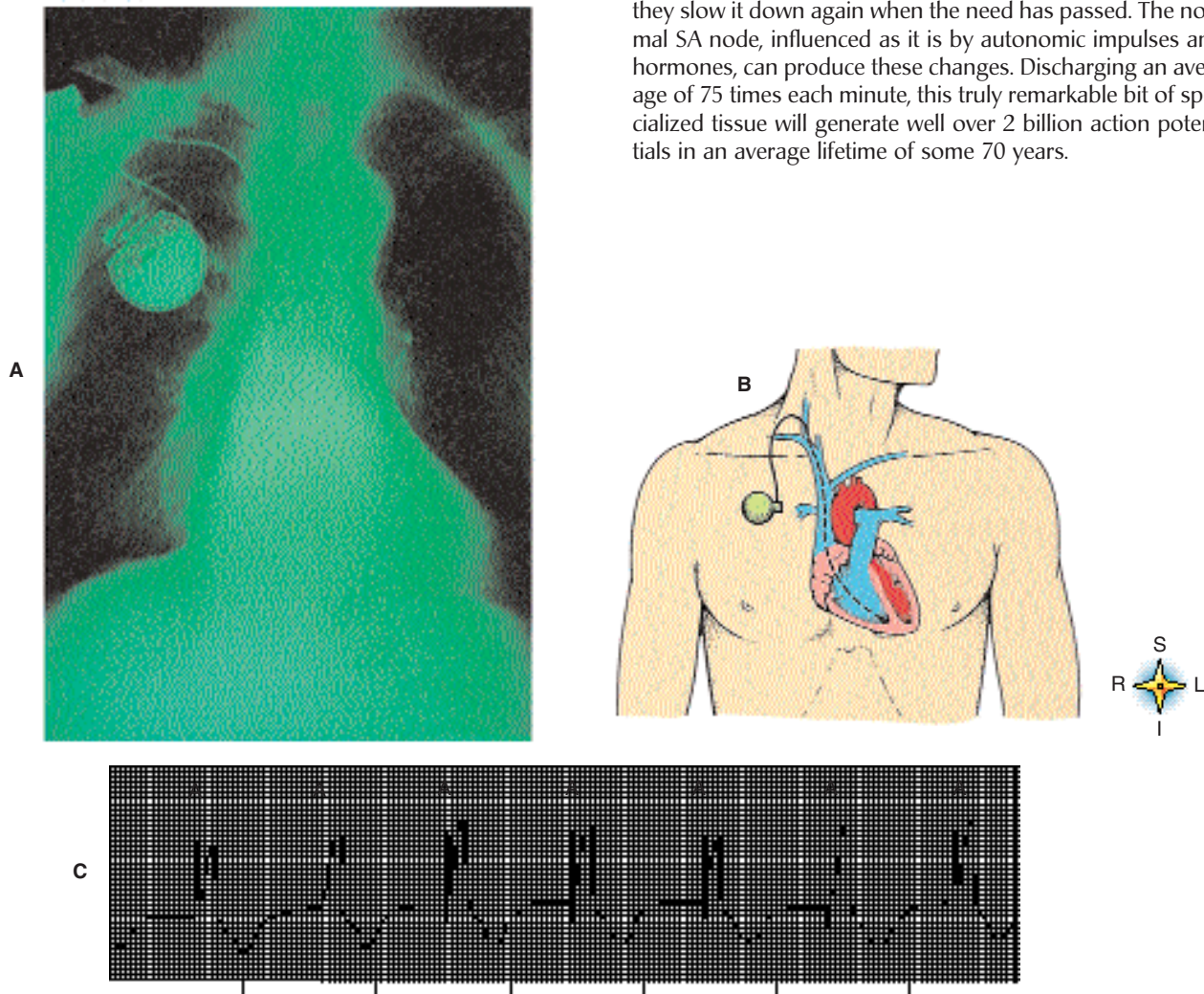
Artificial Cardiac Pacemakers

Everyone has heard about **artificial pacemakers**, devices that electrically stimulate the heart at a set rhythm (continuously discharging pacemakers) or those that fire only when the heart rate decreases below a preset minimum (demand pacemakers). They do an excellent job of maintaining a steady heart rate and of keeping many individuals with damaged hearts alive for many years. Hundreds of thousands of people currently have permanently implanted cardiac pacemakers.

Several types of artificial pacemakers have been designed to deliver an electrical stimulus to the heart muscle. The stimulus passes through electrodes that are sewn directly to the epicardium on the outer surface of the heart or are inserted by a catheter into a heart chamber, such as the right ventricle, and placed in contact with the endocardium. Modern pacemakers generate a stimulus that lasts from 0.08 to 2 msec and produces a very low current output.

One common method of inserting a permanent pacemaker is by the *transvenous approach*. In this procedure, a small incision is made just above the right clavicle and the electrode is threaded into the jugular vein and then advanced to the apex of the right ventricle. Figure A shows the battery-powered stimulus generator, which is placed in a pocket beneath the skin on the right side of the chest just below the clavicle. The proximal end of the electrical lead, or catheter, is then directed through the subcutaneous tissues and attached to the power pack. Figure B shows the tip of the electrical lead in the apex of the right ventricle. Figure C shows the ECG of an artificially paced heart. Notice the uniform, rhythmic “pacemaker spikes” that trigger each heartbeat.

Although life saving, these devices must be judged inferior to the heart’s own natural pacemaker. Why? Because they cannot speed up the heartbeat when necessary (for example, to make strenuous physical activity possible), nor can they slow it down again when the need has passed. The normal SA node, influenced as it is by autonomic impulses and hormones, can produce these changes. Discharging an average of 75 times each minute, this truly remarkable bit of specialized tissue will generate well over 2 billion action potentials in an average lifetime of some 70 years.



Artificial cardiac pacemaker. A, Battery-powered stimulus generator placed below the skin of the chest. B, The electrical lead extends from the stimulus generator into the right ventricle. C, Pacemaker spikes (A) characterize the ECG of an artificially paced heart.

ELECTROCARDIOGRAM (ECG)
Electrocardiography

Impulse conduction generates tiny electrical currents in the heart that spread through surrounding tissues to the surface of the body. This fact has great clinical importance. Why? Because from the skin, visible records of the heart's electrical activity can be made with an instrument called an *electrocardiograph*. Skilled interpretation of these records may sometimes make the difference between life and death.

The *electrocardiogram* (ECG or EKG) is a graphic record of the heart's electrical activity, its conduction of impulses. It is not a record of the heart's contractions but of the electrical events that precede them. To produce an electrocardiogram, electrodes of a recording voltmeter (electrocardiograph) are attached to the limbs and/or chest of the subject (Figure 19-2, A). Changes in voltage, which represent changes in the heart's electrical ac-

tivity, are observed as deflections of a line drawn on paper or traced on a video monitor.

Figure 19-3 explains the basic theory behind electrocardiography. To keep things simple, a single cardiac muscle fiber is shown with the two electrodes of a recording voltmeter nearby. Before the action potential reaches either electrode, there is no difference in charge between the electrodes and thus no change in voltage is recorded on the voltmeter graph (Figure 19-3, A). As an action potential reaches the first electrode, the external surface of the sarcolemma becomes relatively negative and so the voltmeter records a difference in charge between the two electrodes as an upward deflection of the pen on the recording chart (Figure 19-3, B). When the action potential also reaches the second electrode, the pen returns to the zero baseline because there is no difference in charge between the two electrodes (Figure 19-3, C). As the

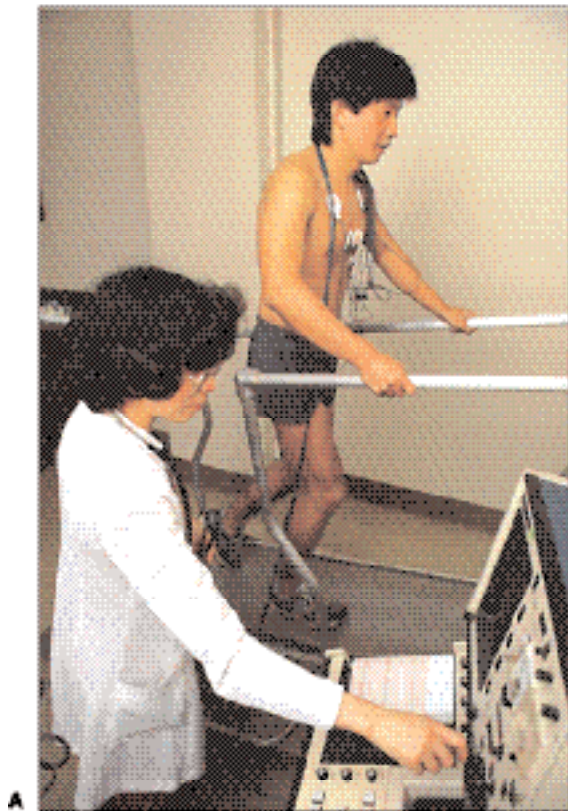
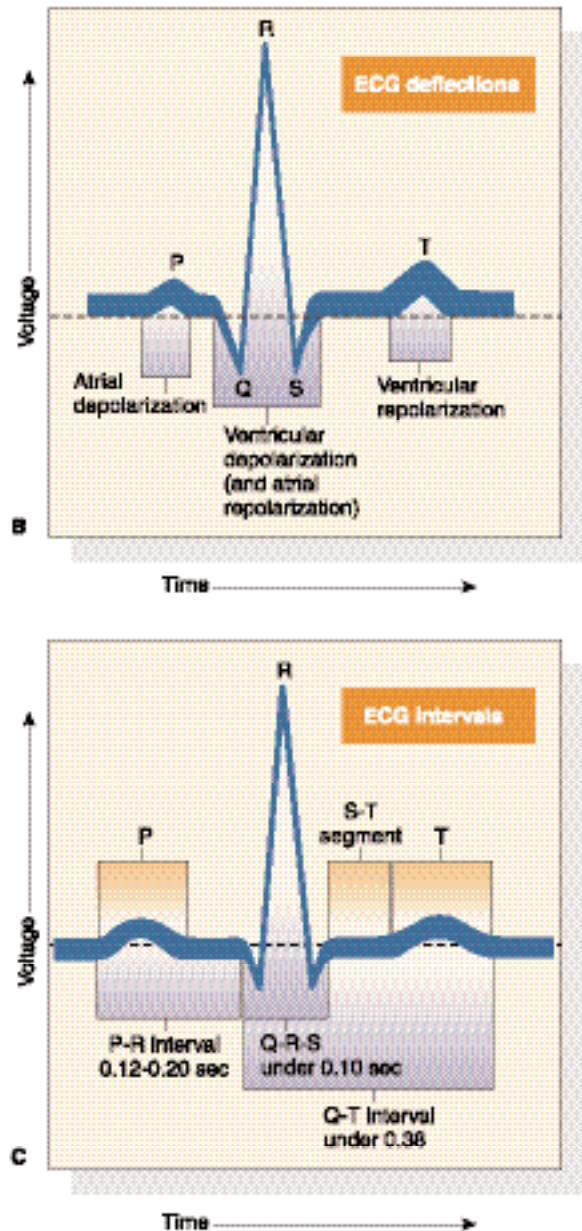


Figure 19-2 Electrocardiogram. A, A nurse monitors a patient's ECG as he exercises on a treadmill. B, Idealized ECG deflections represent depolarization and repolarization of cardiac muscle tissue. C, Principal ECG intervals between P, QRS, and T waves. Note that the P-R interval is measured from the start of the P wave to the start of the Q wave.



end of the action potential passes the first electrode, the sarcolemma is again relatively positive on its outer surface, causing the pen to again deflect away from the baseline. This time, because the direction of the negative and positive electrodes is reversed, the pen now deflects downward rather than upward (Figure 19-3, *D*). After the end of the action potential also passes the second electrode, the pen again returns to the zero baseline (Figure 19-3, *E*). In short, depolarization of cardiac muscle causes a deflection of the graphed line; repolarization causes a deflection in the opposite direction. Electrocardiography electrodes are normally quite some distance from myocardial tissue but, given the massive size of the myocardial syncytium, it should not be surprising that even cutaneous electrodes can detect changes in the heart's polarity.

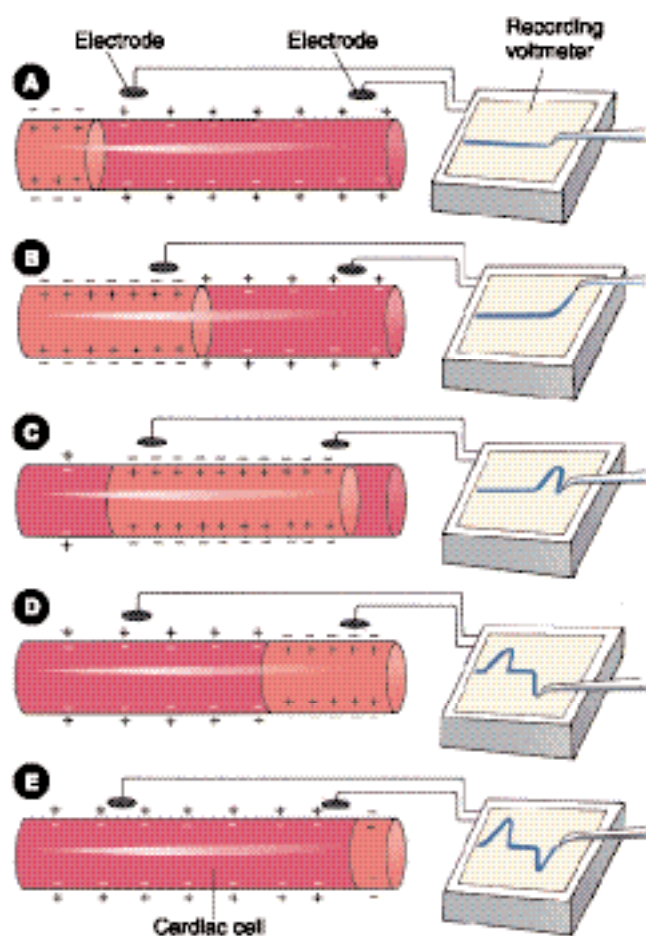


Figure 19-3 The basic theory of electrocardiography. A, A single cardiac muscle fiber at rest. There is no difference in charge between two electrodes of a recording voltmeter—so the pen remains at 0 millivolts, the baseline. B, An action potential reaches the first electrode and the external surface of the sarcolemma becomes relatively negative. The difference in charge between the two electrodes produces an upward deflection of the pen on the recording chart. C, The action potential then reaches the second electrode and the pen returns to the baseline because there is no difference in charge between the electrodes. D, As the end of the action potential passes the first electrode, the sarcolemma is again relatively positive on its outer surface, causing the pen to deflect downward. E, After the end of the action potential also passes the second electrode, there is no difference in charge and the pen again returns to the baseline.

ECG Waves

Because electrocardiography is far too complex a subject to explain fully here, normal ECG *deflection waves* and the ECG *intervals* between them shall be only briefly discussed. As shown in Figures 19-2, *B*, and 19-4, the normal ECG is composed of deflection waves called the *P wave*, *QRS complex*, and *T wave*. (The letters do not stand for any words but were chosen as an arbitrary sequence of the alphabet.)

P Wave. Briefly, the *P wave* represents depolarization of the atria. That is, the *P wave* is the deflection caused by the passage of an electrical impulse from the SA node through the musculature of both atria.

QRS Complex. The *QRS complex* represents depolarization of the ventricles. Depolarization of the ventricles is a complex process, involving depolarization of the interventricular septum and the subsequent spread of depolarization by the Purkinje fibers through the lateral ventricular walls. Rather than getting mired in a detailed explanation, let us simplify matters by stating that all three deflections of the *QRS complex* (*Q*, *R*, and *S*) represent the entire process of ventricular depolarization.

At the same time that the ventricles are depolarizing, the atria are repolarizing. As we explained earlier, we should expect to see a deflection that is opposite in direction to the *P wave* that represented depolarization. However, the massive ventricular depolarization that is occurring at the same time overshadows the voltage fluctuation produced by atrial repolarization. Thus, we can say that the *QRS complex* represents both ventricular depolarization and atrial repolarization.

T Wave. The *T wave* reflects repolarization of the ventricles. In atria, the first part of the myocardium to depolarize is the first to repolarize. In ventricles, on the other hand, the first part of the myocardium to depolarize is the last to repolarize. Thus ECG deflections for both depolarization and repolarization are in the same direction.

Sometimes, an additional *U wave* may be seen in the electrocardiogram. The *U wave*, when visible, appears as a tiny “hump” at the end of the *T wave*. The *U wave* results from repolarization of Purkinje fibers in the papillary muscle of the ventricular myocardium.

ECG Intervals

The principal ECG intervals between *P*, *QRS*, and *T* waves are shown in Figure 19-3, *C*. Measurement of these intervals can provide valuable information concerning the rate of conduction of an action potential through the heart (Box 19-2). Figure 19-4 summarizes the relationship between the electrical events of the myocardium and the ECG recordings.



1. List the principal structures of the heart's conduction system.
2. What are the three types of deflection waves seen in a typical ECG?
3. What event does each type of ECG wave represent?

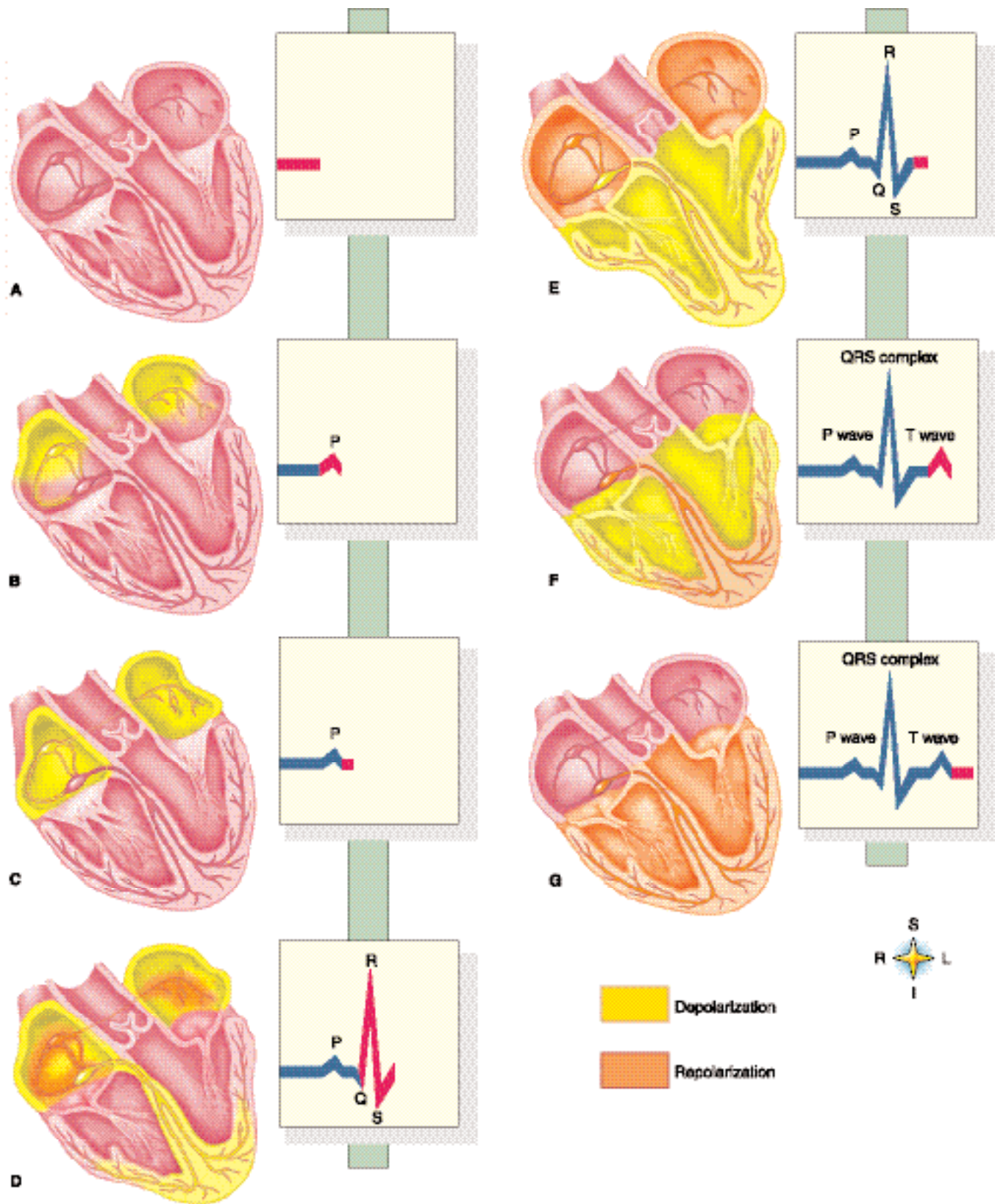


Figure 19-4 Events represented by the electrocardiogram (ECG). It is impossible to illustrate the invisible, dynamic events of heart conduction in a few cartoon panels or “snapshots,” but the sketches here give you an idea of what is happening in the heart as an ECG is recorded. **A**, The heart wall is completely relaxed, with no change in electrical activity, so the ECG remains constant. **B**, P wave occurs when the AV node and atrial walls depolarize. **C**, Atrial walls are completely depolarized and thus no change is recorded in the ECG. **D**, The QRS complex occurs as the atria repolarize and the ventricular walls depolarize. **E**, The atrial walls are now completely repolarized and the ventricular walls are now completely depolarized and thus no change is seen in the ECG. **F**, The T wave appears on the ECG when the ventricular walls repolarize. **G**, Once the ventricles are completely repolarized, we are back at the baseline of the ECG—essentially back where we began in part A of this diagram. Note that depolarization triggers contraction in the affected muscle tissue. Thus cardiac muscle contraction occurs *after* depolarization begins.

Box 19-2

Cardiac Dysrhythmia

Various conditions such as inflammation of the endocardium (endocarditis) or myocardial infarction (heart attack) can damage the heart's conduction system and thereby disturb the normal rhythmical beating of the heart (Figure A). The term **dysrhythmia** refers to an abnormality of heart rhythm.

One kind of dysrhythmia is called a **heart block**. In AV node block, impulses are blocked from getting through to the ventricular myocardium, resulting in the ventricles contracting at a much slower rate than normal. On an ECG, there may be a large interval between the P wave and the R peak of the QRS complex (Figure B). *Complete heart block* occurs when the P waves do not match up at all with the QRS complexes—as in an ECG that shows two or more P waves for every QRS complex. A physician may treat heart block by implanting in the heart an *artificial pacemaker* (see Box 19-1).

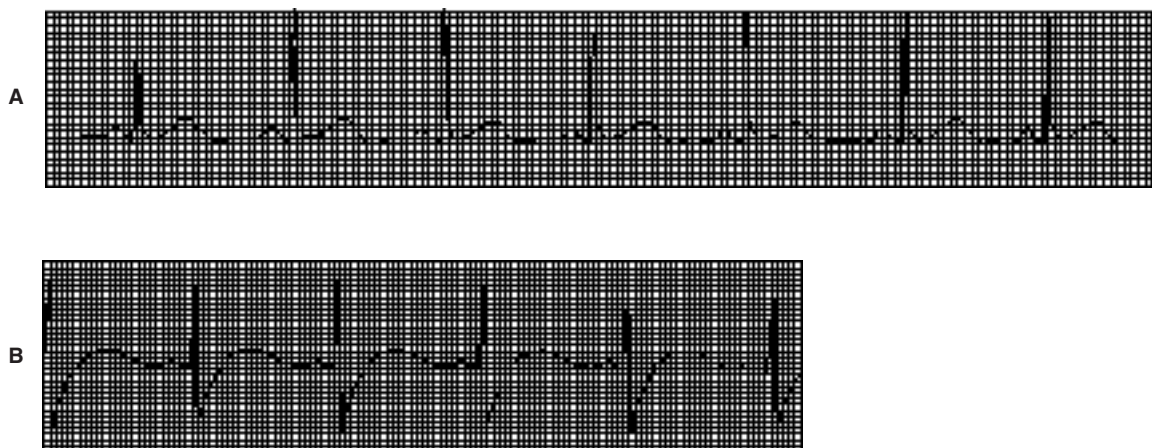
Bradycardia is a slow heart rhythm—below 60 beats per minute (Figure C). Slight bradycardia is normal during sleep and in conditioned athletes while they are awake (but at rest). Abnormal bradycardia can result from improper autonomic nervous control of the heart or from a damaged SA node. If the problem is severe, artificial pacemakers can be used to increase the heart rate by taking the place of the SA node.

Tachycardia is a very rapid heart rhythm—more than 100 beats per minute (Figure D). Tachycardia is normal during and after exercise and during the stress response. Abnormal tachycardia can result from improper autonomic control of the heart, blood loss or shock, the action of drugs and toxins, fever, and other factors.

Sinus dysrhythmia is a variation in heart rate during the breathing cycle. Typically, the rate increases during inspiration and decreases during expiration. The causes of sinus dysrhythmia are not clear. This phenomenon is common in young people and usually does not require treatment.

Premature contractions, or *extrasystoles*, are contractions that occur before the next expected contraction in a series of cardiac cycles. For example, *premature atrial contractions (PACs)* may occur shortly after the ventricles contract—seen as early P waves on the ECG (Figure E). Premature atrial contractions often occur with lack of sleep, too much caffeine or nicotine, alcoholism, or heart damage. Ventricular depolarizations that appear earlier than expected are called *premature ventricular contractions (PVCs)*. PVCs appear on the ECG as early, wide QRS complexes without a preceding related P wave. PVCs are caused by a variety of circumstances that include stress, electrolyte imbalance, acidosis, hypoxemia, ventricular enlargement, or drug reactions. Occasional PVCs are not clinically significant in otherwise healthy individuals. However, in people with heart disease they may reduce cardiac output.

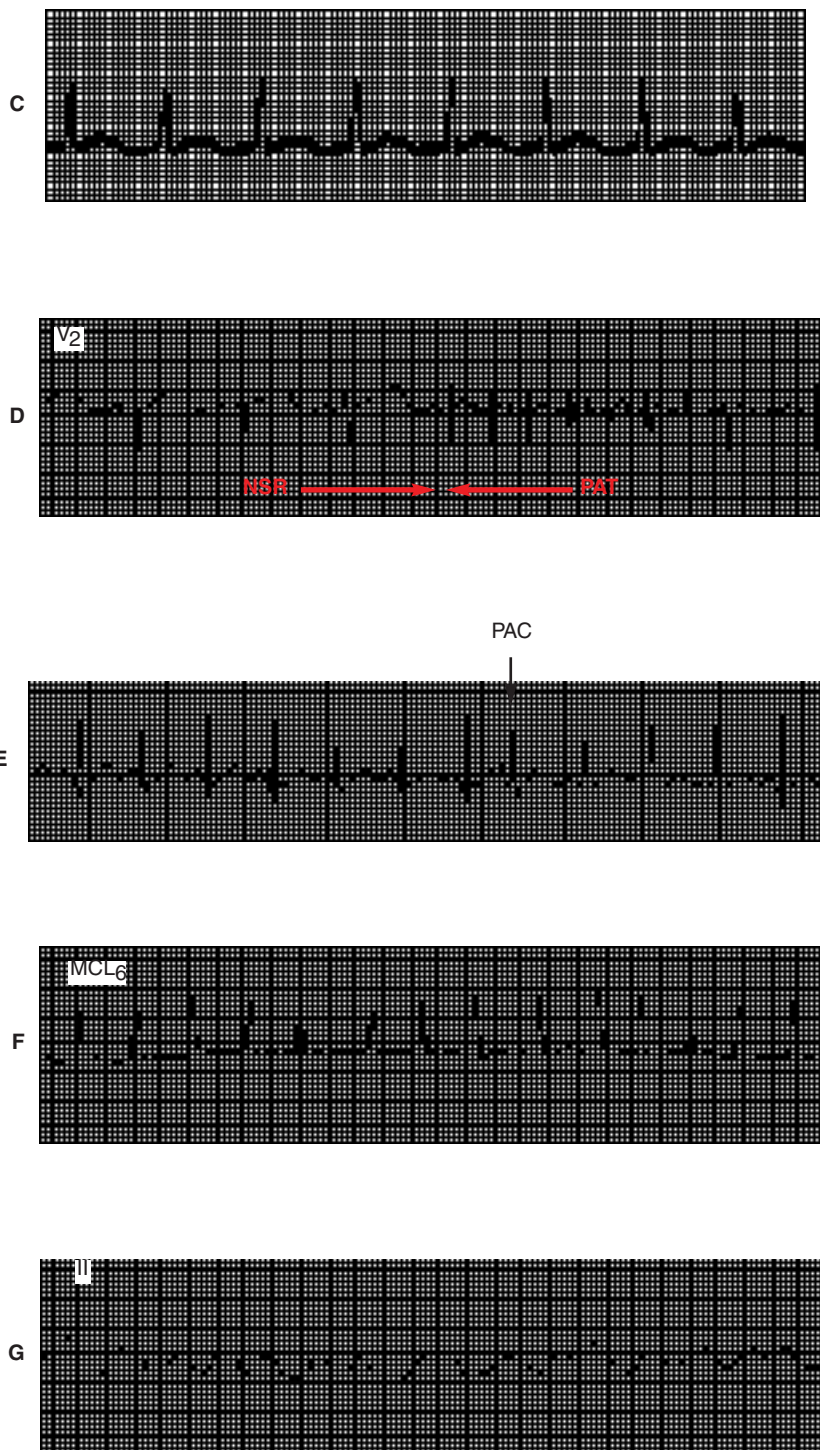
Frequent premature contractions can lead to fibrillation, a condition in which cardiac muscle fibers contract out of step with each other. This event can be seen in an ECG as the absence of regular P waves or abnormal QRS and T waves. In fibrillation, the affected heart chambers do not effectively pump blood. *Atrial fibrillation* occurs commonly in mitral stenosis, rheumatic heart disease, and infarction of the atrial myocardium (Figure F). This condition can be treated with drugs such as digoxin (a *digitalis* preparation) or by *defibrillation*—application of electrical shock to force cardiac muscle fibers to contract in unison. *Ventricular fibrillation* is an immediately life-threatening condition in which the lack of ventricular pumping suddenly stops the flow of blood to vital tissues (Figure G). Unless ventricular fibrillation is corrected immediately by defibrillation or some other method, death may occur within minutes.



ECG strip chart recording. A, Normal ECG. B, AV node block. Very slow ventricular contraction (25 to 45 beats/min at rest); P waves widely separated from peaks of QRS complexes.

Box 19-2

Cardiac Dysrhythmia—cont'd



ECG strip chart recording—cont'd. C, Bradycardia. Slow heart rhythm (less than 60 beats/min); no disruption of normal rhythm pattern. D, Tachycardia. Rapid heart rhythm (greater than 100 beats/min); no disruption of normal rhythm pattern. *NSR*, Normal sinus rhythm; *PAT*, paroxysmal (sudden) atrial tachycardia. E, Premature atrial contraction (PAC). Unexpected, early P wave that is different from normal P waves; PR interval may be shorter or longer than normal; normal QRS complex; more than 6 PACs per minute may precede atrial fibrillation. F, Atrial fibrillation. Irregular, rapid atrial depolarizations; P wave rapid (greater than 300/min) with irregular QRS complexes (150 to 170 beats/min). G, Ventricular fibrillation. Complete disruption of normal heart rhythm.

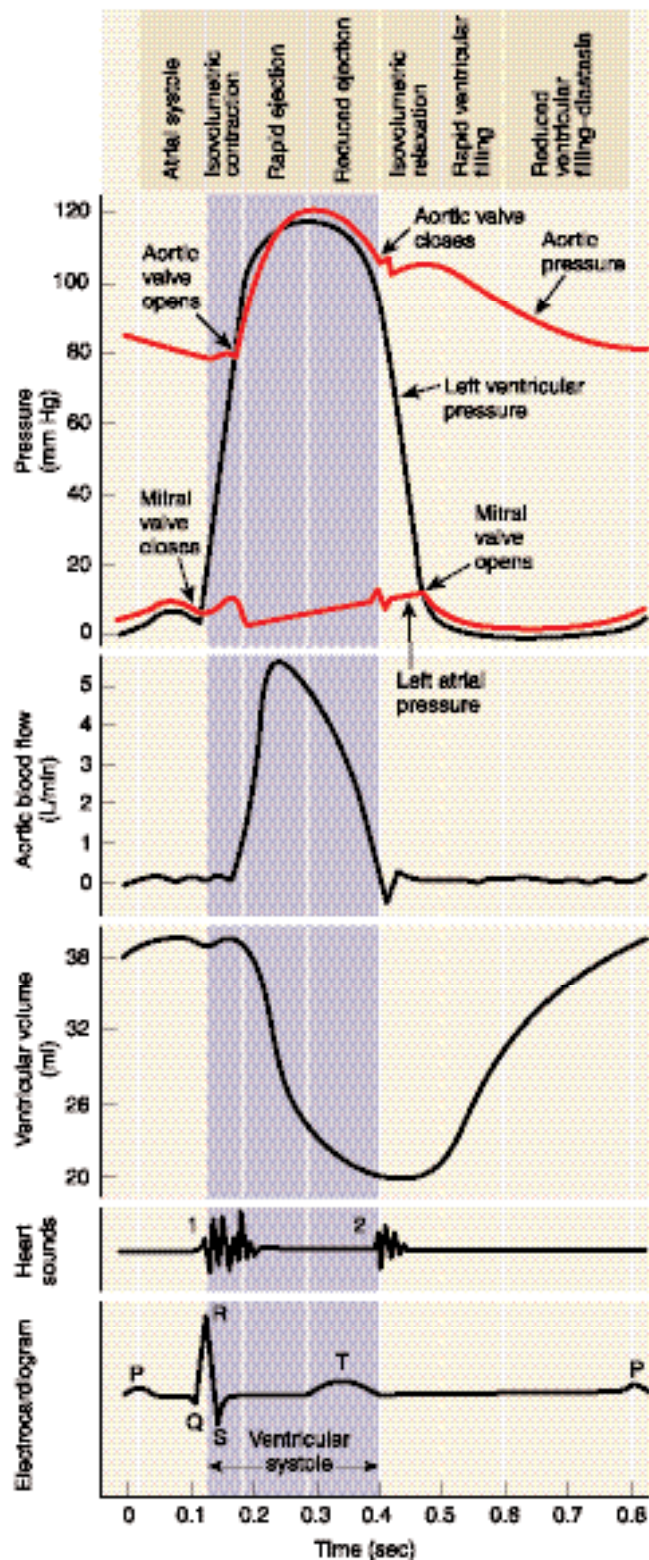


Figure 19-5 Composite chart of heart function. This chart is a composite of several diagrams of heart function (cardiac pumping cycle, blood pressure, blood flow, volume, heart sounds, and ECG), all adjusted to the same time scale. Although it appears daunting at first glance, you will find it a valuable reference tool as you proceed through this chapter.

CARDIAC CYCLE

The term *cardiac cycle* means a complete heartbeat, or pumping cycle, consisting of contraction (*systole*) and relaxation (*diastole*) of both atria and both ventricles. The two atria contract simultaneously. Then, as the atria relax, the two ventricles contract and relax, instead of the entire heart contracting as a unit. This gives a kind of pumping action to the movements of the heart. The atria remain relaxed during part of the ventricular relaxation and then start the cycle over again. The cycle as a whole is often divided into time intervals for discussion and study. The following sections describe several of the important events of the cardiac cycle. As you read through these sections, refer frequently to Figure 19-5, which is a composite chart that graphically illustrates and integrates changes in pressure gradients in the left atrium, left ventricle, and aorta with ECG and heart sound recordings. Aortic blood flow and changes in ventricular volume are also shown. Refer also to Figure 19-6, which shows the major phases of the cardiac cycle.

Atrial Systole

The contracting force of the atria completes the emptying of blood out of the atria into the ventricles. Atrioventricular (or cuspid) valves are necessarily open during this phase; the ventricles are relaxed and filling with blood. The semilunar valves are closed so that blood does not reenter from the pulmonary artery or aorta. This period of the cycle begins with the P wave of the ECG. Passage of the electrical wave of depolarization is then followed almost immediately by actual contraction of the atrial musculature.

Isovolumetric Ventricular Contraction

Iso is a combining form denoting equality or uniformity and *volumetric* denotes measurement of volume. Thus *isovolumetric* is a term that means “having the same measured volume.” During the brief period of isovolumetric ventricular contraction, that is, between the start of ventricular systole and the opening of the semilunar valves, ventricular volume remains constant, or uniform, as the pressure increases rapidly. The onset of ventricular systole coincides with the R wave of the ECG and the appearance of the first heart sound.

Ejection

The semilunar valves open and blood is ejected from the heart when the pressure gradient in the ventricles exceeds the pressure in the pulmonary artery and aorta. An initial, shorter phase, called *rapid ejection*, is characterized by a marked increase in ventricular and aortic pressure and in aortic blood flow (Box 19-3). The T wave of the ECG appears during the later, longer phase of *reduced ejection* (characterized by a less abrupt decrease in ventricular volume). A considerable quantity of blood, called the **residual volume**, normally remains in the ventricles at the end of the ejection period. In heart failure the residual volume remaining in the ventricles may greatly exceed that ejected during systole.

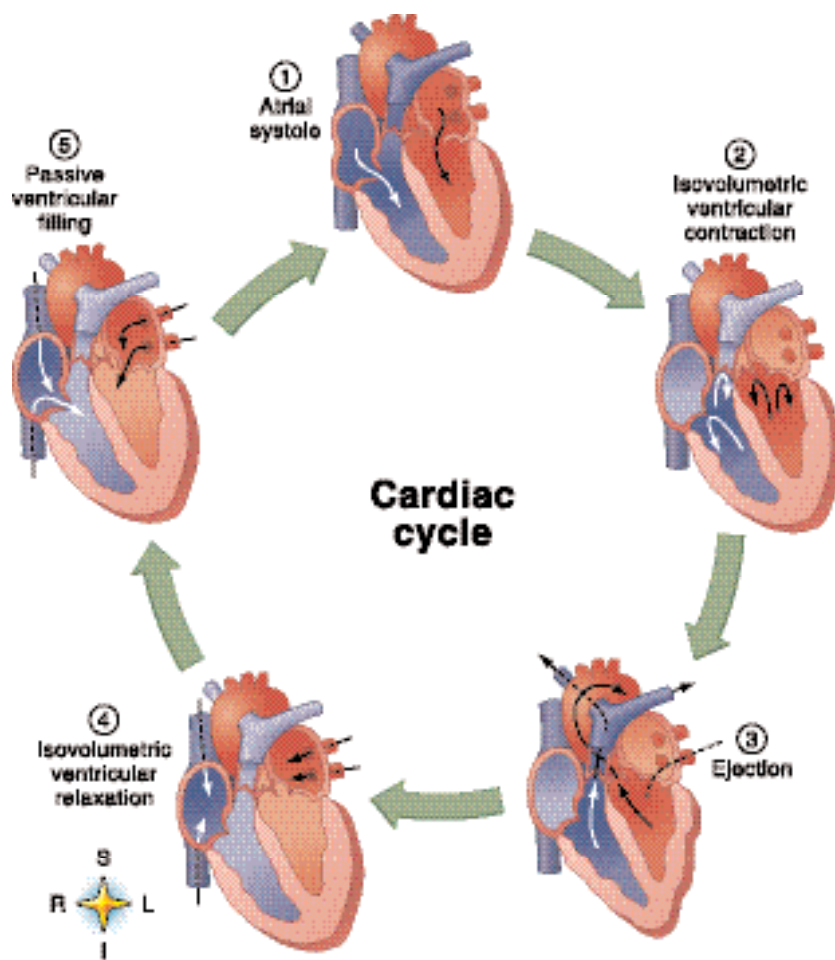


Figure 19-6 The cardiac cycle. The five steps of the heart’s pumping cycle described in the text are shown as a series of changes in the heart wall and valves.

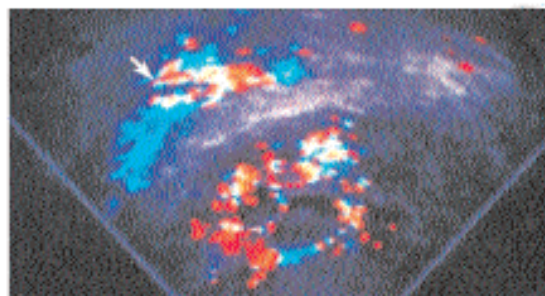


Box 19-3 DIAGNOSTIC STUDY

Doppler Ultrasonography

Doppler ultrasonography uses ultrasound to record the direction of blood flow through the heart. These sound waves are reflected off red blood cells as they pass through the heart, allowing the velocity of blood to be calculated as it travels through the heart chambers.

Color Doppler mapping is a variation that converts recorded flow frequencies into different colors. These color images are then superimposed on M-mode or 2-D echocardiograms, allowing more detailed evaluation of disorders (see Figure).



Color Doppler mapping.

Isovolumetric Ventricular Relaxation

Ventricular diastole, or relaxation, begins with this period of the cardiac cycle. It is the period between closure of the semilunar valves and opening of the atrioventricular valves. At the end of ventricular ejection the semilunar valves close

so that blood cannot reenter the ventricular chambers from the great vessels. The atrioventricular valves do not open until the pressure in the atrial chambers increases above that in the relaxing ventricles. The result is a dramatic fall in intraventricular pressure but no change in volume. Both sets of

valves are closed, and the ventricles are relaxing. The second heart sound is heard during this period.

Passive Ventricular Filling

Return of venous blood increases intraatrial pressure until the atrioventricular valves are forced open and blood rushes into the relaxing ventricles. The rapid influx lasts about 0.1 second and results in a dramatic increase in ventricular volume. The term *diastasis* is often used to describe a later, longer period of slow ventricular filling at the end of ventricular diastole. The abrupt inflow of blood that occurred immediately after opening of the atrioventricular valves is followed by a slow but continuous flow of venous blood into the atria and then through the open atrioventricular valves into the ventricles. Diastasis lasts about 0.2 second and is characterized by a gradual increase in ventricular pressure and volume.

HEART SOUNDS

The heart makes certain typical sounds during each cardiac cycle that are described as sounding like “lubb-dupp” through a stethoscope. The first, or systolic, sound is caused primarily by the contraction of the ventricles and also by vibrations of the closing atrioventricular, or cuspid, valves. It is longer and lower than the second, or diastolic, sound, which is short, sharp, and is caused by vibrations of the closing semilunar valves (see Figure 19-5).

Heart sounds have clinical significance, since they give information about the valves of the heart. Any variation from normal in the sounds indicates imperfect functioning of the valves. *Heart murmur* is one type of abnormal sound heard frequently. It may signify incomplete closing of the valves (valvular insufficiency) or stenosis (constriction, or narrowing) of them.



1. Using Figure 19-6, describe the major events of the cardiac cycle.
2. As the ventricles contract, their volume remains constant for a period of time. Can you explain why the volume does not begin to decrease immediately?

PRIMARY PRINCIPLE OF CIRCULATION

Blood circulates for the same reason that any fluid flows—whether it is water in a river, in a garden hose, or fluid in hospital tubing, or blood in vessels. A fluid flows because a pressure gradient exists between different parts of its volume (Figure 19-7).

This primary fluid flow principle derives from Newton’s first and second laws of motion. In essence, these laws state the following principles:

1. A fluid does not flow when the pressure is the same in all parts of it

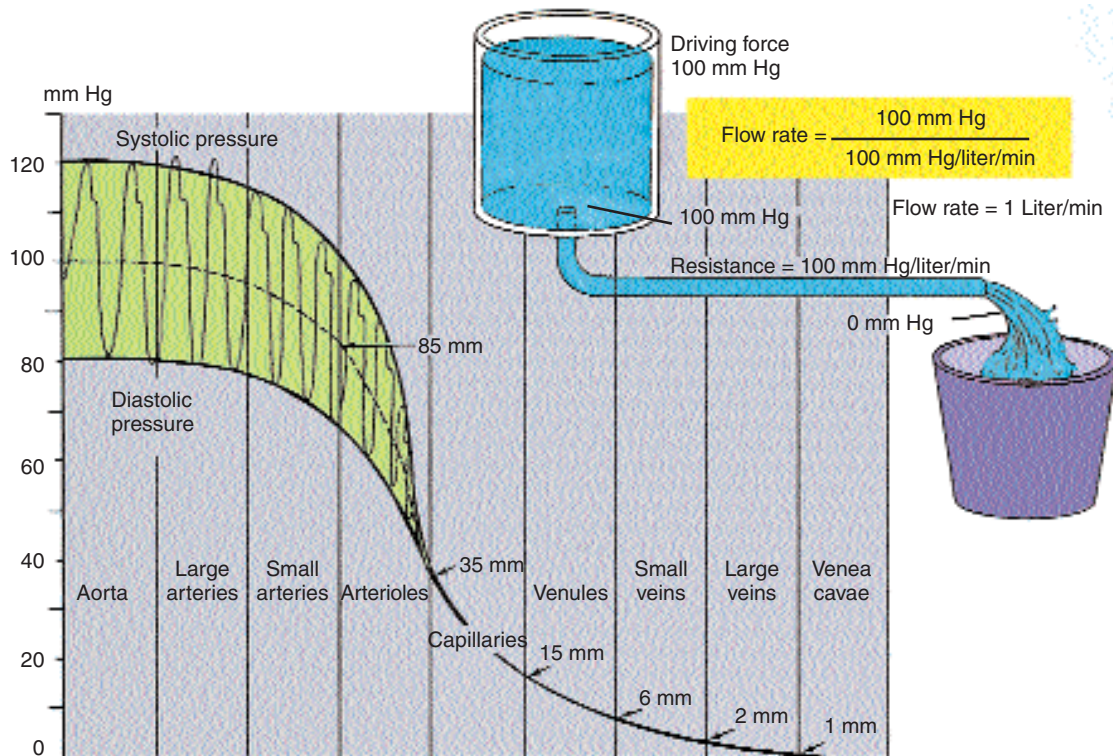


Figure 19-7 The primary principle of circulation. Fluid always travels from an area of high pressure to an area of low pressure. Water flows from an area of high pressure in the tank (100 mm Hg) toward the area of low pressure above the bucket (0 mm Hg). Blood tends to move from an area of high average pressure at the beginning of the aorta (100 mm Hg) toward the area of lowest pressure at the end of the venae cavae (0 mm Hg). Blood flow between any two points in the circulatory system can always be predicted by the pressure gradient.

2. A fluid flows only when its pressure is higher in one area than in another, and it flows always from its higher pressure area toward its lower pressure area

In brief, then, the primary principle about circulation is this: blood circulates from the left ventricle and returns to the right atrium of the heart because a blood pressure gradient exists between these two structures. By blood pressure gradient, we mean the difference between the blood pressure in one structure and the blood pressure in another. For example, a typical normal blood pressure in the aorta, as the left ventricle contracts pumping blood into it, is 120 mm Hg; as the left ventricle relaxes, it decreases to 80 mm Hg. The mean, or average, blood pressure therefore in the aorta in this instance is 100 mm Hg.

Figure 19-7 shows the systolic and diastolic pressures in the arterial system and illustrates the progressive fall in pressure to 0 mm Hg by the time blood reaches the venae cavae and right atrium. The progressive fall in pressure as blood passes through the circulatory system is directly related to resistance. Resistance to blood flow in the aorta is almost zero. Although the pumping action of the heart causes fluctuations in aortic blood pressure (systolic 120 mm Hg; diastolic 80 mm Hg), the mean pressure remains almost constant, dropping perhaps only 1 or 2 mm Hg. The greatest drop in pressure (about 50 mm Hg) occurs across the arterioles because they present the greatest resistance to blood flow.

$P_1 - P_2$ is often used to stand for a pressure gradient, with P_1 the symbol for the higher pressure and P_2 the symbol for the lower pressure. For example, blood enters the arterioles at 85 mm Hg and leaves at 35 mm Hg. Which is P_1 ? P_2 ? What

is the blood pressure gradient? It would cause blood to flow from the arterioles into capillaries.

ARTERIAL BLOOD PRESSURE

According to the primary principle of circulation, high pressure in the arteries must be maintained to keep blood flowing through the cardiovascular system. The chief determinant of arterial blood pressure is the volume of blood in the arteries. Arterial blood volume is directly proportional to arterial pressure. This means that an increase in arterial blood volume tends to increase arterial pressure, and, conversely, a decrease in arterial volume tends to decrease arterial pressure.

Many factors determine arterial pressure through their influence on arterial volume. Two of the most important—*cardiac output* and *peripheral resistance*—are directly proportional to blood volume (Figure 19-8).

CARDIAC OUTPUT

Cardiac output (CO) is determined by the volume of blood pumped out of the ventricles by each beat (*stroke volume* or SV) and by heart rate (HR). Because contraction of the heart is called *systole*, the volume of blood pumped by one contraction is known as *systolic discharge*. Stroke volume means the same thing, the amount of blood pumped by one stroke (contraction) of the ventricle.

Stroke volume, or volume pumped per heartbeat, is one of two major factors that determine CO. CO can be computed by the following simple equation:

$$SV \text{ (volume/beat)} \times HR \text{ (beat/min)} = CO \text{ (volume/min)}$$

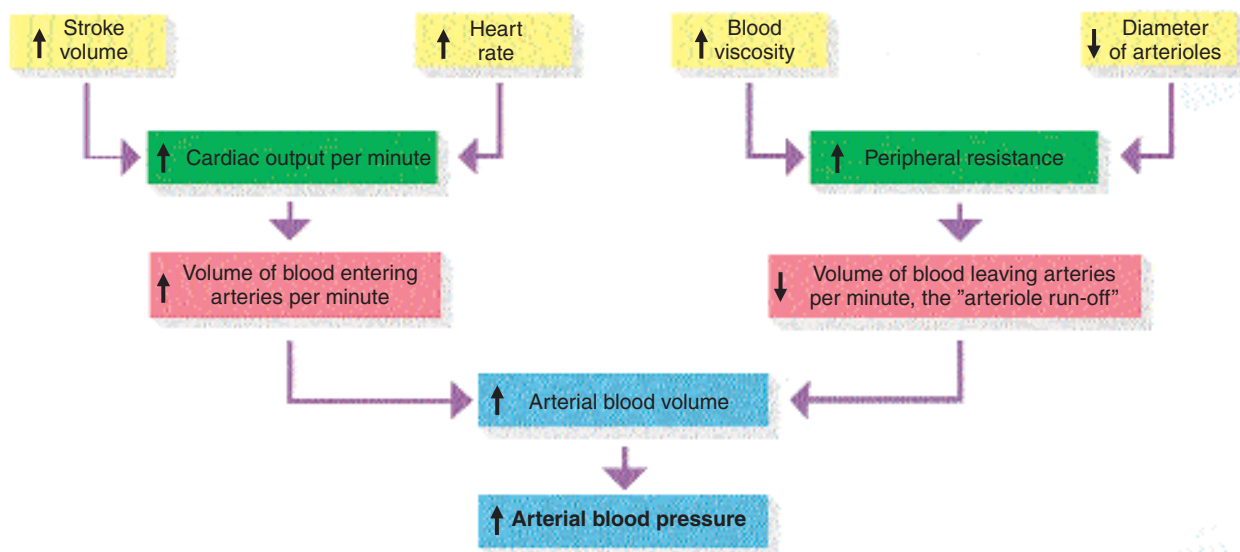


Figure 19-8 Relationship between arterial blood volume and blood pressure. Arterial blood pressure is directly proportional to arterial blood volume. Cardiac output (CO) and peripheral resistance (PR) are directly proportional to arterial blood volume, but for opposite reasons: CO affects blood *entering* the arteries and PR affects blood *leaving* the arteries. If cardiac output increases, the amount of blood entering the arteries increases and tends to increase the volume of blood in the arteries. If peripheral resistance increases, it decreases the amount of blood leaving the arteries, which tends to increase the amount of blood left in them. Thus an increase in either CO or PR results in an increase in arterial blood volume, which increases arterial blood pressure.

Thus the greater the stroke volume, the greater the CO (but only if the heart rate remains constant). In practice, computing the CO is far from simple. It requires introducing a catheter into the right side of the heart (cardiac catheterization) and solving a computation known as *Fick's formula*.

Because the heart's rate and stroke volume determine its output, anything that changes the rate of the heartbeat or its stroke volume tends to change CO, arterial blood volume, and blood pressure in the same direction. In other words, anything that makes the heart beat faster or anything that makes it beat stronger (increases its stroke volume) *tends* to increase CO and therefore arterial blood volume and pressure. Conversely, anything that causes the heart to beat more slowly or more weakly tends to decrease CO, arterial volume, and blood pressure. But do not overlook the word *tends* in the preceding sentences. A change in heart rate or stroke volume does not always change the heart's output, or the amount of blood in the arteries, or the blood pressure. To see whether this is true, do the following simple arithmetic, using the simple formula for computing CO. Assume a normal rate of 72 beats per minute and a normal stroke volume of 70 ml. Next, suppose the rate drops to 60 and the stroke volume increases to 100. Does the decrease in heart rate actually cause a decrease in CO in this case? Clearly not—the CO increases. Do you think it is valid, however, to say that a slower rate tends to decrease the heart's output? By itself, without any change in any other factor, would not a slowing of the heartbeat cause CO volume, arterial volume, and blood pressure to fall?

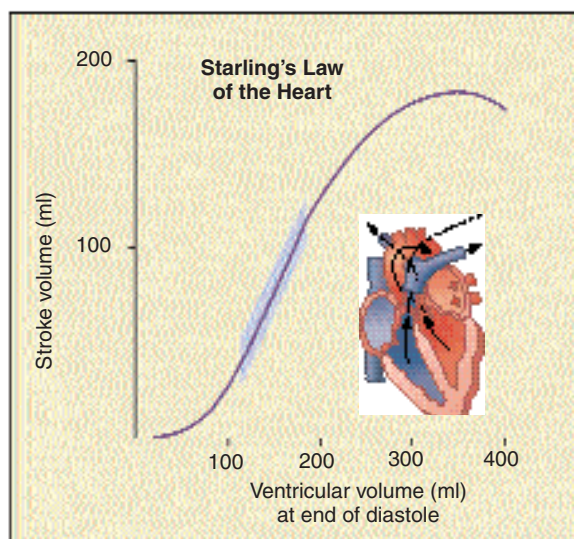


Figure 19-9 Starling's Law of the Heart. This curve represents the relationship between the stroke volume and the ventricular volume at the end of diastole. The range of values observed in a typical heart is shaded. Notice that if the ventricle has an abnormally large volume at the end of diastole (*far right portion of the curve*), the stroke volume cannot compensate.

Factors That Affect Stroke Volume

Mechanical, neural, and chemical factors regulate the strength of the heartbeat and therefore its stroke volume. One mechanical factor that helps determine stroke volume is the length of myocardial fibers at the beginning of ventricular contraction.

Many years ago, an English physiologist named Ernest Starling described a principle that later became known as **Starling's Law of the Heart**. In this principle he stated the factor he had observed as the main regulator of heartbeat strength in experiments performed on denervated animal hearts. Starling's Law of the Heart is this: within limits, the longer, or more stretched, the heart fibers at the beginning of contraction, the stronger is their contraction. Compare this concept with the length-tension relationship in skeletal muscle described in Chapter 11 (p. 325).

The factor determining how stretched the animal hearts were at the beginning of contractions was, as you might deduce, the amount of blood in the hearts at the end of diastole. The more blood returned to the hearts per minute, the more stretched were their fibers, the stronger were their contractions, and the larger was the volume of blood they ejected with each contraction. If, however, too much blood stretched the hearts beyond a certain critical point, they seemed to lose their elasticity. They then contracted less vigorously, similar to how a band of elastic, stretched too much, rebounds with less force (Figure 19-9).

Although Starling's Law of the Heart was first described in animal experiments, most physiologists agree that it operates in humans as a major regulator of stroke volume under ordinary conditions. Operation of Starling's Law of the Heart ensures that increased amounts of blood returned to the heart will be pumped out of it. It automatically adjusts CO to venous return under usual conditions. Factors that influence the amount of blood returned to the heart—venous return—are discussed in a later section.

Factors That Affect Heart Rate

Although the sinoatrial node normally initiates each heartbeat, the rate it sets is not an unalterable one. Various factors can and do change the rate of the heartbeat. One major modifier of sinoatrial node activity—and therefore of the heart rate—is the ratio of sympathetic and parasympathetic impulses conducted to the node per minute. Autonomic control of heart rate is the result of opposing influences between parasympathetic (chiefly vagus) and sympathetic (cardiac nerve) stimulation. The results of parasympathetic stimulation on the heart are inhibitory and are mediated by vagal release of acetylcholine, whereas sympathetic (stimulatory) effects result from the release of norepinephrine at the distal end of the cardiac nerve.

Cardiac Pressoreflexes. Receptors sensitive to changes in pressure (baroreceptors) are located in two places near the heart (Figure 19-10). Called the *aortic baroreceptors* and *carotid baroreceptors*, they send afferent nerve fibers to cardiac

control centers in the medulla oblongata. These stretch receptors, located in the aorta and carotid sinus, constitute a very important heart rate control mechanism because of their effect on the autonomic cardiac control centers—and therefore on parasympathetic and sympathetic outflow. Baroreceptors operate with integrators in the cardiac control centers in negative feedback loops called *pressoreflexes* or *baroreflexes* that oppose changes in pressure by adjusting heart rate.

Carotid Sinus Reflex. The carotid sinus is a small dilation at the beginning of the internal carotid artery just above the branching of the common carotid artery to form the internal and external carotid arteries (see Figure 19-10). The sinus lies just under the sternocleidomastoid muscle at the level of the upper margin of the thyroid cartilage. Sensory (afferent) fibers from carotid sinus baroreceptors (pressure sensors) run through the carotid sinus nerve (of Hering) and on through the glossopharyngeal (or ninth cranial) nerve. These nerves relay feedback information to an integrator area of the medulla called the *cardiac control center*. If the integrators in the cardiac control center detect an increase in blood pressure above the set point, then a correction signal is sent to the SA node via efferent parasympathetic fibers in the vagus (tenth cranial) nerve. Acetylcholine released by vagal fibers decreases the rate of SA node firing, thus decreasing the heart rate back toward the set point. The vagus is said to act as a “brake” on the heart—a situation called *vagal inhibition*. Figure 19-11 summarizes this negative feedback loop.

Aortic Reflex. Sensory (afferent) nerve fibers also extend from baroreceptors located in the wall of the arch of the aorta through the aortic nerve and then through the vagus (tenth cranial) nerve to terminate in the cardiac control center of the medulla (see Figure 19-10).

If blood pressure within the aorta or carotid sinus suddenly increases beyond the set point, it stimulates the aortic or carotid baroreceptors, as shown in Figure 19-11. Stimulation of these stretch receptors causes the cardiac control center to increase vagal inhibition, thus slowing the heart and returning blood pressure back toward the normal set point. A decrease in aortic or carotid blood pressure usually allows some acceleration of the heart via correction signals through the cardiac nerve. More details of pressoreflex activity are included later in the chapter as part of a mechanism that tends to maintain or restore homeostasis of arterial blood pressure.

Other Reflexes That Influence Heart Rate. Reflexes involving such important factors as emotions, exercise, hormones, blood temperature, pain, and stimulation of various exteroceptors also influence heart rate. Anxiety, fear, and anger often make the heart beat faster. Grief, in contrast, tends to slow it. Emotions produce changes in the heart rate through the influence of impulses from the “higher centers” in the cerebrum via the hypothalamus. Such impulses can influence activity of the cardiac control centers.

In exercise the heart normally accelerates. The mechanism is not definitely known, but it is thought to include impulses from the cerebrum through the hypothalamus to the cardiac center. Epinephrine is the hormone most noted as a cardiac accelerator.

Increased blood temperature or stimulation of skin heat receptors tends to increase the heart rate, and decreased blood temperature or stimulation of skin cold receptors tends to slow it. Sudden, intense stimulation of pain receptors in such visceral structures as the gallbladder, ureters, or intestines can result in such slowing of the heart that fainting may result.

Reflexive increases in heart rate often result from an increase in sympathetic stimulation of the heart. Sympathetic impulses originate in the cardiac control center of the medulla and reach the heart via sympathetic fibers (contained in the middle, superior, and inferior cardiac nerves). Norepinephrine released as a result of sympathetic stimulation increases heart rate and strength of cardiac muscle contraction.

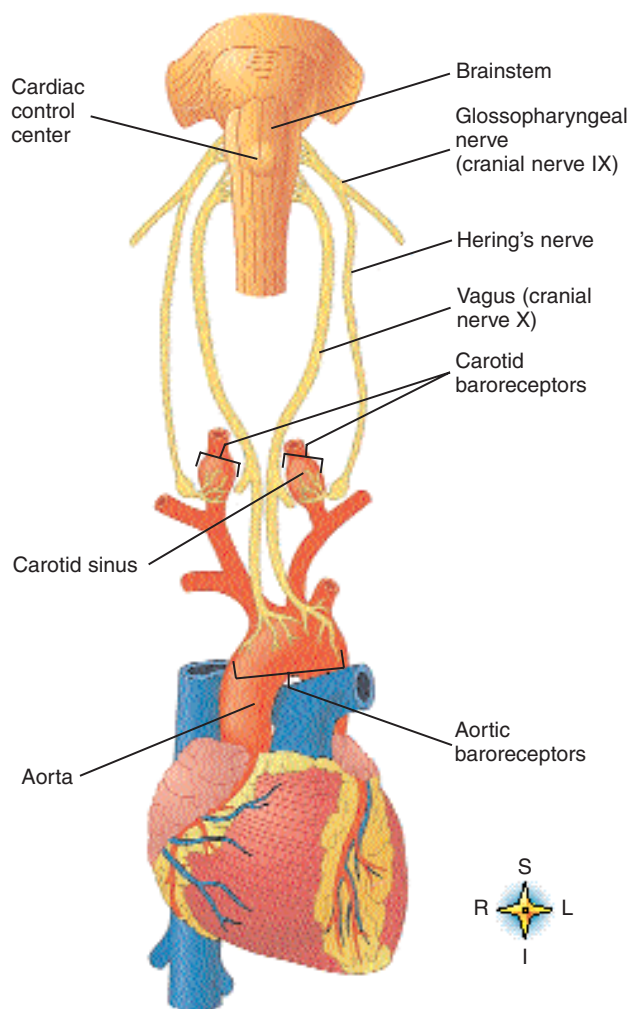


Figure 19-10 Cardiac baroreceptors and the pressoreflexes. Location of aortic and carotid baroreceptors and the sensory nerves that carry feedback information about blood pressure back to the central nervous system.



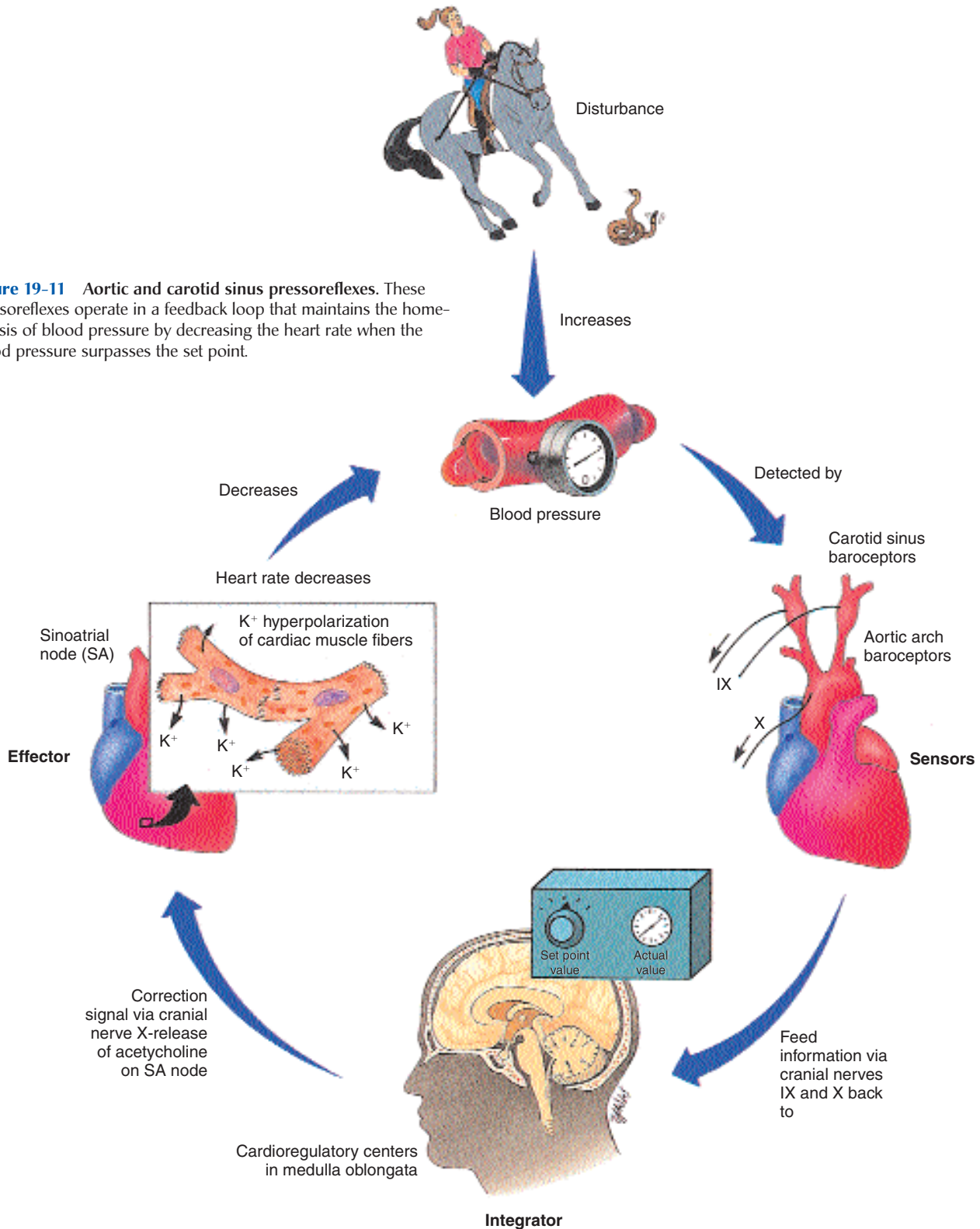
1. State the primary principle of circulation.
2. Relate stroke volume and heart rate to cardiac output.
3. If the amount of blood returned to the heart increases, what happens to the stroke volume? What principle explains this?
4. How does the body use a pressoreflex to counteract an abnormal increase in heart rate? An abnormal decrease in heart rate?

PERIPHERAL RESISTANCE

How Resistance Influences Blood Pressure

Peripheral resistance helps determine arterial blood pressure. Specifically, arterial blood pressure tends to vary directly with peripheral resistance. Peripheral resistance means the resistance to blood flow imposed by the force of friction between blood and the walls of its vessels. Friction develops

Figure 19-11 Aortic and carotid sinus pressoreflexes. These pressoreflexes operate in a feedback loop that maintains the homeostasis of blood pressure by decreasing the heart rate when the blood pressure surpasses the set point.



partly because of a characteristic of blood—its *viscosity*, or stickiness—and partly from the small diameter of arterioles and capillaries. The resistance offered by arterioles, in particular, accounts for almost half of the total resistance in systemic circulation. The muscular coat with which arterioles are vested allows them to constrict or dilate and thus change the amount of resistance to blood flow. Peripheral resistance helps determine arterial pressure by controlling the rate of “arteriole runoff,” the amount of blood that runs out of the arteries into the arterioles. The greater the resistance, the less the arteriole runoff, or outflow, tends to be—and therefore the more blood left in the arteries, the higher the arterial pressure tends to be.

Blood viscosity stems mainly from the red blood cells but also partly from the protein molecules present in blood. An increase in either blood protein concentration or red blood cell count tends to increase viscosity, and a decrease in either tends to decrease it (Box 19-4).

Vasomotor Control Mechanism


Blood distribution patterns, as well as blood pressure, can be influenced by factors that control changes in the diameter of arterioles. Such factors might be said to constitute the vasomotor control mechanism. Like most physiological control mechanisms, it consists of many parts. An area in the medulla called the *vasomotor center*, or *vasoconstrictor center*, will, when stimulated, initiate an impulse outflow via sympathetic fibers that ends in the smooth muscle surrounding resistance vessels, arterioles, venules, and veins of the “blood reservoirs,” causing their constriction. Thus the vasomotor control mechanism plays a role both in the maintenance of the general blood pressure and in the distribution of blood to areas of special need.

The main blood reservoirs are the venous plexuses and sinuses in the skin and abdominal organs (especially in the liver and spleen). In other words, blood reservoirs are the venous networks in most parts of the body—all but those in the skeletal muscles, heart, and brain. Figure 19-12 shows that the volume of blood in the systemic veins and venules in a resting adult is extremely large compared to the volume in other vessels of the body. The term *reservoir* is apt, because the systemic veins and venules serve as a kind of slowly moving stockpile or reserve of blood. Blood can quickly be moved out of blood reservoirs and “shifted” to arteries that

supply heart and skeletal muscles when increased activity demands (Figure 19-13). A change in either arterial blood’s oxygen or carbon dioxide content sets a chemical vasomotor control mechanism in operation. A change in arterial blood pressure initiates a *vasomotor pressoreflex*.

Vasomotor Pressoreflexes. A sudden increase in arterial blood pressure stimulates aortic and carotid baroreceptors—the same ones that initiate cardiac reflexes. Not only does this stimulate the cardiac control center to reduce heart rate (see Figure 19-11), but also it inhibits the vasoconstrictor center. More impulses per second go out over parasympathetic fibers to the heart and to blood vessels. As a result, the heartbeat slows, and arterioles and the venules of the blood reservoirs dilate. Because sympathetic vasoconstrictor impulses predominate at normal arterial pressures, inhibition of these is considered the major mechanism of vasodilation. The nervous pathways involved in this mechanism are illustrated in Figure 19-14.

A decrease in arterial pressure causes the aortic and carotid baroreceptors to send more impulses to the medulla’s vasoconstrictor centers, thereby stimulating them. These centers then send more impulses via the sympathetic fibers to stimulate vascular smooth muscle and cause vasoconstriction. This squeezes more blood out of the blood reservoirs, increasing the amount of venous blood return to the heart. Eventually, this extra blood is redistributed to more active structures such as skeletal muscles and heart because their arterioles become dilated largely from the operation of a local mechanism (discussed later). Thus the vasoconstrictor pressoreflex and the local vasodilating mechanism together serve as an important device for shifting blood from reservoirs to structures that need it more (Box 19-5). It is an especially valuable mechanism during exercise (see Figure 19-13).



Box 19-4 HEALTH MATTERS

Changes in Blood Viscosity

Under normal circumstances, blood viscosity changes very little. But under certain abnormal conditions, such as marked anemia or hemorrhage, a decrease in blood viscosity may be the crucial factor lowering peripheral resistance and arterial pressure, even to the point of circulatory failure.

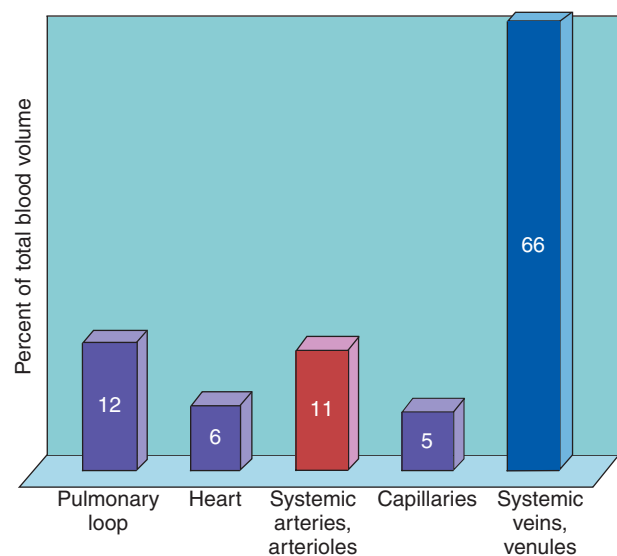


Figure 19-12 Relative blood volumes. The relative volumes of blood at rest in different parts of the adult cardiovascular system expressed as percentages of total blood volume. Notice that at rest most of the body’s blood supply is in the systemic veins and venules.

Vasomotor Chemoreflexes. Chemoreceptors located in the aortic and carotid bodies are particularly sensitive to excess blood carbon dioxide (**hypercapnia**) and somewhat less sensitive to a deficiency of blood oxygen (**hypoxia**) and to decreased arterial blood pH. When one or

more of these conditions stimulates the chemoreceptors, their fibers transmit more impulses to the medulla's vasoconstrictor centers, and vasoconstriction of arterioles and venous reservoirs soon follows (Figure 19-15). This *chemoreceptor reflex* functions as an emergency mecha-

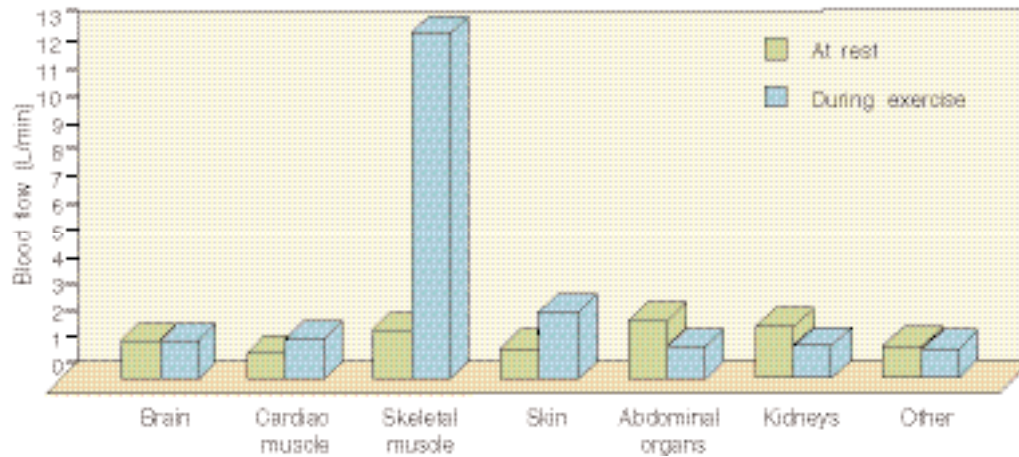


Figure 19-13 Changes in local blood flow during exercise. The *left bar* in each pair of bars shows the resting blood flow; the *right bar* shows the flow during exercise. During exercise, the vasomotor center of the medulla sends sympathetic signals to certain blood vessels to change diameter and thus shunt blood away from "maintenance" organs, such as the digestive organs in the abdomen, and toward the skeletal muscles. Notice that blood flow in the brain is held constant.

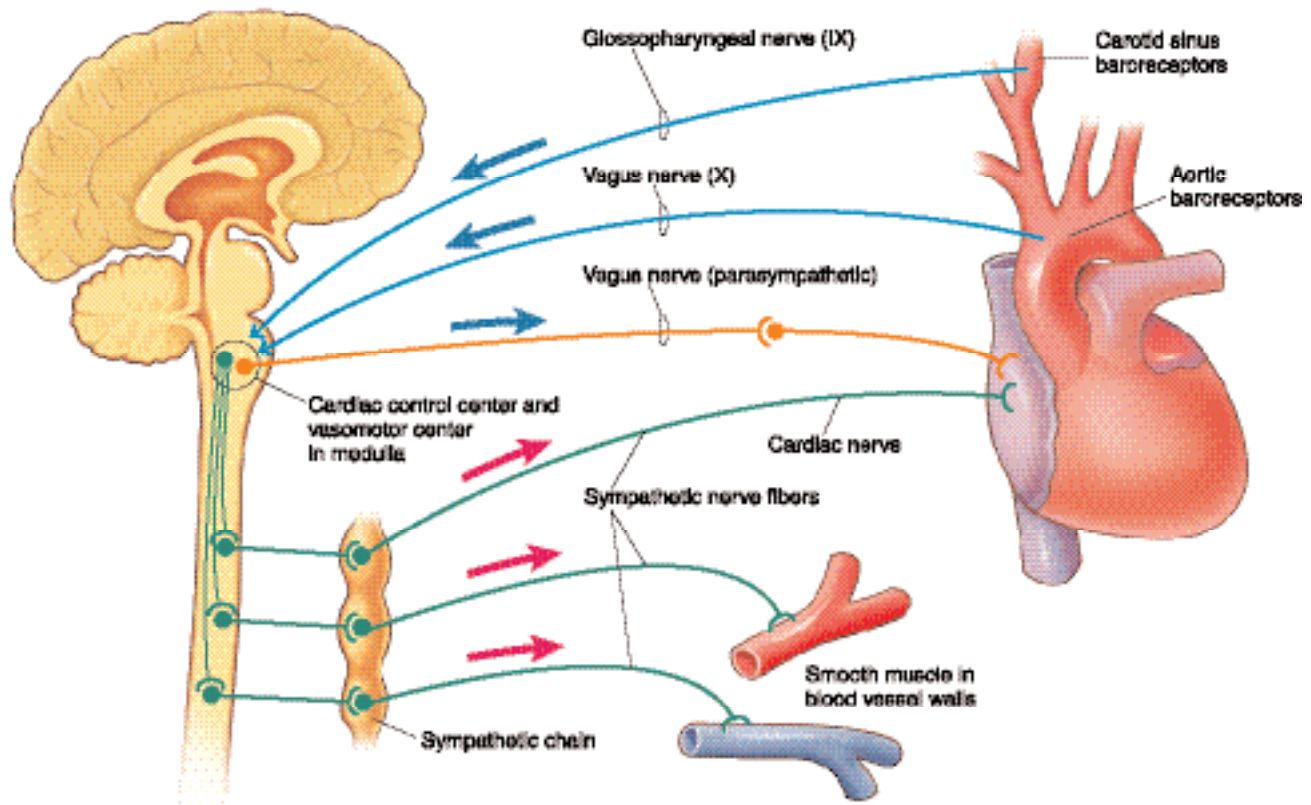


Figure 19-14 Vasomotor pressoreflexes. Carotid sinus and aortic baroreceptors detect changes in blood pressure and feed the information back to the cardiac control center and the vasomotor center in the medulla. In response, these control centers alter the ratio between sympathetic and parasympathetic output. If the pressure is too high, a dominance of parasympathetic impulses will reduce it by slowing heart rate, reducing stroke volume, and dilating blood "reservoir" vessels. If the pressure is too low, a dominance of sympathetic impulses will increase it by increasing heart rate and stroke volume and constricting reservoir vessels.

nism when hypoxia or hypercapnia endangers the stability of the internal environment.

Medullary Ischemic Reflex. The medullary ischemic reflex mechanism is said to exert powerful control of blood vessels during emergency situations when blood flow to the brain drops below normal. When the blood supply to the medulla becomes inadequate (**ischemic**), its neurons suffer from both oxygen deficiency and carbon dioxide excess. But, presumably, it is hypercapnia that intensely and directly stimulates the vasoconstrictor centers to bring about marked arteriole and venous constriction (see Figure 19-15). If the oxygen supply to the medulla decreases below a certain level, its neurons, of course, cannot function, and the medullary ischemic reflex cannot operate.

Vasomotor Control by Higher Brain Centers. Impulses from centers in the cerebral cortex and in the hypothalamus are believed to be transmitted to the vasomotor centers in the medulla and to thereby help control vasoconstriction and dilation. Evidence supporting this view is that vasoconstriction and a rise in arterial blood pressure characteristically accompany emotions of intense fear or anger. Also, laboratory experiments on animals in which stimulation of the posterior or lateral parts of the hypothalamus

leads to vasoconstriction support the belief that higher brain centers influence the vasomotor centers in the medulla.

Local Control of Arterioles

Several kinds of local mechanisms operate to produce vasodilation in localized areas. Although not all these mechanisms are clearly understood, they are known to function in times of increased tissue activity. For example, they probably account for the increased blood flow into skeletal muscles during exercise. They also operate in ischemic tissues, serving as a homeostatic mechanism that tends to restore normal blood flow. Some locally produced substances, such as nitric oxide, activate the local vasodilator mechanism, whereas others, such as *endothelin*, constrict the arterioles. Local vasodilation is also referred to as *reactive hyperemia*.



1. Peripheral resistance is affected by two major factors: blood viscosity and what else?
2. If the diameter of the arteries decreases, what effect does it have on peripheral resistance?
3. In general, how do vasomotor pressoreflexes affect the flow of blood?
4. What is a chemoreflex? How do chemoreflexes affect the flow of blood?

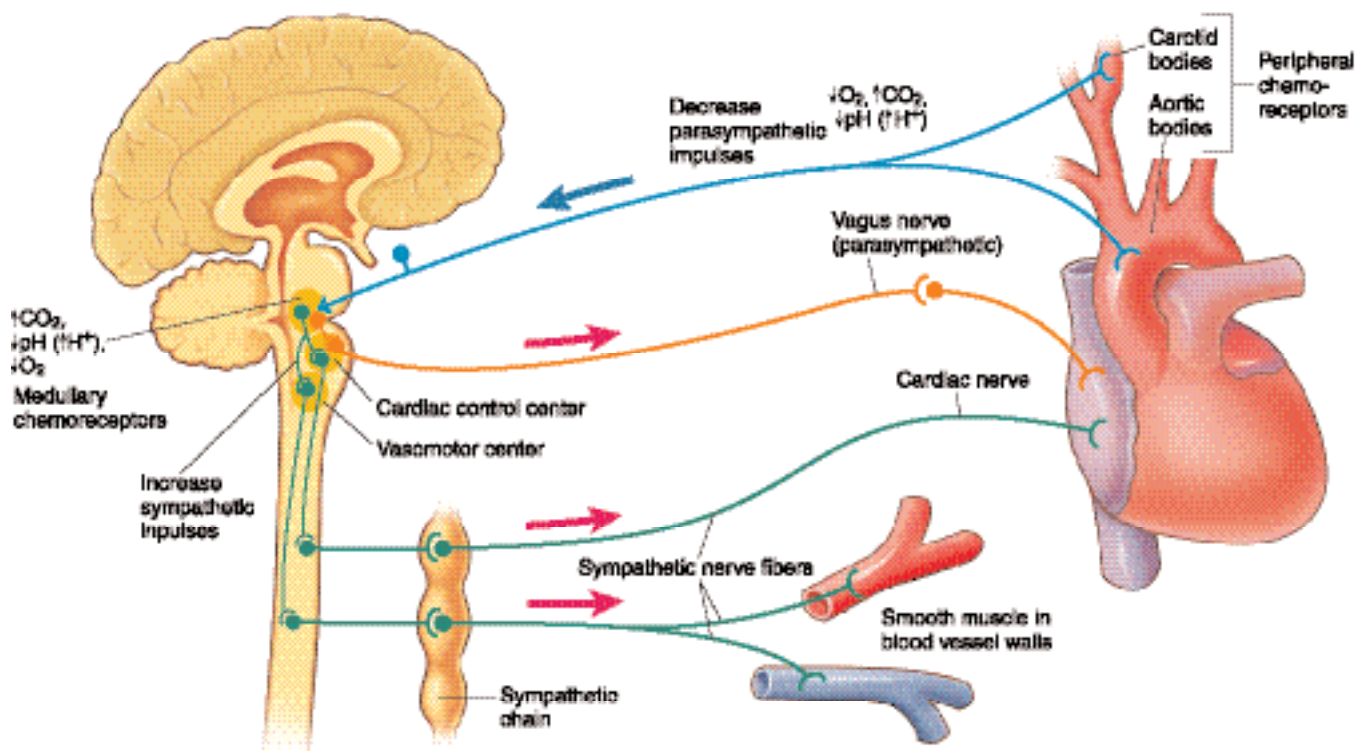


Figure 19-15 Vasomotor chemoreflexes. Chemoreceptors in the carotid and aortic bodies, as well as chemoreceptive neurons in the vasomotor center of the medulla itself, detect increases in carbon dioxide (CO_2), decreases in blood oxygen (O_2), and decreases in pH (which is really an increase in H^+). This information feeds back to the cardiac control center and the vasomotor control center of the medulla, which, in turn, alter the ratio of parasympathetic and sympathetic output. When O_2 drops, CO_2 increases, and/or pH drops, a dominance of sympathetic impulses increases heart rate and stroke volume and constricts reservoir vessels, in response.



Box 19-5 SPORTS AND FITNESS

The Cardiovascular System and Exercise

Exercise produces short-term and long-term changes in the cardiovascular system.

Short-term changes involve negative feedback mechanisms that maintain set point levels of blood oxygen and glucose, as well as other physiological variables. Because moderate to strenuous use of skeletal muscles greatly increases the body's overall rate of metabolism, oxygen and glucose are used up at a faster rate. This requires an increase in transport of oxygen and glucose by the cardiovascular system to maintain normal set point levels of these substances. One response by the cardiovascular system is to increase the cardiac output (CO) from 5 to 6 L/min at rest to up to 30 to 40 L/min during strenuous exercise. This represents a five- to eight-fold increase in the blood output of the heart! Such an increase is accomplished by a reflexive increase in heart rate (see Figure 19-11) coupled with an increase in stroke volume (see Figure 19-9). Exercise can also trigger a reflexive change in local distribution of blood flow to various tissues, shown in

Figure 19-13, that results in a larger share of blood flow going to the skeletal muscles than to some other tissues.

Long-term changes in the cardiovascular system come only when moderate to strenuous exercise occurs regularly over a long period of time. Evidence indicates that 20 to 30 minutes of moderate aerobic exercise such as cycling or running three times a week produces profound, health-promoting changes in the cardiovascular system. Among these long-term cardiovascular changes are an increase in the mass and contractility of the myocardial tissue, an increase in the number of capillaries in the myocardium, a lower resting heart rate, and decreased peripheral resistance during rest. The lower resting heart rate coupled with the increase in stroke volume possible with increased myocardial mass and contractility produce a greater range of CO, and, coupled with the other listed effects, a greater maximum CO. Exercise is also known to decrease the risk of various cardiovascular disorders, including arteriosclerosis, hypertension, and heart failure.

VENOUS RETURN TO THE HEART

Venous return refers to the amount of blood that is returned to the heart by way of the veins. Various factors influence venous return, including the operation of venous pumps that maintains the pressure gradients necessary to keep blood moving into the central veins (e.g., venae cavae) and from there into the atria of the heart. Changes in the total volume of blood in the vessels can also alter venous return. Venous return and total blood volume are discussed in the paragraphs that follow.

VENOUS PUMPS

One important factor that promotes the return of venous blood to the heart is the blood-pumping action of respirations and skeletal muscle contractions. Both actions produce their facilitating effect on venous return by increasing the pressure gradient between the peripheral veins and the venae cavae (central veins).

The process of inspiration increases the pressure gradient between peripheral and central veins by decreasing central venous pressure and also by increasing peripheral venous pressure. Each time the diaphragm contracts, the thoracic cavity necessarily becomes larger and the abdominal cavity smaller. Therefore the pressures in the thoracic cavity, in the thoracic portion of the vena cava, and in the atria decrease, and those in the abdominal cavity and the abdominal veins increase. As Figure 19-16, A, shows, this change in pressure between expiration and inspiration acts as a “respiratory pump” that moves blood along the venous route. Deeper respirations intensify these effects and therefore tend to increase venous return to the heart more than normal respirations. This is part of the reason why the principle is true that

increased respirations and increased circulation tend to go hand in hand.

Skeletal muscle contractions serve as “booster pumps” for the heart. The skeletal muscle pump promotes venous return in the following way. As each skeletal muscle contracts, it squeezes the soft veins scattered through its interior, thereby “milking” the blood in them upward, or toward the heart (Figure 19-16, B). The closing of the semilunar valves present in veins prevents blood from falling back as the muscle relaxes. Their flaps catch the blood as gravity pulls backward on it (Figure 19-17). The net effect of skeletal muscle contraction plus venous valvular action, therefore, is to move venous blood toward the heart, to increase the venous return.

The value of skeletal muscle contractions in moving blood through veins is illustrated by a common experience. Who has not noticed how much more uncomfortable and tiring standing still is than walking? After several minutes of standing quietly, the feet and legs feel “full” and swollen. Blood has accumulated in the veins because the skeletal muscles are not contracting and squeezing it upward. The repeated contractions of the muscles when walking, on the other hand, keep the blood moving in the veins and prevent the discomfort of distended veins.

TOTAL BLOOD VOLUME

The return of venous blood to the heart can be influenced by factors that change the total volume of blood in the closed circulatory pathway. Stated simply, the more the total volume of blood, the greater the volume of blood returned to the heart. What mechanisms can increase or decrease the total volume of blood? The mechanisms that change total blood volume most quickly, making them most useful in

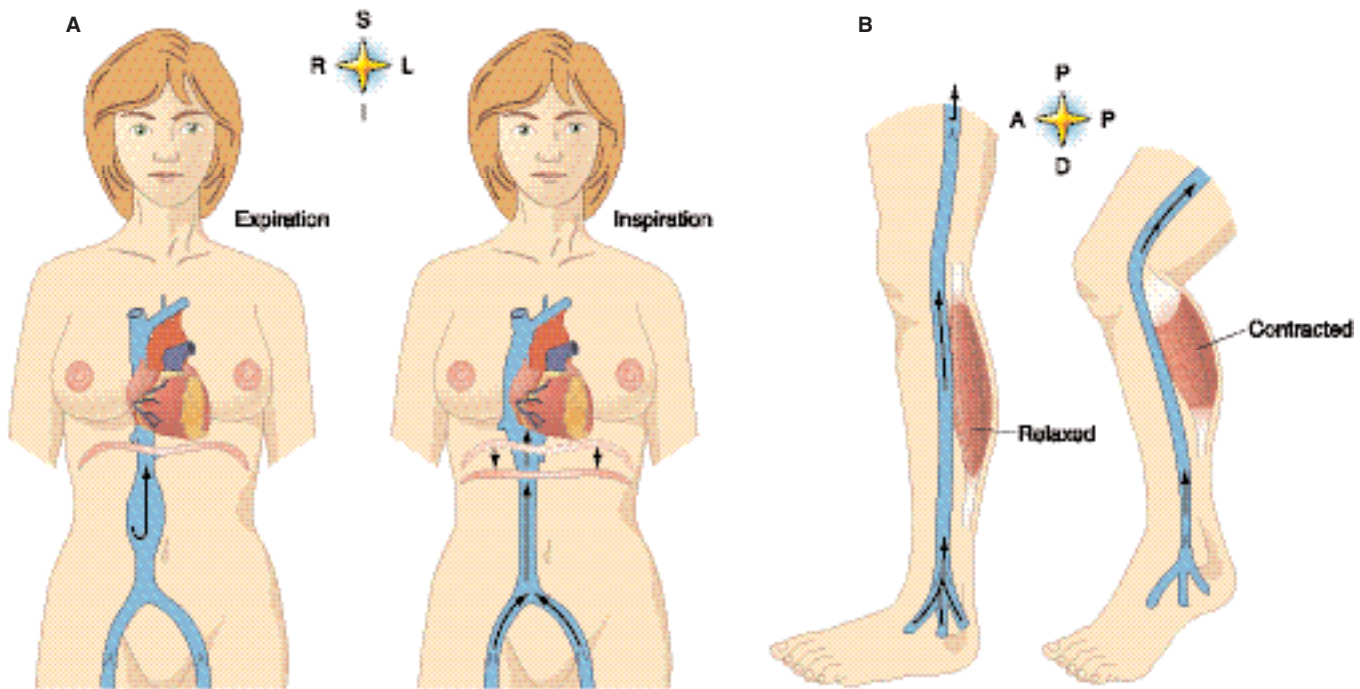


Figure 19-16 Venous pumping mechanisms. **A**, The respiratory pump operates by alternately decreasing thoracic pressure during inspiration (thus pulling venous blood into the central veins) and increasing pressure in the thorax during expiration (thus pushing central venous blood into the heart). **B**, The skeletal muscle pump operates by the alternate increase and decrease in peripheral venous pressure that normally occur when the skeletal muscles are used for the activities of daily living. Both pumping mechanisms rely on the presence of semilunar valves in the veins to prevent backflow during the low-pressure points in the pumping cycle (see Figure 19-17).

maintaining constancy of blood flow, are those that cause water to quickly move into the plasma (increasing total blood volume) or out of the plasma (decreasing total blood volume). Most of the mechanisms that accomplish such changes in plasma volume operate by altering the body's retention of water.

Capillary Exchange and Total Blood Volume

We begin our discussion of fluid movement into and out of the blood plasma with a brief overview of **capillary exchange**—the exchange of materials between plasma in the capillaries and the surrounding interstitial fluid of the systemic tissues.

According to a principle first proposed by Ernest Starling, several factors govern the movement of fluid (and solutes contained in the fluid) back and forth across a capillary wall—a principle now known as **Starling's Law of the Capillaries**. These factors, illustrated in Figure 19-18, include inwardly directed forces and outwardly directed forces. It is the balance between these forces that determines whether fluids will move into or out of the plasma at any particular point.

One type of force, osmotic pressure, tends to promote diffusion of fluid into the plasma. Osmotic pressure generated by blood colloids (large solute particles such as plasma proteins) in the plasma that cannot cross the vessel wall tends to draw water osmotically into the plasma. At the arterial end of a capillary (and in some thin-walled arterioles) the potential osmotic pressure is small and thus generates only a small, inwardly directed force. However, there is a

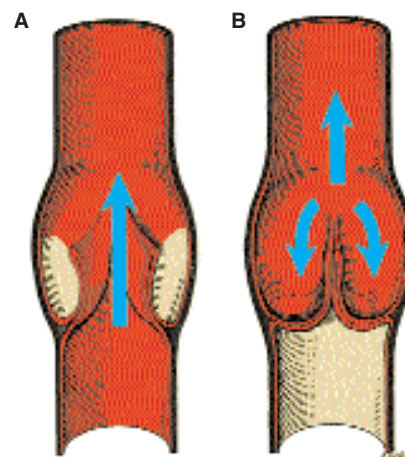


Figure 19-17 Semilunar valves. In veins, semilunar valves aid circulation by preventing backflow of venous blood when pressure in a local area is low. **A**, Local high blood pressure pushes the flaps of the valve to the side of the vessel, allowing easy flow. **B**, When pressure below the valve drops, blood begins to flow backward but fills the “pockets” formed by the valve flaps, pushing the flaps together and thus blocking further backward flow.

much larger outwardly directed force operating at the arterial end of a capillary, namely, a hydrostatic pressure gradient. Recall from Chapter 4 that hydrostatic pressure gradients promote filtration across a barrier with filtration pores, such as the capillary wall. At the arterial end of a capillary the

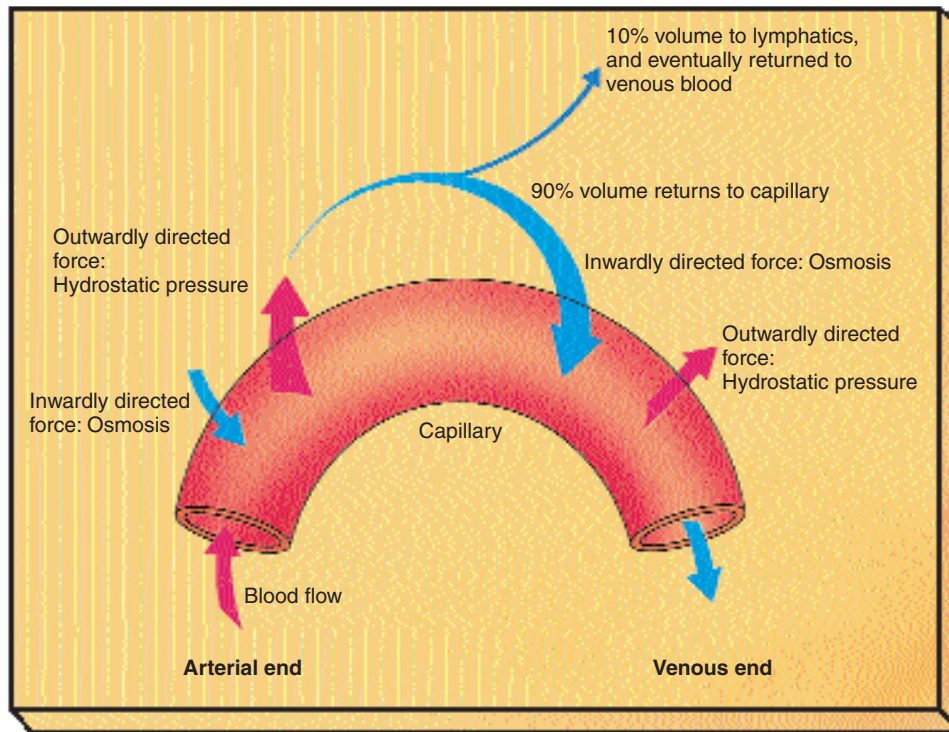


Figure 19-18 Starling's Law of the Capillaries. At the arterial end of a capillary the outward driving force of blood pressure is larger than the inwardly directed force of osmosis—thus fluid moves out of the vessel. At the venous end of a capillary the inward driving force of osmosis is greater than the outwardly directed force of hydrostatic pressure—thus fluid enters the vessel. About 90% of the fluid leaving the capillary at the arterial end is recovered by the blood before it leaves the venous end. The remaining 10% is recovered by the venous blood eventually, by way of the lymphatic vessels (see Chapter 20).

blood pressure in the vessel is much greater than the hydrostatic pressure of the interstitial fluid (IF), thus generating a very large, outwardly directed force. In short, the stronger outwardly directed forces at the arterial end of a capillary drive fluids out of the blood vessel and into the surrounding IF—producing a net loss of blood volume.

At the venous end of the capillary, however, the loss of water has increased the blood colloid osmotic pressure—promoting osmosis of water back into the plasma. This inwardly directed force is much larger than the hydrostatic pressure gradient, which has dissipated somewhat with the loss of water at the arterial end of the vessel. In short, the capillary recovers much of the fluid it lost—recovering some of the previously lost blood volume.

If you look carefully at Figure 19-18, you will notice that about 10% of the fluid lost at the arterial end of a capillary is not recovered by the forces operating in Starling's Law of the Capillaries. Does that mean that there is a constant loss of blood volume? No. Notice that the 10% fluid loss is recovered by the lymphatic system and returned to the venous blood before it reaches the heart. Details of how the lymphatic system accomplishes this fluid recovery are discussed in the next chapter. For now, we will simply state that if the lymphatic system operates normally and the osmotic and hydrostatic pressure gradients remain relatively constant, there is no net loss of blood volume resulting from capillary

exchange. If any of these factors changes, however, fluid retention by the blood will be affected.

Changes in Total Blood Volume

You have already studied the primary mechanisms for altering water retention in the body—they are the endocrine reflexes outlined in Chapter 16. One is the *ADH mechanism*. Recall that ADH (antidiuretic hormone) is released by the neurohypophysis (posterior pituitary) and acts on the kidneys in a way that reduces the amount of water lost by the body. ADH does this by increasing the amount of water that the kidneys reabsorb from urine before the urine is excreted from the body. The more ADH is secreted, the more water will be reabsorbed into the blood, and the greater the blood plasma volume will become. The ADH mechanism can be triggered by various factors such as input from baroreceptors and input from osmoreceptors (which detect the balance between water and solutes in the internal environment).

Another mechanism that changes blood plasma volume is the *renin-angiotensin mechanism* of aldosterone secretion. You may want to turn to Figure 16-26 to see that the enzyme renin is released when blood pressure in the kidney is low. Renin triggers a series of events that leads to the secretion of aldosterone, a hormone of the adrenal cortex. Aldosterone promotes sodium retention by the kidney, which in turn stimulates the osmotic flow of water from kidney tubules

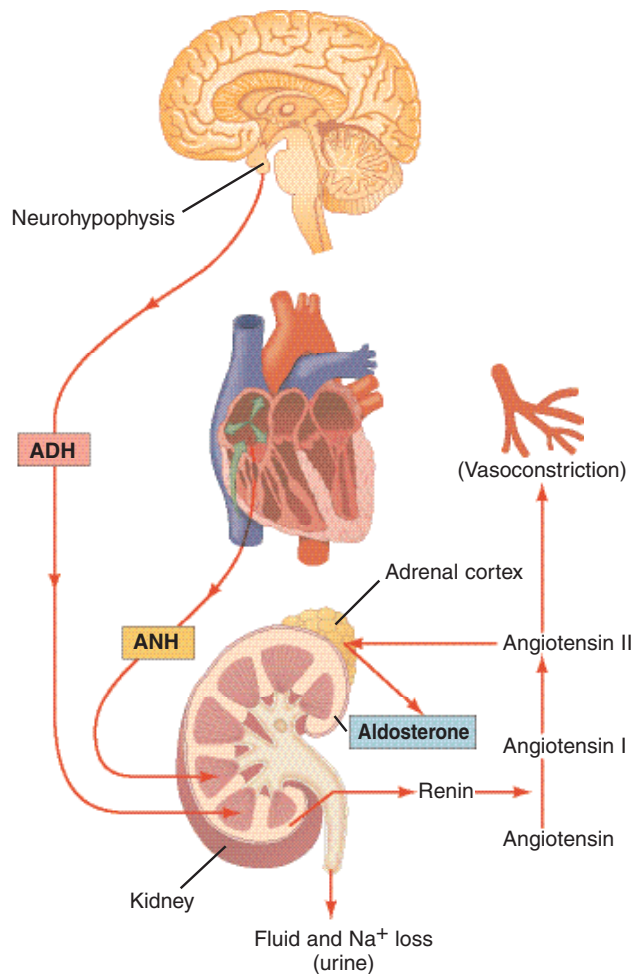


Figure 19-19 Three mechanisms that influence total plasma volume. The antidiuretic hormone (ADH) mechanism and renin-angiotensin and aldosterone mechanisms tend to increase water retention and thus increase total plasma volume. The atrial natriuretic hormone (ANH) mechanism antagonizes these mechanisms by promoting water loss and thus promoting a decrease in total plasma volume.

back into the blood plasma—but only when ADH is present to permit the movement of water. Thus low blood pressure increases the secretion of aldosterone, which, in turn, stimulates retention of water and thus an increase in blood volume. Another effect of the renin-angiotensin mechanism is the vasoconstriction of blood vessels caused by an intermediate compound called *angiotensin II*. This complements the volume-increasing effects of the mechanism and thus also promotes an increase in overall blood flow.

Yet another mechanism that can change blood plasma volume and thus venous return of blood to the heart is the *ANH mechanism*. Recall that ANH (atrial natriuretic hormone) is secreted by specialized cells in the atrial wall in response to overstretching. Overstretching of the atrial wall, of course, occurs when venous return to the heart is abnormally high. ANH adjusts venous return back down to its set point value by promoting the loss of water from the plasma and the resulting decrease in blood volume. ANH accom-

plishes this feat by increasing urine sodium loss, which causes water to follow osmotically. Sodium loss also inhibits the secretion of ADH. ANH may also have other complementary effects such as promoting vasodilation of blood reservoirs.

Thus various mechanisms influence blood volume and therefore venous return. These primary mechanisms are summarized in Figure 19-19. The ANH mechanism opposes ADH, renin-angiotensin, and aldosterone mechanisms to produce a balanced, precise control of blood volume. Precision of blood volume control contributes to precision in controlling venous return, which, in turn, contributes to precision in the overall control of blood circulation.



1. What is meant by the term *venous return*?
2. Briefly describe how the *respiratory pump* and *skeletal muscle pump* work.
3. How does Starling's Law of the Capillaries explain capillary exchange?
4. What three hormonal mechanisms work together to regulate blood volume?

MEASURING BLOOD PRESSURE

ARTERIAL BLOOD PRESSURE

Blood pressure is measured with the aid of an apparatus known as a *sphygmomanometer*, which makes it possible to measure the amount of air pressure equal to the blood pressure in an artery. The measurement is made in terms of how many millimeters (mm) high the air pressure raises a column of mercury (Hg) in a glass tube.

The sphygmomanometers originally consisted of a rubber cuff attached by a rubber tube to a compressible bulb and by another tube to a column of mercury that was marked off in millimeters (Figure 19-20). Pressure in the cuff and rubber tube pushes the column of mercury to a new height; therefore pressure can be expressed as *millimeters of mercury (mm Hg)*. Because of the hazardous nature of mercury, many sphygmomanometers in use today have mercury-free mechanical or electronic pressure sensors that are calibrated to the mercury scale.

The cuff of the sphygmomanometer is wrapped around the arm over the brachial artery, and air is pumped into the cuff by means of the bulb. In this way, air pressure is exerted against the outside of the artery. Air is added until the air pressure exceeds the blood pressure within the artery or, in other words, until it compresses the artery. At this time, no pulse can be heard through a stethoscope placed over the brachial artery at the bend of the elbow along the inner margin of the biceps muscle. By slowly releasing the air in the cuff the air pressure is decreased until it approximately equals the blood pressure within the artery. At this point, the vessel opens slightly and a small spurt of blood comes through, producing sharp “tapping” sounds. This is followed by increasingly louder sounds that suddenly change. They become more muffled, then disappear altogether. These sounds are called *Korotkoff sounds* (Box 19-6). Health pro-

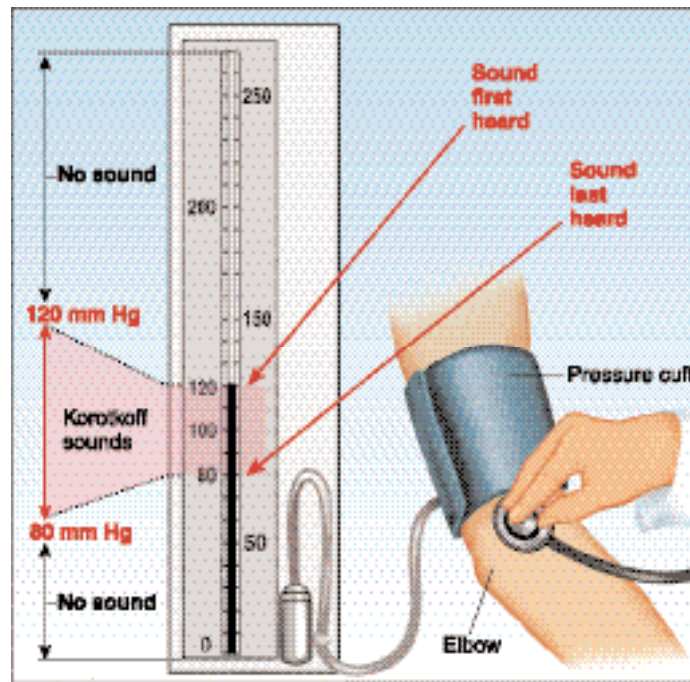


Figure 19-20 Sphygmomanometer. This mercury-filled pressure sensor is used in clinical and research settings to quickly and accurately measure arterial blood pressure.

professionals train themselves to hear these different sounds and simultaneously to read the column of mercury, because the first tapping sound appears when the column of mercury indicates the **systolic blood pressure**. Systolic pressure is the force with which the blood is pushing against the artery walls when the ventricles are contracting. The lowest point at which the sounds can be heard, just before they disappear, is approximately equal to the **diastolic pressure**, or the force of the blood when the ventricles are relaxed. Systolic pressure gives valuable information about the force of the left ventricular contraction, and diastolic pressure gives valuable information about the resistance of the blood vessels.

Blood in the arteries of the average adult exerts a pressure equal to that required to raise a column of mercury about 120 mm (or a column of water over 5 feet) high in a glass tube during systole of the ventricles and 80 mm high during their diastole. For the sake of brevity, this is expressed as a blood pressure of 120 over 80 (120/80). The first, or upper, figure represents systolic pressure and the second represents diastolic pressure. From the figures just given, we observe that blood pressure fluctuates considerably during each heartbeat. During ventricular systole, the force is great enough to raise the mercury column 40 mm higher than during ventricular diastole. This difference between systolic and diastolic pressure is called **pulse pressure**. It characteristically increases in arteriosclerosis, mainly because systolic pressure increases more than diastolic pressure. Pulse pressure increases even more markedly in aortic valve insufficiency because of both a rise in systolic and a fall in diastolic pressure.

Be aware that although the classic method of indirect arterial blood pressure assessment involves a column of mercury, and the units in which blood pressure is expressed is based on the height of a mercury column, very few modern sphygmomanometers actually use mercury. The reason is twofold. First, mercury is a very hazardous substance and should the mercury column break, an environmental health hazard would exist. Second, it is much more cost effective to use cheap and durable electronic pressure sensors that are calibrated to the “mercury scale” than to build fragile columns of mercury.

Blood pressure can also be measured directly in various ways. For example, a small tube called a cannula with a removable pointed insert can be pushed directly into a vessel, the insert withdrawn, and connected to a manometer or electronic pressure sensor. A long, flexible tube called a catheter can likewise be placed in blood vessels or even into a chamber of the heart. These techniques can be used in critical care, but the more indirect methods described previously are more practical for routine screening and clinical evaluation.

BLOOD PRESSURE AND ARTERIAL VERSUS VENOUS BLEEDING

Because blood exerts a comparatively high pressure in arteries and a very low pressure in veins, it gushes forth with considerable force from a cut artery but seeps in a slow, steady stream from a vein. As we have just seen, each ventricular contraction raises arterial blood pressure to the systolic level, and each ventricular relaxation lowers it to the diastolic

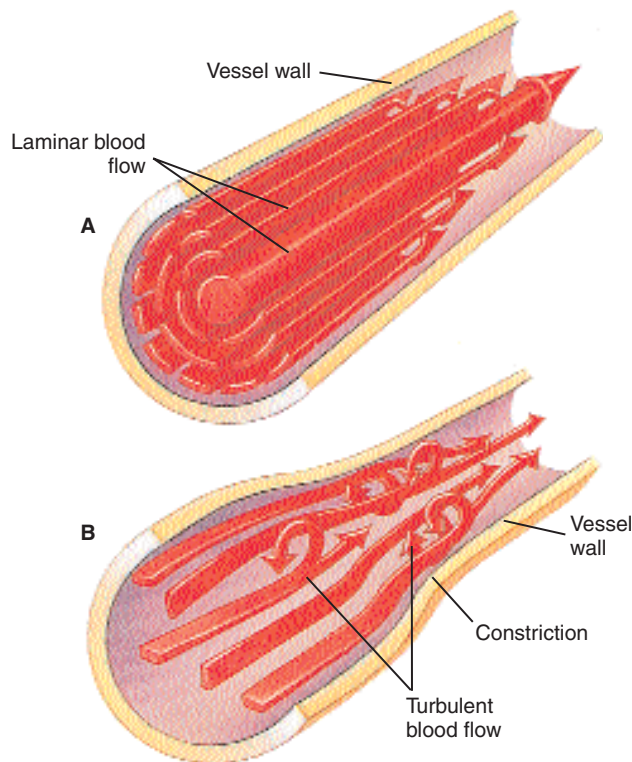


Box 19-6 DIAGNOSTIC STUDY

Focus on Turbulent Blood Flow

Understanding the mechanics of fluid flow through blood vessels is to be able to see the bigger picture of hemodynamics.

Normally, blood flows through vessels with smooth walls that taper or enlarge only slightly, if at all. The manner in which a fluid, in this case blood, flows through a smooth vessel is termed **laminar flow**. The Latin word *lamina* means layer, an apt description of the way fluids flow through tubes in concentric, cylindrical layers—as you can see in Figure A. Because of friction against the inner face of the vessel wall, the outer layers of blood flow more slowly than the inner layers. Laminar flow is the normal pattern of blood flow in most healthy vessels.



Listening for bruits.

When smooth, laminar flow is disrupted by branching of a vessel, a sharp turn, or a sudden constriction or obstruction, the flow of blood becomes turbulent. **Turbulent flow**, shown in Figure B, occurs normally at heart valves and contributes to the first and second heart sounds described on p. 604.

Significant turbulent flow in most vessels is not normal. If the sounds of turbulence are detected in a peripheral vessel, an abnormal or unusual constriction may exist. Such constrictions could be temporary and intentional, as in the case of an inflated blood pressure cuff pressing on an artery and creating turbulence. The sounds of turbulence generated by such blood pressure measurements are called *Korotkoff sounds*. However, some sounds of turbulence are caused by constrictions that are a serious threat to one's health. For example, in the previous chapter we discussed the fact that arteriosclerosis can cause such a narrowing of a vessel's channel and can ultimately lead to death by ischemia, thrombosis, or other mechanisms (see Figure 18-35). Figure C shows a stethoscope being used to listen for low-pitched blowing sounds called **bruits** (BROO-eez) that can occur in the carotid arteries. Bruits may result from obstruction caused by arteriosclerosis or abnormally increased pulse pressure. A usually benign, but abnormal, condition known as venous hum produces a humming noise in the internal jugular vein. Venous hum is common in children and probably results from vigorous myocardial contraction.



level. As the ventricles contract, the blood spurts forth forcefully from the increased pressure in the artery, but as the ventricles relax, the flow ebbs to almost nothing because of the fall in pressure. In other words, blood escapes from an artery in spurts because of the alternate raising and lowering of arterial blood pressure but flows slowly and steadily from a vein because of the low, practically constant pressure. A uniform, instead of a pulsating, pressure exists in the capillaries and veins. Why? Because the arterial walls, being elas-

tic, continue to squeeze the blood forward while the ventricles are in diastole. Therefore blood enters capillaries and veins under a relatively steady pressure (see Figure 19-7).

MINUTE VOLUME OF BLOOD

The volume of blood circulating through the body per minute (*minute volume*) is determined by the magnitude of the blood pressure gradient and the peripheral resistance (Figure 19-21).

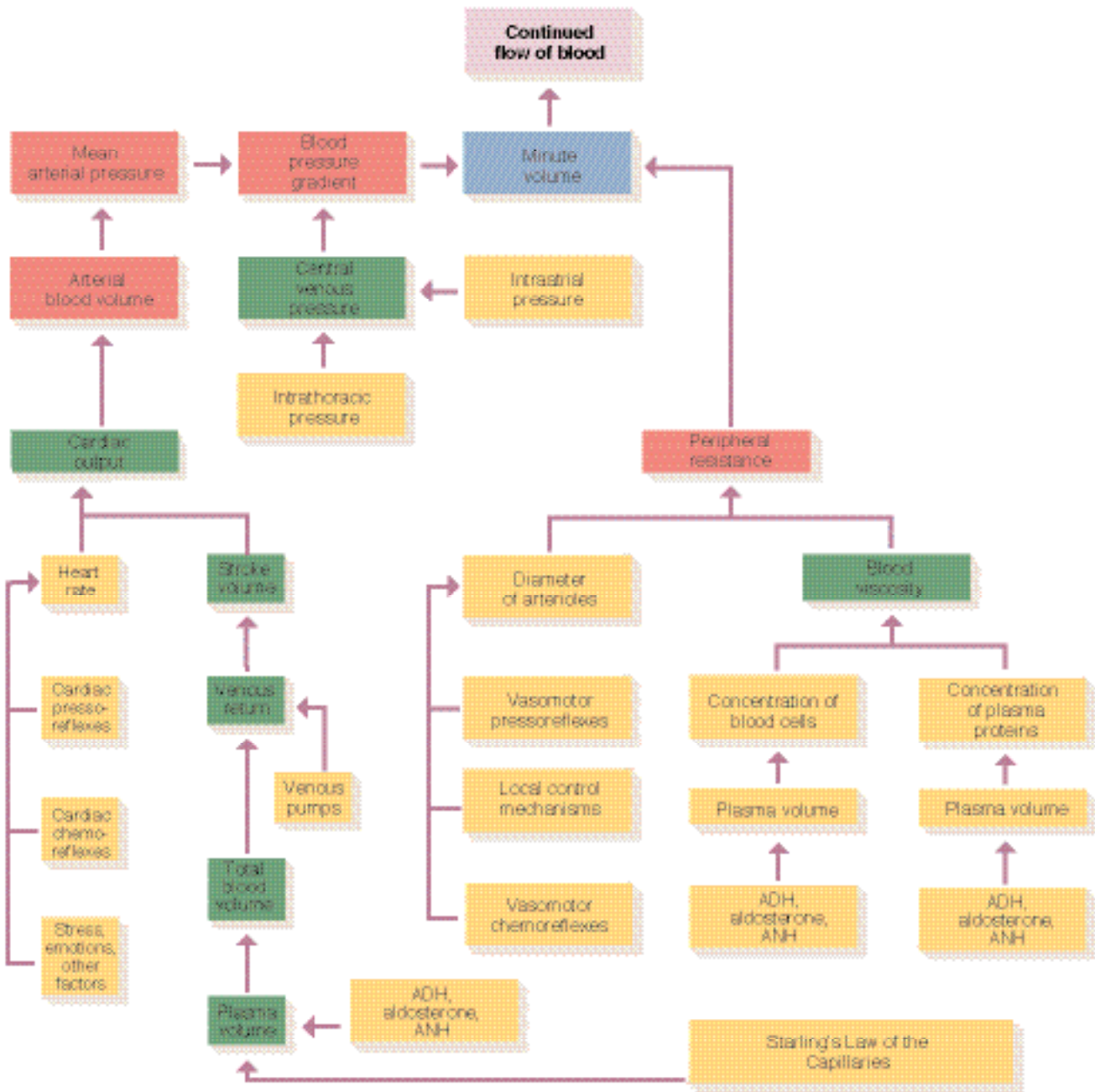


Figure 19-21 Factors that influence the flow of blood. The flow of blood, expressed as volume of blood flowing per minute (or *minute volume*), is determined by various factors. This chart shows only some of the major factors that influence blood flow. Notice that some factors appear more than once in the chart, indicating that they can influence blood flow in several ways.

A nineteenth century physiologist and physicist, Poiseuille (pwah-soo-EE), described the relation between these three factors—pressure gradient, resistance, and minute volume—with a mathematical equation known as *Poiseuille’s law*. In general, but with certain modifications, it applies to blood circulation. We can state it in a simplified form as follows: the volume of blood circulated per minute is directly related to mean arterial pressure minus central venous pressure and inversely related to resistance:

$$\text{Volume of blood circulated per minute} = \frac{\text{Mean arterial pressure} - \text{Central venous pressure}}{\text{Resistance}}$$

$$\text{Minute volume} = \frac{\text{Pressure gradient}}{\text{Resistance}}$$

This mathematical relationship needs qualifying with regard to the influence of peripheral resistance on circulation.

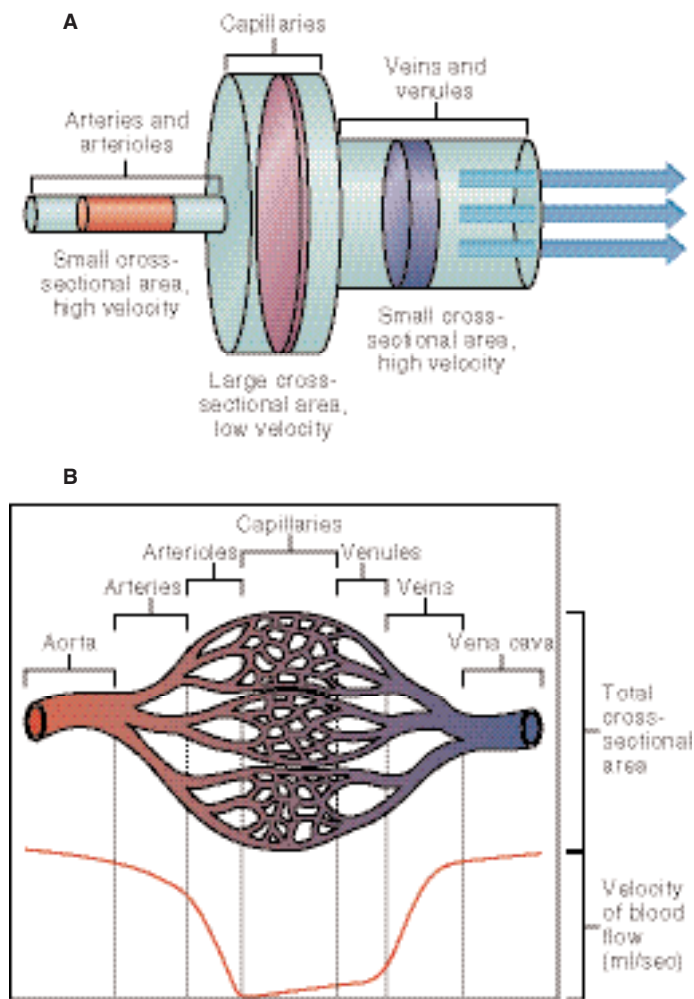


Figure 19-22 Relationship between cross-sectional area and velocity of blood flow. As you can see in the simple diagram (A) and the blood vessel chart (B), blood flows with great speed in the large arteries. However, branching of arterial vessels increases the total cross-sectional area of the arterioles and capillaries, reducing the flow rate. When capillaries merge into venules and venules merge into veins, the total cross-sectional area decreases, causing the flow rate to increase.

For instance, according to the equation, an increase in peripheral resistance tends to decrease blood flow. (Why? Increasing peripheral resistance increases the denominator of the fraction in the preceding equation. Increasing the denominator of any fraction necessarily does what to its value? It decreases the value of the fraction.)

Increased peripheral resistance, however, has a secondary action that opposes its primary tendency to decrease blood flow. An increase in peripheral resistance hinders or decreases arteriole runoff. This, of course, tends to increase the volume of blood left in the arteries and so tends to increase arterial pressure. Note also that increasing arterial pressure tends to increase the value of the fraction in Poiseuille's equation. Therefore it tends to increase circulation. In short, to say unequivocally what the effect of

an increased peripheral resistance will be on circulation is impossible. It depends also on arterial blood pressure—whether it increases, decreases, or stays the same when peripheral resistance increases. The clinical condition *arteriosclerosis with hypertension* (high blood pressure) illustrates this point. Both peripheral resistance and arterial pressure are increased in this condition. If resistance were to increase more than arterial pressure, circulation (that is, volume of blood flow per minute) would decrease. But if arterial pressure increases proportionately to resistance, circulation remains normal.

Think

1. What is meant by the term *minute volume*?
2. How is minute volume related to peripheral resistance?

VELOCITY OF BLOOD FLOW

The speed with which the blood flows, that is, distance per minute, through its vessels is governed in part by the physical principle that when a liquid flows from an area of one cross-sectional size to an area of larger size, its velocity slows in the area with the larger cross section (Figure 19-22). For example, a narrow river whose bed widens flows more slowly through the wide section than through the narrow section. In terms of the blood vascular system, the total cross-sectional area of all arterioles together is greater than that of the arteries. Therefore blood flows more slowly through arterioles than through arteries. Likewise, the total cross-sectional area of all capillaries together is greater than that of all arterioles, and therefore capillary flow is slower than arteriole flow. The venule cross-sectional area, on the other hand, is smaller than the capillary cross-sectional area. Therefore blood velocity increases in venules and again in veins, which have a still smaller cross-sectional area. In short, the most rapid blood flow takes place in arteries and the slowest in capillaries. Can you think of a valuable effect stemming from the fact that blood flows most slowly through the capillaries?

PULSE

MECHANISM

Pulse is defined as the alternate expansion and recoil of an artery. Two factors are responsible for the existence of a pulse that can be felt:

1. Intermittent injections of blood from the heart into the aorta, which alternately increase and decrease the pressure in that vessel. If blood poured steadily out of the heart into the aorta, the pressure there would remain constant, and there would be no pulse.
2. The elasticity of the arterial walls, which allows them to expand with each injection of blood and then recoil. If the vessels were fashioned from rigid mater-

ial such as glass, there would still be an alternate raising and lowering of pressure within them with each systole and diastole of the ventricles, but the walls could not expand and recoil, and therefore no pulse could be felt.

PULSE WAVE

Each ventricular systole starts a new pulse that proceeds as a wave of expansion throughout the arteries and is known as the pulse wave. It gradually dissipates as it travels, disappearing entirely in the capillaries. The pulse wave felt at the common carotid artery in the neck is large and powerful, rapidly following the first heart sound. Figure 19-23 shows that the carotid pulse wave begins during ventricular systole. Note that the closure of the aortic valve produces a detectable *dicrotic notch* in the pulse wave. However, the pulse felt in the radial artery at the wrist does not coincide with the contraction of the ventricles. It follows each contraction by an appreciable interval (the length of time required for the pulse wave to travel from an aorta to the radial artery). The farther from the heart the pulse is taken, therefore, the longer that interval is.

Almost everyone is aware of the diagnostic importance of the pulse. It reveals important information about the cardiovascular system, heart action, blood vessels, and circulation.

Not everyone is aware of the basic functional role of the pulse wave, however. The pulse wave actually conserves energy produced by the pumping action of the heart. The great force of pressure with which blood is ejected from the heart during ventricular systole expands the wall of the aorta. The stretched aortic wall now has potential energy stored in it—just as a stretched rubber band has potential energy. During ventricular diastole, the elastic nature of the aortic wall allows it to recoil. This recoil exerts pressure on the blood and thus keeps it moving. If the wall of the aorta was inelastic, it would not alternately expand and recoil—and would thus not keep blood moving continuously. Instead, arterial blood would simply spurt, then stop, then spurt, then stop, and so on.

WHERE PULSE CAN BE FELT

The pulse can be felt wherever an artery lies near the surface and over a bone or other firm background. Some of the spe-

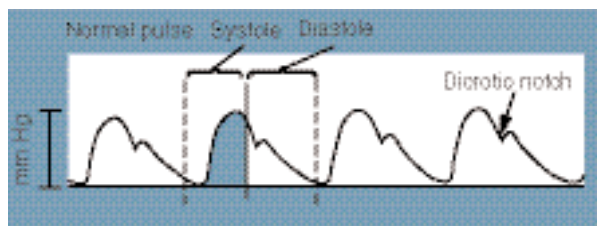


Figure 19-23 Normal carotid pulse wave. This series of pulse waves shows rhythmic increases and decreases in pressure as measured at the common carotid artery in the neck. The dicrotic notch represents the pressure fluctuation generated by closure of the aortic valve.

cific locations where the pulse point is most easily felt are listed below and shown in Figure 19-24.

- **Radial artery**—at wrist
- **Temporal artery**—in front of ear or above and to outer side of eye
- **Common carotid artery**—along anterior edge of sternocleidomastoid muscle at level of lower margin of thyroid cartilage
- **Facial artery**—at lower margin of lower jawbone on a line with corners of mouth and in groove in mandible about one third of way forward from angle
- **Brachial artery**—at bend of elbow along inner margin of biceps muscle
- **Popliteal artery**—behind the knee
- **Posterior tibial artery**—behind the medial malleolus (inner “ankle bone”)
- **Dorsalis pedis artery**—on the dorsum (upper surface) of the foot

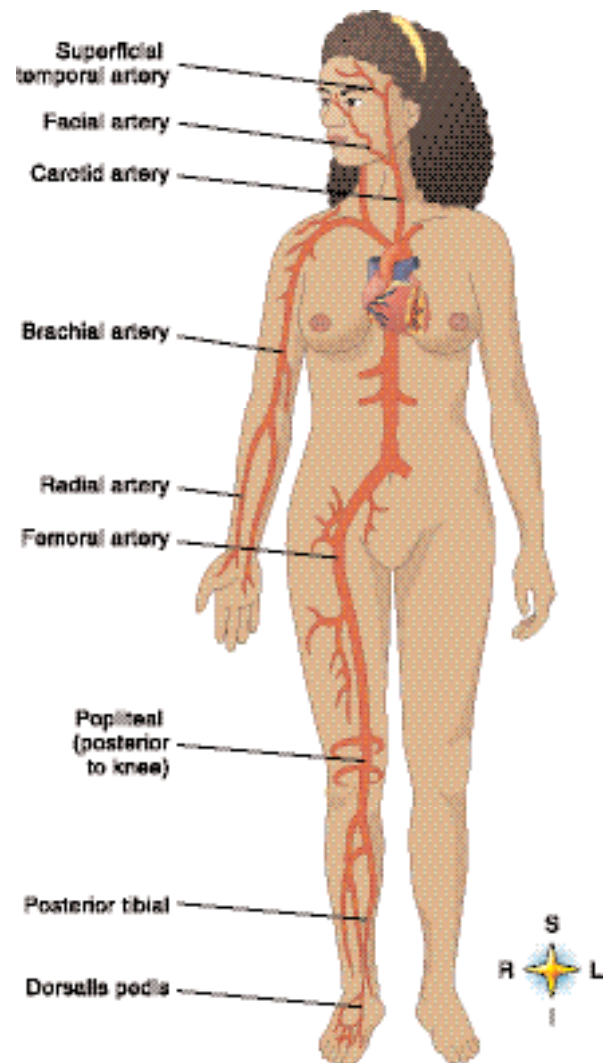


Figure 19-24 Pulse points. Each pulse point is named after the artery with which it is associated.

There are six important pressure points that can be used to stop arterial bleeding:

1. **Temporal artery**—in front of ear
2. **Facial artery**—same place as pulse is taken
3. **Common carotid artery**—point where pulse is taken, with pressure back against spinal column
4. **Subclavian artery**—behind medial third of clavicle, pressing against first rib
5. **Brachial artery**—few inches above elbow on inside of arm, pressing against humerus
6. **Femoral artery**—in middle of groin, where artery passes over pelvic bone; pulse can also be felt here

In trying to stop arterial bleeding by pressure, one must always remember to apply the pressure at the pulse point, or *pressure point*, that lies between the bleeding part and the heart. Why? Blood flows from the heart through the arteries to the part. Pressure between the heart and bleeding

point therefore cuts off the source of the blood flow to that point.

VENOUS PULSE

A detectable pulse exists in the large veins only. It is most prominent in the veins near the heart because of changes in venous blood pressure brought about by alternate contraction and relaxation of the atria of the heart. The clinical significance of venous pulse is not as great as that of arterial pulse and thus it is less often measured.



1. What device is used in clinical settings to measure arterial blood pressure?
2. Which is more important for assessing health—the systolic pressure or the diastolic pressure?
3. In which type of vessel is blood most likely to be flowing at a very slow rate—an artery, a capillary, or a vein?
4. Without being specific, where are pulse points normally located in the body?

CYCLE OF LIFE

Cardiovascular Physiology

Changes in the function of the heart and blood vessels usually parallel the structural changes in these organs over the life span. For example, changes at the time of birth that adapt the circulatory system to life outside the womb cause changes in the blood pressure gradients that alter the flow of blood in many parts of the body. Likewise, the degenerative changes associated with aging reduce the heart's ability to maintain cardiac output and the ability of arteries to withstand high pressure.

Among the most apparent changes in the function of the cardiovascular system associated with the progression through the life cycle are changes in arterial blood pressure. In a newborn, normal arterial blood pressure is only about 90/55 mm Hg—much lower than the arterial pressure of

120/80 mm Hg in most healthy young adults. In older adults, arterial blood pressures commonly reach 150/90 mm Hg.

Another commonly observed change in cardiovascular function relates to heart rate. The heart rates of infants and children are typically more variable than those in adults. Compared to adults, children often exhibit very large increases in heart rate in response to stressors such as illness, pain, tension, and exercise. Whereas a typical resting heart rate for adults is about 72 beats per minute, the resting heart rate of a newborn can range from 120 to 170 beats per minute, and the resting heart rate of a preschooler can range from 80 to 160 beats per minute. In older adults, resting heart rates range from lows of around 40 beats per minute to 100 beats per minute.

THE BIG PICTURE

Blood Flow and the Whole Body

As stated in this chapter and many times throughout this book, one of the essential concepts of homeostasis is the fact that our internal environment is a renewable fluid. If we could not maintain the chemical nature and other characteristics of our internal fluid environment, we would not survive. To maintain the constancy of the internal fluid, we must be able to shift nutrients, gases, hormones, waste products, agents of immunity, and other materials around in the body. As certain materials are depleted in one tissue and new materials enter the internal environment in another tissue, redistribution must occur. What better way than in a system of circulating fluid? This fluid shifts materials from place to place and redistributes heat and pressure. Recall from your study of

the integumentary and muscular systems that shifting the flow of blood to or from warm tissues at the proper time is essential to maintaining the homeostasis of body temperature. As we will learn in a later chapter, the ability of our blood to increase or decrease blood pressure in the kidney has a great impact on that organ's vital function of filtering the internal environment. Understanding the basic mechanisms of almost any system in the body requires an understanding of the dynamics of blood flow.

What we have seen in this chapter is a wonderfully complex array of mechanisms that work together in concert with the actions of other systems to maintain the constancy of the *milieu intérieur*—the internal environment.

MECHANISMS OF DISEASE

Disorders of Cardiovascular Physiology

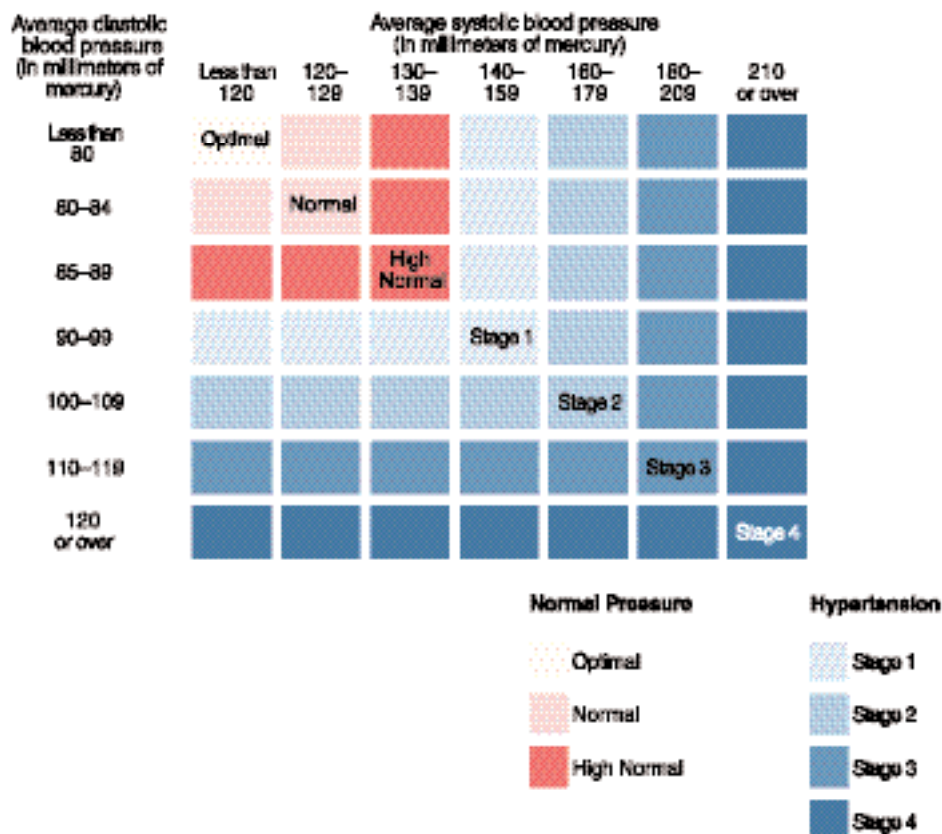
Hypertension

The largest number of office visits to physicians is due to a condition called **hypertension (HTN)**, or high blood pressure. More than 60 million cases of HTN have been diagnosed in the United States. This condition occurs when the force of blood exerted by the arterial blood vessel exceeds a blood pressure of 140/90 mm Hg. Ninety percent of HTN cases are classified as *primary-essential*, or idiopathic, with no single known causative etiology. Another classification, secondary (HTN), is caused by kidney disease or hormonal problems, or induced by oral contraceptives, pregnancy, or other causes.

Another way of classifying hypertension is illustrated in the accompanying chart adapted from the National High Blood Pressure Education Program (Figure 19-25). This system uses systolic and diastolic blood pressure values to classify hypertension into four stages according to severity. The guidelines that accompany this scheme emphasize the belief that there is no precise distinction between normal and abnormal values—thus even those in the high normal range may be treated as having HTN.

Many risk factors have been identified in the development of HTN. Genetic factors play a large role. There is an increased susceptibility or predisposition with a family history of HTN. Males experience higher rates of HTN at an earlier age than women, and HTN in blacks far exceeds that of whites in the United States. There is also a direct relationship between age and high blood pressure. This is because as age advances, the blood vessels become less compliant and there is a higher incidence of atherosclerotic plaque build-up. HTN can also be fatal if undetected in women taking oral contraceptives. Risk factors include high stress levels, obesity, calcium deficiencies, high levels of alcohol and caffeine intake, smoking, and lack of exercise.

There are many potential complications of untreated HTN. The risk of ischemic heart disease and heart failure, kidney failure, and stroke are some examples. As many as 400,000 people per year experience a stroke. Because HTN manifests minimal or no overt signs, it is known as the “silent killer.” Headaches, dizziness, and fainting have been reported but are not always symptomatic of HTN. Regular screenings at the worksite and screening booths in malls and in hospitals often help to identify asymptomatic HTN.



Source: National High Blood Pressure Education Program

Figure 19-25 Classification of hypertension. This chart is adapted from the National High Blood Pressure Education Program.

Heart Failure

Heart failure is the inability of the heart to pump enough blood to sustain life. Heart failure can be the result from many different heart diseases. Valve disorders can reduce the pumping efficiency of the heart enough to cause heart failure. **Cardiomyopathy** (kar-dee-o-my-OP-ath-ee), or disease of the myocardial tissue, may reduce pumping effectiveness. A specific event such as myocardial infarction can result in myocardial damage that causes heart failure. Dysrhythmias such as complete heart block or ventricular fibrillation can also impair the pumping effectiveness of the heart and thus cause heart failure.

Congestive heart failure (CHF) or, simply, *left-side heart failure*, is the inability of the left ventricle to pump blood effectively. Most often, such failure results from myocardial infarction caused by coronary artery disease. It is called *congestive heart failure* because it decreases pumping pressure in the systemic circulation, which in turn causes the body to retain fluids. Portions of the systemic circulation thus become congested with extra fluid. As previously stated, left-side heart failure also causes congestion of blood in the pulmonary circulation (termed *pulmonary edema*)—possibly leading to right-side heart failure.

Failure of the right side of the heart, or *right-side heart failure*, accounts for about one fourth of all cases of heart failure. Right-side heart failure often results from the progression of disease that begins in the left side of the heart. Failure of the left side of the heart results in reduced pumping of blood returning from the lungs. Blood backs up into the pulmonary circulation, then into the right side of the heart—causing an increase in pressure that the right side of the heart simply cannot overcome. Right-side heart failure can also be caused by lung disorders that obstruct normal pulmonary blood flow and thus overload the right side of the heart—a condition called **cor pulmonale** (kor pul-mon-AHL-ee).

Circulatory Shock

The term **circulatory shock** refers to the failure of the circulatory system to adequately deliver oxygen to the tissues, resulting in the impairment of cell function throughout the body. If left untreated, circulatory shock may lead to death. Circulatory failure has many causes, all of which somehow reduce the flow of blood through the blood vessels of the body. Because of the variety of causes, circulatory shock is often classified into the following types:

- **Cardiogenic** (kar-dee-o-JEN-ik) **shock** results from any type of heart failure, such as that after severe myocardial infarction (heart attack), heart infections,

and other heart conditions. Because the heart can no longer pump blood effectively, blood flow to the tissues of the body decreases or stops.

- **Hypovolemic** (hye-po-vo-LEE-mik) **shock** results from the loss of blood volume in the blood vessels (hypovolemia means “low blood volume”). Reduced blood volume results in low blood pressure and reduced flow of blood to tissues. Hemorrhage is a common cause of blood volume loss leading to hypovolemic shock. Hypovolemia can also be caused by loss of interstitial fluid, causing a drain of blood plasma out of the vessels and into the tissue spaces. Loss of interstitial fluid is common in chronic diarrhea or vomiting, dehydration, intestinal blockage, severe or extensive burns, and other conditions.
- **Neurogenic** (noo-ro-JEN-ik) **shock** results from widespread dilation of blood vessels caused by an imbalance in autonomic stimulation of smooth muscle in vessel walls. You may recall from Chapter 14 that autonomic effectors such as smooth muscle tissues are controlled by a balance of stimulation from the sympathetic and parasympathetic divisions of the autonomic nervous system. Normally, sympathetic stimulation maintains the muscle tone that keeps blood vessels at their usual diameter. If sympathetic stimulation is disrupted by an injury to the spinal cord or medulla, depressive drugs, emotional stress, or some other factor, blood vessels dilate significantly. Widespread vasodilation reduces blood pressure, thus reducing blood flow.
- **Anaphylactic** (an-a-fi-LAK-tik) **shock** results from an acute type of allergic reaction called anaphylaxis. Anaphylaxis causes the same kind of blood vessel dilation characteristic of neurogenic shock.
- **Septic shock** results from complications of septicemia, a condition in which infectious agents release toxins into the blood. The toxins often dilate blood vessels, causing shock. The situation is usually made worse by the damaging effects of the toxins on tissues combined with the increased cell activity caused by the accompanying fever. One type of septic shock is *toxic shock syndrome* (TSS), which usually results from staphylococcal infections that begin in the vagina of menstruating women and spread to the blood.

The body has numerous mechanisms that compensate for the changes that occur during shock. However, these mechanisms may fail to compensate for changes that occur in severe cases, and this failure often results in death.

CASE STUDY

Mr. Charles Simpson, age 20, is injured in an industrial accident resulting in a crushed pelvis, ruptured spleen, and associated blood loss. His past medical history is negative for previous trauma or chronic illness and positive for usual childhood diseases. Before the accident Mr. Simpson was in good health and exercised daily. His height is 6 feet and weight 165 pounds.

At midnight a 10-ton forklift fell and pinned Mr. Simpson at the pelvis for about 20 minutes. Paramedics at the scene began intravenous lactated Ringer's solution at 150 ml/hr. Vital signs were heart rate 120 beats per minute, blood pressure 90/70, respirations 46. Mr. Simpson was awake and complained of pelvic, back, and abdominal pain. His toes were mottled, pedal pulses were absent, radial pulses weak, brachial and carotid pulses palpable. The electrocardiogram monitor reflected tachycardia, and he became short of breath with conversation. Skin was cold and clammy, with numerous pinpoint hemorrhages present over his upper thorax, face, and neck.

Mr. Simpson was transported to the hospital, where his injuries were diagnosed and immediate treatment for shock initiated. After stabilization, his ruptured spleen was surgically corrected, he was placed in pelvic traction to stabilize his fractures, and was admitted to the intensive care unit.

- Mr. Simpson's extracellular fluid volume deficit occurred as a result of which primary mechanism?
 - Decreased intake of fluids and electrolytes
 - Excessive loss of blood and fluids
 - Shifts of fluids and electrolytes into nonaccessible areas
- Which of Mr. Simpson's signs are the result of compensatory mechanisms directed at maintaining cardiac output?
 - Increased heart rate and oliguria
 - Decreased blood pressure and sodium loss
 - Respiratory acidosis and decreased heart rate
 - All of the above
- The mechanism MOST responsible for Mr. Simpson's tachycardia is:
 - Hypoxemia caused by atelectasis
 - Anxiety as a result of traumatic injuries and pain
 - A physiological change in response to decreased blood pressure
 - Reaction to blood transfusion
- Based on the information provided, what type of shock did Mr. Simpson experience?
 - Cardiogenic
 - Hypovolemic
 - Neurogenic
 - Anaphylactic

CHAPTER SUMMARY

- Vital role of the cardiovascular system in maintaining homeostasis depends on the continuous and controlled movement of blood through the capillaries
- Numerous control mechanisms help to regulate and integrate the diverse functions and component parts of the cardiovascular system to supply blood in response to specific body area needs

HEMODYNAMICS

- Hemodynamics—collection of mechanisms that influence the dynamic (active and changing) circulation of blood
- Circulation of different volumes of blood per minute is essential for healthy survival
- Circulation control mechanisms must accomplish two functions
 - Maintain circulation
 - Vary volume and distribution of the blood circulated

THE HEART AS A PUMP

- Conduction system (Figure 19-1)
 - Four of the major structures that compose the conduction system of the heart
 - Sinoatrial node (SA node)
 - Atrioventricular node (AV node)
 - AV bundle (bundle of His)
 - Purkinje system
 - Conduction system structures are more highly specialized than ordinary cardiac muscle tissue and permit only rapid conduction of an action potential through the heart
 - SA node (pacemaker)
 - Initiates each heartbeat and sets its pace
 - Specialized pacemaker cells in the node possess an intrinsic rhythm
 - Sequence of cardiac stimulation
 - After being generated by the SA node, each impulse travels throughout the muscle fibers of both atria and the atria begin to contract
 - As the action potential enters the AV node from the right atrium, its conduction slows to allow complete contraction of both atrial chambers before the impulse reaches the ventricles
 - After the AV node, conduction velocity increases as the impulse is relayed through the AV bundle into the ventricles
 - Right and left branches of the bundle fibers and Purkinje fibers conduct the impulses throughout

the muscles of both ventricles, stimulating them to contract almost simultaneously

- B. Electrocardiogram (ECG or EKG)
 1. Graphic record of the heart's electrical activity, its conduction of impulses; a record of the electrical events that precede the contractions of the heart
 2. To produce an ECG (Figure 19-2)
 - a. Electrodes of an electrocardiograph are attached to the subject
 - b. Changes in voltage are recorded that represent changes in the heart's electrical activity
 3. Normal ECG (Figures 19-3 and 19-4) is composed of
 - a. P wave—represents depolarization of the atria
 - b. QRS complex—represents depolarization of the ventricles and repolarization of the atria
 - c. T wave—represents repolarization of the ventricles; may also have a U wave that represents repolarization of the papillary muscle
 - d. Measurement of the intervals between P, QRS, and T waves can provide information about the rate of conduction of an action potential through the heart
- C. Cardiac cycle—a complete heartbeat consisting of contraction (systole) and relaxation (diastole) of both atria and both ventricles; the cycle is often divided into time intervals (Figures 19-5 and 19-6)
 1. Atrial systole
 - a. Contraction of atria completes emptying blood out of the atria into the ventricles
 - b. AV valves are open; SL valves are closed
 - c. Ventricles are relaxed and filling with blood
 - d. This cycle begins with the P wave of the ECG
 2. Isovolumetric ventricular contraction
 - a. Occurs between the start of ventricular systole and the opening of the SL valves
 - b. Ventricular volume remains constant as the pressure increases rapidly
 - c. Onset of ventricular systole coincides with the R wave of the ECG and the appearance of the first heart sound
 3. Ejection
 - a. SL valves open and blood is ejected from the heart when the pressure gradient in the ventricles exceeds the pressure in the pulmonary artery and aorta
 - b. Rapid ejection—initial, short phase is characterized by a marked increase in ventricular and aortic pressure and in aortic blood flow
 - c. Reduced ejection—characterized by a less abrupt decrease in ventricular volume, coincides with the T wave of the ECG
 4. Isovolumetric ventricular relaxation
 - a. Ventricular diastole begins with this phase
 - b. Occurs between closure of the SL valves and opening of the AV valves
 - c. A dramatic fall in intraventricular pressure but no change in volume
- D. Heart sounds
 1. Systolic sound—first sound, believed to be caused primarily by the contraction of the ventricles and by vibrations of the closing AV valves
 2. Diastolic sound—short, sharp sound; thought to be caused by vibrations of the closing of SL valves
 3. Heart sounds have clinical significance because they give information about the functioning of the valves of the heart
- E. The second heart sound is heard during this period
5. Passive ventricular filling
 - a. Returning venous blood increases intraatrial pressure until the AV valves are forced open and blood rushes into the relaxing ventricles
 - b. Influx lasts approximately 0.1 second and results in a dramatic increase in ventricular volume
 - c. Diastasis—later, longer period of slow ventricular filling at the end of ventricular diastole lasting approximately 0.2 second; characterized by a gradual increase in ventricular pressure and volume

PRIMARY PRINCIPLE OF CIRCULATION

- A. Blood flows because a pressure gradient exists between different parts of its bed; this is based on Newton's first and second laws of motion (Figure 19-7)
- B. Blood circulates from the left ventricle to the right atrium of the heart because a blood pressure gradient exists between these two structures
- C. P_1-P_2 is the symbol used to stand for a pressure gradient, with P_1 representing the higher pressure and P_2 the lower pressure

ARTERIAL BLOOD PRESSURE

- A. Primary determinant of arterial blood pressure is the volume of blood in the arteries; a direct relationship exists between arterial blood volume and arterial pressure (Figure 19-8)
- B. Cardiac output (CO)—determined by stroke volume and heart rate
 1. General principles and definitions
 - a. Stroke volume (SV)—volume pumped per heartbeat
 - b. CO (volume/min)—SV (volume/beat) times HR (beats/min)
 - c. In practice, CO is computed by Fick's formula
 - d. Heart rate and stroke volume determine CO, so anything that changes either also tends to change CO, arterial blood volume, and blood pressure in the same direction
 2. Stroke volume—influenced mainly by Starling's Law of the Heart (Figure 19-9)
 - a. Within limits, the longer, or more stretched, the heart fibers at the beginning of contraction, the stronger the contraction

- b. The amount of blood in the heart at the end of diastole determines the amount of stretch placed on the heart fibers
3. Factors that affect heart rate—SA node normally initiates each heartbeat; however, various factors can and do change the rate of the heartbeat
 - a. Cardiac pressoreflexes—aortic baroreceptors and carotid baroreceptors, located in the aorta and carotid sinus, are extremely important because they affect the autonomic cardiac control center, and therefore parasympathetic and sympathetic outflow, to aid in control of blood pressure (Figures 19-10 and 19-11)
 - (1) Carotid sinus reflex
 - (a) Carotid sinus is located at the beginning of the internal carotid artery
 - (b) Sensory fibers from carotid sinus baroreceptors run through the carotid sinus nerve and the glossopharyngeal nerve to the cardiac control center
 - (c) Parasympathetic impulses leave the cardiac control center, travel through the vagus nerve to reach the SA node
 - (2) Aortic reflex—sensory fibers extend from baroreceptors located in the wall of the arch of the aorta through the aortic nerve and through the vagus nerve to terminate in the cardiac control center
 - b. Other reflexes that influence heart rate—various important factors influence the heart rate; reflexive increases in heart rate often result from increased sympathetic stimulation of the heart
 - (1) Anxiety, fear, and anger often increase heart rate
 - (2) Grief tends to decrease heart rate
 - (3) Emotions produce changes in heart rate through the influence of impulses from the cerebrum via the hypothalamus
 - (4) Exercise—heart rate normally increases
 - (5) Increased blood temperature or stimulation of skin heat receptors increases heart rate
 - (6) Decreased blood temperature or stimulation of skin cold receptors decreases heart rate
- C. Peripheral resistance—resistance to blood flow imposed by the force of friction between blood and the walls of its vessels
 1. How resistance influences blood pressure
 - a. Arterial blood pressure tends to vary directly with peripheral resistance
 - b. Friction due to viscosity and small diameter of arterioles and capillaries
 - c. Muscular coat of arterioles allows them to constrict or dilate and change the amount of resistance to blood flow
 - d. Peripheral resistance helps determine arterial pressure by controlling the amount of blood that runs from the arteries to the arterioles; increased resistance, decreased arteriole runoff leads to higher arterial pressure
 2. Vasomotor control mechanism—controls changes in the diameter of arterioles; plays role in maintenance of the general blood pressure and in distribution of blood to areas of special need (Figures 19-12 and 19-13)
 - a. Vasomotor pressoreflexes (Figure 19-14)
 - (1) Sudden increase in arterial blood pressure stimulates aortic and carotid baroreceptors; results in arterioles and venules of the blood reservoirs dilating
 - (2) Decrease in arterial blood pressure results in stimulation of vasoconstrictor centers, causing vascular smooth muscle to constrict
 - b. Vasomotor chemoreflexes (Figure 19-15)—chemoreceptors located in aortic and carotid bodies are sensitive to hypercapnia, hypoxia, and decreased arterial blood pH
 - c. Medullary ischemic reflex—acts during emergency situation when there is decreased blood flow to the medulla; causes marked arteriole and venous constriction
 - d. Vasomotor control by higher brain centers—impulses from centers in cerebral cortex and hypothalamus are transmitted to vasomotor centers in medulla to help control vasoconstriction and dilation
 3. Local control of arterioles—several local mechanisms produce vasodilation in localized areas; referred to as reactive hyperemia

VENOUS RETURN TO HEART

- A. Venous pumps—blood-pumping action of respirations and skeletal muscle contractions facilitate venous return by increasing pressure gradient between peripheral veins and venae cavae (Figure 19-16)
 1. Respirations—inspiration increases the pressure gradient between peripheral and central veins by decreasing central venous pressure and also by increasing peripheral venous pressure
 2. Skeletal muscle contractions—promote venous return by squeezing veins through a contracting muscle and milking the blood toward the heart
 3. Semilunar valves in veins prevent backflow (Figure 19-17)
- B. Total blood volume—changes in total blood volume change the amount of blood returned to the heart
 1. Capillary exchange—governed by Starling's Law of the Capillaries (Figure 19-18)
 - a. At arterial end of capillary, outward hydrostatic pressure is strongest force; moves fluid out of plasma and into IF
 - b. At venous end of capillary, inward osmotic pressure is strongest force; moves fluid into plasma from IF; 90% of fluid lost by plasma at arterial end is recovered

- c. Lymphatic system recovers fluid not recovered by capillary and returns it to the venous blood before it is returned to the heart
- 2. Changes in total blood volume—mechanisms that change total blood volume most quickly are those that cause water to quickly move into or out of the plasma (Figure 19-19)
 - a. ADH mechanism—decreases the amount of water lost by the body by increasing the amount of water that kidneys reabsorb from urine before the urine is excreted from the body; triggered by input from baroreceptors and osmoreceptors
 - b. Renin-angiotensin mechanism
 - (1) Renin—released when blood pressure in kidney is low; leads to increased secretion of aldosterone, which stimulates retention of sodium, causing increased retention of water and an increase in blood volume
 - (2) Angiotensin II—intermediate compound that causes vasoconstriction, which complements the volume-increasing effects of renin and promotes an increase in overall blood flow
 - c. ANH mechanism—adjusts venous return from an abnormally high level by promoting the loss of water from plasma, causing a decrease in blood volume; increases urine sodium loss, which causes water to follow osmotically

MEASURING BLOOD PRESSURE

- A. Arterial blood pressure
 - 1. Measured with the aid of a sphygmomanometer and stethoscope; listen for Korotkoff sounds as the pressure in the cuff is gradually decreased (Figure 19-20)
 - 2. Systolic blood pressure—force of the blood pushing against the artery walls while ventricles are contracting
 - 3. Diastolic blood pressure—force of the blood pushing against the artery walls when ventricles are relaxed
 - 4. Pulse pressure—difference between systolic and diastolic blood pressure
- B. Relation to arterial and venous bleeding
 - 1. Arterial bleeding—blood escapes from artery in spurts due to alternating increase and decrease of arterial blood pressure
 - 2. Venous bleeding—blood flows slowly and steadily due to low, practically constant pressure

MINUTE VOLUME OF BLOOD (Figure 19-21)

- A. Minute volume—determined by the magnitude of the blood pressure gradient and peripheral resistance
- B. Poiseuille's Law—Minute volume = Pressure gradient ÷ Resistance

VELOCITY OF BLOOD FLOW

- A. Velocity of blood is governed by the physical principle that when a liquid flows from an area of one cross-sectional size to an area of larger size, its velocity decreases in the area with the larger cross section (Figure 19-22)
- B. Blood flows more slowly through arterioles than arteries because total cross-sectional area of arterioles is greater than that of arteries and capillary blood flow is slower than arteriole blood flow
- C. Venule cross-sectional area is smaller than capillary cross-sectional area, causing blood velocity to increase in venules and then veins with a still smaller cross-sectional area

PULSE

- A. Mechanism
 - 1. Pulse—alternate expansion and recoil of an artery
 - a. Clinical significance: reveals important information regarding the cardiovascular system, blood vessels, and circulation
 - b. Physiological significance: expansion stores energy released during recoil, conserving energy generated by the heart and maintaining relatively constant blood flow
 - 2. Existence of pulse is due to two factors
 - a. Alternating increase and decrease of pressure in the vessel
 - b. Elasticity of arterial walls allows walls to expand with increased pressure and recoil with decreased pressure
- B. Pulse wave (Figure 19-23)
 - 1. Each pulse that starts with ventricular contraction and proceeds as a wave of expansion throughout the arteries
 - 2. Gradually dissipates as it travels, disappearing in the capillaries
- C. Where pulse can be felt—wherever an artery lies near the surface and over a bone or other firm background (Figure 19-24)
- D. Venous pulse—detectable pulse exists only in large veins; most prominent near the heart; not of clinical importance

THE BIG PICTURE: BLOOD FLOW AND THE WHOLE BODY

- A. Blood flow shifts materials from place to place and redistributes heat and pressure
- B. Vital to maintaining homeostasis of internal environment

REVIEW QUESTIONS

1. Identify, locate, and describe the function of each of the following structures: SA node, AV node, AV bundle, and Purkinje fibers.
2. What does an electrocardiogram measure and record? List the normal ECG deflection waves and intervals. What do the various ECG waves represent?
3. What is meant by the term *cardiac cycle*?
4. List the “phases” of the cardiac cycle and briefly describe the events that occur in each.
5. What is meant by the term *residual volume* as it applies to the heart?
6. Describe and explain the origin of the heart sounds.
7. What blood vessels present the greatest resistance to blood flow?
8. What is the primary determinant of arterial blood pressure?
9. List the two most important factors that indirectly determine arterial pressure by their influence on arterial volume.
10. How is cardiac output determined?
11. List and give the effect of several factors such as grief or pain on heart rate.
12. What mechanisms control peripheral resistance? Cite an example of the operation of one or more parts of this mechanism to increase resistance and to decrease it.
13. What are the components of the vasomotor control mechanism?
14. Explain how antidiuretic hormone can change the total blood volume.
15. What is the effect of low blood pressure in relation to aldosterone and antidiuretic hormone secretion?
16. Describe the measurement of arterial blood pressure.
17. Identify the eight locations where the pulse point is most easily felt. List the six pressure points at which pressure can be applied to stop arterial bleeding distal to that point.
18. Describe the various types of cardiac dysrhythmias.

CRITICAL THINKING QUESTIONS

1. What is an ectopic pacemaker? What would be the effect on the heart rate if an ectopic pacemaker took over for the SA node?
2. State in your own words the primary principle of circulation. How does it govern the various mechanisms involved in blood flow?
3. What is Starling’s Law of the Heart? What role does *venous return* play in Starling’s law?
4. Explain how the heart rate is a good example of dual innervation in the autonomic nervous system. Include the nerves and neurotransmitters involved.
5. Explain how blood pressure in the brain is kept from getting too high.
6. What is arterial runoff? What is the relationship of arteriole runoff and peripheral resistance?
7. Explain what occurs when the carbon dioxide level in the medulla rises above set point.
8. By the time blood gets to the veins, almost all the pressure from the contracting ventricles has been lost. What mechanisms does the body use to assist in returning blood to the heart?
9. Explain the forces that act on capillary exchange on both the arterial and venous ends of the capillary. Is the recovery of fluid at the venous end 100% effective?
10. State in your own words Poiseuille’s law. What is the equation that expresses this law? According to the equation, an increase in resistance would lower the minute volume. Give an example of where this would not be the case.
11. What two factors determine blood viscosity? What does viscosity mean? Give an example of a condition in which blood viscosity decreases. Explain its effects on resistance, arterial pressure, and circulation.