Function of the Heart

chapter 17

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Key Terms

afterload (p. 331)
cardiac cycle (p. 325)
cardiac reserve (p. 329)
chronotropic effect (p. 331)
diastole (p. 325)
dromotropic effect (p. 331)
ejection fraction (p. 330)
heart failure (p. 327)
inotropic effect (p. 329)
preload (p. 330)
pulmonary edema (PE) (p. 332)
Starling's law of the heart (p. 329)
stroke volume (p. 329)
systole (p. 325)

Objective

1. Define *cardiac cycle* with respect to systole and diastole.
2. Describe the autonomic innervation of the heart, including:
   - Define *cardiac output*.
   - Describe the effect of Starling's law of the heart on myocardial contraction.
   - Describe the inotropic effect on myocardial contraction.
   - Explain how changes in heart rate and/or stroke volume change cardiac output.
3. Define specific clinical vocabulary used to describe cardiac function, including:
   - Define *preload* (end diastolic volume) and explain how it affects cardiac output.
   - Define *afterload* and identify the major factor that determines afterload.
4. Define *heart failure* and differentiate between right-sided and left-sided heart failure.

The heart functions as a pump that supplies blood to every cell in the body. Moreover, the heart is an adaptable pump. For example, the heart alters its pumping activity to meet the demands of day-to-day physiological functions such as eating, exercise, and responding to changes in environmental temperature; it also adapts to disease. How does the heart know when to beat faster or slower, or weaker or stronger? The coordinated and adaptable heart is the focus of the first part of this chapter, "The Coordinated and Adaptable Pump." The second part of this chapter, "Heart Talk," defines terminology that is commonly used in clinical situations. The third part, "The Failing Heart: When the Heart Can't Pump," describes the failing heart.

THE COORDINATED AND ADAPTABLE PUMP

CARDIAC CYCLE

The **cardiac cycle** is the sequence of events that occurs during one heartbeat. A cardiac cycle is a coordinated contraction and relaxation of the chambers of the heart. Contraction of the heart muscle (myocardium) is called **systole** (SIS-toh-lee). Contraction of the heart muscle during systole pumps blood out of a chamber. Relaxation of the myocardium is called **diastole** (dye-ASS-toh-lee). Blood fills a chamber during diastole.

Atrial and ventricular muscle activity is closely coordinated. For example, during atrial systole, the ventricles are in diastole. In this way, when the atria contract, they pump blood into the relaxed ventricles.

The cardiac cycle has three stages (Figure 17-1):

- *Atrial systole.* The atria contract (systole) and pump blood into the ventricles. During atrial systole, the atrioventricular (AV) valves are open and the ventricles are relaxed.
- *Ventricular systole.* At the end of atrial systole, the ventricles contract; this is called *ventricular systole.* As ventricular contractions begin, blood is forced against the AV valves, causing them to snap shut. The blood pushes the semilunar valves open, allowing blood to flow into the pulmonary artery and aorta.
- *Diastole.* For a brief period during the cardiac cycle, both the atria and the ventricles are in diastole. As the chambers relax, blood flows into the atria. Because the AV valves are open at this time, much of this blood also flows passively into the ventricles. The period of diastole therefore is a period of filling (of blood); atrial systole follows. The cycle then repeats itself.

How long does the cardiac cycle last? With a heart rate of 70 beats/min, the duration of the cardiac cycle is 0.8 second. All chambers rest for 0.4 second. As heart rate increases, the duration of the cardiac cycle

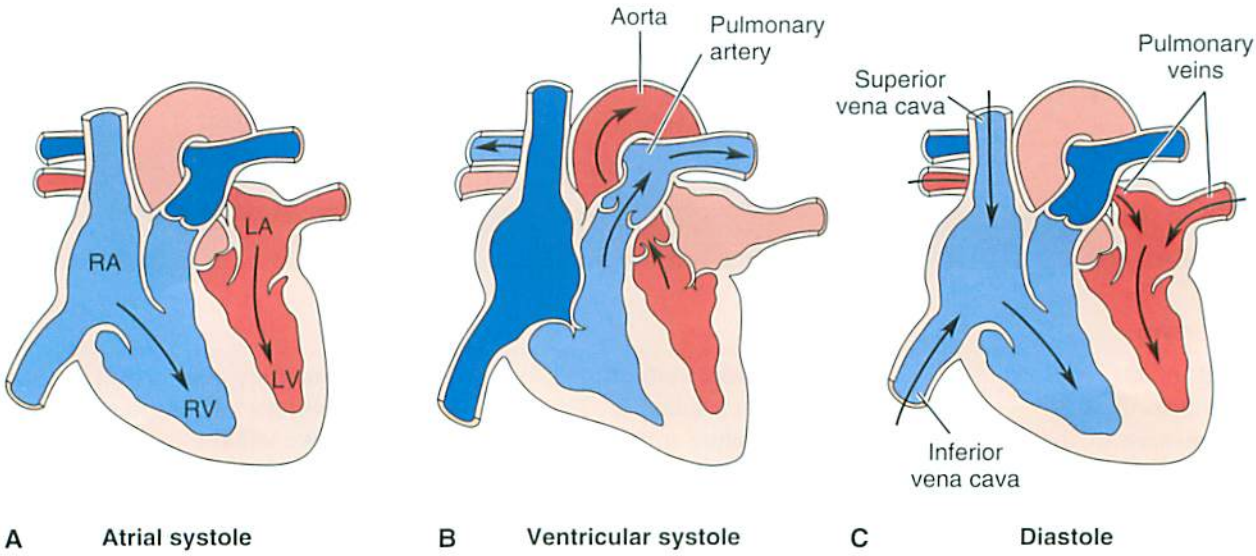

FIGURE 17-1 Stages of the cardiac cycle. **A,** Atrial systole. **B,** Ventricular systole. **C,** Diastole.

shortens. With a dramatic increase in heart rate, the period of rest (diastole) may shorten so much that cardiac function is compromised. Why? First, because of a decreased filling time, the amount of blood that enters the ventricles decreases so that less blood is pumped. Second, because coronary blood flow to the myocardium occurs during diastole, the diminished period of diastole decreases coronary blood flow.

NOTE: The valves of the heart open and close in response to pressure changes. Think of the valves during the three stages of the cardiac cycle. Are they open or closed?

? Re-Think

1. Define systole and diastole.
2. Why is it important that the atria and ventricles do not contract and relax at the same time?

2+2 Sum It Up!

The cardiac cycle is the sequence of events that occurs during one heartbeat. The cardiac cycle has three stages: atrial systole, ventricular systole, and diastole. Cardiac muscle contraction is called *systole;* cardiac muscle relaxation is called *diastole*. The duration of the cardiac cycle (resting heart) is 0.8 second; duration changes with a change in heart rate.

AUTONOMIC CONTROL OF THE HEART

The autonomic nervous system (ANS) plays an important role in coordinating and adapting cardiac function.

WHY THE AUTONOMIC NERVOUS SYSTEM?

In the previous chapter, we learned that specialized cardiac tissue displays automaticity and rhythmicity. The electrical signal, the cardiac impulse, arises within the sinoatrial (SA) node and then spreads throughout the heart, causing the heart muscle to contract. If the heart is capable of initiating its own cardiac impulse, why are the autonomic nerves needed? Although the ANS does not cause the cardiac impulse, it can affect the rate at which the cardiac impulse is fired and the speed at which it travels throughout the heart. The ANS can also make the heart muscle contract more forcefully; thus, the ANS can change the pumping activity of the heart. For example, if a person suddenly sprints down the street, his or her heart autonomically or automatically responds to the increased need for more oxygenated blood; it beats faster and stronger.

AUTONOMIC WIRING

As described in Chapter 12 the ANS has two branches: the sympathetic and parasympathetic branches. Refer to Figure 17-2 and note the autonomic wiring of the heart. Sympathetic nerves supply the SA node, AV node, and ventricular myocardium (the sarcomere represents heart muscle). Parasympathetic nerves, also called the *vagus nerve*, innervate the SA node and the AV node; there is no parasympathetic innervation of the ventricular myocardium.

AUTONOMIC FIRING

What happens when the ANS fires?

Sympathetic Stimulation

- Increases SA node activity, thus increasing heart rate
- Increases the speed at which the cardiac impulse travels from the SA node throughout the His-Purkinje system
- Increases the force of myocardial contraction

There are four clinically important points regarding excess sympathetic activity on the heart and blood vessels:

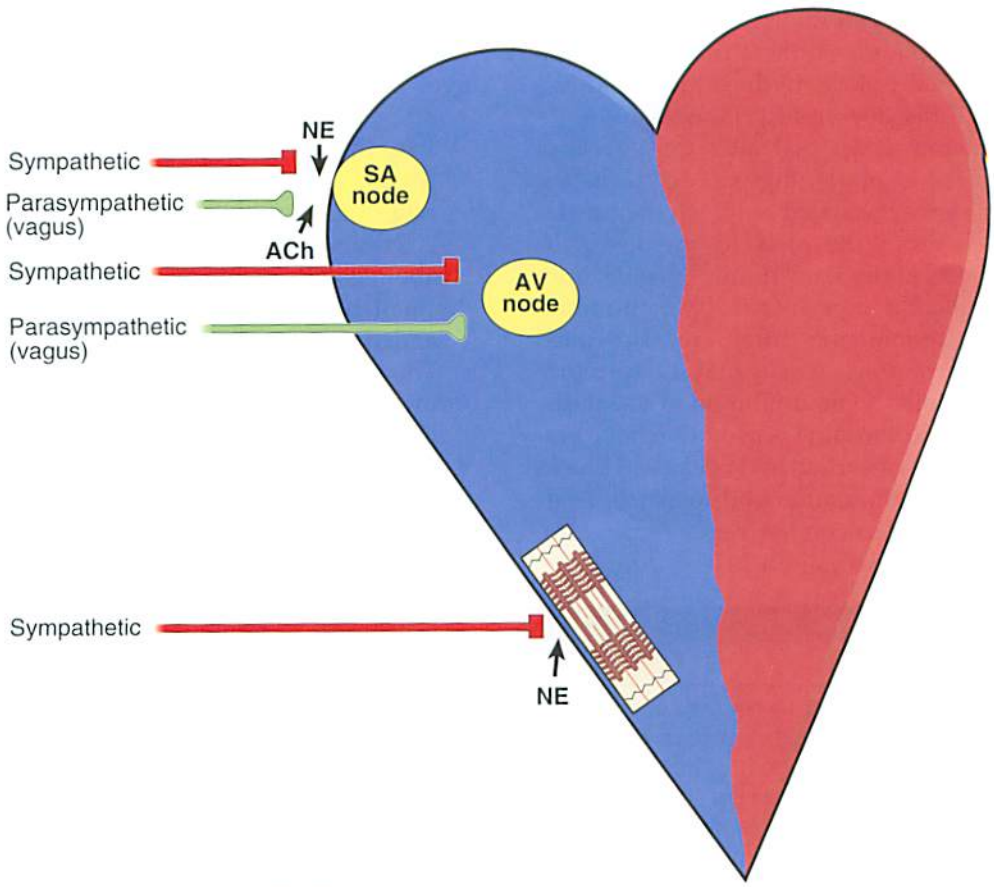

FIGURE 17-2 Autonomic innervation of the heart.

- Excess sympathetic activity produces the "fight-or-flight" response. You will often observe these symptoms (racing and pounding heart) in people who are very anxious, such as during panic attacks. The symptoms are frightening, and the person is convinced of an impending heart attack. Many trips to the emergency room (ER) are panic related.
- Excess sympathetic activity plays a key role in some disease states. For example, most of the signs and symptoms of circulatory shock are caused by excess sympathetic firing. Similarly, the progressive deterioration of patients with **heart failure** is in large measure caused by persistent sympathetic nerve stimulation. Some of the drugs used to treat heart failure are aimed at minimizing the sympathetic effects.
- Excess sympathetic activity often causes tachydysrhythmias ("fast" rhythm disorders).
- You will be giving drugs that resemble or block the effects of sympathetic activity. For example, epinephrine (Adrenalin) and dopamine increase heart rate and myocardial contractile force. Because the effects mimic stimulation of the sympathetic nervous system (SNS), these drugs are called *sympathomimetic drugs*. You will also be giving drugs that block the sympathetic effects, including beta- and alpha-adrenergic blockers.

Parasympathetic (Vagus Nerve) Stimulation

- Decreases SA node activity, thereby decreasing heart rate
- Decreases the speed at which the cardiac impulse travels from the SA node to and through the AV node
- Exerts no effect on the strength of myocardial contraction because there are no parasympathetic (vagal) fibers innervating the ventricular myocardium

There are three clinically important points about parasympathetic (vagal) nervous system activity:

- In the resting heart, vagal tone is more intense than sympathetic activity. For example, the SA node would like to fire at a rate of 90 beats/min. However, the "braking" or inhibiting effect of the vagus nerve slows SA node firing to a rate of 72 beats/min, the normal resting heart rate. If the vagus nerve were interrupted, the heart rate would increase to 90 beats/min.
- Certain conditions (heart attack) and drugs (digoxin) can cause excess vagal discharge. Excess vagal discharge causes *bradycardia* (bray-dee-KAR-dee-ah) (<60 beats/min) and increases the tendency of the heart to develop life-threatening electrical rhythm disturbances. Excess vagal discharge also slows the conduction of the cardiac impulse through the heart,

causing heart block (a condition in which the signal has difficulty traveling from the atria to the ventricles). The abnormally slow rhythms are called *bradydysrhythmias* (bray-dee-diss-RITH-mee-ahs).

- You will be giving drugs that alter the effects of vagal activity. For example, digoxin decreases the heart rate and slows the speed at which the cardiac impulse travels from the atria to the ventricle. Because the effects of digoxin "mimic" vagal stimulation, it is called a *vagomimetic* (vag-oh-mihm-EHT-ik) *drug*. Vagomimetic drugs are also called *parasympathomimetic drugs*. A drug may also produce effects that are similar to an inhibition of vagal discharge. For example, atropine is used to relieve bradycardia (following a heart attack) because it blocks the effects of vagal stimulation and increases heart rate. Because of its effects on the vagus nerve, atropine is called a *vagolytic* (vag-oh-LIT-ik) *drug*.

Do You Know...

Why a Patient May "Brady-Down"?

Sometimes a patient may respond to fear or anxiety by an intense discharge of the parasympathetic (vagus) nerves. Vagal discharge to the heart causes a dangerous bradycardia and decline in cardiac output and blood pressure. The administration of a vagolytic drug, such as atropine, relieves the vagal effects and restores the heart rate to normal.

Re-Think

1. If the electrical signal (cardiac impulse) arises within the SA node spontaneously, why is the heart innervated by the ANS?
2. Describe a sympathomimetic effect on heart rate and force of myocardial contraction.
3. Describe a vagomimetic effect on heart rate.

CARDIAC OUTPUT

To understand how the heart alters its pumping activity, you must understand cardiac output. Cardiac output is the amount of blood pumped by each ventricle in 1 minute. The normal cardiac output is about 5 L/min. Because the total blood volume is about 5 L (5000 mL), the entire blood volume is pumped through the heart every minute.

Two factors determine cardiac output: heart rate and stroke volume.

Cardiac Output = Heart Rate × Stroke Volume

Heart Rate

The heart rate (HR) is the number of times the heart beats each minute. The heart rate is caused by the rhythmic firing of the SA node. The normal adult resting heart rate is between 60 and 100 beats/min, with an average of 72 beats/min. Resting heart rates differ for many reasons, including size, gender, and age.

- *Size.* Size affects heart rate; generally, the larger the size, the slower the rate. Our feathered and furry friends dramatically illustrate this point. The heart rate of a hummingbird, for example, is more than 200 beats/min, whereas that of a grizzly bear is only about 30 beats/min. Why do heart rates differ? The small hummingbird has a very high metabolism and therefore requires a large amount of oxygen. The metabolism of a grizzly is much slower, requiring less oxygen. Similarly, a tiny baby has a faster heart rate than a larger adult.
- *Gender.* Women have slightly faster heart rates than men.
- *Age.* Generally, the younger the person, the faster the rate. The normal adult heart rate, for example, is 70 to 80 beats/min, whereas a normal child's heart rate is around 100 beats/min. An infant's heart rate is about 120 beats/min, and fetal heart rates are about 140 beats/min.

In addition to the variation in heart rate according to size, age, and gender, a person's heart rate can change for a variety of other reasons, such as exercise, stimulation of the autonomic nerves, hormonal influence, pathology, and various medications.

- *Exercise.* Exercise increases heart rate. Check your pulse as you exercise and note the increase; also note the decrease in pulse when you rest. At rest, the heart rate may be 65 beats/min but may increase to well over 100 beats/min with exercise.
- *Stimulation of the autonomic nerves.* Firing of the sympathetic nerve stimulates the SA node, causing an increase in heart rate. Stimulation of the parasympathetic (vagus) nerve slows the rate.
- *Hormonal influence.* Several hormones affect heart rate. Epinephrine and norepinephrine (adrenal gland hormones) and thyroid hormone increase heart rate.
- *Pathology.* Certain disease states can affect heart rate. For example, a sick SA node may fire too slowly, thereby slowing the heart too much. Vagal discharge following a heart attack (myocardial infarction) slows the rate, predisposing the heart to lethal rhythm disorders. A high fever and persistent sympathetic activity can increase heart rate, overworking the heart and causing it to fail.
- *Medications.* Certain drugs can affect heart rate. Digoxin slows heart rate, whereas epinephrine and dopamine increase heart rate. Heavy coffee drinkers often experience palpitations (the heart feels "jumpy," as if it has extra beats) because of the stimulatory effect of caffeine on the heart. Because some drugs can profoundly alter heart rate, heart rate must be monitored when these drugs are used. For example, digoxin should not be administered if the heart rate is less than 60 beats/min.

Stroke Volume

Stroke volume is the second factor affecting cardiac output. **Stroke volume** is the amount of blood pumped by the ventricle per beat. An average resting stroke volume is 60 to 80 mL/beat (about 2 ounces). At rest, the ventricles pump out only about 67% of the blood in the ventricles. Therefore, if the ventricles can be made to contract more forcefully, a greater percentage of the blood can be pumped per beat. In other words, a greater force of contraction can increase stroke volume.

? Re-Think

1. What two factors determine cardiac output?
2. Explain why cardiac output can remain unchanged if heart rate changes.

How to Change Stroke Volume

The stroke volume can be altered in two ways: through Starling's law of the heart and through an inotropic effect.

Starling's Law of the Heart. **Starling's law of the heart** depends on the degree of stretch of the myocardial fibers. The greater the stretch, the stronger the force of contraction. For example, an increase in the amount of blood entering the ventricle, the venous return, causes the ventricle to stretch (Figure 17-3). Stretch increases the force of contraction, which in turn increases stroke volume. Conversely, a decrease in the amount of blood entering the ventricles causes less stretch. As a result, the force of contraction decreases, thereby decreasing stroke volume.

What is the purpose of Starling's law of the heart? It allows the heart to pump out the same amount of blood it receives. In other words, Starling's law allows the heart to match cardiac output with venous return (of blood).

Do You Know...

About Atrial Kick?

About 70% of blood flows passively from the atria into the ventricles. Atrial contraction (systole) accounts for the remaining 30%; it is called the *atrial kick*. With atrial fibrillation the contribution from atrial kick is missing and cardiac output declines. Atrial kick—it's a good thing!

Inotropic Effect. A second way to increase stroke volume is by strengthening the force of myocardial contraction without stretching the myocardial fibers. This is called a positive **inotropic** (in-o-TROH-pik) **effect.** Stimulation of the heart muscle by sympathetic nerves causes a positive inotropic effect. Certain hormones and drugs, such as epinephrine, also cause this effect. Digoxin is the most famous of the positive inotropic drugs. Some medications cause a negative inotropic effect. A negative inotropic effect is a decrease in the force of contraction, resulting in a weaker myocardial contraction. Excessive dosing with a negative inotropic agent can depress the myocardium to the point of failure.

CHANGING CARDIAC OUTPUT

Because cardiac output is determined by heart rate and stroke volume, cardiac output can be altered by changing either or both. The healthy heart can increase cardiac output four to five times the resting cardiac output; an Olympic athlete can do even better! The capacity to increase cardiac output above the resting cardiac output is called the **cardiac reserve.** A person with a diseased heart may have little cardiac reserve and therefore becomes easily fatigued with mild exercise.

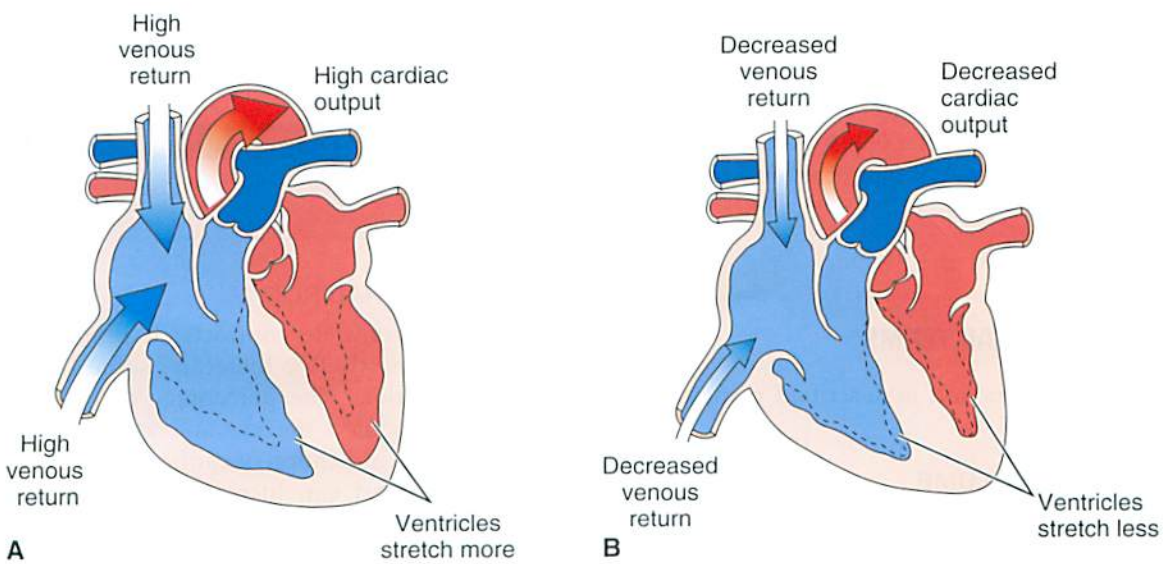

FIGURE 17-3 Starling's law of the heart, matching venous return with cardiac output. **A,** A large venous return. **B,** A smaller venous return.

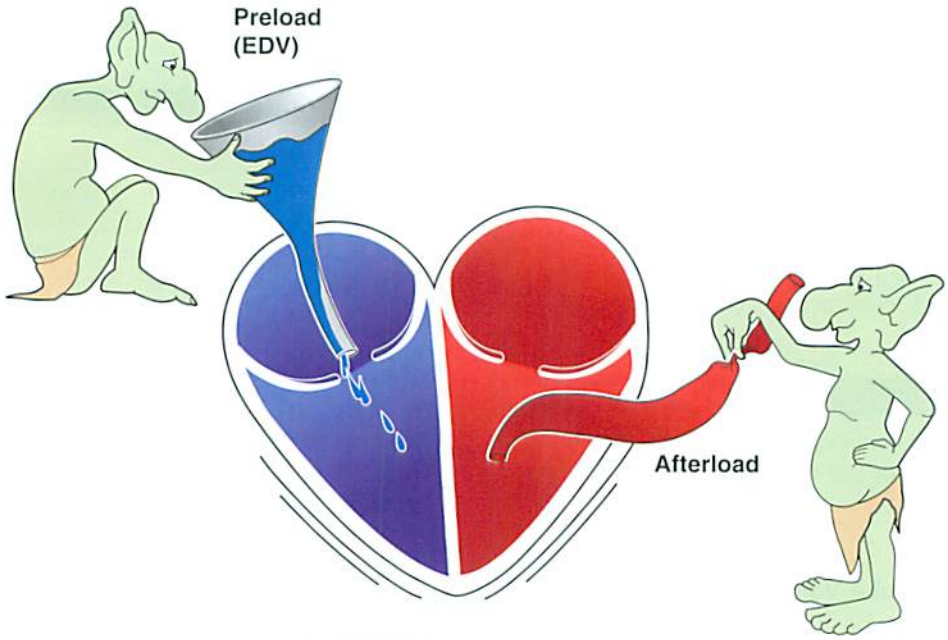

FIGURE 17-4 Preload and afterload.

? Re-Think

1. Explain how a Starling effect changes force of myocardial contraction.
2. Explain how an inotropic effect changes force of myocardial contraction.

2+2 Sum It Up!

Cardiac output is determined by multiplying heart rate by stroke volume. The heart rate is the number of times the heart beats each minute. The normal adult resting heart rate is between 60 and 100 beats/min (average, 72 beats/min). Heart rates differ for three main reasons: size, gender, and age. A person's heart rate may also change in response to exercise, stimulation of the ANS, hormonal influence, pathology, and various medications. Stroke volume is the amount of blood ejected by the ventricle in one beat. Stroke volume can be altered through Starling's law of the heart or an inotropic effect.

The following major sections, "Heart Talk" and "The Failing Heart: When the Heart Can't Pump," are clinically focused and may be omitted in nonmedical courses.

HEART TALK

HEART TALK: CLINICAL TERMS

Clinically, cardiac function is described using special vocabulary.

END-DIASTOLIC VOLUME

The end-diastolic volume (EDV) refers to the amount of blood in the ventricle at the end of its resting phase (diastole). Remember that the ventricle fills with blood during diastole. The EDV determines how much the ventricle is stretched and is the basis of Starling's law of the heart.

PRELOAD

The **preload** is the amount of blood in the ventricles at the end of diastole; this is the same as the EDV. Note in Figure 17-4 that the filling of the right heart by the funnel illustrates preload. An increased preload stretches the ventricles, causing a stronger force of contraction. The stronger contraction increases stroke volume and cardiac output. Drugs can also affect preload. For example, a drug may dilate the veins, causing blood to pool in the veins and decreasing the amount of blood returned to the heart, thus decreasing preload, stroke volume, and cardiac output. Another drug may constrict the veins; this increases blood flow to the ventricles, thereby increasing preload, stroke volume, and cardiac output.

? Re-Think

Explain why EDV, preload, and the Starling effect "make" the same point?

EJECTION FRACTION

When the ventricle contracts, it pumps about 67% of its volume (EDV); therefore, some blood remains in the ventricle. The percentage of the EDV that is pumped is called the **ejection fraction**. The ejection fraction is an indication of cardiac health. For example, a healthy heart can increase its ejection fraction to 90% with exercise. A weakened failing heart is characterized by a decrease in ejection fraction, perhaps as low as 30%.

AFTERLOAD

Afterload refers to resistance or opposition to the flow of blood. For example, for the left ventricle to pump blood into the aorta, it must push against blood that is already in the aorta. The aortic blood pressure is the resistance or the afterload. Note in Figure 17-4 that the pinched aorta represents an increased afterload, demanding that the left ventricle work harder to overcome the resistance. The pinched aorta can represent a number of clinical conditions such as aortic valve stenosis and systemic hypertension. If the person develops high blood pressure, afterload increases and the ventricle must work harder to pump blood into the aorta. Like any other muscle that overworks, the left ventricular myocardium enlarges or hypertrophies. The enlarged left ventricle will eventually fail as a pump.

What about afterload and the right ventricle? Its afterload is determined by the pressure within the pulmonary artery. If pulmonary artery pressure rises (increased afterload), the right ventricle must work harder to pump blood and therefore hypertrophies. Right ventricular hypertrophy frequently occurs in response to chronic lung diseases such as emphysema and asthma. The elevation in pulmonary artery pressure and right ventricular hypertrophy is called *cor pulmonale* (kohr pul-mah-NAL-ee). Cor pulmonale often causes the right ventricle to fail as a pump.

Afterload can be altered by drugs. For example, a drug that relaxes and dilates the blood vessels in the peripheral circulation can lower blood pressure and therefore decrease the afterload. The reduction in afterload reduces the work of the heart. Conversely, a drug that constricts blood vessels in the periphery increases afterload, thereby increasing the workload of the heart.

INOTROPIC EFFECT

An inotropic effect is a change in myocardial contraction that is not caused by stretch. A positive inotropic effect is an increase in contractile force, whereas a negative inotropic effect is a decrease in contractile force. Sympathetic nerve stimulation and hormones such as epinephrine and norepinephrine cause a positive inotropic effect.

CHRONOTROPIC EFFECT

A **chronotropic** (KRON-oh-TROH-pik) **effect** is a change in heart rate. Anything that increases heart rate exerts a positive chronotropic effect, whereas anything that decreases heart rate exerts a negative chronotropic effect. Sympathetic nerve stimulation causes a positive chronotropic effect, whereas vagal (parasympathetic) stimulation causes a negative chronotropic effect.

DROMOTROPIC EFFECT

A **dromotropic** (DROM-oh-TROH-pik) **effect** is a change in the speed at which the cardiac impulse travels from the SA node through the AV node and the His-Purkinje system. If the speed of the cardiac impulse increases, it is called a positive dromotropic effect; a decrease in the speed causes a negative dromotropic effect. Sympathetic nerve stimulation causes a positive dromotropic effect, whereas vagal (parasympathetic) stimulation causes a negative dromotropic effect. The negative dromotropic effect may be so pronounced that the person develops a heart block.

HEART TALK: RECEPTOR LANGUAGE

Heart talk often involves the autonomic receptors of the heart and their responses to autonomic stimulation and to drugs. (Review Chapter 12.)

BETA$_1$-ADRENERGIC RECEPTOR ACTIVATION

The sympathetic nerves innervate the SA node, AV node, His-Purkinje system, and ventricular myocardium. The neurotransmitter for the adrenergic neuron is norepinephrine. The cardiac adrenergic receptors for norepinephrine are called *beta$_1$-adrenergic receptors*. Activation of the beta$_1$ receptors causes a positive chronotropic effect, a positive dromotropic effect, and a positive inotropic effect. The increase in heart rate and stroke volume increases cardiac output. A drug that activates beta$_1$-adrenergic receptors is called a *beta$_1$-adrenergic agonist*. Examples include dopamine and epinephrine. Note that beta$_1$-adrenergic receptor activation is the same as a sympathomimetic effect.

BETA$_1$-ADRENERGIC RECEPTOR BLOCKADE

Blockade of the beta$_1$-adrenergic receptors prevents cardiac beta$_1$-adrenergic receptor activation. People taking beta$_1$-adrenergic blockers, such as propranolol, will not increase their heart rate when their sympathetic nerves fire as in exercise or stress. If a person is tachycardic (heart rate >100 beats/min) from excessive sympathetic nervous stimulation, a beta$_1$-adrenergic blocker can decrease the heart rate and the force of myocardial contraction; this results in a decrease in cardiac output and blood pressure.

? Re-Think

1. Why is a beta$_1$-adrenergic agonist called a *sympathomimetic drug?*
2. What is the name of the neurotransmitter that activates a beta$_1$-adrenergic receptor?

MUSCARINIC (CHOLINERGIC) RECEPTOR ACTIVATION

The parasympathetic (vagus) nerves supply the SA and the AV nodes. The neurotransmitter for the cholinergic neuron is acetylcholine (ACh). The cardiac cholinergic receptors for ACh are called *muscarinic receptors*. Activation of the muscarinic receptors causes a negative chronotropic effect and a negative

dromotropic effect. There is no effect on myocardial contractile force because there is no parasympathetic innervation to the ventricular myocardium. The administration of a cholinergic (muscarinic) agonist drug causes a negative chronotropic effect and a negative dromotropic effect. Note that muscarinic receptor activation is the same as a parasympathomimetic or vagomimetic effect.

MUSCARINIC (CHOLINERGIC) RECEPTOR BLOCKADE

The muscarinic receptors can also be blocked. Muscarinic-blocking drugs act by blocking the effects of ACh at the muscarinic receptors. Muscarinic blockade relieves the inhibiting effects of ACh at the receptors, thereby increasing heart rate and increasing the speed of the cardiac impulse from the atria to the ventricles. Atropine is an example of a muscarinic blocker that is often used to relieve bradycardia and heart block.

Note the duplication in terminology (your worst terminology nightmare!). The muscarinic receptors are activated by ACh. Because ACh is secreted by a cholinergic fiber, a muscarinic agonist is also called a *cholinergic agonist*. A muscarinic blocker is also called an *antimuscarinic agent*, a *cholinergic blocker*, or an *anticholinergic agent*. Unfortunately this terminology is used frequently in pharmacology, so you need to start building your vocabulary now.

? Re-Think

1. Why is a muscarinic agonist called a *vagomimetic drug?*
2. What is the name of the neurotransmitter that activates the muscarinic receptors?

2+2 Sum It Up!

Cardiac function is often described using special vocabulary, such as EDV, preload, ejection fraction, and afterload. Inotropic, chronotropic, and dromotropic effects describe changes in cardiac contractile force, heart rate, and conduction velocity through the heart. Cardiac function is also described in receptor terminology, specifically the beta$_1$-adrenergic and muscarinic receptors.

THE FAILING HEART: WHEN THE HEART CAN'T PUMP

The heart functions as a double pump. The right ventricle pumps blood to the lungs for oxygenation and the left ventricle pumps blood into the aorta for distribution to the systemic circulation. What happens when either or both pumps fail?

Do You Know...

Why BNP Is Used in the Assessment of Heart Failure?

Brain natriuretic peptide (BNP) is secreted by the walls of the ventricles in response to stretch. The failing heart is characterized by pooling of blood in the cardiac chambers (cardiac dilation), thereby increasing the secretion of BNP.

A new class of drugs that mimic BNP is also used to treat heart failure. The BNP-like drug increases the excretion of sodium in the urine, suppresses the renin-angiotensin system, and decreases sympathetic activity.

LEFT HEART FAILURE

When the left ventricle fails to pump blood into the aorta, two things happen: blood backs up in the lungs, and the heart is unable to pump a sufficient amount of blood to the systemic circulation.

BACKWARD FAILURE

What happens when the blood backs up (Figure 17-5)? The blood backs up into the structures "behind" the left ventricle—namely, the left atrium, pulmonary veins, and, most importantly, the pulmonary capillaries. The pooled blood increases the pressure within the pulmonary capillaries and forces fluid into the lungs. The presence of fluid in the lungs impairs oxygenation of blood. The accumulation of fluid within the lungs is called **pulmonary edema (PE)**. The signs and symptoms of PE are exertional dyspnea (difficulty breathing upon exertion), cyanosis (bluish appearance), blood-tinged sputum and cough, orthopnea (or-THOP-nee-ah) (inability to breathe while lying down), tachycardia, and restlessness. Because these symptoms are largely caused by the backup of blood behind the failed ventricle, the condition is called *backward failure*. Note that the signs and symptoms of left-sided heart failure are predominantly respiratory!

Do You Know...

About Two- and Three-Pillow Dyspnea?

A patient with heart failure may develop pulmonary edema and dyspnea. There is a simple way to determine the severity of the dyspnea: ask the patient how many pillows he uses at night. A patient with no pulmonary edema can lie flat and use a single pillow. As fluid collects in the alveoli (lungs), a patient must sit up to breathe (orthopnea). Thus, he tends to use two or three pillows, depending on the severity of the edema and dyspnea—hence the name *two-* or *three-pillow dyspnea*.

FORWARD FAILURE

There is also a "forward" component to left heart failure. For example, if the damaged left ventricle cannot pump enough blood to the systemic circulation, all the organs of the body receive inadequate oxygen. The decreased cardiac output causes additional signs and symptoms to develop. For example, the kidneys

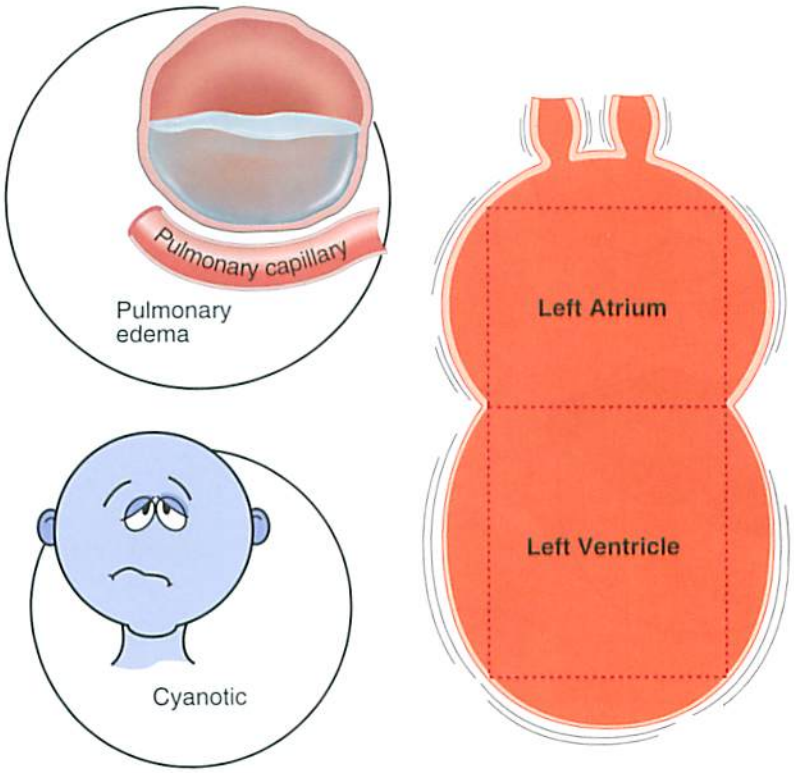

FIGURE 17-5 Left-sided heart failure.

filter less water for excretion as urine. The kidneys also reabsorb excess salt and water, causing an increase in blood volume and edema formation. The decrease in cardiac output also stimulates the sympathetic nervous system. Sympathetic activity stimulates the heart and blood vessels in a way that temporarily improves cardiac output. Over time, however, the improvement diminishes and additional signs of heart failure develop. The heart begins to show the wear and tear of continuous and excessive sympathetic activity.

What usually causes the left heart to fail? Two common causes are myocardial infarction (MI) and chronic hypertension (increased blood pressure). If a person suffers an acute MI, a part of the left ventricular myocardium may be destroyed. The damaged myocardium is unable to contract and fails as a pump. More commonly, the left heart fails in response to chronic hypertension. Increased blood pressure overworks the heart, eventually causing the left ventricle to enlarge (left ventricular hypertrophy) and then fail.

? Re-Think

1. Why does left-sided heart failure cause pulmonary edema?
2. Differentiate between forward and backward heart failure.
3. Explain why a positive inotropic drug, such as digoxin, relieves backward and forward heart failure.

RIGHT HEART FAILURE

When the right ventricle fails (Figure 17-6), blood backs up into the veins that return blood to the right heart. Blood backs up into the superior vena cava, thereby slowing venous drainage from the head via the jugular veins. The congestion of the jugular veins causes jugular vein distention (JVD), which is visible as the veins in the neck pulsate. Blood also backs up into the veins that drain the liver, spleen, and digestive organs, causing hepatomegaly (enlarged liver), splenomegaly (enlarged spleen), and digestive symptoms. Right-sided failure is also characterized by ankle, or pedal, edema; ankle edema can be so severe that the skin remains indented when you depress an area of skin with your thumb. This "indentation" response is called *pitting edema*.

The right heart most often fails as a consequence of left-sided failure; when one side of the heart fails, the other side will eventually fail. Another common cause is chronic lung disease, such as emphysema. The diseased lungs make it difficult for the right ventricle to pump blood into the pulmonary circulation. The overworked right ventricle becomes enlarged (right ventricular hypertrophy) and eventually fails.

? Re-Think

1. Why does right-sided heart failure cause JVD, hepatomegaly, and pedal edema rather than pulmonary edema?
2. Translate this into lay terminology. A patient with an ejection fraction of 35% was given a drug (digoxin) that exerted positive inotropic, negative dromotropic, and negative chronotropic effects.

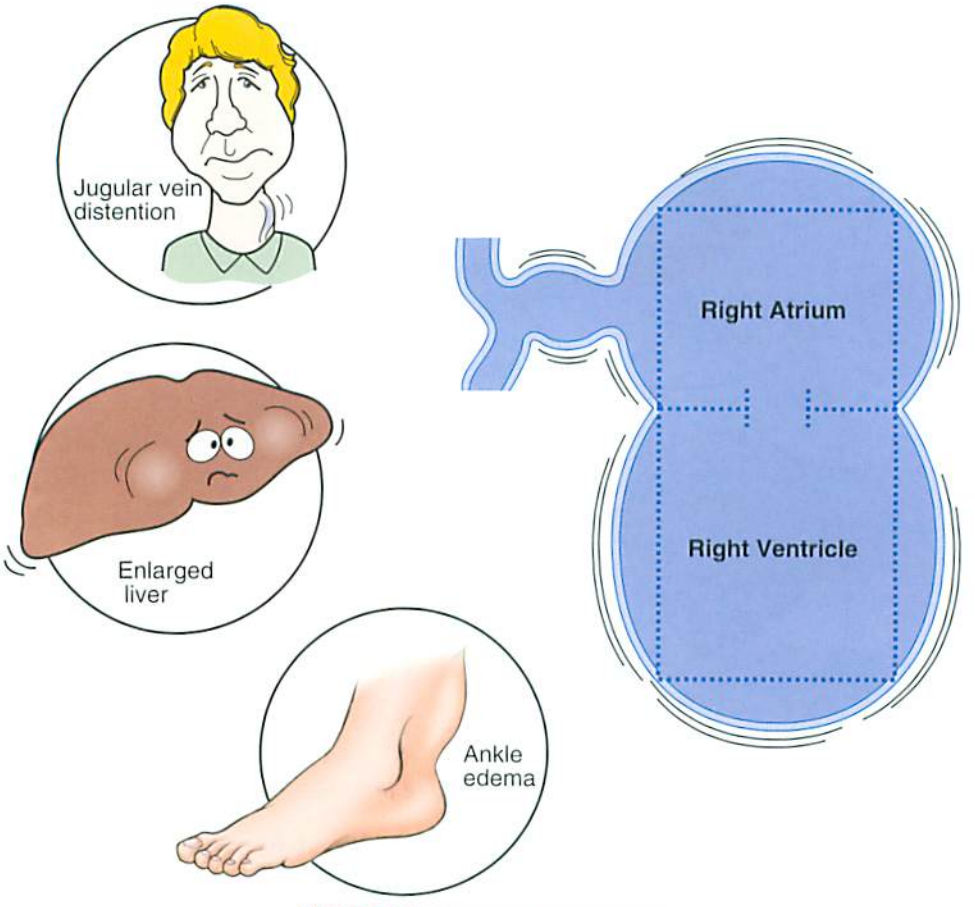

FIGURE 17-6 Right-sided heart failure.

Do You Know...

Why CVP Is Elevated in Heart Failure?

Central venous pressure (CVP) is considered a direct measurement of the pressure in the right atrium and superior vena cava (the large vein that empties blood into the right atrium). The CVP reading is obtained by threading a catheter through the subclavian vein into the superior vena cava. When the heart fails, blood backs up in the vena cava, thereby elevating venous pressure. Thus, an elevated CVP is indicative of a failing heart.

Let's see if you can follow the rationale for the treatment in the following case studies.

- *Case 1.* A patient in left-sided heart failure and pulmonary edema is given a positive inotropic drug, a potent diuretic, oxygen, and morphine. The rationale for this is that the failing heart muscle contracts weakly. A positive inotropic drug such as digoxin or dopamine strengthens myocardial contraction. The increased force of contraction results in an increased cardiac output, decreased pooling of blood in the pulmonary capillaries (decreases pulmonary edema), and increased blood flow to the kidney, which results in greater excretion of urine. A potent diuretic, such as furosemide (Lasix), blocks the absorption of sodium and water by the kidney, thereby ridding the body of excess water and decreasing the edema. The oxygen and morphine improve oxygenation and relieve the anxiety caused by poor oxygenation.
- *Case 2.* An older patient with symptoms of right-sided failure and a fast heart rate is given a $beta_1$-adrenergic blocker. The rationale for this is that heart failure is characterized by excess sympathetic nerve stimulation. Long-term sympathetic stimulation damages the heart, causing the heart to fail eventually. The administration of a $beta_1$-adrenergic receptor blocker diminishes the response to sympathetic activity and protects the heart from further damage.
- *Case 3.* An older patient has mild right-sided failure and is given an angiotensin-converting enzyme (ACE) inhibitor. An ACE inhibitor decreases afterload, acts like a diuretic, and decreases the secretion of aldosterone from the adrenal cortex. The decreased afterload decreases the workload of the heart. The diuretic effect excretes water and relieves the edema. The decrease in the secretion of aldosterone has two effects: it causes diuresis, and it prevents myocardial damage. (Chronic secretion of aldosterone causes structural damage, called *remodeling*, to the heart muscle.)

Clearly, the healthy heart is a magnificent, adaptable pump. Pump failure adversely affects the functioning of every organ system and is a common cause of disability and death.

2+2 Sum It Up!

The heart functions as a double pump. Either or both pumps can fail. When the left heart fails, fluid backs up into the lungs, causing pulmonary edema. The failing left heart is also unable to pump sufficient blood to the organs of the body; the result is poor tissue oxygenation. Right-sided failure causes JVD, ankle edema, and congestion of the abdominal organs. The treatment of heart failure is aimed at strengthening of myocardial contractile force, removing excess water, decreasing the work of the heart, and protecting the heart from excess sympathetic nerve activity.

As You Age

1. Contrary to popular opinion, no significant age-related decline occurs in resting cardiac output. When cardiac output declines, it is a result of age-related disease processes such as arteriosclerosis.
2. An age-related decline occurs in exercise cardiac output. The heart cannot respond as quickly or as forcefully to the increased workload of the exercised heart. Exertion, sudden movements, and changes in position may cause a decrease in cardiac output, resulting in dizziness, loss of balance, and falls.
3. Several structural changes in the heart contribute to the impaired response to exercise: heart muscle loses elasticity and becomes more rigid; heart valves become thicker and more rigid; the number of pacemaker cells decreases; and the aging heart cells have a decreased ability to use oxygen.
4. An age-related increase occurs in blood pressure, which increases the work the heart must do to pump blood into the systemic circulation.

MEDICAL TERMINOLOGY AND DISORDERS Disorders of the Heart

Medical Term	Word Parts	Word Part Meaning or Derivation	Description
Words			
bradycardia	brady-	abnormally slow	**Bradycardia** is *an abnormally slow heart rate (<60 beats/min).*
	-cardi/o-	heart	
	-ia	condition of	
cardiogram	cardi/o-	heart	A **cardiogram** is *a recording of the electrical events of the heart.*
	-gram	record	
ectopic	ec-	out of	An ectopic beat is one that originates outside the SA node. Examples include premature atrial and ventricular beats.
	-top/o-	place	
	-ic	condition of	
pericardium	peri-	around	The **pericardium** is *a slinglike membrane that surrounds the heart.*
	-cardi/o-	heart	
	-ium	tissue or structure	
tachycardia	tachy-	fast	**Tachycardia** is *an abnormally fast heart rate (>120 beats/min).*
	-cardi/o-	heart	
	-ia	condition of	
Disorders			
angina pectoris	angina	From the Greek word *ankhone,* meaning "to strangle"	**Angina pectoris,** also called *chest pain,* signals a diminished coronary blood flow to the heart. There are three forms of angina pectoris: 1. **Chronic stable angina (or exertional angina)** is triggered by physical activity and/or emotional excitement. The decreased coronary flow is due to changes associated with fatty plaque buildup (atherosclerosis). 2. **Variant angina (Prinzmetal's angina, vasospastic angina)** is caused by diminished blood flow resulting from spasm of the coronary arteries. 3. **Unstable angina** is a medical emergency; the diminished blood flow is due to severe coronary artery disease and complicated by vasospasm, rupture of the plaques, and formation of thrombi.
	pectoris	From the Latin word *pectus,* meaning "chest"	

Continued

MEDICAL TERMINOLOGY AND DISORDERS Disorders of the Heart—cont'd

Medical Term	Word Parts	Word Part Meaning or Derivation	Description
coronary artery disease and atherosclerosis	coronary	Related to the veins and arteries	Also referred to as **CAD,** or **coronary heart disease (CHD).** Coronary blood flow is reduced because of a progressive narrowing of the coronary blood vessels that deprives the myocardium of O_2 and nutrients. Diminished coronary blood flow is most often caused by **atherosclerosis,** an inflammation-induced accumulation of fatty plaques on the inner lining of the coronary blood vessels. With time the fatty plaques reduce the elasticity of the arteries, causing them to thicken and harden *(sclerosis);* the plaque reduces coronary blood flow and can eventually rupture, causing thrombotic occlusion of the coronary artery. Untreated, atherosclerosis commonly progresses to myocardial infarction. A **myocardial infarction (MI),** or **"heart attack,"** refers to *the death or necrosis* **(infarction)** *of the heart muscle* **(myocardium)** *caused by insufficient oxygenated blood.*
	athero/o-	From the Greek word *ather/o,* meaning "oatmeal" or "gruel"	
	-scler/o-	harden	
	-osis	condition of	
carditis	cardi/o-	heart	**Carditis** is *inflammation of the heart;* this includes the following conditions: endocarditis, myocarditis, and pericarditis. **Endocarditis** is *an inflammation of the endocardium, the inner lining of the heart and valves.* It is most often caused by bacterial infection; bacteria enter the blood from any infected part of the body, particularly infected gums and dental procedures. **Myocarditis** is *an inflammation of the heart muscle and is most often associated with viral infection.* **Pericarditis** is *an inflammation of the outer lining of the heart (pericardium).* Pericarditis may be accompanied by the accumulation of serous or purulent exudates in the pericardial space, which, in turn, may cause **cardiac tamponade** *(external compression of the heart)* and heart failure.
	-itis	inflammation	
cardiomegaly	cardi/o-	heart	**Cardiomegaly,** *an enlarged heart,* is not a disease, but a consequence of an underlying disorder. There are two types of cardiomegaly: 1. **Dilative cardiomegaly** develops when the heart weakens and becomes dilated by the increased blood volume, as in heart failure. 2. **Hypertrophic cardiomegaly** refers to the hypertrophy (growth in size) of the myocardium in response to an increased workload.
	-megal/o-	large	
	-y	condition of	
cardiomyopathy	cardi/o-	heart	**Cardiomyopathy** is a *disease of the heart muscle that results in weak and insufficient pumping activity.* In its earliest stage, cardiomyopathy is often asymptomatic, but it gradually progresses to cardiomegaly and heart failure. There are four types: 1. In **dilated cardiomyopathy,** the cardiac chambers dilate. 2. In **hypertrophic cardiomyopathy,** the myocardium thickens or hypertrophies. 3. In **ischemic cardiomyopathy,** the myocardium thins and weakens because of a poor blood supply. 4. In **restrictive cardiomyopathy,** the thickened myocardium prevents ventricular filling.
	-my/o-	muscle	
	-path	disease of	
	-y	condition of	
congenital heart defects	con-	together or with	*Heart and defects of the great vessels that are present from birth* are called **congenital heart defects.** The term includes structural defects, such as septal defects, valvular disorders, absence or incomplete development of cardiac structures, and disturbances in cardiac rhythm. Congenital heart defects are also classified as **cyanotic** or **acyanotic,** depending upon the degree of O_2 saturation. Examples of congenital heart defects are septal defects, patent ductus arteriosus, tetralogy of Fallot, transposition of the great vessels, tricuspid atresia, and coarctation of the aorta. Septal defects create **shunts** (left-to-right, right-to-left) because of the pressure differences within the chambers. A **ventriculoseptal defect (VSD)** is the most common congenital heart defect.
	-gen/o-	origin	
	-al	pertaining to	

MEDICAL TERMINOLOGY AND DISORDERS Disorders of the Heart—cont'd

Medical Term	Word Parts	Word Part Meaning or Derivation	Description
dysrhythmia	dys-	faulty	*Any disturbance in normal cardiac rhythm* is referred to a **dysrhythmia.** Dysrhythmias are classified in several ways. According to rate: normal sinus rhythm, bradycardia, and tachycardia. According to mechanism: automaticity, reentry, junctional, and fibrillation. According to origin: atrial, junctional, ventricular, heart blocks, and arrhythmias that cause sudden death. **Bradydysrhythmias** (*brady-* = slow) are *rhythm disturbances that are characterized by excessively slow activity.* Slow electrical conduction through the heart causes varying degrees of heart block (first, second, complete). **Tachydysrhythmias** (*tachy-* = fast) are *rhythm disturbances that are characterized by excessive electrical activity.* Fast rhythm disorders include tachycardia, flutter, and fibrillation. These dysrhythmias may be atrial, ventricular, or both. Generally the ventricular rhythm disorders are more acute, demanding prompt attention.
	-rhythm/o-	rhythm	
	-ia	condition of	
heart failure			**Described in the text.**
valvular disorders	valvul/o-	valve	Some examples of **valvular disorders** include semilunar and AV valves, which are incompletely formed, too narrow, or incompetent. Tricuspid atresia is an example of an incompletely formed valve. **Valvular stenosis** (i.e., mitral valve stenosis) is a narrowing of a valve restricting the flow of blood. An **incompetent valve** is a leaky valve; it does not do its job, which is to prevent backflow of blood.
	-ar	pertaining to	

Get Ready for Exams!

Summary Outline

The heart pumps blood through the blood vessels, supplying the cells of the body with oxygen and nutrients, and carrying away the waste products of metabolism. The heart functions in a coordinated and adaptable manner to perform its tasks.

I. The Coordinated and Adaptable Pump

A. Cardiac Cycle
   1. The cardiac cycle is a sequence of events that occurs during one heartbeat.
   2. The events of the cardiac cycle include atrial and ventricular systole (contraction) and diastole (relaxation).

B. Heart: Autonomic Control
   1. The autonomic nervous system (ANS) allows the heart to respond to changing body needs.
   2. Stimulation of the sympathetic nerves increases heart rate (SA node), conduction velocity (AV node), and contractile force (myocardium).
   3. Stimulation of the parasympathetic nerves (vagus) decreases heart rate and conduction velocity.

C. Cardiac Output (CO)
   1. CO is the amount of blood pumped by the ventricle in 1 minute.
   2. CO is determined by heart rate (HR) and stroke volume (SV).
   3. Many factors can change HR and/or SV.

D. How Stroke Volume Can Be Changed
   1. SV can be changed by Starling's law of the heart (stretch).
   2. SV can be changed by an inotropic effect (nonstretch).

II. Heart Talk

A. Heart Talk: Clinical Terms
   1. Includes the definition and description of commonly used clinical terms such as *preload, afterload, ejection fraction, inotropic effect, chronotropic effect,* and *dromotropic effect*

B. Heart Talk: Receptor Terminology
   1. Includes the definitions of *beta$_1$-adrenergic receptor activation, beta$_1$-adrenergic receptor blockade, muscarinic receptor activation,* and *muscarinic receptor blockade*

III. The Failing Heart: When the Heart Can't Pump

A. Left Heart Failure
   1. The left heart can fail, producing symptoms caused by a backup of blood into the pulmonic circulation (pulmonary edema); referred to as *backward failure.*
   2. The failing left heart is unable to pump enough blood to the systemic circulation, producing symptoms related to poor tissue oxygenation; referred to as *forward failure.*

B. Right Heart Failure

C. Blood backs up behind the failed right ventricle, causing jugular vein distention, hepatomegaly, splenomegaly, digestive problems, and ankle edema.

Review Your Knowledge

Matching: Cardiac Function Terms

Directions: Match the following words with their descriptions below. Some words may be used more than once.

a. inotropic effect
b. cardiac output
c. stroke volume
d. diastole
e. systole
f. Starling's law of the heart

1. ___ 5000 mL/min
2. ___ 70 mL/beat
3. ___ Stroke volume times heart rate
4. ___ Phase of the cardiac cycle that refers to myocardial contraction
5. ___ Phase of the cardiac cycle during which the ventricles fill with blood
6. ___ Phase of the cardiac cycle that refers to myocardial relaxation
7. ___ Change in myocardial contraction that is caused by stretching of the heart muscle
8. ___ Change in myocardial contraction that is not caused by stretching of the heart muscle
9. ___ Amount of blood pumped by the ventricle in one beat
10. ___ Amount of blood pumped by the ventricle in 1 minute

Matching: Loads and Effects

Directions: Match the following words with their descriptions below. Some words may be used more than once.

a. afterload
b. preload
c. ejection fraction
d. dromotropic effect
e. chronotropic effect

1. ___ Amount of blood in the ventricle at the end of its resting phase
2. ___ Percentage of the end-diastolic volume (EDV) pumped by the ventricle
3. ___ Arteriolar constriction and hypertension cause this to increase.
4. ___ Forms the basis of Starling's law of the heart
5. ___ The effect of a drug that changes heart rate
6. ___ Digoxin slows the speed of the cardiac impulse through the conduction system, thereby causing a heart block.
7. ___ Same as end-diastolic volume (EDV)
8. ___ May decline from 67% to 30% in the failing heart

Multiple Choice

1. Which of the following statements is correct about cardiac output?
   a. Cardiac output is determined by the heart rate and pulse.
   b. Stimulation of the sympathetic nerves decreases cardiac output.
   c. Vagal discharge increases cardiac output.
   d. Cardiac output is determined by heart rate and stroke volume.
2. Which statement is true of ventricular diastole?
   a. Blood is ejected from the ventricles.
   b. The semilunar valves are open.
   c. The atrioventricular valves are closed.
   d. Blood fills the ventricles.
3. Increased return of blood to the heart stretches the heart muscle, thereby
   a. stimulating the vagus nerve.
   b. increasing stroke volume.
   c. closing the atrioventricular valves.
   d. increasing coronary blood flow.
4. Ventricular systole refers to
   a. ventricular depolarization.
   b. the opening of the valves of the ventricles.
   c. ventricular filling.
   d. contraction of the ventricular myocardium.
5. Which of the following is least related to the vagus nerve?
   a. Parasympathetic
   b. Slows heart rate
   c. Positive inotropic effect
   d. Autonomic nerve
6. Which of the following is least characteristic of sympathetic nerve stimulation?
   a. Negative inotropic effect
   b. Increased heart rate
   c. Increased stroke volume
   d. Increased cardiac output
7. Which of the following is least related to bradycardia?
   a. <60 beats/min
   b. Vagal discharge
   c. Negative chronotropic effect
   d. $Beta_1$-adrenergic receptor activation
8. Which of the following is least apt to increase cardiac output?
   a. Increased heart rate
   b. Increased stroke volume
   c. Increased venous return (Starling's law of the heart)
   d. Vagal discharge

Go Figure

1. **According to Figure 17-1**
   a. The semilunar valves are open during atrial systole.
   b. The AV valves are closed during ventricular systole.
   c. Blood is pumped out of the atria during ventricular systole.
   d. The AV valves remain closed during atrial systole.
2. **According to Figure 17-2**
   a. The neurotransmitter for the vagus nerve is norepinephrine.
   b. The vagus nerve is a sympathetic nerve.
   c. The ventricular myocardium is innervated only by a sympathetic nerve.
   d. The SA node is subject only to vagal effects.