

| Cardiac Cycle

By the end of this section, you will be able to:

- Describe the relationship between blood pressure and blood flow
- Summarize the events of the cardiac cycle
- Compare atrial and ventricular systole and diastole
- Relate heart sounds detected by auscultation to action of heart's valves

The period of time that begins with contraction of the atria and ends with ventricular relaxation is known as the **cardiac cycle** (Figure 19.27). The period of contraction that the heart undergoes while it pumps blood into circulation is called **systole**. The period of relaxation that occurs as the chambers fill with blood is called **diastole**. Both the atria and ventricles undergo systole and diastole, and it is essential that these components be carefully regulated and coordinated to ensure blood is pumped efficiently to the body.

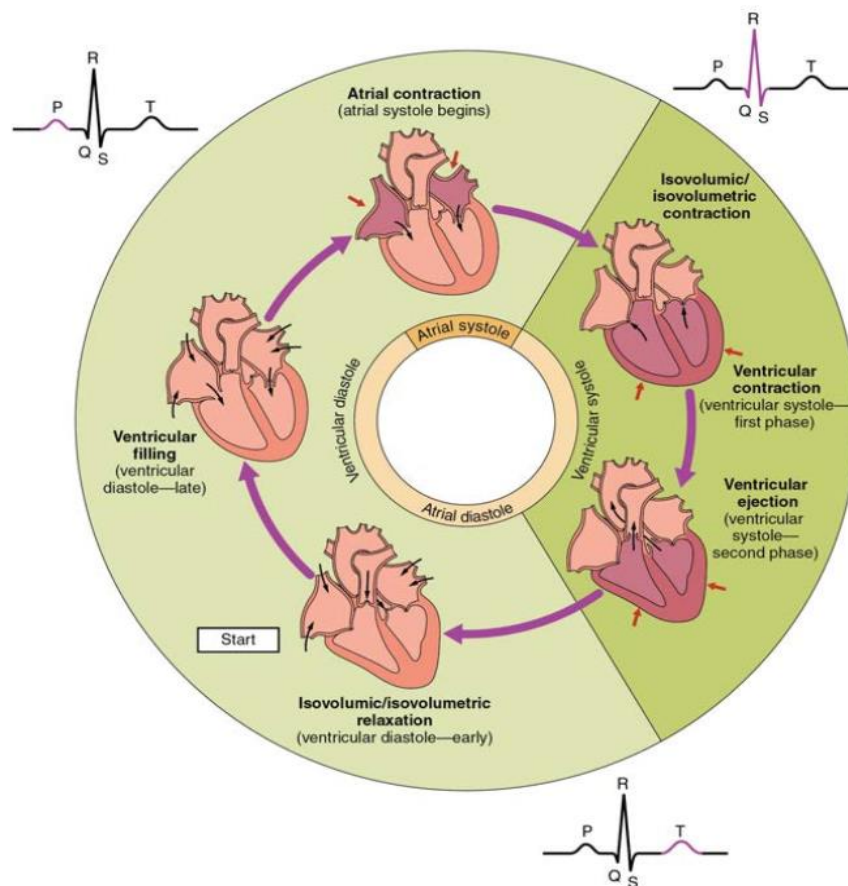


Figure 19.27 Overview of the Cardiac Cycle The cardiac cycle begins with atrial systole and progresses to ventricular systole, atrial diastole, and ventricular diastole, when the cycle begins again. Correlations to the ECG are highlighted.

Pressures and Flow

Fluids, whether gases or liquids, are materials that flow according to pressure gradients—that is, they move from regions that are higher in pressure to regions that are lower in pressure. Accordingly, when the heart chambers are relaxed (diastole), blood will flow into the atria from the veins, which are higher in pressure. As blood flows into the atria, the pressure will rise, so the blood will initially move passively from the atria into the ventricles. When the action potential triggers the muscles in the atria to contract (atrial systole), the pressure within the atria rises further, pumping blood into the ventricles.

During ventricular systole, pressure rises in the ventricles, pumping blood into the pulmonary trunk from the right ventricle and into the aorta from the left ventricle. Again, as you consider this flow and relate it to the conduction pathway, the elegance of the system should become apparent.

Phases of the Cardiac Cycle

At the beginning of the cardiac cycle, both the atria and ventricles are relaxed (diastole). Blood is flowing into the right atrium from the superior and inferior venae cavae and the coronary sinus. Blood flows into the left atrium from the four pulmonary veins. The two atrioventricular valves, the tricuspid and mitral valves, are both open, so blood flows unimpeded from the atria and into the ventricles. Approximately 70–80 percent of ventricular filling occurs by this method. The two semilunar valves, the pulmonary and aortic valves, are closed, preventing backflow of blood into the right and left ventricles from the pulmonary trunk on the right and the aorta on the left.

Atrial Systole and Diastole

Contraction of the atria follows depolarization, represented by the P wave of the ECG. As the atrial muscles contract from the superior portion of the atria toward the atrioventricular septum, pressure rises within the atria and blood is pumped into the ventricles through the open atrioventricular (tricuspid, and mitral or bicuspid) valves. At the start of atrial systole, the ventricles are normally filled with approximately 70–80 percent of their capacity due to inflow during diastole. Atrial contraction, also referred to as the “atrial kick,” contributes the remaining 20–30 percent of filling (see [Figure 19.27](#)). Atrial systole lasts approximately 100 ms and ends prior to ventricular systole, as the atrial muscle returns to diastole.

Ventricular Systole

Ventricular systole (see [Figure 19.27](#)) follows the depolarization of the ventricles and is represented by the QRS complex in the ECG. It may be conveniently divided into two phases, lasting a total of 270 ms. At the end of atrial systole and just prior to atrial contraction, the ventricles contain approximately 130 mL blood in a resting adult in a standing position. This volume is known as the **end diastolic volume (EDV)** or **preload**.

Initially, as the muscles in the ventricle contract, the pressure of the blood within the chamber rises, but it is not yet high enough to open the semilunar (pulmonary and aortic) valves and be ejected from the heart. However, blood pressure quickly rises above that of the atria that are now relaxed and in diastole. This increase in pressure causes blood to flow back toward the atria, closing the tricuspid and mitral valves. Since blood is not being ejected from the ventricles at this early stage, the volume of blood within the chamber remains constant. Consequently, this initial phase of ventricular systole is known as **isovolumic contraction**, also called isovolumetric contraction (see [Figure 19.27](#)).

In the second phase of ventricular systole, the **ventricular ejection phase**, the contraction of the ventricular muscle has raised the pressure within the ventricle to the point that it is greater than the pressures in the pulmonary trunk and the aorta. Blood is pumped from the heart, pushing open the pulmonary and aortic semilunar valves. Pressure generated by the left ventricle will be appreciably greater than the pressure generated by the right ventricle, since the existing pressure in the aorta will be so much higher. Nevertheless, both ventricles pump the same amount of blood. This quantity is referred to as stroke volume. Stroke volume will normally be in the range of 70–80 mL. Since ventricular systole began with an EDV of approximately 130 mL of blood, this means that there is still 50–60 mL of blood remaining in the ventricle following contraction. This volume of blood is known as the **end systolic volume (ESV)**.

Ventricular Diastole

Ventricular relaxation, or diastole, follows repolarization of the ventricles and is represented by the T wave of the ECG. It too is divided into two distinct phases and lasts approximately 430 ms.

During the early phase of ventricular diastole, as the ventricular muscle relaxes, pressure on the remaining blood within the ventricle begins to fall. When pressure within the ventricles drops below pressure in both the pulmonary trunk and aorta, blood flows back toward the heart, producing the dicrotic notch (small dip) seen in blood pressure tracings. The semilunar valves close to prevent backflow into the heart. Since the atrioventricular valves remain closed at this point, there is no change in the volume of blood in the ventricle, so the early phase of ventricular diastole is called the **isovolumic ventricular relaxation phase**, also called isovolumetric ventricular relaxation phase (see [Figure 19.27](#)).

In the second phase of ventricular diastole, called late ventricular diastole, as the ventricular muscle relaxes, pressure on the blood within the ventricles drops even further. Eventually, it drops below the pressure in the atria. When this occurs, blood flows from the atria into the ventricles, pushing open the tricuspid and mitral valves. As pressure drops within the ventricles, blood flows from the major veins into the relaxed atria and from there into the ventricles. Both chambers are in diastole, the atrioventricular valves are open, and the semilunar valves remain closed (see [Figure 19.27](#)). The cardiac cycle is complete.

[Figure 19.28](#) illustrates the relationship between the cardiac cycle and the ECG.

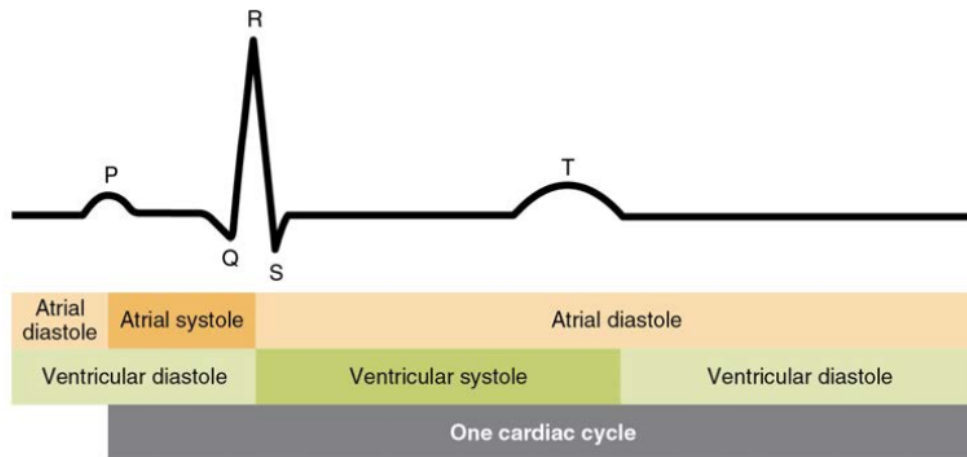


Figure 19.28 Relationship between the Cardiac Cycle and ECG Initially, both the atria and ventricles are relaxed (diastole). The P wave represents depolarization of the atria and is followed by atrial contraction (systole). Atrial systole extends until the QRS complex, at which point, the atria relax. The QRS complex represents depolarization of the ventricles and is followed by ventricular contraction. The T wave represents the repolarization of the ventricles and marks the beginning of ventricular relaxation.

Heart Sounds

One of the simplest, yet effective, diagnostic techniques applied to assess the state of a patient's heart is auscultation using a stethoscope.

In a normal, healthy heart, there are only two audible **heart sounds**: S₁ and S₂. S₁ is the sound created by the closing of the atrioventricular valves during ventricular contraction and is normally described as a “lub,” or first heart sound. The second heart sound, S₂, is the sound of the closing of the semilunar valves during ventricular diastole and is described as a “dub” (Figure 19.29). In both cases, as the valves close, the openings within the atrioventricular septum guarded by the valves will become reduced, and blood flow through the opening will become more turbulent until the valves are fully closed. There is a third heart sound, S₃, but it is rarely heard in healthy individuals. It may be the sound of blood flowing into the atria, or blood sloshing back and forth in the ventricle, or even tensing of the chordae tendineae. S₃ may be heard in youth, some athletes, and pregnant women. If the sound is heard later in life, it may indicate congestive heart failure, warranting further tests. Some cardiologists refer to the collective S₁, S₂, and S₃ sounds as the “Kentucky gallop,” because they mimic those produced by a galloping horse. The fourth heart sound, S₄, results from the contraction of the atria pushing blood into a stiff or hypertrophic ventricle, indicating failure of the left ventricle. S₄ occurs prior to S₁ and the collective sounds S₄, S₁, and S₂ are referred to by some cardiologists as the “Tennessee gallop,” because of their similarity to the sound produced by a galloping horse with a different gait. A few individuals may have both S₃ and S₄, and this combined sound is referred to as S₇.

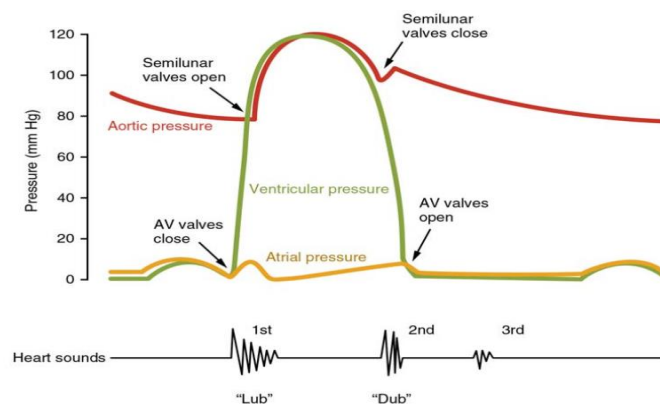


Figure 19.29 Heart Sounds and the Cardiac Cycle In this illustration, the x-axis reflects time with a recording of the heart sounds. The y-axis represents pressure.

The term **murmur** is used to describe an unusual sound coming from the heart that is caused by the turbulent flow of blood. Murmurs are graded on a scale of 1 to 6, with 1 being the most common, the most difficult sound to detect, and the least serious. The most severe is a 6. Phonocardiograms or auscultograms can be used to record both normal and abnormal sounds using specialized electronic stethoscopes.

During auscultation, it is common practice for the clinician to ask the patient to breathe deeply. This procedure not only allows for listening to airflow, but it may also amplify heart murmurs. Inhalation increases blood flow into the right side of the heart and may increase the amplitude of right-sided heart murmurs. Expiration partially restricts blood flow into the left side of the heart and may amplify left-sided heart murmurs. **Figure 19.30** indicates proper placement of the bell of the stethoscope to facilitate auscultation.

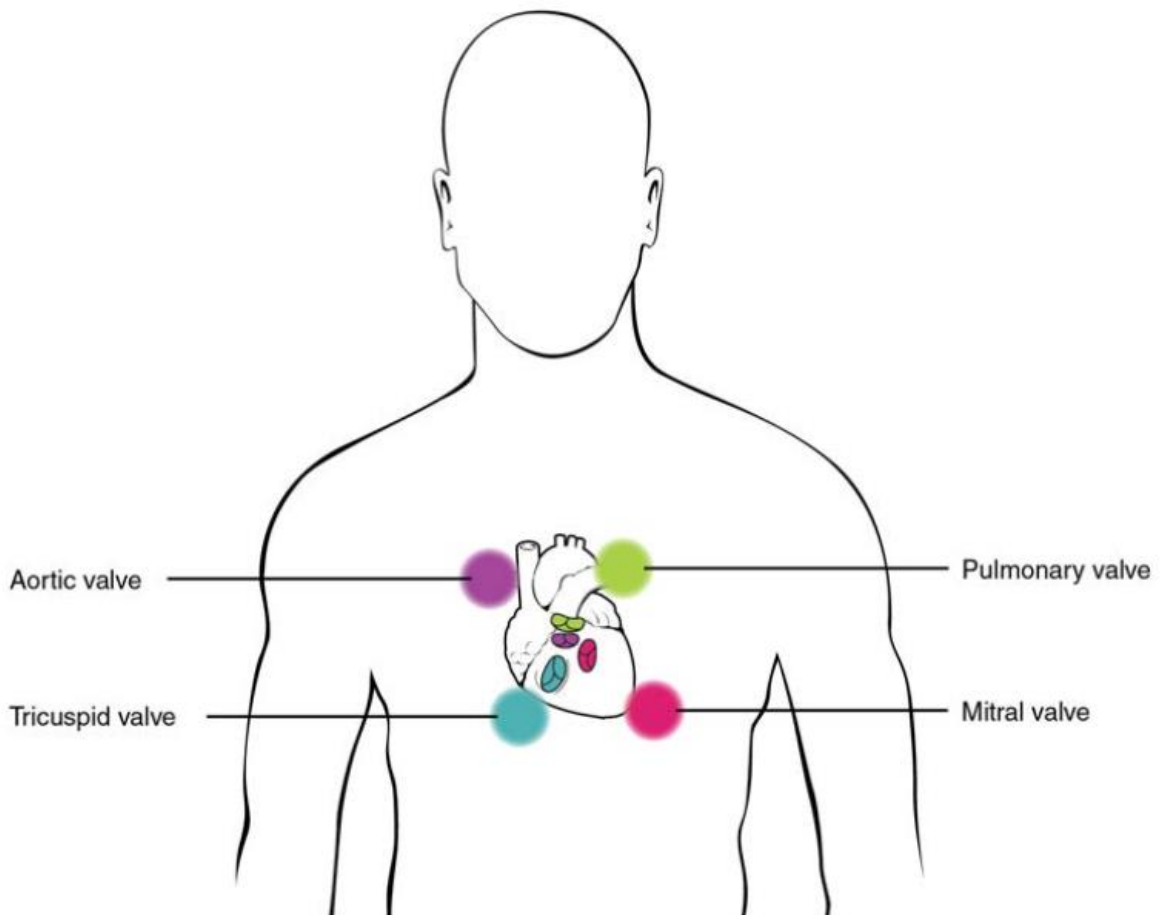


Figure 19.30 Stethoscope Placement for Auscultation Proper placement of the bell of the stethoscope facilitates auscultation. At each of the four locations on the chest, a different valve can be heard.

By the end of this section, you will be able to:

- Relate heart rate to cardiac output
- Describe the effect of exercise on heart rate
- Identify cardiovascular centers and cardiac reflexes that regulate heart function
- Describe factors affecting heart rate
- Distinguish between positive and negative factors that affect heart contractility
- Summarize factors affecting stroke volume and cardiac output
- Describe the cardiac response to variations in blood flow and pressure

The autorhythmicity inherent in cardiac cells keeps the heart beating at a regular pace; however, the heart is regulated by and responds to outside influences as well. Neural and endocrine controls are vital to the regulation of cardiac function. In addition, the heart is sensitive to several environmental factors, including electrolytes.

Resting Cardiac Output

Cardiac output (CO) is a measurement of the amount of blood pumped by each ventricle in one minute. To calculate this value, multiply **stroke volume (SV)**, the amount of blood pumped by each ventricle, by **heart rate (HR)**, in contractions per minute (or beats per minute, bpm). It can be represented mathematically by the following equation:

$$CO = HR \times SV$$

SV is normally measured using an echocardiogram to record EDV and ESV, and calculating the difference: $SV = EDV - ESV$. SV can also be measured using a specialized catheter, but this is an invasive procedure and far more dangerous to the patient. A mean SV for a resting 70-kg (150-lb) individual would be approximately 70 mL. There are several important variables, including size of the heart, physical and mental condition of the individual, sex, contractility, duration of contraction, preload or EDV, and afterload or resistance. Normal range for SV would be 55–100 mL. An average resting HR would be approximately 75 bpm but could range from 60–100 in some individuals.

Using these numbers, the mean CO is 5.25 L/min, with a range of 4.0–8.0 L/min. Remember, however, that these numbers refer to CO from each ventricle separately, not the total for the heart. Factors influencing CO are summarized in **Figure 19.31**.

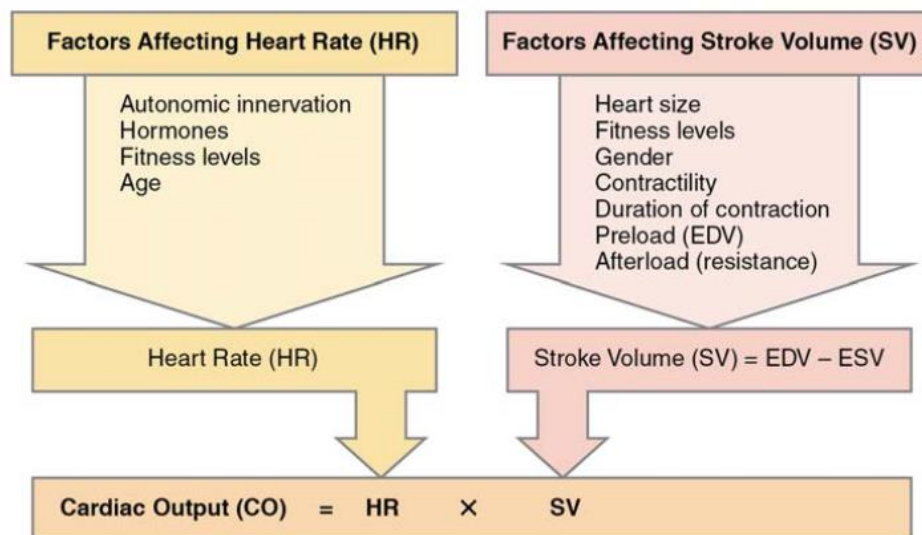


Figure 19.31 Major Factors Influencing Cardiac Output Cardiac output is influenced by heart rate and stroke volume, both of which are also variable.

SVs are also used to calculate **ejection fraction**, which is the portion of the blood that is pumped or ejected from the heart with each contraction. To calculate ejection fraction, SV is divided by EDV. Despite the name, the ejection fraction is normally expressed as a percentage. Ejection fractions range from approximately 55–70 percent, with a mean of 58 percent.

Exercise and Maximum Cardiac Output

In healthy young individuals, HR may increase to 150 bpm during exercise. SV can also increase from 70 to approximately 130 mL due to increased strength of contraction. This would increase CO to approximately 19.5 L/min, 4–5 times the resting rate. Top cardiovascular athletes can achieve even higher levels. At their peak performance, they may increase resting CO by 7–8 times.

Since the heart is a muscle, exercising it increases its efficiency. The difference between maximum and resting CO is known as the **cardiac reserve**. It measures the residual capacity of the heart to pump blood.

Heart Rates

HRs vary considerably, not only with exercise and fitness levels, but also with age. Newborn resting HRs may be 120 bpm. HR gradually decreases until young adulthood and then gradually increases again with age.

Maximum HRs are normally in the range of 200–220 bpm, although there are some extreme cases in which they may reach higher levels. As one ages, the ability to generate maximum rates decreases. This may be estimated by taking the maximal value of 220 bpm and subtracting the individual's age. So a 40-year-old individual would be expected to hit a maximum rate of approximately 180, and a 60-year-old person would achieve a HR of 160.

Correlation Between Heart Rates and Cardiac Output

Initially, physiological conditions that cause HR to increase also trigger an increase in SV. During exercise, the rate of blood returning to the heart increases. However as the HR rises, there is less time spent in diastole and consequently less time for the ventricles to fill with blood. Even though there is less filling time, SV will initially remain high. However, as HR continues to increase, SV gradually decreases due to decreased filling time. CO will initially stabilize as the increasing HR compensates for the decreasing SV, but at very high rates, CO will eventually decrease as increasing rates are no longer able to compensate for the decreasing SV. Consider this phenomenon in a healthy young individual. Initially, as HR increases from resting to approximately 120 bpm, CO will rise. As HR increases from 120 to 160 bpm, CO remains stable, since the increase in rate is offset by decreasing ventricular filling time and, consequently, SV. As HR continues to rise above 160 bpm, CO actually decreases as SV falls faster than HR increases. So although aerobic exercises are critical to maintain the health of the heart, individuals are cautioned to monitor their HR to ensure they stay within the **target heart rate** range of between 120 and 160 bpm, so CO is maintained. The target HR is loosely defined as the range in which both the heart and lungs receive the maximum benefit from the aerobic workout and is dependent upon age.

Cardiovascular Centers

Nervous control over HR is centralized within the two paired cardiovascular centers of the medulla oblongata (**Figure 19.32**). The cardioaccelerator regions stimulate activity via sympathetic stimulation of the cardioaccelerator nerves, and the cardioinhibitory centers decrease heart activity via parasympathetic stimulation as one component of the vagus nerve, cranial nerve X. During rest, both centers provide slight stimulation to the heart, contributing to **autonomic tone**. This is a similar concept to tone in skeletal muscles. Normally, vagal stimulation predominates as, left unregulated, the SA node would initiate a sinus rhythm of approximately 100 bpm.

Both sympathetic and parasympathetic stimulations flow through a paired complex network of nerve fibers known as the **cardiac plexus** near the base of the heart. The cardioaccelerator center also sends additional fibers, forming the cardiac nerves via sympathetic ganglia (the cervical ganglia plus superior thoracic ganglia T1–T4) to both the SA and AV nodes, plus additional fibers to the atria and ventricles. The ventricles are more richly innervated by sympathetic fibers than parasympathetic fibers. Sympathetic stimulation causes the release of the neurotransmitter norepinephrine (NE) at the neuromuscular junction of the cardiac nerves. NE shortens the repolarization period, thus speeding the rate of depolarization

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and contraction, which results in an increase in HR. It opens chemical- or ligand-gated sodium and calcium ion channels, allowing an influx of positively charged ions.

NE binds to the beta-1 receptor. Some cardiac medications (for example, beta blockers) work by blocking these receptors, thereby slowing HR and are one possible treatment for hypertension. Overprescription of these drugs may lead to bradycardia and even stoppage of the heart.

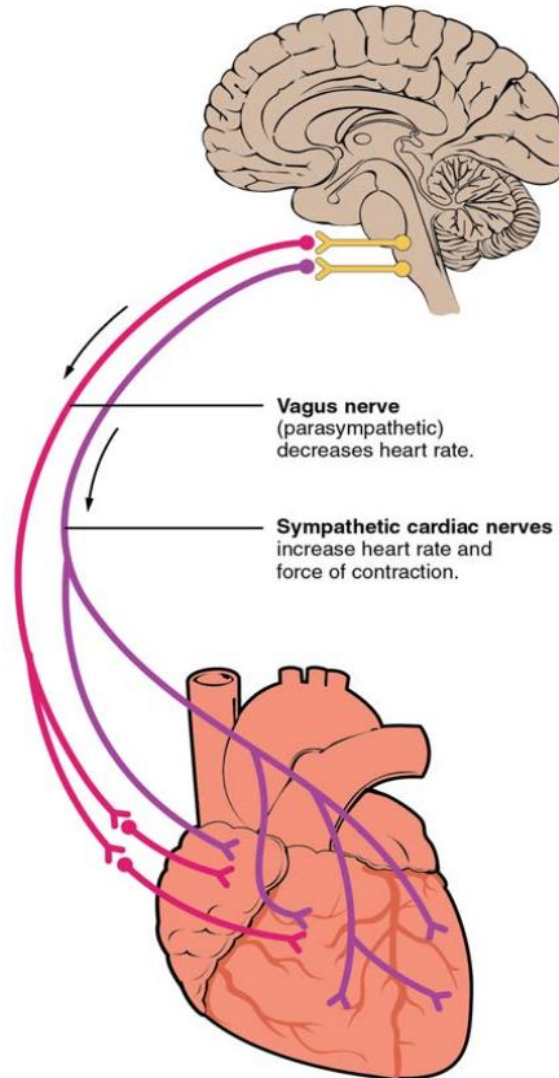


Figure 19.32 Autonomic Innervation of the Heart Cardioaccelerator and cardioinhibitory areas are components of the paired cardiac centers located in the medulla oblongata of the brain. They innervate the heart via sympathetic cardiac nerves that increase cardiac activity and vagus (parasympathetic) nerves that slow cardiac activity.

Parasympathetic stimulation originates from the cardioinhibitory region with impulses traveling via the vagus nerve (cranial nerve X). The vagus nerve sends branches to both the SA and AV nodes, and to portions of both the atria and ventricles. Parasympathetic stimulation releases the neurotransmitter acetylcholine (ACh) at the neuromuscular junction. ACh slows HR by opening chemical- or ligand-gated potassium ion channels to slow the rate of spontaneous depolarization, which extends repolarization and increases the time before the next spontaneous depolarization occurs. Without any nervous stimulation, the SA node would establish a sinus rhythm of approximately 100 bpm. Since resting rates are considerably less than this, it becomes evident that parasympathetic stimulation normally slows HR. This is similar to an individual driving a car with one foot on the brake pedal. To speed up, one need merely remove one's foot from the break and let the engine increase speed. In the case of the heart, decreasing parasympathetic stimulation decreases the release of ACh, which allows HR to increase up to approximately 100 bpm. Any increases beyond this rate would require sympathetic stimulation. **Figure 19.33** illustrates the effects of parasympathetic and sympathetic stimulation on the normal sinus rhythm.

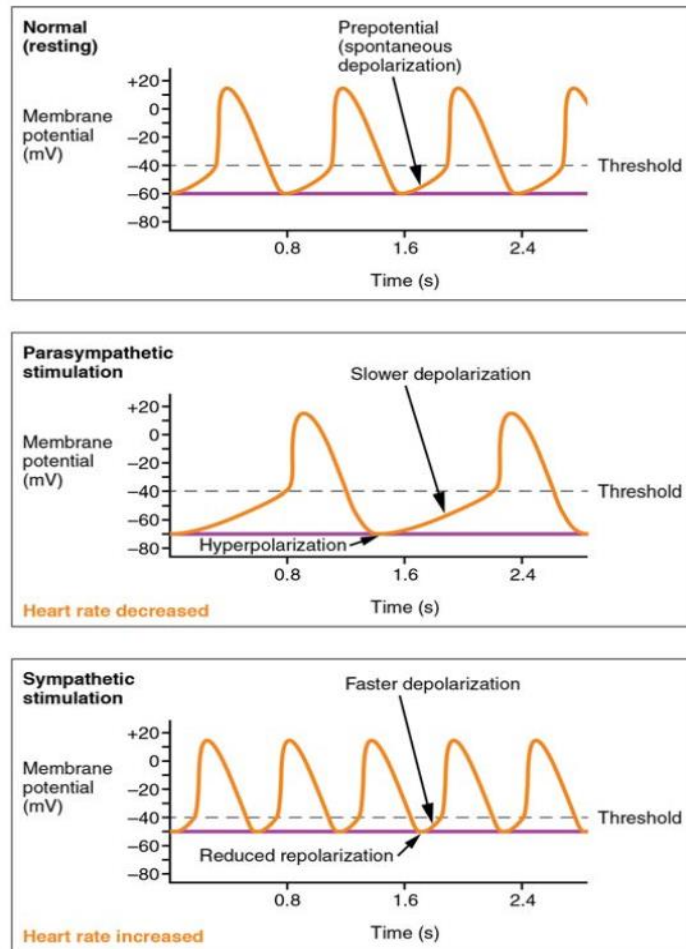


Figure 19.33 Effects of Parasympathetic and Sympathetic Stimulation on Normal Sinus Rhythm The wave of depolarization in a normal sinus rhythm shows a stable resting HR. Following parasympathetic stimulation, HR slows. Following sympathetic stimulation, HR increases.

Input to the Cardiovascular Center

The cardiovascular center receives input from a series of visceral receptors with impulses traveling through visceral sensory fibers within the vagus and sympathetic nerves via the cardiac plexus. Among these receptors are various proprioceptors, baroreceptors, and chemoreceptors, plus stimuli from the limbic system. Collectively, these inputs normally enable the cardiovascular centers to regulate heart function precisely, a process known as **cardiac reflexes**. Increased physical activity results in increased rates of firing by various proprioceptors located in muscles, joint capsules, and tendons. Any such increase in physical activity would logically warrant increased blood flow. The cardiac centers monitor these increased rates of firing, and suppress parasympathetic stimulation and increase sympathetic stimulation as needed in order to increase blood flow.

Similarly, baroreceptors are stretch receptors located in the aortic sinus, carotid bodies, the venae cavae, and other locations, including pulmonary vessels and the right side of the heart itself. Rates of firing from the baroreceptors represent blood pressure, level of physical activity, and the relative distribution of blood. The cardiac centers monitor baroreceptor firing to maintain cardiac homeostasis, a mechanism called the **baroreceptor reflex**. With increased pressure and stretch, the rate of baroreceptor firing increases, and the cardiac centers decrease sympathetic stimulation and increase parasympathetic stimulation. As pressure and stretch decrease, the rate of baroreceptor firing decreases, and the cardiac centers increase sympathetic stimulation and decrease parasympathetic stimulation.

There is a similar reflex, called the **atrial reflex** or **Bainbridge reflex**, associated with varying rates of blood flow to the atria. Increased venous return stretches the walls of the atria where specialized baroreceptors are located. However, as the atrial baroreceptors increase their rate of firing and as they stretch due to the increased blood pressure, the cardiac center responds by increasing sympathetic stimulation and inhibiting parasympathetic stimulation to increase HR. The opposite is also true.

Increased metabolic byproducts associated with increased activity, such as carbon dioxide, hydrogen ions, and lactic acid, plus falling oxygen levels, are detected by a suite of chemoreceptors innervated by the glossopharyngeal and vagus nerves. These chemoreceptors provide feedback to the cardiovascular centers about the need for increased or decreased blood flow, based on the relative levels of these substances.

The limbic system can also significantly impact HR related to emotional state. During periods of stress, it is not unusual to identify higher than normal HRs, often accompanied by a surge in the stress hormone cortisol. Individuals experiencing extreme anxiety may manifest panic attacks with symptoms that resemble those of heart attacks. These events are typically transient and treatable. Meditation techniques have been developed to ease anxiety and have been shown to lower HR effectively. Doing simple deep and slow breathing exercises with one's eyes closed can also significantly reduce this anxiety and HR.

Other Factors Influencing Heart Rate

Using a combination of autorhythmicity and innervation, the cardiovascular center is able to provide relatively precise control over HR. However, there are a number of other factors that have an impact on HR as well, including epinephrine, NE, and thyroid hormones; levels of various ions including calcium, potassium, and sodium; body temperature; hypoxia; and pH balance (Table 19.1 and Table 19.2). After reading this section, the importance of maintaining homeostasis should become even more apparent.

Major Factors Increasing Heart Rate and Force of Contraction

Factor	Effect
Cardioaccelerator nerves	Release of norepinephrine
Proprioceptors	Increased rates of firing during exercise
Chemoreceptors	Decreased levels of O ₂ ; increased levels of H ⁺ , CO ₂ , and lactic acid
Baroreceptors	Decreased rates of firing, indicating falling blood volume/pressure
Limbic system	Anticipation of physical exercise or strong emotions
Catecholamines	Increased epinephrine and norepinephrine
Thyroid hormones	Increased T ₃ and T ₄
Calcium	Increased Ca ²⁺
Potassium	Decreased K ⁺
Sodium	Decreased Na ⁺
Body temperature	Increased body temperature

Table 19.1

Major Factors Increasing Heart Rate and Force of Contraction

Factor	Effect
Nicotine and caffeine	Stimulants, increasing heart rate

Table 19.1

Factors Decreasing Heart Rate and Force of Contraction

Factor	Effect
Cardioinhibitor nerves (vagus)	Release of acetylcholine
Proprioreceptors	Decreased rates of firing following exercise
Chemoreceptors	Increased levels of O ₂ ; decreased levels of H ⁺ and CO ₂
Baroreceptors	Increased rates of firing, indicating higher blood volume/pressure
Limbic system	Anticipation of relaxation
Catecholamines	Decreased epinephrine and norepinephrine
Thyroid hormones	Decreased T ₃ and T ₄
Calcium	Decreased Ca ²⁺
Potassium	Increased K ⁺
Sodium	Increased Na ⁺
Body temperature	Decrease in body temperature

Table 19.2

Epinephrine and Norepinephrine

The catecholamines, epinephrine and NE, secreted by the adrenal medulla form one component of the extended fight-or-flight mechanism. The other component is sympathetic stimulation. Epinephrine and NE have similar effects: binding to the beta-1 receptors, and opening sodium and calcium ion chemical- or ligand-gated channels. The rate of depolarization is increased by this additional influx of positively charged ions, so the threshold is reached more quickly and the period of repolarization is shortened. However, massive releases of these hormones coupled with sympathetic stimulation may actually lead to arrhythmias. There is no parasympathetic stimulation to the adrenal medulla.

Thyroid Hormones

In general, increased levels of thyroid hormone, or thyroxin, increase cardiac rate and contractility. The impact of thyroid hormone is typically of a much longer duration than that of the catecholamines. The physiologically active form of thyroid hormone, T₃ or triiodothyronine, has been shown to directly enter cardiomyocytes and alter activity at the level of the genome. It also impacts the beta adrenergic response similar to epinephrine and NE described above. Excessive levels of thyroxin may trigger tachycardia.

Calcium

Calcium ion levels have great impacts upon both HR and contractility; as the levels of calcium ions increase, so do HR and contractility. High levels of calcium ions (hypercalcemia) may be implicated in a short QT interval and a widened T wave in the ECG. The QT interval represents the time from the start of depolarization to repolarization of the ventricles, and includes the period of ventricular systole. Extremely high levels of calcium may induce cardiac arrest. Drugs known as calcium channel blockers slow HR by binding to these channels and blocking or slowing the inward movement of calcium ions.

Caffeine and Nicotine

Caffeine and nicotine are not found naturally within the body. Both of these nonregulated drugs have an excitatory effect on membranes of neurons in general and have a stimulatory effect on the cardiac centers specifically, causing an increase in HR. Caffeine works by increasing the rates of depolarization at the SA node, whereas nicotine stimulates the activity of the sympathetic neurons that deliver impulses to the heart.

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Although it is the world's most widely consumed psychoactive drug, caffeine is legal and not regulated. While precise quantities have not been established, "normal" consumption is not considered harmful to most people, although it may cause disruptions to sleep and acts as a diuretic. Its consumption by pregnant women is cautioned against, although no evidence of negative effects has been confirmed. Tolerance and even physical and mental addiction to the drug result in individuals who routinely consume the substance.

Nicotine, too, is a stimulant and produces addiction. While legal and nonregulated, concerns about nicotine's safety and documented links to respiratory and cardiac disease have resulted in warning labels on cigarette packages.

Factors Decreasing Heart Rate

HR can be slowed when a person experiences altered sodium and potassium levels, hypoxia, acidosis, alkalosis, and hypothermia (see [Table 19.1](#)). The relationship between electrolytes and HR is complex, but maintaining electrolyte balance is critical to the normal wave of depolarization. Of the two ions, potassium has the greater clinical significance. Initially, both hyponatremia (low sodium levels) and hypernatremia (high sodium levels) may lead to tachycardia. Severely high hypernatremia may lead to fibrillation, which may cause CO to cease. Severe hyponatremia leads to both bradycardia and other arrhythmias. Hypokalemia (low potassium levels) also leads to arrhythmias, whereas hyperkalemia (high potassium levels) causes the heart to become weak and flaccid, and ultimately to fail.

Heart muscle relies exclusively on aerobic metabolism for energy. Hypoxia (an insufficient supply of oxygen) leads to decreasing HRs, since metabolic reactions fueling heart contraction are restricted.

Acidosis is a condition in which excess hydrogen ions are present, and the patient's blood expresses a low pH value. Alkalosis is a condition in which there are too few hydrogen ions, and the patient's blood has an elevated pH. Normal blood pH falls in the range of 7.35–7.45, so a number lower than this range represents acidosis and a higher number represents alkalosis. Recall that enzymes are the regulators or catalysts of virtually all biochemical reactions; they are sensitive to pH and will change shape slightly with values outside their normal range. These variations in pH and accompanying slight physical changes to the active site on the enzyme decrease the rate of formation of the enzyme-substrate complex, subsequently decreasing the rate of many enzymatic reactions, which can have complex effects on HR. Severe changes in pH will lead to denaturation of the enzyme.

The last variable is body temperature. Elevated body temperature is called hyperthermia, and suppressed body temperature is called hypothermia. Slight hyperthermia results in increasing HR and strength of contraction. Hypothermia slows the rate and strength of heart contractions. This distinct slowing of the heart is one component of the larger diving reflex that diverts blood to essential organs while submerged. If sufficiently chilled, the heart will stop beating, a technique that may be employed during open heart surgery. In this case, the patient's blood is normally diverted to an artificial heart-lung machine to maintain the body's blood supply and gas exchange until the surgery is complete, and sinus rhythm can be restored. Excessive hyperthermia and hypothermia will both result in death, as enzymes drive the body systems to cease normal function, beginning with the central nervous system.

Stroke Volume

Many of the same factors that regulate HR also impact cardiac function by altering SV. While a number of variables are involved, SV is ultimately dependent upon the difference between EDV and ESV. The three primary factors to consider are preload, or the stretch on the ventricles prior to contraction; the contractility, or the force or strength of the contraction itself; and afterload, the force the ventricles must generate to pump blood against the resistance in the vessels. These factors are summarized in [Table 19.1](#) and [Table 19.2](#).

Preload

Preload is another way of expressing EDV. Therefore, the greater the EDV is, the greater the preload is. One of the primary factors to consider is **filling time**, or the duration of ventricular diastole during which filling occurs. The more rapidly the heart contracts, the shorter the filling time becomes, and the lower the EDV and preload are. This effect can be partially overcome by increasing the second variable, contractility, and raising SV, but over time, the heart is unable to compensate for decreased filling time, and preload also decreases.

With increasing ventricular filling, both EDV or preload increase, and the cardiac muscle itself is stretched to a greater degree. At rest, there is little stretch of the ventricular muscle, and the sarcomeres remain short. With increased ventricular filling, the ventricular muscle is increasingly stretched and the sarcomere length increases. As the sarcomeres reach their optimal lengths, they will contract more powerfully, because more of the myosin heads can bind to the actin on the thin filaments, forming cross bridges and increasing the strength of contraction and SV. If this process were to continue and the sarcomeres stretched beyond their optimal lengths, the force of contraction would decrease. However, due to the physical constraints of the location of the heart, this excessive stretch is not a concern.

The relationship between ventricular stretch and contraction has been stated in the well-known **Frank-Starling mechanism** or simply Starling's Law of the Heart. This principle states that, within physiological limits, the force of heart contraction is directly proportional to the initial length of the muscle fiber. This means that the greater the stretch of the ventricular muscle (within limits), the more powerful the contraction is, which in turn increases SV. Therefore, by increasing preload, you increase the second variable, contractility.

Otto Frank (1865–1944) was a German physiologist; among his many published works are detailed studies of this important heart relationship. Ernest Starling (1866–1927) was an important English physiologist who also studied the heart.

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Although they worked largely independently, their combined efforts and similar conclusions have been recognized in the name “Frank-Starling mechanism.”

Any sympathetic stimulation to the venous system will increase venous return to the heart, which contributes to ventricular filling, and EDV and preload. While much of the ventricular filling occurs while both atria and ventricles are in diastole, the contraction of the atria, the atrial kick, plays a crucial role by providing the last 20–30 percent of ventricular filling.

Contractility

It is virtually impossible to consider preload or ESV without including an early mention of the concept of contractility. Indeed, the two parameters are intimately linked. Contractility refers to the force of the contraction of the heart muscle, which controls SV, and is the primary parameter for impacting ESV. The more forceful the contraction is, the greater the SV and smaller the ESV are. Less forceful contractions result in smaller SVs and larger ESVs. Factors that increase contractility are described as **positive inotropic factors**, and those that decrease contractility are described as **negative inotropic factors** (ino- = “fiber;” -tropic = “turning toward”).

Not surprisingly, sympathetic stimulation is a positive inotrope, whereas parasympathetic stimulation is a negative inotrope. Sympathetic stimulation triggers the release of NE at the neuromuscular junction from the cardiac nerves and also stimulates the adrenal cortex to secrete epinephrine and NE. In addition to their stimulatory effects on HR, they also bind to both alpha and beta receptors on the cardiac muscle cell membrane to increase metabolic rate and the force of contraction. This combination of actions has the net effect of increasing SV and leaving a smaller residual ESV in the ventricles. In comparison, parasympathetic stimulation releases ACh at the neuromuscular junction from the vagus nerve. The membrane hyperpolarizes and inhibits contraction to decrease the strength of contraction and SV, and to raise ESV. Since parasympathetic fibers are more widespread in the atria than in the ventricles, the primary site of action is in the upper chambers. Parasympathetic stimulation in the atria decreases the atrial kick and reduces EDV, which decreases ventricular stretch and preload, thereby further limiting the force of ventricular contraction. Stronger parasympathetic stimulation also directly decreases the force of contraction of the ventricles.

Several synthetic drugs, including dopamine and isoproterenol, have been developed that mimic the effects of epinephrine and NE by stimulating the influx of calcium ions from the extracellular fluid. Higher concentrations of intracellular calcium ions increase the strength of contraction. Excess calcium (hypercalcemia) also acts as a positive inotropic agent. The drug digitalis lowers HR and increases the strength of the contraction, acting as a positive inotropic agent by blocking the sequestering of calcium ions into the sarcoplasmic reticulum. This leads to higher intracellular calcium levels and greater strength of contraction. In addition to the catecholamines from the adrenal medulla, other hormones also demonstrate positive inotropic effects. These include thyroid hormones and glucagon from the pancreas.

Negative inotropic agents include hypoxia, acidosis, hyperkalemia, and a variety of synthetic drugs. These include numerous beta blockers and calcium channel blockers. Early beta blocker drugs include propranolol and pronethalol, and are credited with revolutionizing treatment of cardiac patients experiencing angina pectoris. There is also a large class of dihydropyridine, phenylalkylamine, and benzothiazepine calcium channel blockers that may be administered decreasing the strength of contraction and SV.

Afterload

Afterload refers to the tension that the ventricles must develop to pump blood effectively against the resistance in the vascular system. Any condition that increases resistance requires a greater afterload to force open the semilunar valves and pump the blood. Damage to the valves, such as stenosis, which makes them harder to open will also increase afterload. Any decrease in resistance decreases the afterload. **Figure 19.34** summarizes the major factors influencing SV, **Figure 19.35** summarizes the major factors influencing CO, and **Table 19.3** and **Table 19.4** summarize cardiac responses to increased and decreased blood flow and pressure in order to restore homeostasis.

Factors Affecting Stroke Volume (SV)			
	Preload	Contractility	Afterload
Raised due to:	<ul style="list-style-type: none"> fast filling time increased venous return <p>Increases end diastolic volume, Increases stroke volume</p>	<ul style="list-style-type: none"> sympathetic stimulation epinephrine and norepinephrine high intracellular calcium ions high blood calcium level thyroid hormones glucagon <p>Decreases end systolic volume, Increases stroke volume</p>	<ul style="list-style-type: none"> increased vascular resistance semilunar valve damage <p>Increases end systolic volume Decreases stroke volume</p>
Lowered due to:	<ul style="list-style-type: none"> decreased thyroid hormones decreased calcium ions high or low potassium ions high or low sodium low body temperature hypoxia abnormal pH balance drugs (i.e., calcium channel blockers) <p>Decreases end diastolic volume, Decreases stroke volume</p>	<ul style="list-style-type: none"> parasympathetic stimulation acetylcholine hypoxia hyperkalemia <p>Increases end systolic volume Decreases stroke volume</p>	<ul style="list-style-type: none"> decreased vascular resistance <p>Decreases end systolic volume Increases stroke volume</p>

Figure 19.34 Major Factors Influencing Stroke Volume Multiple factors impact preload, afterload, and contractility, and are the major considerations influencing SV.

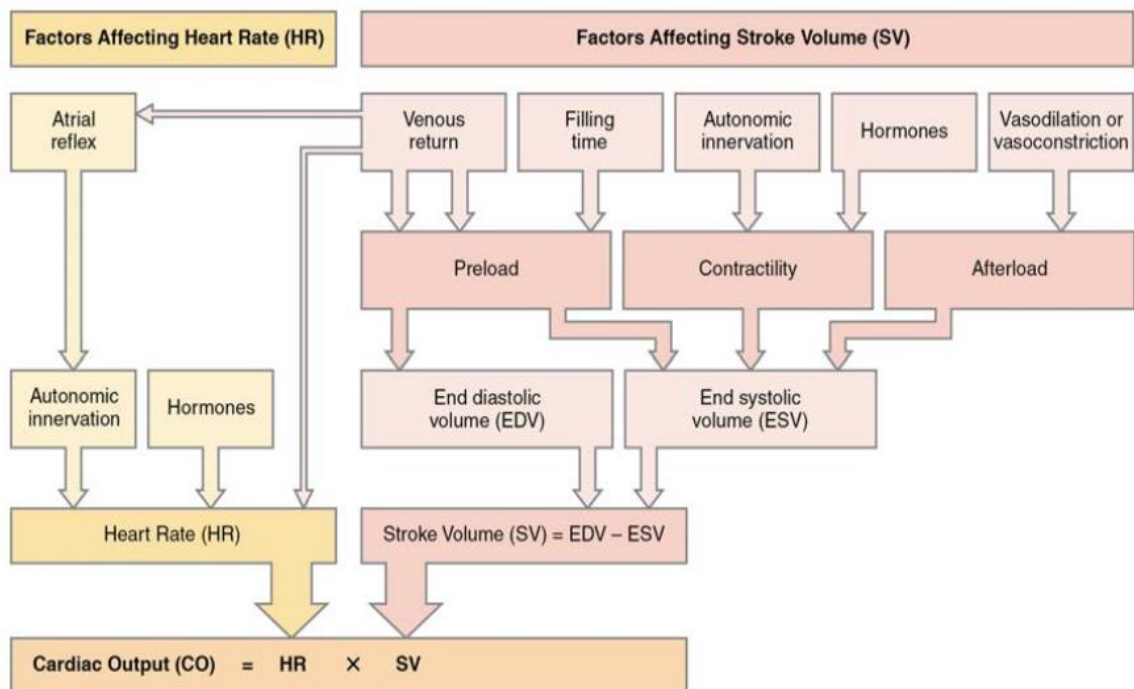


Figure 19.35 Summary of Major Factors Influencing Cardiac Output The primary factors influencing HR include autonomic innervation plus endocrine control. Not shown are environmental factors, such as electrolytes, metabolic products, and temperature. The primary factors controlling SV include preload, contractility, and afterload. Other factors such as electrolytes may be classified as either positive or negative inotropic agents.

Cardiac Response to Decreasing Blood Flow and Pressure Due to Decreasing Cardiac Output

	Baroreceptors (aorta, carotid arteries, venae cavae, and atria)	Chemoreceptors (both central nervous system and in proximity to baroreceptors)
Sensitive to	Decreasing stretch	Decreasing O ₂ and increasing CO ₂ , H ⁺ , and lactic acid
Target	Parasympathetic stimulation suppressed	Sympathetic stimulation increased
Response of heart	Increasing heart rate and increasing stroke volume	Increasing heart rate and increasing stroke volume
Overall effect	Increasing blood flow and pressure due to increasing cardiac output; hemostasis restored	Increasing blood flow and pressure due to increasing cardiac output; hemostasis restored

Table 19.3

Cardiac Response to Increasing Blood Flow and Pressure Due to Increasing Cardiac Output

	Baroreceptors (aorta, carotid arteries, venae cavae, and atria)	Chemoreceptors (both central nervous system and in proximity to baroreceptors)
Sensitive to	Increasing stretch	Increasing O ₂ and decreasing CO ₂ , H ⁺ , and lactic acid
Target	Parasympathetic stimulation increased	Sympathetic stimulation suppressed
Response of heart	Decreasing heart rate and decreasing stroke volume	Decreasing heart rate and decreasing stroke volume
Overall effect	Decreasing blood flow and pressure due to decreasing cardiac output; hemostasis restored	Decreasing blood flow and pressure due to decreasing cardiac output; hemostasis restored

Table 19.4

| Development of the Heart

By the end of this section, you will be able to:

- Describe the embryological development of heart structures
- Identify five regions of the fetal heart
- Relate fetal heart structures to adult counterparts

The human heart is the first functional organ to develop. It begins beating and pumping blood around day 21 or 22, a mere three weeks after fertilization. This emphasizes the critical nature of the heart in distributing blood through the vessels and the vital exchange of nutrients, oxygen, and wastes both to and from the developing baby. The critical early development of the heart is reflected by the prominent **heart bulge** that appears on the anterior surface of the embryo.

The heart forms from an embryonic tissue called **mesoderm** around 18 to 19 days after fertilization. Mesoderm is one of the three primary germ layers that differentiates early in development that collectively gives rise to all subsequent tissues and organs. The heart begins to develop near the head of the embryo in a region known as the **cardiogenic area**. Following chemical signals called factors from the underlying endoderm (another of the three primary germ layers), the cardiogenic area begins to form two strands called the **cardiogenic cords** (Figure 19.36). As the cardiogenic cords develop, a lumen rapidly develops within them. At this point, they are referred to as **endocardial tubes**. The two tubes migrate together and fuse to form a single **primitive heart tube**. The primitive heart tube quickly forms five distinct regions. From head to tail, these include the truncus arteriosus, bulbus cordis, primitive ventricle, primitive atrium, and the sinus venosus. Initially, all

venous blood flows into the sinus venosus, and contractions propel the blood from tail to head, or from the sinus venosus to the truncus arteriosus. This is a very different pattern from that of an adult.

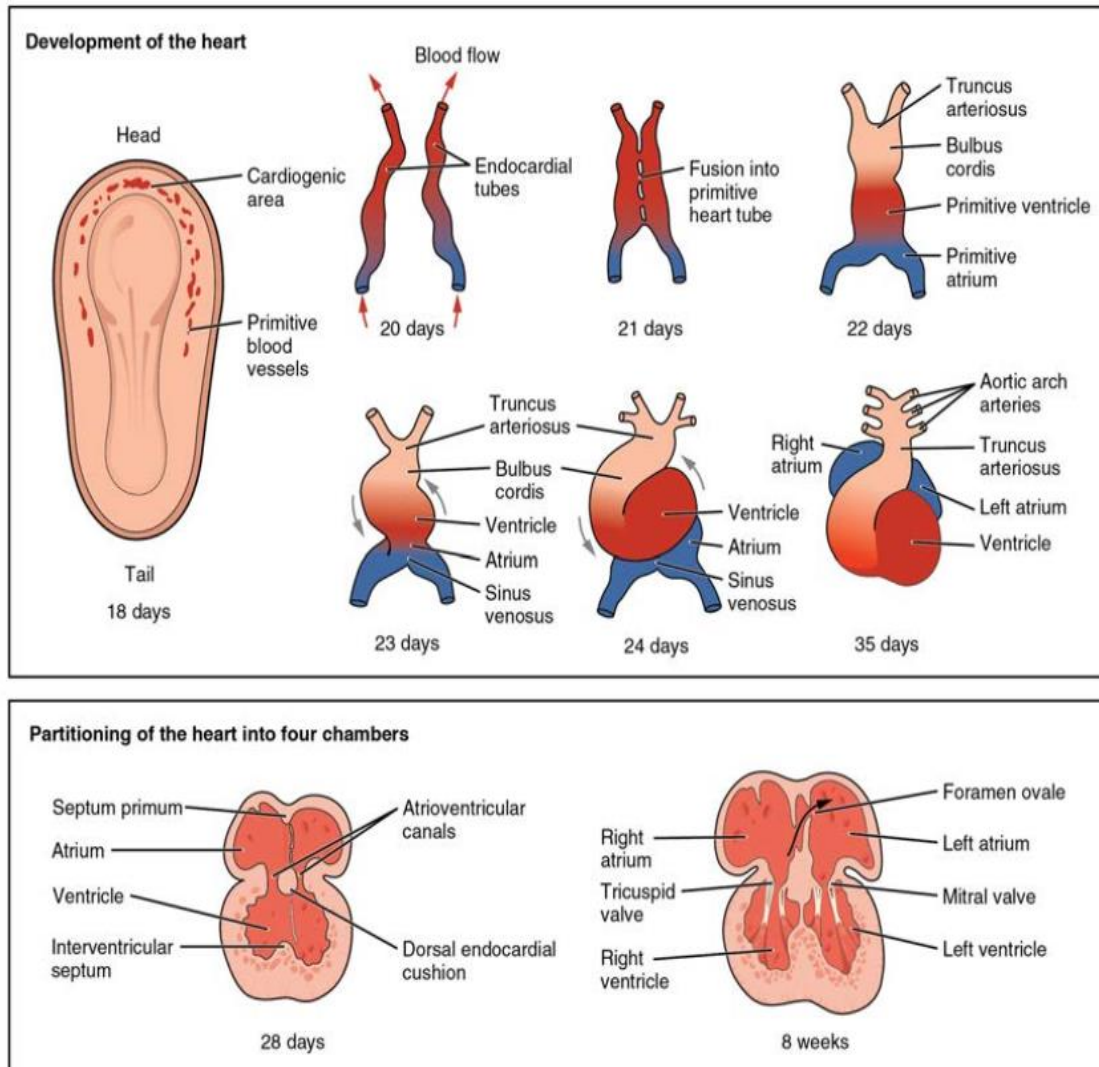


Figure 19.36 Development of the Human Heart This diagram outlines the embryological development of the human heart during the first eight weeks and the subsequent formation of the four heart chambers.

The five regions of the primitive heart develop into recognizable structures in a fully developed heart. The **truncus arteriosus** will eventually divide and give rise to the ascending aorta and pulmonary trunk. The **bulbus cordis** develops into the right ventricle. The **primitive ventricle** forms the left ventricle. The **primitive atrium** becomes the anterior portions of both the right and left atria, and the two auricles. The **sinus venosus** develops into the posterior portion of the right atrium, the SA node, and the coronary sinus.

As the primitive heart tube elongates, it begins to fold within the pericardium, eventually forming an S shape, which places the chambers and major vessels into an alignment similar to the adult heart. This process occurs between days 23 and 28. The remainder of the heart development pattern includes development of septa and valves, and remodeling of the actual chambers. Partitioning of the atria and ventricles by the interatrial septum, interventricular septum, and atrioventricular septum is complete by the end of the fifth week, although the fetal blood shunts remain until birth or shortly after. The atrioventricular valves form between weeks five and eight, and the semilunar valves form between weeks five and nine.

KEY TERMS

- afterload** force the ventricles must develop to effectively pump blood against the resistance in the vessels
- anastomosis** (plural = anastomoses) area where vessels unite to allow blood to circulate even if there may be partial blockage in another branch
- anterior cardiac veins** vessels that parallel the small cardiac arteries and drain the anterior surface of the right ventricle; bypass the coronary sinus and drain directly into the right atrium
- anterior interventricular artery** (also, left anterior descending artery or LAD) major branch of the left coronary artery that follows the anterior interventricular sulcus
- anterior interventricular sulcus** sulcus located between the left and right ventricles on the anterior surface of the heart
- aortic valve** (also, aortic semilunar valve) valve located at the base of the aorta
- artificial pacemaker** medical device that transmits electrical signals to the heart to ensure that it contracts and pumps blood to the body
- atrial reflex** (also, called Bainbridge reflex) autonomic reflex that responds to stretch receptors in the atria that send impulses to the cardioaccelerator area to increase HR when venous flow into the atria increases
- atrioventricular (AV) node** clump of myocardial cells located in the inferior portion of the right atrium within the atrioventricular septum; receives the impulse from the SA node, pauses, and then transmits it into specialized conducting cells within the interventricular septum
- atrioventricular bundle branches** (also, left or right bundle branches) specialized myocardial conductile cells that arise from the bifurcation of the atrioventricular bundle and pass through the interventricular septum; lead to the Purkinje fibers and also to the right papillary muscle via the moderator band
- atrioventricular bundle** (also, bundle of His) group of specialized myocardial conductile cells that transmit the impulse from the AV node through the interventricular septum; form the left and right atrioventricular bundle branches
- atrioventricular septum** cardiac septum located between the atria and ventricles; atrioventricular valves are located here
- atrioventricular valves** one-way valves located between the atria and ventricles; the valve on the right is called the tricuspid valve, and the one on the left is the mitral or bicuspid valve
- atrium** (plural = atria) upper or receiving chamber of the heart that pumps blood into the lower chambers just prior to their contraction; the right atrium receives blood from the systemic circuit that flows into the right ventricle; the left atrium receives blood from the pulmonary circuit that flows into the left ventricle
- auricle** extension of an atrium visible on the superior surface of the heart
- autonomic tone** contractile state during resting cardiac activity produced by mild sympathetic and parasympathetic stimulation
- autorhythmicity** ability of cardiac muscle to initiate its own electrical impulse that triggers the mechanical contraction that pumps blood at a fixed pace without nervous or endocrine control
- Bachmann's bundle** (also, interatrial band) group of specialized conducting cells that transmit the impulse directly from the SA node in the right atrium to the left atrium
- Bainbridge reflex** (also, called atrial reflex) autonomic reflex that responds to stretch receptors in the atria that send impulses to the cardioaccelerator area to increase HR when venous flow into the atria increases
- baroreceptor reflex** autonomic reflex in which the cardiac centers monitor signals from the baroreceptor stretch receptors and regulate heart function based on blood flow
- bicuspid valve** (also, mitral valve or left atrioventricular valve) valve located between the left atrium and ventricle; consists of two flaps of tissue

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- bulbus cordis** portion of the primitive heart tube that will eventually develop into the right ventricle
- bundle of His** (also, atrioventricular bundle) group of specialized myocardial conductile cells that transmit the impulse from the AV node through the interventricular septum; form the left and right atrioventricular bundle branches
- cardiac cycle** period of time between the onset of atrial contraction (atrial systole) and ventricular relaxation (ventricular diastole)
- cardiac notch** depression in the medial surface of the inferior lobe of the left lung where the apex of the heart is located
- cardiac output (CO)** amount of blood pumped by each ventricle during one minute; equals HR multiplied by SV
- cardiac plexus** paired complex network of nerve fibers near the base of the heart that receive sympathetic and parasympathetic stimulations to regulate HR
- cardiac reflexes** series of autonomic reflexes that enable the cardiovascular centers to regulate heart function based upon sensory information from a variety of visceral sensors
- cardiac reserve** difference between maximum and resting CO
- cardiac skeleton** (also, skeleton of the heart) reinforced connective tissue located within the atrioventricular septum; includes four rings that surround the openings between the atria and ventricles, and the openings to the pulmonary trunk and aorta; the point of attachment for the heart valves
- cardiogenic area** area near the head of the embryo where the heart begins to develop 18–19 days after fertilization
- cardiogenic cords** two strands of tissue that form within the cardiogenic area
- cardiomyocyte** muscle cell of the heart
- chordae tendineae** string-like extensions of tough connective tissue that extend from the flaps of the atrioventricular valves to the papillary muscles
- circumflex artery** branch of the left coronary artery that follows coronary sulcus
- coronary arteries** branches of the ascending aorta that supply blood to the heart; the left coronary artery feeds the left side of the heart, the left atrium and ventricle, and the interventricular septum; the right coronary artery feeds the right atrium, portions of both ventricles, and the heart conduction system
- coronary sinus** large, thin-walled vein on the posterior surface of the heart that lies within the atrioventricular sulcus and drains the heart myocardium directly into the right atrium
- coronary sulcus** sulcus that marks the boundary between the atria and ventricles
- coronary veins** vessels that drain the heart and generally parallel the large surface arteries
- diastole** period of time when the heart muscle is relaxed and the chambers fill with blood
- ejection fraction** portion of the blood that is pumped or ejected from the heart with each contraction; mathematically represented by SV divided by EDV
- electrocardiogram (ECG)** surface recording of the electrical activity of the heart that can be used for diagnosis of irregular heart function; also abbreviated as EKG
- end diastolic volume (EDV)** (also, preload) the amount of blood in the ventricles at the end of atrial systole just prior to ventricular contraction
- end systolic volume (ESV)** amount of blood remaining in each ventricle following systole
- endocardial tubes** stage in which lumens form within the expanding cardiogenic cords, forming hollow structures
- endocardium** innermost layer of the heart lining the heart chambers and heart valves; composed of endothelium reinforced with a thin layer of connective tissue that binds to the myocardium
- endothelium** layer of smooth, simple squamous epithelium that lines the endocardium and blood vessels
- epicardial coronary arteries** surface arteries of the heart that generally follow the sulci

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epicardium innermost layer of the serous pericardium and the outermost layer of the heart wall

Frank-Starling mechanism relationship between ventricular stretch and contraction in which the force of heart contraction is directly proportional to the initial length of the muscle fiber

filling time duration of ventricular diastole during which filling occurs

foramen ovale opening in the fetal heart that allows blood to flow directly from the right atrium to the left atrium, bypassing the fetal pulmonary circuit

fossa ovalis oval-shaped depression in the interatrial septum that marks the former location of the foramen ovale

great cardiac vein vessel that follows the interventricular sulcus on the anterior surface of the heart and flows along the coronary sulcus into the coronary sinus on the posterior surface; parallels the anterior interventricular artery and drains the areas supplied by this vessel

heart block interruption in the normal conduction pathway

heart bulge prominent feature on the anterior surface of the heart, reflecting early cardiac development

heart rate (HR) number of times the heart contracts (beats) per minute

heart sounds sounds heard via auscultation with a stethoscope of the closing of the atrioventricular valves ("lub") and semilunar valves ("dub")

hypertrophic cardiomyopathy pathological enlargement of the heart, generally for no known reason

inferior vena cava large systemic vein that returns blood to the heart from the inferior portion of the body

interatrial band (also, Bachmann's bundle) group of specialized conducting cells that transmit the impulse directly from the SA node in the right atrium to the left atrium

interatrial septum cardiac septum located between the two atria; contains the fossa ovalis after birth

intercalated disc physical junction between adjacent cardiac muscle cells; consisting of desmosomes, specialized linking proteoglycans, and gap junctions that allow passage of ions between the two cells

internodal pathways specialized conductile cells within the atria that transmit the impulse from the SA node throughout the myocardial cells of the atrium and to the AV node

interventricular septum cardiac septum located between the two ventricles

isovolumic contraction (also, isovolumetric contraction) initial phase of ventricular contraction in which tension and pressure in the ventricle increase, but no blood is pumped or ejected from the heart

isovolumic ventricular relaxation phase initial phase of the ventricular diastole when pressure in the ventricles drops below pressure in the two major arteries, the pulmonary trunk, and the aorta, and blood attempts to flow back into the ventricles, producing the dicrotic notch of the ECG and closing the two semilunar valves

left atrioventricular valve (also, mitral valve or bicuspid valve) valve located between the left atrium and ventricle; consists of two flaps of tissue

marginal arteries branches of the right coronary artery that supply blood to the superficial portions of the right ventricle

mesoderm one of the three primary germ layers that differentiate early in embryonic development

mesothelium simple squamous epithelial portion of serous membranes, such as the superficial portion of the epicardium (the visceral pericardium) and the deepest portion of the pericardium (the parietal pericardium)

middle cardiac vein vessel that parallels and drains the areas supplied by the posterior interventricular artery; drains into the great cardiac vein

mitral valve (also, left atrioventricular valve or bicuspid valve) valve located between the left atrium and ventricle; consists of two flaps of tissue

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moderator band band of myocardium covered by endocardium that arises from the inferior portion of the interventricular septum in the right ventricle and crosses to the anterior papillary muscle; contains conductile fibers that carry electrical signals followed by contraction of the heart

murmur unusual heart sound detected by auscultation; typically related to septal or valve defects

myocardial conducting cells specialized cells that transmit electrical impulses throughout the heart and trigger contraction by the myocardial contractile cells

myocardial contractile cells bulk of the cardiac muscle cells in the atria and ventricles that conduct impulses and contract to propel blood

myocardium thickest layer of the heart composed of cardiac muscle cells built upon a framework of primarily collagenous fibers and blood vessels that supply it and the nervous fibers that help to regulate it

negative inotropic factors factors that negatively impact or lower heart contractility

P wave component of the electrocardiogram that represents the depolarization of the atria

Purkinje fibers specialized myocardial conduction fibers that arise from the bundle branches and spread the impulse to the myocardial contraction fibers of the ventricles

pacemaker cluster of specialized myocardial cells known as the SA node that initiates the sinus rhythm

papillary muscle extension of the myocardium in the ventricles to which the chordae tendineae attach

pectinate muscles muscular ridges seen on the anterior surface of the right atrium

pericardial cavity cavity surrounding the heart filled with a lubricating serous fluid that reduces friction as the heart contracts

pericardial sac (also, pericardium) membrane that separates the heart from other mediastinal structures; consists of two distinct, fused sublayers: the fibrous pericardium and the parietal pericardium

pericardium (also, pericardial sac) membrane that separates the heart from other mediastinal structures; consists of two distinct, fused sublayers: the fibrous pericardium and the parietal pericardium

positive inotropic factors factors that positively impact or increase heart contractility

posterior cardiac vein vessel that parallels and drains the areas supplied by the marginal artery branch of the circumflex artery; drains into the great cardiac vein

posterior interventricular artery (also, posterior descending artery) branch of the right coronary artery that runs along the posterior portion of the interventricular sulcus toward the apex of the heart and gives rise to branches that supply the interventricular septum and portions of both ventricles

posterior interventricular sulcus sulcus located between the left and right ventricles on the anterior surface of the heart

preload (also, end diastolic volume) amount of blood in the ventricles at the end of atrial systole just prior to ventricular contraction

prepotential depolarization (also, spontaneous depolarization) mechanism that accounts for the autorhythmic property of cardiac muscle; the membrane potential increases as sodium ions diffuse through the always-open sodium ion channels and causes the electrical potential to rise

primitive atrium portion of the primitive heart tube that eventually becomes the anterior portions of both the right and left atria, and the two auricles

primitive heart tube singular tubular structure that forms from the fusion of the two endocardial tubes

primitive ventricle portion of the primitive heart tube that eventually forms the left ventricle

pulmonary arteries left and right branches of the pulmonary trunk that carry deoxygenated blood from the heart to each of the lungs

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pulmonary capillaries capillaries surrounding the alveoli of the lungs where gas exchange occurs: carbon dioxide exits the blood and oxygen enters

pulmonary circuit blood flow to and from the lungs

pulmonary trunk large arterial vessel that carries blood ejected from the right ventricle; divides into the left and right pulmonary arteries

pulmonary valve (also, pulmonary semilunar valve, the pulmonic valve, or the right semilunar valve) valve at the base of the pulmonary trunk that prevents backflow of blood into the right ventricle; consists of three flaps

pulmonary veins veins that carry highly oxygenated blood into the left atrium, which pumps the blood into the left ventricle, which in turn pumps oxygenated blood into the aorta and to the many branches of the systemic circuit

QRS complex component of the electrocardiogram that represents the depolarization of the ventricles and includes, as a component, the repolarization of the atria

right atrioventricular valve (also, tricuspid valve) valve located between the right atrium and ventricle; consists of three flaps of tissue

semilunar valves valves located at the base of the pulmonary trunk and at the base of the aorta

septum primum flap of tissue in the fetus that covers the foramen ovale within a few seconds after birth

septum (plural = septa) walls or partitions that divide the heart into chambers

sinoatrial (SA) node known as the pacemaker, a specialized clump of myocardial conducting cells located in the superior portion of the right atrium that has the highest inherent rate of depolarization that then spreads throughout the heart

sinus rhythm normal contractile pattern of the heart

sinus venosus develops into the posterior portion of the right atrium, the SA node, and the coronary sinus

small cardiac vein parallels the right coronary artery and drains blood from the posterior surfaces of the right atrium and ventricle; drains into the great cardiac vein

spontaneous depolarization (also, prepotential depolarization) the mechanism that accounts for the autorhythmic property of cardiac muscle; the membrane potential increases as sodium ions diffuse through the always-open sodium ion channels and causes the electrical potential to rise

stroke volume (SV) amount of blood pumped by each ventricle per contraction; also, the difference between EDV and ESV

sulcus (plural = sulci) fat-filled groove visible on the surface of the heart; coronary vessels are also located in these areas

superior vena cava large systemic vein that returns blood to the heart from the superior portion of the body

systemic circuit blood flow to and from virtually all of the tissues of the body

systole period of time when the heart muscle is contracting

T wave component of the electrocardiogram that represents the repolarization of the ventricles

target heart rate range in which both the heart and lungs receive the maximum benefit from an aerobic workout

trabeculae carneae ridges of muscle covered by endocardium located in the ventricles

tricuspid valve term used most often in clinical settings for the right atrioventricular valve

truncus arteriosus portion of the primitive heart that will eventually divide and give rise to the ascending aorta and pulmonary trunk

valve in the cardiovascular system, a specialized structure located within the heart or vessels that ensures one-way flow of blood

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ventricle one of the primary pumping chambers of the heart located in the lower portion of the heart; the left ventricle is the major pumping chamber on the lower left side of the heart that ejects blood into the systemic circuit via the aorta and receives blood from the left atrium; the right ventricle is the major pumping chamber on the lower right side of the heart that ejects blood into the pulmonary circuit via the pulmonary trunk and receives blood from the right atrium

ventricular ejection phase second phase of ventricular systole during which blood is pumped from the ventricle