

5.4 The Heart as a Pump

Learning Objectives

- Describe the anatomical location of the heart and identify its major external features
- Define diastole and systole
- Trace the flow of blood from the venae cavae to the aorta
- Name the four valves of the heart and describe their location
- Describe the origin of the first and second heart sound
- Be able to determine whether flow is turbulent or laminar
- Write Laplace's law for a sphere
- Define stroke volume and ejection fraction
- Describe the general function of the SA node
- Describe the general structure of the electrical conduction system of the heart

THE HEART IS LOCATED IN THE CENTER OF THE THORACIC CAVITY

The heart is located in the middle of the thoracic cavity, oriented obliquely, with the apex of the heart pointing down and to the left, as shown in [Figures 5.4.1 and 5.4.2](#). It is suspended within a tough fibrous sac, the **pericardium**, by its connections to the great vessels: the superior and inferior venae cavae, the pulmonary artery and veins, and the aorta. The pericardium is fused to the diaphragm, and so downward movement of the diaphragm during inspiration pulls the heart into a more vertical orientation. The heart sits atop the diaphragm and its **apex** is close to the anterior surface of the thoracic cavity. With every beat, the heart twists forward and the apex taps against the chest wall, producing the **apex beat**. This can be felt in the fifth left intercostal space. The heart itself is built around a ring of fibrous tissue called the **annulus fibrosus** that separates the atria from the ventricles and contains the four heart valves, as shown in [Figure 5.4.6](#). The right and left atria are two thin-walled sacs that collect blood either from the veins (right atria) or from the lungs (left atria), whereas the ventricles are thick-walled muscular sacs that propel blood through the arteries. We introduce the function of the heart here by tracing the flow of blood through it.

THE HEART IS A MUSCLE

516 The heart consists of three types of muscle tissue: atrial muscle, ventricular muscle, and specialized muscle

tissue that coordinates electrical signals through the heart. Like other muscles, the main function of the atrial and ventricular muscle fibers is to contract and produce force. These muscle fibers surround hollow cavities that are filled with blood. Therefore, their contraction produces a pressure within the cavity, and this pressure drives flow. The period of contraction of the heart muscle is called **systole**; its period of relaxation is called **diastole**. The mechanism of contraction is similar to that of skeletal muscles that we have already discussed (Chapters 3.4–3.7), except that the duration of cardiac contractions is longer. As we will discuss later, entirely different mechanisms regulate cardiac force than those that regulate the force of skeletal muscle.

The conductive fibers contract only weakly because they contain few contractile elements. Instead, they are specialized to conduct an electrical signal through the heart so as to coordinate cardiac contraction. Cardiac muscle fibers also conduct electrical signals, but they coordinate contraction locally rather than coordinating the activity of the entire heart. The heart is actually two pumps in series, both of which consist of an atrium and a ventricle. Uncoordinated contractions would have the pumps operating against each other. Therefore, coordination of mechanical activity of the chambers is essential for efficient pumping.

CONTRACTION OF CARDIAC MUSCLE PRODUCES A PRESSURE WITHIN THE CHAMBER

Contraction of cardiac muscle produces a tension within its walls that generates a pressure within the chamber. The relationship between tension and pressure in the heart is complicated because the geometry is not simple and because the wall tension is not uniform. Nevertheless, some approximations can be made. We consider first a thin-walled sphere.

LAW OF LAPLACE FOR THIN-WALLED SPHERES

Consider a sphere as shown in [Figure 5.4.3](#). We draw an imaginary plane that bisects the sphere. The sphere is in mechanical equilibrium, meaning that the total forces on any part of the sphere sum to zero. The pressure difference between the inside and outside of the sphere is P . Really there are two pressures, but the outside pressure is taken to be zero gauge pressure (i.e., equal to the ambient pressure). The net pressure force on the upper

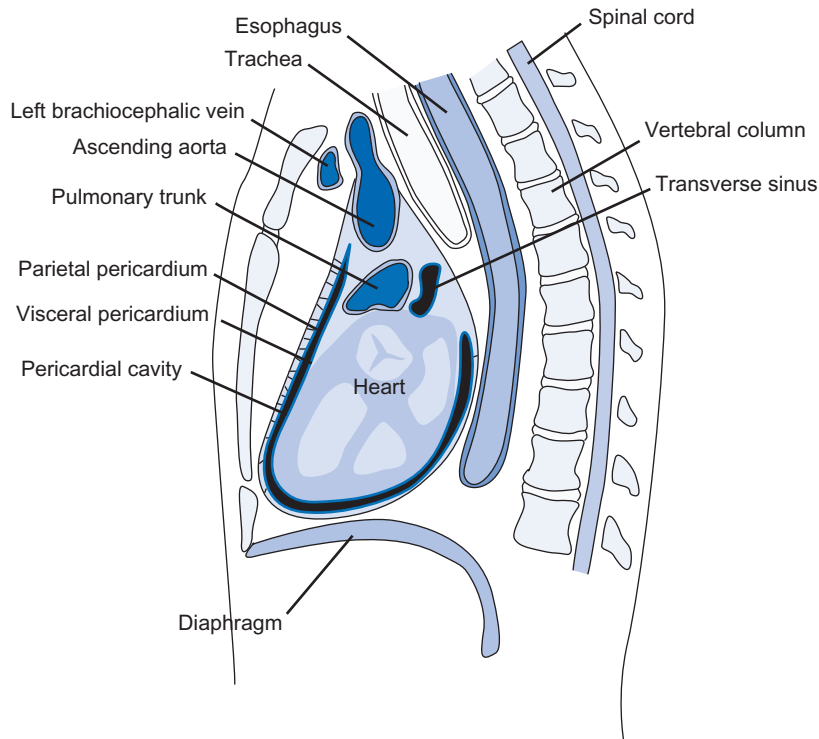


FIGURE 5.4.1 Location of the heart in the center of the chest. The figure shows a transverse section. The left ventricle actually lies behind and to the left of the right ventricle. (Source: Redrawn from C. Rosse and P. Gaddum-Rosse, *Textbook of Anatomy*, Lippincott Raven, New York, NY, 1997.)

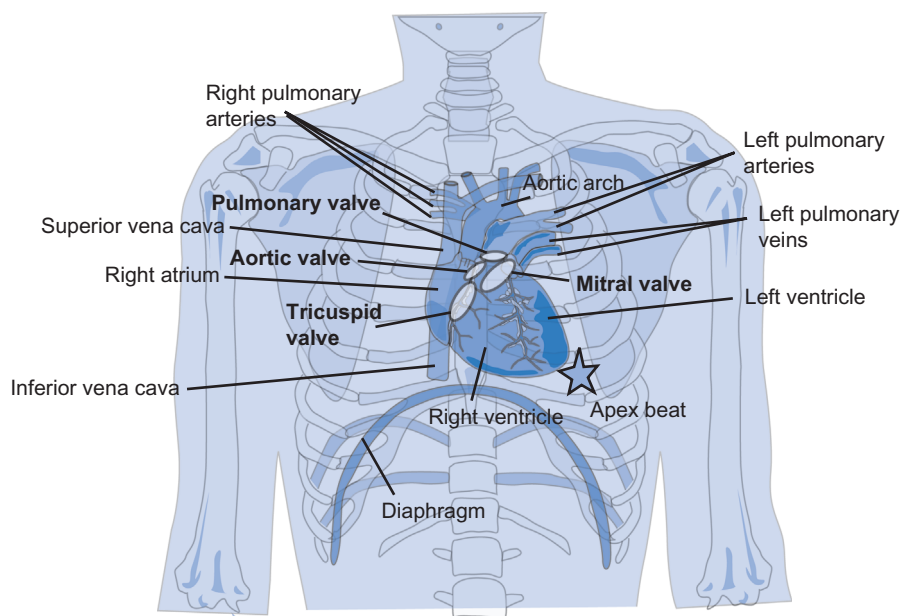


FIGURE 5.4.2 Location of the heart in the center of the chest. The figure shows a frontal, surface view of the heart. Its major axis lies obliquely to the plane of symmetry. The connective tissue ring containing the four heart valves (shown schematically as light blue rings) serves as a structural base for the heart. The tricuspid, mitral, aortic, and pulmonary valves are all grouped in this connective tissue ring set in an oblique plane beneath the sternum, at right angles to the major axis of the heart. The apex of the heart taps against the chest wall, causing the apex beat in the fifth left intercostal space.

hemisphere is thus the pressure, P , times the area of the hemisphere exposed to the pressure: $A = \pi r^2$. This pressure is balanced by the tension in the walls, which is the force per unit distance. The tension per unit length, T , acts all the way around the equator shown in the figure, so that the total tension is $F = T \times 2\pi r$. At mechanical equilibrium these two forces, the pressure force and the tension, are equal:

$$[5.4.1] \quad \begin{aligned} P\pi r^2 &= T 2\pi r \\ P &= \frac{2T}{r} \end{aligned}$$

This last equation is the Law of Laplace for thin-walled spheres.

THE LAW OF LAPLACE FOR THICK-WALLED SPHERES

The model here is a hollow sphere with an inner radius r_1 and outer radius r_2 , and wall thickness $w = r_2 - r_1$. We define here the **wall stress**, which is the force per unit area in the wall. Recall that the wall tension in the thin-wall model was the force per unit length. Thus we have

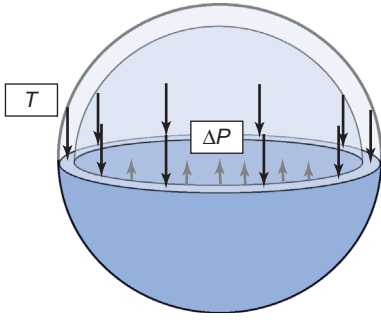


FIGURE 5.4.3 Schematic diagram of the component forces experienced by one hemisphere of a thin-walled sphere. The force on the upper hemisphere by the walls of the lower hemisphere is the tension times the circumference and is directed downward. The force of the contents of the sphere on the upper hemisphere is the pressure times the area and is directed upward. At mechanical equilibrium the total force on the upper hemisphere is zero.

$$\begin{aligned} \sigma &= \frac{F}{A} \\ T &= \frac{F}{l} \\ A &= w l \\ \sigma &= \frac{T}{w} \end{aligned} \quad [5.4.2]$$

where σ is the wall stress, F is the force, A is the area, T is the tension, l is the length, and w is the width or, in this case, the wall thickness.

The condition of mechanical equilibrium demands that the internal pressure times the area must balance the wall force:

$$P_1 \pi r_1^2 = \int_{r_1}^{r_2} \sigma(r) 2 \pi r dr \quad [5.4.3]$$

This cannot be integrated unless we know the form of $\sigma(r)$. In general, $\sigma(r)$ is not constant with radius or with time and consists of two components: an elastic component having to do with the passive resistance of the heart muscle to stretch and an active component that produces the high pressures in the chamber to drive blood flow. If we assume $\sigma(r)$ is constant with r , we obtain:

$$P_1 \pi r_1^2 = \sigma \pi [r_2^2 - r_1^2] \quad [5.4.4]$$

Substituting in from Eqn [5.4.2] for $\sigma = T/w = T/(r_2 - r_1)$, we get

$$P_1 = \frac{T}{r_2 - r_1} \frac{\pi (r_2^2 - r_1^2)}{\pi r_1^2} = T \frac{r_2 + r_1}{r_1^2} \quad [5.4.5]$$

This form of Laplace's law reverts to the thin-walled case when $r_2 = r_1$.

The point of these formulas is to emphasize that development of tension within the walls of the heart produces a pressure within the chamber. Because the heart is not a sphere but is more like a prolate spheroid, and

the fact that the wall stress is not constant with r , the equations derived here are only approximately valid.

An alternate form of the Law of Laplace keeps σ in the equation. Taking Eqn [5.4.4], we have

$$\begin{aligned} P_1 &= \frac{\sigma \pi [r_2^2 - r_1^2]}{\pi r_1^2} \\ [5.4.6] \quad P_1 &= \frac{\sigma (r_2 - r_1) (r_2 + r_1)}{r_1^2} \\ P_1 &\approx \frac{2 \sigma w}{r_1} \end{aligned}$$

This has a practical consequence for dilated hearts. When hearts enlarge, as in dilated cardiomyopathy, to achieve the same pressure the wall stress must also increase. To reduce the wall stress, the thickness of the heart wall also increases. Thus, dilated hearts are also hypertrophied: they have thicker walls.

BLOOD IS PUMPED THROUGH FOUR CHAMBERS

A surface view of the heart is shown in Figure 5.4.4; Figure 5.4.5 shows a cross-section of the heart with the direction of blood flow. Blood flow is unidirectional, from the veins to the arteries. Blood comes to the heart by collection into successively larger veins, finally flowing into the **superior vena cava**, which drains the head, neck, and arms, and the **inferior vena cava**, which drains the abdominal organs and the lower limbs. Blood from the venae cavae and the **coronary sinus** (the main drain of blood that supplies the heart) enters the **right atrium**. The right atrium is one of four chambers within the heart that collect blood and, by its contraction, propels blood forward. The right atrium is a thin-walled chamber that serves mainly as a blood reservoir and gateway to the right ventricle. Its contraction only modestly enhances flow into the ventricle.

When the heart is relaxed, blood that enters the right atrium continues to flow into the **right ventricle**. The **tricuspid valve** between the right atrium and right ventricle ensures that blood flow is unidirectional. This valve derives its name from its three cusps. The valve consists of a ring of connective tissue in the wall between atrium and ventricle, to which thin flaps of connective tissue are attached. Upon contraction of the right ventricle, the tricuspid valve closes and prevents blood from flowing back into the right atrium. The valve is kept from inverting by a series of cords, the **chordae tendineae**, that connect the valve to the **papillary muscles** on the opposite ventricular wall. These papillary muscles also contract, preventing **prolapse** of the valve (its inversion into the right atrium). Thus, the tricuspid valve is closed by increasing pressure within the right ventricle, and it opens when pressure in the right atrium exceeds that in the right ventricle. This valve, like the other three cardiac valves, is pressure operated.

The right ventricle in human adults is about 0.5 cm thick. It resembles a pocket sewn onto the larger left

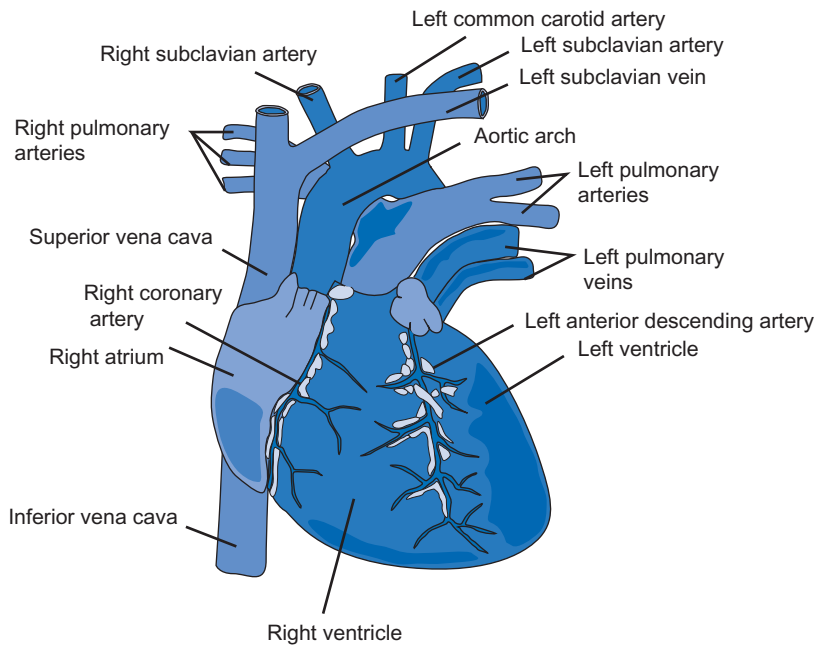


FIGURE 5.4.4 Surface view of the heart and its associated veins and arteries. Blood exits the heart by the pulmonary arteries and the aorta. The coronary arteries originate at the base of the aorta. The blood enters the heart through the large veins, the superior and inferior venae cavae. The large veins and pulmonary arteries contain blood with lower oxygen content because it is returning from the tissues where oxygen has been extracted. The pulmonary veins and aorta contain blood that has recently passed through the lungs without going to the tissues, and so it has a higher oxygen content. Vessels containing lower oxygen content are shown in lighter colour; vessels with higher oxygen content are shown in darker colour.

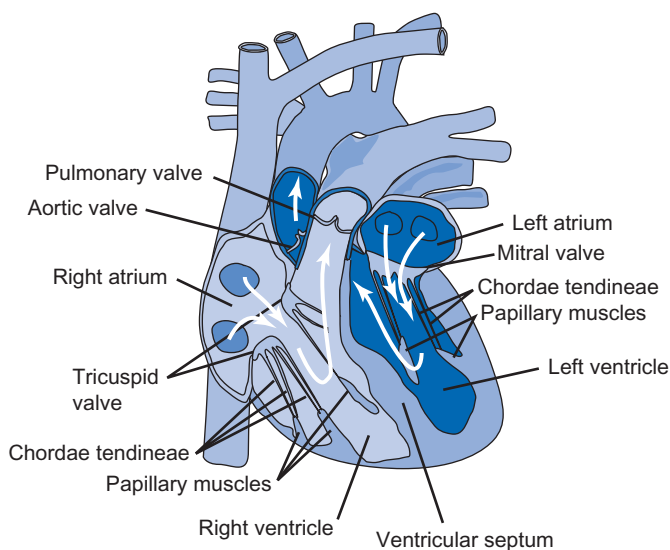


FIGURE 5.4.5 Sectional view of the heart showing the direction of blood flow. This is a schematic construct because no section of the heart simultaneously shows the ventricles and all four valves. During diastole, blood enters the right atrium and flows into the right ventricle through the tricuspid valve. During systole, the right ventricle contracts, closing the tricuspid valve and sending blood through the pulmonary valve to the pulmonary circulation. Blood returning from the lungs enters the left atrium and flows through the mitral valve into the left ventricle. During systole, the mitral valve also closes and contraction of the left ventricle sends blood through the aortic valve into the systemic circulation. Thus, the valves produce unidirectional flow through the heart. The chordae tendineae anchor the valve flaps to the papillary muscles in the ventricles, preventing valve prolapse.

ventricle. The part of the left ventricle between the two ventricles is called the **septum**. Contraction of the free anterior wall of the right ventricle squeezes the blood between it and the septum, forcing the blood out through the **pulmonary artery to the lungs**. The

pulmonary artery has a valve, the **pulmonary valve**, that prevents blood from running back into the right ventricle during diastole. The pulmonary valve consists of three baggy cusps. It opens when pressure within the right ventricle exceeds that in the pulmonary artery, and it snaps shut when the pressure in the pulmonary artery exceeds that in the right ventricle.

The blood in the right heart comes from the peripheral tissues and so it is depleted of oxygen and carries extra waste CO_2 that was produced by the tissues. In the lungs, the blood loses its extra CO_2 and replenishes its O_2 content. The reoxygenated blood returns to the heart through the **pulmonary veins**, flowing into the **left atrium**. Blood that enters the left atrium flows directly into the **left ventricle** during diastole. Left atrial contraction precedes left ventricular contraction, and this helps load the ventricle with blood prior to its contraction. The flow is kept unidirectional here by the **mitral valve**. As with the other atrioventricular valve, the margins of the cusps are tethered by chordae tendineae to two papillary muscles on the wall of the ventricle.

Contraction of the left ventricle decreases its chamber's length and diameter. Contraction begins at the apex of the heart and proceeds up the ventricle. In this way, the ventricle pushes blood out into the systemic circulation through the **aorta**. Because the pressure in the systemic circulation is much higher than in the pulmonary circulation, the left ventricle must provide more pressure to push blood into the high-pressure system. Thus, the left ventricular walls are about three times thicker than those of the right ventricle. The aorta contains another three-cusp valve, the **aortic valve**. The aortic valve and the pulmonary valves are called the **semi-lunar valves** because of their shape. When pressure within the left ventricle exceeds that in the aorta, the valve opens and blood rushes forward. As the pressure within the ventricle falls upon relaxation (diastole), the aortic valve

snaps shut, preventing backflow from the aorta to the left ventricle.

Although blood flow through the heart is sequential, the contractions of the chambers are not: both atria contract nearly simultaneously, followed shortly thereafter by the simultaneous contractions of the ventricles. At any time, the blood pumped out by the left ventricle is blood that was pumped out by the right ventricle a couple of beats ago, but both contract at the same time.

THE FOUR VALVES ARE NEARLY COPLANAR

A fibrous connective tissue ring called the **annulus fibrosus** contains all four of the heart valves. [Figure 5.4.5](#) does not adequately show this because otherwise it becomes nearly impossible to show the valves and their connectivity with clarity. [Figure 5.4.6](#) shows a cross-section of the heart at the level of the annulus fibrosus.

CLOSURE OF THE VALVES PRODUCES THE HEART SOUNDS

Valves are pressure operated. Greater pressure on the outflow side of a heart valve causes it to close. The cusps snap back as they check the movement of the refluxing blood. The sudden movement of the cusps produces a brief turbulence that transmits a vibration to the chest wall. These vibrations are audible to the ear pressed against the chest and are better heard through a **stethoscope**. The procedure of carefully monitoring sounds within the body is called **auscultation** (from the Latin “auscultare”, meaning “to listen”). The sounds can be recorded by a microphone and displayed graphically in a tracing called a **phonocardiogram**. There are a total of four heart sounds, but two are normally clearly audible. The **first heart sound** corresponds to the closure of the tricuspid and mitral valves. It has a frequency of about 100 Hz. The second heart sound, at a slightly higher frequency, corresponds to closure of the aortic and

pulmonary valves. [Figure 5.4.7](#) shows a phonocardiogram overlaying a trace of ventricular volume and pressure during the cardiac cycle.

ADDITIONAL TURBULENCE CAUSES HEART MURMURS

Laminar flow is streamlined flow, and it is silent. Chaotic flow is also called turbulent flow, and it is noisy. The occurrence of turbulent flow is often estimated from the Reynolds number, named after Osborne Reynolds, who studied the patterns of flows in tubes by injecting a thin stream of visible dye into the moving fluid. The Reynolds number is given as

$$[5.4.7] \quad Re = \frac{2a <V> \rho}{\eta}$$

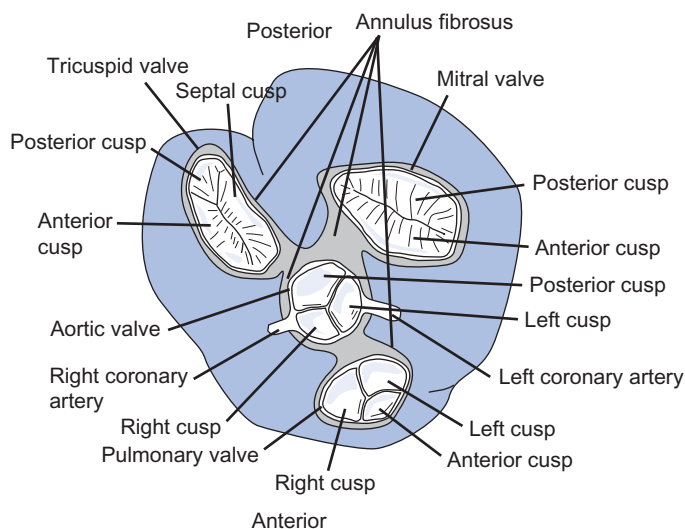
where Re is the Reynolds number, a is the radius of the tube, $<V>$ is the average velocity, ρ is the density, and η is the viscosity. Thus, the Reynolds number is a dimensionless number that is a ratio of the inertial forces to the viscous forces. Turbulence normally occurs when $Re \sim 2000$. Abnormal movement of fluid can produce turbulence in the heart, causing additional sounds that can be heard by auscultation. These are **heart murmurs**. Examples include **aortic regurgitation**. In this case, the aortic valve incompletely seals the left ventricle from the aorta during ventricular relaxation. Blood squirts back through the leaky valve into the ventricle, driven by the higher pressure in the aorta. The squirting produces a noise, or **bruit**.

SUMMARY OF THE CONTRACTILE EVENTS IN THE CARDIAC CYCLE

VENTRICULAR FILLING

The ventricles fill over a period of about 0.45 s. Note in [Figure 5.4.7](#) that while the left ventricle fills, its pressure decreases. This occurs because the ventricles are still recoiling from systole. During ventricular filling, both the tricuspid and mitral valves are open, whereas the

FIGURE 5.4.6 Surface view of the top of the heart with the atria dissected away. The annulus fibrosus, the connective tissue ring that separates the two atria from the two ventricles, is shown in gray. This ring approximates a plane in which all four valves (shown in white) are embedded. The tricuspid valve separates the right atria from the right ventricle. It has three cusps: an anterior cusp, a septal cusp, and a posterior cusp. The mitral valve separates the left atria and ventricle, and it has an anterior and posterior cusp. The pulmonary valve and aortic valve are located in the pulmonary artery and aorta, respectively. Each of these have three cusps. The valves open in a definite sequence during the pumping action of the heart. (Source: Adapted from C. Rosse and P. Gaddum-Rosse, *Textbook of Anatomy*, Lippincott Raven, New York, NY, 1997.)



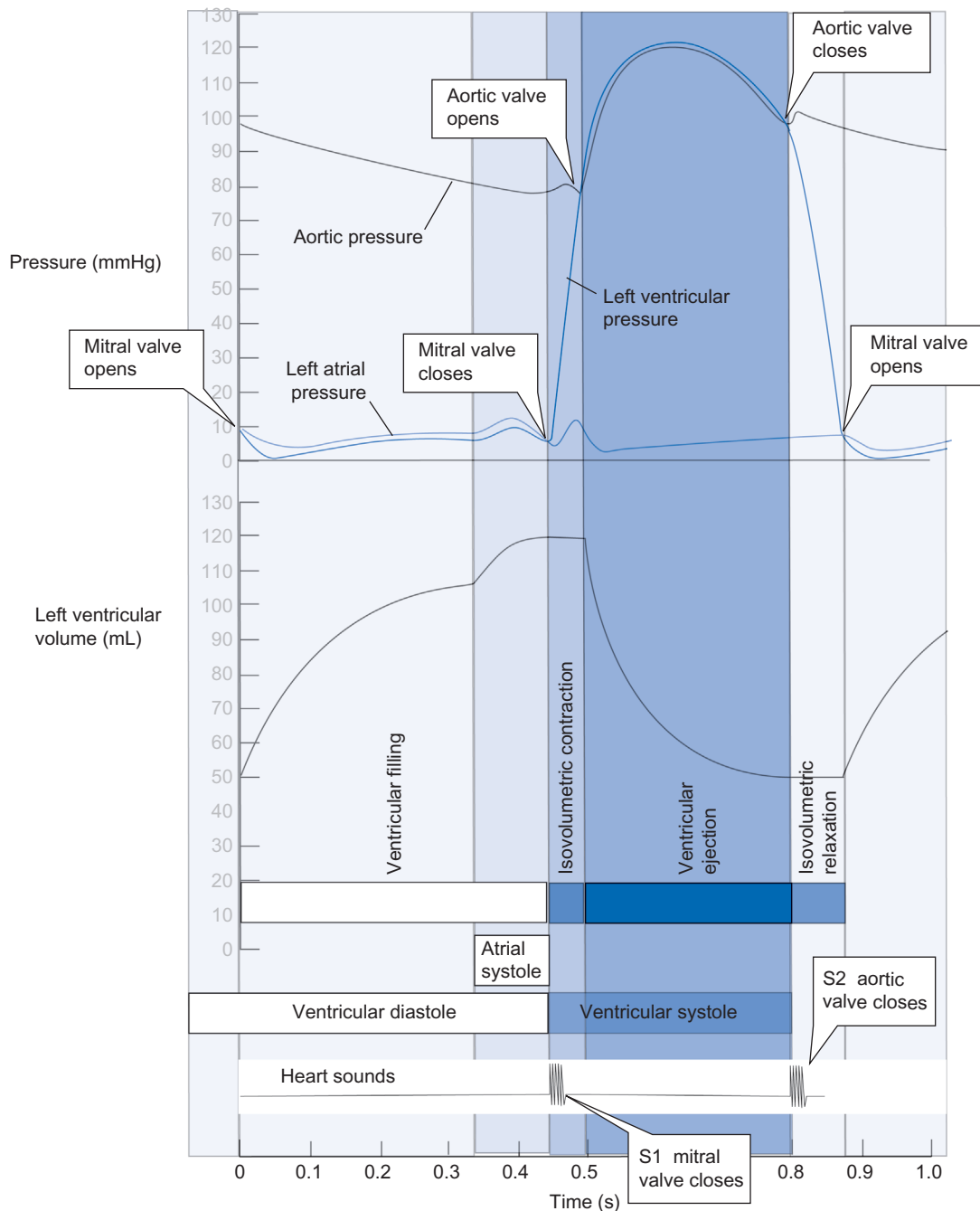


FIGURE 5.4.7 Sequence of some events during the heart cycle. The cycle consists of one period of left ventricular diastole (relaxation) followed by a phase of left ventricular systole (contraction). Because there is a lag between contraction and development of pressure, these periods do not precisely correspond to the periods of left ventricular filling and ejection, as shown. The mitral valve opens when left atrial pressure exceeds left ventricular pressure and closes when contraction of the ventricle raises left ventricular pressure to equal, and then exceed, left atrial pressure. Closure of the mitral valve contributes to the first heart sound, S1. Similarly, the aortic valve opens when contraction of the heart increases pressure to levels above aortic diastolic pressure. Continued contraction causes ejection of blood from the left ventricle. On relaxing, the pressure in the left ventricle falls. The first phase is isovolumetric relaxation. When left ventricular pressure falls below aortic pressures, the aortic valves snap shut, contributing to the second heart sound, S2.

aortic and pulmonary valves are closed. Ventricular filling is completed by contraction of the atria during the last 0.12 s. The contraction of the atria is not really necessary, but without it blood can stagnate in the atria, leading to clots, which can be released into the circulation and cause strokes or heart attacks. The volume of blood in the left ventricle at the end of ventricular

filling is called the **end-diastolic volume (EDV)**, which is about 120 mL in the adult human. The corresponding pressure, the end-diastolic pressure (EDP), is about 4–7 mmHg.

- ventricles fill for about 0.45 s;
- mitral and tricuspid valves open;

- aortic and pulmonary valves close;
- End-diastolic volume ≈ 120 mL;
- End-diastolic pressure $\approx 4\text{--}7$ mmHg.

ISOVOLUMETRIC CONTRACTION

Ventricular contraction occurs over about 0.35 s and consists of two phases. The first phase is a brief isovolumetric contraction, lasting about 0.05 s and a longer ejection phase of about 0.30 s. The isovolumetric contraction causes left ventricular pressure to rise above atrial pressure, which closes the mitral valve and produces the first heart sound. The aortic valve opens at the end of isovolumetric contraction when left ventricular pressure exceeds aortic pressure.

- ventricular contraction for about 0.05 s;
- mitral and tricuspid valves closed;
- aortic and pulmonary valves closed.

EJECTION

When ventricular pressure rises further to exceed aortic pressure or pulmonary artery pressure, the aortic and pulmonary valves open and blood flows from the ventricles into the systemic circulation or the pulmonary circulation. The ejection phase of the cardiac cycle ends when the aortic valve snaps shut, producing the heart's second sound. At the end of the ejection phase the volume of blood remaining in the left ventricle, its end-systolic volume (ESV), is about 50 mL. Thus, the **stroke volume**, the volume of blood ejected with each heart beat, is the difference between the end-diastolic volume and the end-systolic volume = $120\text{ mL} - 50\text{ mL} = \text{about } 70\text{ mL}$. The **ejection fraction** is the fraction of the EDV that is ejected. The typical value for the ejection fraction is $70/120 = 0.58$.

- ejection takes about 0.30 s;
- mitral and tricuspid valves closed;
- aortic and pulmonary valves open;
- peak pressure of about 25 mmHg (pulmonary circulation) or 120 mmHg (systemic circulation).

ISOVOLUMETRIC RELAXATION

When the aortic and pulmonary valves close, the heart relaxes isovolumetrically because both the outflow valves and inflow valves are closed, and so no fluid moves across them. This phase lasts until the intraventricular pressure falls below the pressure in the atria, at which time the mitral and tricuspid valves open again. The isovolumetric relaxation lasts about 0.08 s. The volume throughout this phase is the ESV, approximately 50 mL. The atrioventricular (AV) valves open at an atrial pressure of about 7 mmHg.

- isovolumetric relaxation lasts about 0.08 s;
- aortic and pulmonary valves closed;
- mitral and tricuspid valves closed.

AN AUTOMATIC ELECTRICAL SYSTEM CONTROLS THE CONTRACTION OF THE HEART

THE HEART BEAT ORIGINATES IN THE SA NODE, THE HEART'S PACEMAKER

The heart contains its own rhythm generator or **pacemaker** and will beat on its own when isolated from the body and perfused appropriately, or when it is transplanted from a donor to a recipient. The normal pacemaker activity is located in a specialized group of cells that comprise the **sinoatrial node** or **SA node**. The SA node consists of a small strip of modified muscle tissue, about $20\text{ mm} \times 4\text{ mm}$, on the posterior wall of the atrium near the superior vena cava. Cells in the SA node have an unstable membrane potential that spontaneously depolarizes to produce an action potential about once per second. This action potential is conducted through the atrial tissue to the AV node, as shown in Figure 5.4.8. The wave of electrical activity that spreads outward from the SA node is called the **cardiac impulse**. Other parts of the heart also have intrinsic rhythm, but the SA node has the higher intrinsic frequency, so it entrains all parts of the heart to its rhythm.

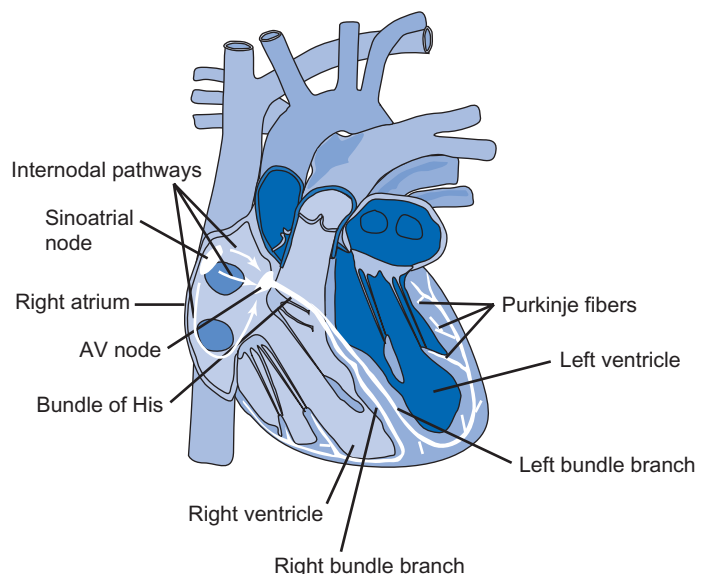


FIGURE 5.4.8 Conduction system of the heart. The heart beat originates in the SA node at the junction of the venae cavae with the right atria. Excitation spreads through the atrial muscle to the AV node, which connects to the bundle of His. This conducts the excitation across the annulus fibrosus, which insulates the ventricles from the atria. The bundle of His separates into a left bundle branch that activates the left ventricle and a right bundle branch. The conducting fibers terminate in wide conducting fibers called Purkinje fibers.

SPECIAL PATHWAYS CONDUCT THE ELECTRICAL SIGNAL FOR CONTRACTION

Effective pumping requires coordinated contraction of the heart's four chambers. This is accomplished through special conducting fibers. Fibers called **Bachmann's bundle** relay the impulse to the left atria. This coordinates right and left atrial contraction. The velocity of conduction in Bachmann's bundle is about 1 m s^{-1} .

THE AV NODE DELAYS THE TRANSMISSION OF THE CARDIAC IMPULSE

The cardiac impulse travels to the AV node through the atrial tissue. The AV node is a small group of cells and connective tissue at the posterior and lower area of the right atrium. The annulus fibrosus forms an insulating band between the atria and the ventricles, and the AV node begins the only electrical pathway across it. Transmission of the cardiac impulse is delayed here for about 0.1 s. This **nodal delay** allows for contraction of the atria and complete filling of the ventricles before beginning contraction of the ventricles. AV nodal cells conduct the impulse at $0.01\text{--}0.05 \text{ m s}^{-1}$. AV nodal cells also have intrinsic rhythm and will take over if transmission from the SA node fails. Because AV nodal cells have a lower frequency, failure of the SA node will cause lower frequency of heart beats.

THE BUNDLE OF HIS AND ITS BRANCHES CONDUCT THE IMPULSE TO THE APEX OF THE HEART

The bundle of His consists of wide, fast-conducting muscle fibers that carry the cardiac impulse through the insulating annulus fibrosus into the fibrous upper part of the ventricular septum. There, it becomes the **left bundle branch** and the **right bundle branch**. The right bundle branch travels along the right side of the septum

and supplies excitation to the right ventricle. The left bundle branch is really two sets of fibers, one anterior and another posterior. These course down the left side of the septum and supply the left ventricle. The bundle fibers terminate in an extensive network of large fibers called **Purkinje fibers**. These fibers conduct the impulse at high velocity, $3\text{--}5 \text{ m s}^{-1}$. They distribute the impulse to the subendocardium, the layer of cells beneath the endocardium. The endocardium lines the cavities of the heart.

HEART CELLS CONDUCT ELECTRICAL SIGNALS TO NEIGHBORING CELLS THROUGH INTERCALATED DISKS

Contractile cells in the heart generally have one or two nuclei and they form a branching network, as shown schematically in [Figure 5.4.9](#). The junction between adjacent contractile cardiac cells is called the **intercalated disk**. It is easily distinguished in both light and electron microscopy because it forms a dark and wide band running across the width of the cell. Here the membranes of two adjacent cells interdigitate and are linked by cytoskeletal elements. In addition, the intercalated disks form a conductive pathway between two adjacent cells—the cells are electrically coupled and excitation of one cell readily passes to the second. All cardiomyocytes have electrical connections to at least two and often more cells. The branches assure that the cells form a **functional syncytium** (from “syn”, meaning “together” and “kyto”, meaning “cell”). Thus electrical activity is conducted along the contractile cells of the heart and local neighbors contract in sequence, one shortly after the other. The ventricular muscle conducts the impulse at about $0.5\text{--}1 \text{ m s}^{-1}$. This local connectivity of the cardiomyocytes coordinates local contraction but not global contraction. Global coordination is provided by the distribution network.

Clinical Applications: Tetralogy of Fallot

The tetralogy of Fallot is a congenital heart defect that consists of four components:

1. Pulmonary stenosis—a narrowing of the outflow of the right ventricle.
2. Overriding aorta—the aorta attaches to both right and left ventricle.
3. Ventricular septal defect—a hole in the septum connects deoxygenated with oxygenated blood.
4. Right ventricular hypertrophy—this is secondary to the defects listed above.

Because of the mixing of deoxygenated with oxygenated blood, due to the ventricular septal defect, and preferential flow of blood from both ventricles through the aorta due to obstructed flow through the pulmonary artery, the blood in the systemic

circulation has low oxygenation. Babies with the condition are called “blue babies” because of the resulting cyanosis.

Surgical treatment originally consisted of constructing a shunt between the subclavian artery and the pulmonary artery, redirecting a large proportion of partially oxygenated blood to the lungs and increasing flow through the pulmonary circulation and greatly relieving symptoms. The procedure was invented by the team of Alfred Blalock, Helen Taussig, and Vivien Thomas at Johns Hopkins in 1944 and is portrayed in the movie *Something the Lord Made*. Although palliative, the Blalock-Thomas-Taussig shunt was not curative. Total surgical repair of tetralogy of Fallot was first successful in 1954, and since then survival rates have increased markedly.

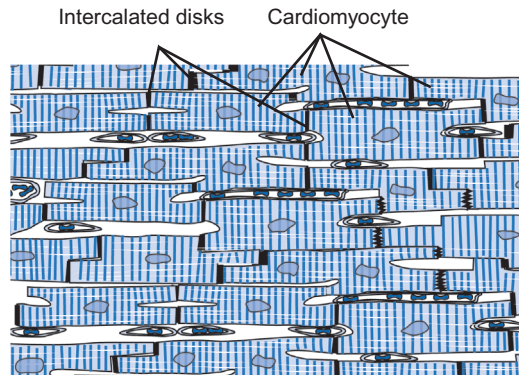


FIGURE 5.4.9 Schematic diagram of the functional syncytium of the heart. Contractile cells have cross striations and connect to each other at intercalated disks, special structures that allow passage of electrical activity from one cell to the next.

SUMMARY

The heart is a four-chambered pump. Blood returns to the heart from the tissues through the superior and inferior venae cavae and enters the right atrium. During ventricular filling, blood drains from the right atrium through the tricuspid valve into the right ventricle. The ventricles are given an extra boost of filling by atrial contraction. When the right ventricle contracts, the increased pressure closes the tricuspid valve and, at higher pressure, the pulmonary valve opens and blood flows into the pulmonary artery to the lungs. Blood returns to the heart through the pulmonary veins, which empty into the left atrium. During ventricular filling, blood leaves the left atrium through the mitral valve into the left ventricle. Contraction of the left ventricle increases the pressure within the ventricle, which closes the mitral valve and opens the aortic valve. Blood is then ejected into the aorta to supply the rest of the body. Thus, the four pressure-operated valves provide for unidirectional flow of blood sequentially through the pulmonary and systemic circulations. The four valves are situated within a fibrous ring, the annulus fibrosus, that approximates a plane that divides and electrically isolates the atria from the ventricles. Simultaneous closure of the tricuspid and mitral valves

produces the first heart sound; closure of the aortic and pulmonary valves causes the second heart sound.

Coordination of the contraction of the heart is provided by a special conduction system that consists of modified muscle tissues rather than nerve. The rhythm of the heart is set by the sinoatrial (SA) node at the posterior of the right atrium near the vena cava. The SA node is the pacemaker because cells in the SA node have the fastest spontaneous rate of depolarization, but other parts of the heart also have spontaneous rhythm. The wave of electrical activity, the cardiac impulse, can be conducted by the contractile cells of the heart because they are electrically coupled at their junctions, the intercalated disks. The cardiac impulse is conducted to the AV node, which delays the impulse and then sends it through the annulus fibrosus by the bundle of His. The bundle of His consists of larger, specialized muscle cells that conduct the impulse more rapidly. The right and left bundle branches derive from the bundle of His and separately conduct the impulse to the right and left ventricles. In this way, the conducting system distributes excitation to the heart muscle to coordinate its contraction from the apex upwards.

REVIEW QUESTIONS

1. Where does blood come from that fills the right atrium? Left atrium? Where does blood go when it is pumped from the right ventricle? Left ventricle?
2. What valve separates right atrium from right ventricle? What valve separates left atrium from left ventricle?
3. Why is the right ventricular wall thinner than the left ventricular wall?
4. What valve prevents backflow in the pulmonary artery? In the aorta?
5. What controls opening and closing of the valves in the heart?
6. What causes the first heart sound? The second? Why are there not more heart sounds?
7. What is a bruit? What causes it?
8. What is the pacemaker of the heart? Where is it? What is the AV node and where is it?