Section 1: Structure of the Heart

Learning Outcomes

18.1 Describe the heart’s location, shape, its four chambers, and the pulmonary and systemic circuits.

18.2 Describe the location and general features of the heart.

18.3 Describe the structure of the pericardium and explain its functions, identify the layers of the heart wall, and describe the structures and functions of cardiac muscle.

18.4 Describe the cardiac chambers and the heart’s external anatomy.
Section 1: Structure of the Heart

Learning Outcomes (continued)

18.5 Describe the major vessels supplying the heart, and cite their locations.

18.6 Trace blood flow through the heart, identifying the major blood vessels, chambers, and heart valves.

18.7 Describe the relationship between the AV and semilunar valves during a heartbeat.

18.8 Define arteriosclerosis, and explain its significance to health.
Module 18.1: The heart has four chambers that pump and circulate blood through the pulmonary and systemic circuits

**Cardiovascular system** = heart and blood vessels transporting blood

**Heart**—directly behind sternum

- **Base**—superior
  - where major vessels are
  - ~1.2 cm (0.5 in.) to left
  - 3rd costal cartilage

- **Apex**—inferior, pointed tip
  - ~12.5 cm (5 in.) from base
  - ~7.5 cm (3 in.) to left
  - 5th intercostal space
Borders of the heart

- Superior border = base
- Right border = right atrium
- Left border = left ventricle, part of left atrium
- Inferior border = right ventricle
Heart = 2-sided pump with 4 chambers

- **Right atrium** receives blood from systemic circuit
- **Right ventricle** pumps blood into pulmonary circuit
- **Left atrium** receives blood from pulmonary circuit
- **Left ventricle** pumps blood into systemic circuit
Module 18.1: Review

A. Describe the location and position of the heart.
B. Compare the base of the heart with the apex.
C. Name the four chambers of the heart.
D. Compare the volume of blood each circuit receives from contraction of the ventricles.

Learning Outcome: Describe the heart’s location, shape, its four chambers, and the pulmonary and systemic circuits.
Module 18.2: The heart is located in the mediastinum and is enclosed by the pericardial cavity

**Mediastinum** = space or region in thorax between the two pleural cavities (between the lungs)
Module 18.2: The pericardium

Pericardium = sac-like structure wrapped around heart

- *Fibrous pericardium*: Outermost layer; dense fibrous tissue that extends to sternum and diaphragm
- *Serous pericardium* (2 layers): Outer parietal layer lines fibrous pericardium; inner serous layer covers surface of the heart
- *Pericardial cavity*: Space between serous layers; contains 15–50 mL of pericardial fluid secreted from serous membranes; lubricates movement of heart.
Module 18.2: The pericardium

Pericardium = sac-like structure wrapped around heart (continued)
- Pericarditis = inflammation of the pericardium
- Cardiac tamponade = excess accumulation of pericardial fluid
Module 18.2: The pericardium

Relationship between heart and pericardium

- Push fist into partly inflated balloon
- Fist = heart
- Wrist = base of heart, with **great vessels**
- Inside of balloon = pericardial cavity
Module 18.2: The pericardium

Superior view of the thorax, showing the positions of the pericardium, pericardial cavity, heart, mediastinum, and three of the great vessels: aorta, superior vena cava, and pulmonary trunk.
Module 18.2: Review

A. Define mediastinum.
B. Describe the heart’s location in the body.
C. Why can cardiac tamponade be a life-threatening condition?

Learning Outcome: Describe the location and general features of the heart.
Module 18.3: The heart wall contains concentric layers of cardiac muscle tissue

Heart wall has 3 layers:

1. **Pericardium**—outer fibrous pericardium (dense fibrous tissue) and 2-layered **serous pericardium** (mesothelium and underlying areolar tissue)
   - **Epicardium** = visceral layer of serous pericardium

2. **Myocardium**—middle layer; concentric layers of cardiac muscle; supporting blood vessels, nerves

3. **Endocardium**—(endo = within); innermost layer; simple squamous epithelium (renamed endothelium inside heart and vessels) and areolar tissue; endocardium is continuous with endothelium in vessels and covers heart valves
The layers of the heart wall

The Pericardium

Parietal Layer of Serous Pericardium
- Dense fibrous tissue
- Areolar tissue
- Mesothelium

Visceral Layer of Serous Pericardium
- Mesothelium
- Areolar tissue

Myocardium

Endocardium
- Endothelium
- Areolar tissue

Connective tissues

Pericardial cavity (contains pericardial fluid)
Module 18.3: The heart wall

Myocardium arrangement:

- Atrial musculature wraps around atria in figure-8 pattern
- Ventricular musculature
  - Superficial layer surrounds both ventricles
  - Deeper layers spiral around and between the ventricles
Module 18.3: The heart wall—cardiac muscle

Cardiac muscle vs. skeletal muscle:
1. Smaller cell size (avg. 10–20 μm diameter; 50–100 μm length)
2. Single, centrally located nucleus
3. **Intercalated discs** = branching interconnections between cells
4. Specialized intercellular connections
Module 18.3: The heart wall—cardiac muscle

Cardiac muscle tissue

- Only in heart
- Striated (from organized myofibrils and aligned sarcomeres)
- Almost totally dependent on aerobic metabolism (need oxygen) for energy
  - Abundant mitochondria and myoglobin (stores O₂)
  - Extensive capillaries

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Cardiac muscle tissue (continued)

- Intercalated discs
  - Intertwined plasma membranes of adjacent cardiac muscle cells
  - Attached by desmosomes and gap junctions
  - Gap junctions allow action potentials to spread cell to cell; allows all interconnected cells to function together as single unit = a functional syncytium
Module 18.3: Review

A. From superficial to deep, name the layers of the heart wall.

B. Describe the tissue layers of the pericardium.

C. Why is it important that cardiac tissue contain many mitochondria and capillaries?

Learning Outcome: Describe the structure of the pericardium and explain its functions, identify the layers of the heart wall, and describe the structures and functions of cardiac muscle.
Module 18.4: The boundaries between the four chambers of the heart can be identified on its external surface

Visible on anterior surface

- All four chambers
- **Auricle** of each atrium (expandable pouch)
- **Coronary sulcus**—groove separating atria and ventricles
- **Anterior interventricular sulcus**—groove marking boundary between the two ventricles
- **Ligamentum arteriosum**—fibrous remnant of fetal connection between aorta and pulmonary trunk
Surface anatomy of the heart (anterior view)

- Aortic arch
- Ligamentum arteriosum
- Ascending aorta
- Superior vena cava
- Auricle
- Right atrium
- Auricle of left atrium
- Right ventricle
- Fat and vessels in the anterior interventricular sulcus
- Coronary sulcus
- Left ventricle
- Anterior interventricular sulcus
- Anterior surface

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Module 18.4: Heart surface anatomy

Visible on posterior surface:

- All four chambers
- *Pulmonary veins* (4) returning blood to left atrium
- *Superior and inferior venae cavae* returning blood to right atrium
- *Coronary sinus*—returns blood from myocardium to right atrium
- *Posterior interventricular sulcus*—groove marking boundary between the two ventricles
Surface anatomy of the heart (posterior view)

- Aortic arch
- Right pulmonary artery
- Superior vena cava
- Right pulmonary veins (superior and inferior)
- Inferior vena cava
- Left atrium
- Right atrium
- Left pulmonary artery
- Left pulmonary veins
- Fat and vessels in the coronary sulcus
- Coronary sinus
- Left ventricle
- Right ventricle
- Posterior interventricular sulcus

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Module 18.4: Review

A. The anterior view of the heart is dominated by which chambers?

B. Which structures collect blood from the myocardium, and into which heart chamber does this blood flow?

C. Name and describe the shallow depressions and grooves found on the heart’s external surface.

Learning Outcome: Describe the cardiac chambers and the heart’s external anatomy.
Module 18.5: The heart has an extensive blood supply

Coronary circulation

- Continuously supplies cardiac muscle (myocardium) with oxygen/nutrients
- **Left and right coronary arteries** arise from ascending aorta; fill when ventricles are relaxed (diastole)
- Myocardial blood flow may increase to 9 times the resting level during maximal exertion
Module 18.5: Coronary circulation

Right coronary artery

- Supplies right atrium, parts of both ventricles, and parts of cardiac (electrical) conducting system
- Follows coronary sulcus (groove between atria and ventricles)
- Main branches:
  - **Marginal arteries**—supply right ventricle
  - **Posterior interventricular (posterior descending) artery**—runs in posterior interventricular sulcus; supplies interventricular septum and adjacent parts of ventricles
Module 18.5: Coronary circulation

Left coronary artery

- Supplies left ventricle, left atrium, interventricular septum
- Main Branches:
  - **Anterior interventricular artery (left anterior descending artery)**—follows anterior interventricular sulcus; supplies interventricular septum and adjacent parts of ventricles
  - **Circumflex artery**—follows coronary sulcus to the left; meets branches of right coronary artery posteriorly; **marginal artery** off circumflex supplies posterior of left ventricle
Coronary circulation, main arteries (anterior view)

- Aortic arch
- Ascending aorta
- Left atrium
- Pulmonary trunk
- Right atrium
- Left ventricle
- Right ventricle
- Left coronary artery
  - Circumflex artery
- Anterior interventricular artery (left anterior descending) artery
- Arterial anastomoses between anterior and posterior interventricular arteries

Right Coronary Artery
Right coronary artery
Marginal arteries
Coronary circulation, main arteries (posterior view)
Coronary circulation—veins (anterior)

- **Great cardiac vein**—in anterior interventricular sulcus
  - Drains area supplied by anterior interventricular artery
  - Empties into coronary sinus posteriorly
- **Anterior cardiac veins**
  - Drain anterior surface of right ventricle
  - Empty directly into the right atrium
Module 18.5: Coronary circulation

Coronary circulation—veins (posterior)

- **Coronary sinus**—expanded vein that empties into right atrium
- **Posterior vein of left ventricle**—drains area supplied by circumflex artery
- **Middle cardiac vein**—drains area supplied by posterior interventricular artery; empties into coronary sinus
- **Small cardiac vein**—drains posterior of right atrium/ventricle; empties into coronary sinus
Module 18.5: Coronary circulation

Blood flow through the coronary circuit is maintained by changing blood pressure and elastic rebound

- Left ventricular contraction forces blood into aorta, elevating blood pressure there, stretching aortic walls
- Left ventricular relaxation—pressure decreases, aortic walls recoil (elastic rebound), pushing blood in both directions
  - Forward into systemic circuit
  - Backward into coronary arteries
Module 18.5: Review

A. Describe the areas of the heart supplied by the right and left coronary arteries.

B. Compare the anterior cardiac veins to the posterior vein of the left ventricle.

C. List the arteries and veins of the heart.

D. Describe what happens to blood flow during elastic rebound.

Learning Outcome: Describe the major vessels supplying the heart, and cite their locations.
Module 18.6: Internal valves control the direction of blood flow between the heart chambers and great vessels

Each side of heart has two chambers: an *atrium* (receives blood) sends blood to a *ventricle* (pumps blood out of heart)

- Right and left atria are separated by the *interatrial septum*
- Right and left ventricles are separated by *interventricular septum* (much thicker)
Module 18.6: Internal valves control the direction of blood flow between the heart chambers and great vessels

- **Atrioventricular (AV) valves**—between each atrium and ventricle
  - Allow only one-way blood flow from atrium into ventricle

- **Semilunar valves**—at exit from each ventricle; allow only one-way blood flow from ventricle out into aorta or pulmonary trunk
Internal anatomy of the heart, showing chambers and heart valves (coronal section)
Module 18.6: Heart valves

Atria

- **Right atrium** receives deoxygenated blood from superior and inferior venae cavae and coronary sinus
  - **Fossa ovalis**—remnant of fetal *foramen ovale* that allowed fetal blood to pass between atria; closes at birth

- **Left atrium** receives oxygenated blood from pulmonary veins

- **Pectinate muscles**—muscular ridges located inside both atria along the anterior atrial wall and in the auricles
Module 18.6: Heart valves

Ventricles

- **Right ventricle**—receives blood from right atrium through tricuspid valve (has three **cusps** or flaps), also called the **right atrioventricular (AV) valve**
  - With contraction, blood exits through the **pulmonary valve** (pulmonary semilunar valve) into the pulmonary trunk
Module 18.6: Heart valves

Ventricles (continued)

- **Left ventricle**—much thicker wall than right ventricle
  - Receives blood from left atrium through **mitral valve**, also called **bicuspid valve** (two cusps) or **left atrioventricular valve**
  - With contraction, blood exits through the aortic valve (aortic semilunar valve) into the ascending aorta
- **Trabeculae carneae**—muscular ridges inside both ventricles
Module 18.6: Heart valves

AV valve structure (tricuspid and mitral valve)

- Each has three (tricuspid) or two (mitral/bicuspid) cusps
- Cusps attach to tendon-like connective tissue bands = chordae tendineae
- Chordae tendineae anchored to thickened cone-shaped papillary muscles
- Moderator band—thickened muscle ridge providing rapid conduction path; tenses papillary muscles just before ventricular contraction; prevents slamming or inversion of AV valve
Internal anatomy of the heart (coronal section)

- **Right Atrium**
  - Fossa ovalis
  - Pectinate muscles
  - Opening of the coronary sinus

- **Right Ventricle**
  - Tricuspid valve or right atrioventricular valve
  - Chordae tendineae
  - Papillary muscles
  - Pulmonary valve, or pulmonary semilunar valve

- **Left Atrium**

- **Left Ventricle**
  - Mitral valve, or left atrioventricular valve, or bicuspid valve
  - Aortic valve (aortic semilunar valve)
  - Trabeculae carneae
  - Interventricular septum

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Comparison between chambers

- Atria have similar workloads; walls about same thickness

- Ventricles have very different loads
  - Right ventricle—thinner wall; sends blood to adjacent lungs (pulmonary circuit)
    - Contraction squeezes against left ventricle, forces blood out pulmonary trunk efficiently; minimal effort, low pressure
Module 18.6: Heart valves

Comparison between chambers (continued)

- Ventricles have very different loads (continued)
  - Left ventricle—very thick wall, rounded chamber
    - 4–6 times the pressure of right; sends blood through entire systemic circuit
    - Contraction decreases diameter and apex-to-base distance
    - Reduces right ventricular volume, aiding its emptying
Module 18.6: Heart valves

- Posterior interventricular sulcus
- Thin wall of left ventricle
- Thin wall of right ventricle
- Fat in anterior interventricular sulcus
Module 18.6: Heart valves

The wall of the left ventricle is much thicker than that of the right ventricle because it must generate tremendous force.

Note the shape and change in size of both ventricles when they contract.
Module 18.6: Review

A. Why is the left ventricle more muscular than the right ventricle?

B. Damage to the semilunar valve on the right side of the heart would affect blood flow to which vessel?

Learning Outcome: Trace blood flow through the heart, identifying the major blood vessels, chambers, and heart valves.
Module 18.7: When the heart beats, the AV valves close before the semilunar valves open, and the semilunar valves close before the AV valves open

When ventricles are relaxed, they fill

- **AV valves**—open
  - Chordae tendineae are loose
- **Semilunar valves**—closed
  - Blood pressure from pulmonary and systemic circuits keeps them closed

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Position of heart valves while ventricles are filling (ventricular relaxation)

Superior view of cardiac valves

- Right ventricle
- Mitral (left AV) valve (open)
- Left ventricle (dilated)
- Tricuspid (right AV) valve (open)
- Aortic valve (closed)
- Pulmonary valve (closed)

Frontal section through left atrium and ventricle

- Pulmonary veins
- Aortic valve (closed)
- Mitral (left AV) valve (open)
- Left ventricle (dilated)
- Right ventricle

KEY
- Oxygenated blood
- Deoxygenated blood

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Module 18.7: Valves control direction of flow

When ventricles contract, they empty

- AV valves—closed
  - Pressure from contracting ventricles pushes cusps together
  - Papillary muscles tighten chordae tendineae so cusps can’t invert into atria; prevents backflow (regurgitation)

- Semilunar valves—open
  - Ventricular pressure overcomes pressure in pulmonary trunk and aorta that held them shut
Position of heart valves while ventricles are emptying (ventricular contraction)
Module 18.6: Valves control direction of flow

Cardiac skeleton (fibrous skeleton)

- Flexible connective tissue frame
  - Interconnected bands of dense connective tissue
  - Encircle heart valves, stabilize their positions
  - Also surrounds base of aorta and pulmonary trunk

- Electrically isolates atrial from ventricular myocardium
Module 18.7: Valves control direction of flow

Pulmonary and aortic (semilunar) valves

- Each has three half-moon shaped cusps
  - Prevent backflow of blood from aorta and pulmonary trunk back into ventricles
  - No muscular brace needed—cusps support each other when closed

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Valvular heart disease (VHD)
- Valve function deteriorates until heart cannot maintain adequate blood flow
- Congenital malformations or heart inflammation (carditis)
- Severe cases may require replacement with prosthetic valve
  - Bioprosthetic valves come from pigs or cows
Module 18.7: Review

A. Define *cardiac regurgitation*.

B. Describe the structural and functional roles of the cardiac skeleton.

C. What do semilunar valves prevent?

*Learning Outcome:* Describe the relationship between the AV and semilunar valves during a heartbeat.
Module 18.8: Arteriosclerosis can lead to coronary artery disease

**Arteriosclerosis** *(arterio-, artery + sclerosis, hardness)*

- Thickening/toughening of arterial walls
- Related complications account for about half of U.S. deaths
  - Coronary artery disease (CAD) = arteriosclerosis of coronary vessels
  - Arteriosclerosis of brain arteries can lead to strokes
Module 18.8: Arteriosclerosis

Atherosclerosis

- formation of lipid deposits in arterial tunica media and damage to endothelium
- Most common form of arteriosclerosis; often associated with elevated blood cholesterol
- Fatty tissue mass (plaque) in vessel; restricts blood flow
Module 18.8: Arteriosclerosis

Risk factors

- Age (elderly)
- Sex (male)
- High blood cholesterol levels
- High blood pressure
- Cigarette smoking
Module 18.8: Arteriosclerosis

Atherosclerosis treatment

- Replace damaged segment of the vessel
- Compressing plaque with balloon angioplasty
  - Catheter inserted past blockage; balloon inflated to press plaque against wall and open vessel
  - Most effective for small, soft plaques
  - Very low surgical mortality rate (about 1%)
  - Very high success rate (>90%)
  - Can be outpatient
Module 18.8: Arteriosclerosis

Coronary artery disease (CAD)

= Areas of partial or complete blockage of coronary circulation

• Reduces blood flow to area (coronary ischemia)
• Usually from atherosclerosis in a coronary artery or associated blood clot (thrombus)
• Seen in digital subtraction angiography (DSA)
• May treat with wire-mesh tube (stent) to hold vessel open
This is a color-enhanced digital subtraction angiography (DSA) scan of a normal heart. The major branches of the left and right coronary arteries are clearly visible.

This is a color-enhanced DSA scan of the heart of a person with advanced CAD. Blood flow to the ventricular myocardium is severely restricted.
Module 18.8: Arteriosclerosis
Module 18.8: Review

A. Compare arteriosclerosis with atherosclerosis.
B. What is coronary ischemia?
C. Describe the purpose of a stent.

*Learning Outcome:* Define arteriosclerosis, and explain its significance to health.
Section 2: Cardiac Cycle

Learning Outcomes

18.9 Explain the complete round of cardiac systole and diastole.

18.10 Explain the events of the cardiac cycle, and relate the heart sounds to specific events.

18.11 Describe an action potential in cardiac muscle, and explain the role of calcium ions.

18.12 Describe the components and functions of the conducting system of the heart.
Section 2: Cardiac Cycle

Learning Outcomes (continued)

18.13 Identify the electrical events shown on an electrocardiogram.

18.14 Describe the factors affecting the heart rate.

18.15 Describe the variables that influence stroke volume.

18.16 Explain how stroke volume and cardiac output are coordinated.
Module 18.9: The cardiac cycle is a complete round of systole and diastole

Cardiac cycle = period between start of one heartbeat and the next; heart rate = number of beats per minute

- Two atria contract first to fill ventricles; two ventricles then contract to pump blood into pulmonary and systemic circuits
Module 18.9: The cardiac cycle is a complete round of systole and diastole

**Cardiac cycle (continued)**

- Two phases:
  - Contraction (**systole**)—blood leaves the chamber
  - Relaxation (**diastole**)—chamber refills
Module 18.9: The cardiac cycle

Sequence of contractions

1. Atria contract together first (atrial systole)
   - Push blood into the ventricles
   - Ventricles are relaxed (diastole) and filling

2. Ventricles contract together next (ventricular systole)
   - Push blood into the pulmonary and systemic circuits
   - Atria are relaxed (diastole) and filling

   Typical cardiac cycle lasts 800 msec
Phases of cardiac cycle for a heart rate of 75 bpm
Module 18.9: Review

A. Define *cardiac cycle*.

B. Give the alternate terms for heart contraction and heart relaxation.

C. Compare the duration of atrial and ventricular systole at a representative heart rate of 75 bpm.

*Learning Outcome*: Explain the complete round of cardiac systole and diastole.
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle, diagrammed for heart rate of 75 bpm

1. Cardiac cycle begins—all four chambers are relaxed (diastole; ventricles are passively refilling)
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

2. **Atrial systole** (100 msec)—atria contract; finish filling ventricles
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

3. **Atrial diastole** (270 msec)—continues until start of next cardiac cycle (through ventricular systole)
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

4. **Ventricular systole—first phase.** Contracting ventricles push AV valves closed but not enough pressure to open semilunar valves (= isovolumetric contraction—no volume change)
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

5. **Ventricular systole—second phase.** Increasing pressure opens semilunar valves; blood leaves ventricle (= ventricular ejection)
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

6. **Ventricular diastole—early.** Ventricles relax and their pressure drops; blood in aorta and pulmonary trunk backflows, closes semilunar valves.
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

7. **Isovolumetric relaxation.** All valves closed; no volume change; blood passively filling atria
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Phases of cardiac cycle (continued)

8. **Ventricular diastole**—late. All chambers relaxed; AV valves open; ventricles fill passively to ~70%
SmartArt Video: The Cardiac Cycle
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Pressure changes in aorta during the cardiac cycle

- Increase in pressure with opening of aortic valve
- Drop in pressure with closing of aortic valve
  - Followed by a short pressure rise as aortic elastic walls recoil
  - Produces a valley in pressure tracing called the dicrotic notch (*dikrotos*, double beating)
Pressure changes in aorta and left chambers during the cardiac cycle. Note the **dicrotic** notch, which occurs when the elastic walls of the aorta expand to receive incoming blood. The peak that follows shows the effect of elastic recoil.

![Graph showing pressure changes in aorta and left chambers during the cardiac cycle.](image-url)
Module 18.10: The cardiac cycle creates pressure gradients that maintain blood flow

Heart sounds

- **S₁** ("lubb")—when AV valves close; marks start of ventricular contraction
- **S₂** ("dupp")—when semilunar valves close
- **S₃** and **S₄**—very faint; rarely heard in adults
  - **S₃**—blood flowing into ventricles
  - **S₄**—atrial contraction
Module 18.10: Review

A. List the phases of the cardiac cycle.
B. What are the two phases of ventricular systole?
C. Is the heart always pumping blood when pressure in the left ventricle is rising? Explain.

Learning Outcome: Explain the events of the cardiac cycle, and relate the heart sounds to specific events.
Module 18.11: Cardiac muscle cell contractions last longer than skeletal muscle fiber contractions primarily because of differences in calcium ion membrane permeability

<table>
<thead>
<tr>
<th>Skeletal Muscle</th>
<th>Cardiac Muscle</th>
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<tbody>
<tr>
<td>• Brief action potential; ends as short twitch contraction begins.</td>
<td>• Long action potential</td>
</tr>
<tr>
<td>• Contraction ends when sarcoplasmic reticulum recovers Ca(^{2+})</td>
<td>• Ca(^{2+}) enters cells over prolonged period</td>
</tr>
<tr>
<td>• Short refractory period ends before peak tension develops</td>
<td>• Long contraction (~250 msec)</td>
</tr>
<tr>
<td>• Twitches can summate; tetanus can occur</td>
<td>• Refractory period continues into relaxation</td>
</tr>
<tr>
<td></td>
<td>• No tetanic contractions occur (heart couldn’t pump blood)</td>
</tr>
</tbody>
</table>
Skeletal muscle fiber contraction

**Skeletal Muscle Fiber**

- **Membrane potential (mV)**
  - Action potential
  - Membrane potential ranges from -85 to +30 mV.

- **Tension**
  - Contraction
  - Tension increases rapidly and then decreases.

**KEY**
- **Absolute refractory period**
- **Relative refractory period**
Cardiac muscle cell contraction

**Cardiac Muscle Cell**

- **Action potential**
- **Contraction**

**Key**
- Absolute refractory period
- Relative refractory period

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Module 18.11: Cardiac muscle cell contraction

Three stages of a cardiac muscle action potential

1. Rapid depolarization
2. Plateau
3. Repolarization
Module 18.11: Cardiac muscle cell contraction

1. **Rapid depolarization**—similar to that in skeletal muscle
   - At threshold, voltage-gated **fast sodium channels** open
   - Massive, rapid $\text{Na}^+$ influx
   - Channels open quickly and very briefly
Module 18.11: Cardiac muscle cell contraction

2. **Plateau**
   - Membrane potential stays near 0 mV due to 2 opposing factors:
     - **Fast sodium channels** close as potential nears +30 mV
     - Cell actively pumps Na\(^+\) out
   - Voltage-gated **slow calcium channels** open—Ca\(^{2+}\) influx (open slowly/stay open ~175 msec)
3. **Repololarization**
   - Slow calcium channels close
   - Slow potassium channels open; $K^+$ rushes out; causes rapid repolarization and restores resting potential
Module 18.11: Review

A. Why does tetany not occur in cardiac muscle?

B. List the three stages of an action potential in a cardiac muscle cell.

C. Describe slow calcium channels and the significance of their activity.

Learning Outcome: Describe an action potential in cardiac muscle, and explain the role of calcium ions.
Module 18.12: Electrical events of pacemaker cells and conducting cells establish the heart rate

**Cardiac output (CO)** = amount of blood pumped from the left ventricle each minute

- Determined by heart rate and stroke volume
- Precisely adjusted to meet needs of tissues
Module 18.12: Cardiac conducting system

To calculate cardiac output:

\[ \text{Cardiac Output} = \text{HR} \times \text{SV} \]

- **Heart rate (HR)** = # contractions/minute (beats per minute)
- **Stroke volume** = volume of blood pumped out of ventricle per contraction

By changing either or both HR and SV, cardiac output is precisely controlled to meet changing needs of tissues.

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Module 18.12: Cardiac conducting system

Autorhythmicity = cardiac muscle’s ability to contract at its own pace independent of neural or hormonal stimulation

Conducting system = network of specialized cardiac muscle cells (pacemaker and conducting cells) that initiate/distribute a stimulus to contract

- Components of conducting system:
  1. Sinoatrial node (SA node)
  2. Internodal pathways
  3. Atrioventricular node (AV node)
  4. AV bundle and bundle branches
  5. Purkinje fibers
Conducting system (continued)

1. **Sinoatrial (SA) node**
   - = pacemaker
   - Each heartbeat begins with action potential generated here
   - In posterior wall of right atrium, near superior vena cava
   - Impulse is initiated here and spreads through adjacent cells
   - Average 60–100 bpm
2. Internodal pathways

- Formed by conducting cells
- Distribute signal through both atria
3. **Atrioventricular (AV) node**
   - At junction between atria and ventricles
   - Relays signals from atria to ventricles
   - Has pacemaker cells that can take over pacing if SA node fails
   - AV pacing is slower—40 to 60 bpm
Conducting system (continued)

4. AV bundle

- Conducting cells transmit signal from AV node down through interventricular septum
- Usually only electrical connection between atria/ventricles
Module 18.12: Cardiac conducting system

Conducting system (continued)

5. Bundle branches

- Right and left branches
- Left bundle branch larger
- Conducting cells transmit signal to apex of heart, then spreading out in ventricular walls
Conducting system (continued)

6. Purkinje fibers
   • Radiate upward through ventricular walls
   • Large-diameter conducting cells
   • Propagate action potentials as fast as myelinated neurons
   • Stimulate ventricular myocardium and trigger contraction
Components of the cardiac conducting system

1. Sinoatrial (SA) node
2. Internodal pathways
3. Atrioventricular (AV) node
4. AV bundle
   - Bundle branches
5. Purkinje fibers

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The cardiac conduction system’s coordination of the cardiac cycle

1. Action potential generated at SA node; atrial activation begins.

   Time = 0

   ECG Tracing

2. Stimulus spreads across atrial surfaces within internodal pathways to AV node.

   Elapsed time = 50 msec

   P wave: atrial depolarization

3. A 100-msec delay occurs at the AV node. During this delay, atrial contraction occurs.

   Elapsed time = 150 msec

   P-R interval: conduction through AV node and AV bundle
The cardiac conduction system’s coordination of the cardiac cycle

4 Impulse travels along interventricular septum within the AV bundle and the bundle branches to the Purkinje fibers and, by the moderator band, to the papillary muscles of the right ventricle. Ventricular contraction begins.

Elapsed time = 175 msec

Q wave: beginning of ventricular depolarization

5 Impulse distributed by Purkinje fibers throughout ventricular myocardium; ventricular contraction completes.

Elapsed time = 225 msec

QRS complex: completion of ventricular depolarization
The cardiac conduction system’s coordination of the cardiac cycle

1. Action potential generated at SA node; atrial activation begins.
   
   Time = 0

   ECG Tracing

2. Stimulus spreads across atrial surfaces within internodal pathways to AV node.
   
   Elapsed time = 50 msec

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   Q wave: beginning of ventricular depolarization

5. Impulse distributed by Purkinje fibers throughout ventricular myocardium; ventricular contraction completes.
   
   Elapsed time = 225 msec

   QRS complex: completion of ventricular depolarization
SmartArt Video: *The Conducting System of the Heart*
Module 18.12: Review

A. Define autorhythmicity.

B. If the cells of the SA node failed to function, how would the heart rate be affected?

C. Why is it important for impulses from the atria to be delayed at the AV node before they pass into the ventricles?

Learning Outcome: Describe the components and functions of the conducting system of the heart.
Module 18.13: Normal and abnormal cardiac activity can be detected in an electrocardiogram

Electrocardiogram (ECG or EKG)

- Recording of heart’s electrical activities from body surface
- To assess performance of nodal, conducting, and contractile components
- If part of heart is damaged by heart attack, may see abnormal ECG pattern
- Appearance varies with placement and number of electrodes (leads)
Explanation of ECG features

**P wave** = atrial depolarization

- Atria begin contracting ~25 msec after P wave starts
Module 18.13: ECG

Explanation of ECG features (continued)

QRS complex = *ventricular depolarization*

- Larger wave due to larger ventricle muscle mass
- Ventricles begin contracting shortly after *R wave* peak
- Atrial repolarization also occurs now but is masked by QRS
Module 18.13: ECG

Explanation of ECG features (continued)

**T wave** = ventricular repolarization
Explanation of ECG features (continued)

P–R interval
- Period from start of atrial depolarization to start of ventricular depolarization
- >200 msec may mean damage to conducting pathways or AV node
Module 18.13: ECG

Explanation of ECG features (continued)

Q–T interval

- Time for ventricles to undergo a single cycle
- May be lengthened by electrolyte disturbances, medications, conduction problems, coronary ischemia, myocardial damage
ECGs valuable for detecting and diagnosing arrhythmias

**Cardiac arrhythmias** = abnormal patterns of cardiac electrical activity

- About 5% of healthy people experience a few abnormal heartbeats each day
- Not a clinical problem unless pumping efficiency is reduced
Cardiac arrhythmias (continued)

- Premature atrial contractions (PACs)
  - Often occur in healthy people
  - Normal atrial rhythm momentarily interrupted by “surprise” atrial contraction
  - Increased incidences caused by stress, caffeine, various drugs that increase permeability of the SA pacemakers
  - Normal ventricular contraction follows the atrial beat
Cardiac arrhythmias (continued)

- **Paroxysmal atrial tachycardia (PAT)**
  - Premature atrial contraction triggers flurry of atrial activity
  - Ventricles keep pace
  - Heart rate jumps to about 180 bpm
Cardiac arrhythmias (continued)

- **Atrial fibrillation**
  - Impulses move over atrial surface at up to 500 bpm
  - Atria quiver—not organized contraction
  - Ventricular rate cannot follow, may remain fairly normal
  - Atria nonfunctional, but ventricles still fill passively
  - Person may not realize there is an arrhythmia
Module 18.13: Arrhythmias

Cardiac arrhythmias (continued)

- Premature ventricular contractions (PVCs)
  - Purkinje cell or ventricular myocardial cell depolarizes; triggers premature contraction
    - Cell responsible called an **ectopic pacemaker** (pacemaker other than the SA node)
  - Single PVCs common, not dangerous
  - Frequency increased by epinephrine, stimulatory drugs, or ionic changes that depolarize cardiac muscle cells

![Premature Ventricular Contractions (PVCs)](image-url)
Cardiac arrhythmias (continued)

- Ventricular tachycardia
  - Also known as VT or V-tach
  - Defined as four or more PVCs without intervening normal beats
  - Multiple PVCs and V-tach may indicate serious cardiac problems
Cardiac arrhythmias (continued)

- Ventricular fibrillation
  - Also known as VF or V-fib
  - Responsible for condition known as cardiac arrest
  - Rapidly fatal because ventricles quiver, but cannot pump any blood
Module 18.13: Review

A. Define *electrocardiogram*.

B. List the important features of the ECG, and indicate what each represents.

C. Why is ventricular fibrillation fatal?

*Learning Outcome*: Identify the electrical events shown on an electrocardiogram.
Module 18.14: The intrinsic heart rate can be altered by autonomic activity

Pacemaker potential

- Pacemaker cells in SA/AV nodes cannot maintain a stable resting membrane potential; membrane drifts toward threshold

  - **Pacemaker potential** = the gradual spontaneous depolarization
Module 18.14: The intrinsic heart rate can be altered by autonomic activity

Pacemaker potential (continued)

- Pacemaker potential in SA node cells occurs 80–100 times/min
  - Establishes heart rate
  - SA node brings AV nodal cells to threshold before they reach it on their own, thus SA node paces the heart

![Diagram of membrane potential and threshold with heart rate and time](image)
Parasympathetic influence

- Parasympathetic stimulation decreases heart rate
- ACh from parasympathetic neurons:
  - Opens K⁺ channels in plasma membrane
  - Hyperpolarizes membrane
  - Slows rate of spontaneous depolarization
  - Lengthens repolarization
Module 18.14: Autonomic effects on heart rate

Sympathetic influence

- Sympathetic stimulation increases heart rate
- Binding of norepinephrine to beta-1 receptors opens ion channels
  - Increases rate of depolarization
  - Decreases repolarization
Module 18.14: Autonomic effects on heart rate

Resting heart rate

- Varies with age, general health, physical conditioning
- Normal range is 60–100 bpm
- **Bradycardia**
  - Heart rate slower than normal (<60 bpm)
- **Tachycardia**
  - Heart rate faster than normal (>100 bpm)
Module 18.14: Autonomic effects on heart rate

Cardiac centers of the medulla oblongata

- **Cardioinhibitory center**
  - Controls parasympathetic neurons; slows heart rate
  - Parasympathetic supply to heart via *vagus nerve* (X); synapse in cardiac plexus
  - Postganglionic fibers to SA/AV nodes, atrial musculature

- **Cardioacceleratory center**
  - Controls sympathetic neurons; increases heart rate
  - Sympathetic innervation to heart via postganglionic fibers in cardiac nerves; innervate nodes, conducting system, atrial and ventricular myocardium
The cardiac centers and innervation of the heart

- Cardioinhibitory center
- Cardioacceleratory center
- Vagal nucleus
- Medulla oblongata
- Vagus nerve (X)
- Spinal cord

**Sympathetic**
- Sympathetic preganglionic fiber
- Sympathetic ganglia (cervical ganglia and superior thoracic ganglia [T1–T4])
- Sympathetic postganglionic fiber
- Cardiac nerve

**Parasympathetic**
- Parasympathetic preganglionic fiber
- Synapses in cardiac plexus
- Parasympathetic postganglionic fibers
Module 18.14: Review

A. Compare bradycardia with tachycardia.
B. Describe the sites and actions of the cardioinhibitory and cardioacceleratory centers.
C. Caffeine has effects on conducting cells and contractile cells that are similar to those of NE. What effect would drinking large amounts of caffeinated beverages have on the heart rate?

Learning Outcome: Describe the factors affecting the heart rate.
Module 18.15: Stroke volume depends on the relationship between end-diastolic volume and end-systolic volume

Stroke volume analogy

- **Stroke volume** can be compared to pumping water with a manual pump
  - Amount pumped varies with pump handle movement
- Heart has two pumps (ventricles) that pump the same volume
  - Can use single pump as a model
Module 18.15: Stroke volume analogy

- As pump handle raises, pressure in cylinder decreases; water enters through one-way valve. Corresponds to passive filling during ventricular diastole.
Module 18.15: Stroke volume analogy

At the start of pumping (cardiac) cycle:

- Water in pump cylinder = blood in ventricle at end of ventricular diastole or the **end-diastolic volume** (EDV)
Module 18.15: Stroke volume analogy

As the pump handle comes down, water is forced out.

- Corresponds to ventricular ejection
Handle fully depressed, water remaining in cylinder = blood in ventricles at end of systole, or end-systolic volume (ESV)

- Amount of water pumped out = stroke volume
- Stroke volume = EDV – ESV
Factors affecting stroke volume

- **End-diastolic volume (EDV)**
  - **Venous return** = amount of venous blood returned to the right atrium
    - Varies directly with blood volume, muscular activity, and rate of blood flow
  - **Filling time** = length of ventricular diastole; the longer it is, the more filling occurs (higher EDV)
Module 18.15: Factors affecting stroke volume

Factors affecting stroke volume (continued)

- **Preload** = amount of myocardial stretch
  - Greater EDV causes greater preload; more stretching causes stronger contractions and more blood being ejected (*Frank-Starling law of the heart*)
Factors affecting stroke volume

- Influences on ESV
  - **Contractility** = amount of force produced during contraction at a given preload
    - Increased by sympathetic stimulation, some hormones (epinephrine, norepinephrine, thyroid hormone, glucagon)
    - Reduced by “beta blockers” and calcium channel blockers
Factors affecting stroke volume (continued)

- **Afterload** = ventricular tension required to open semilunar valves and empty
  - As afterload increases, stroke volume decreases
  - Afterload increases whenever blood flow is restricted, such as with vasoconstriction
Module 18.15: Review

A. Define *end-diastolic volume* (*EDV*) and *end-systolic volume* (*ESV*).

B. What effect would an increase in venous return have on the stroke volume?

C. What effect would an increase in sympathetic stimulation of the heart have on the end-systolic volume (*ESV*)?

*Learning Outcome*: Describe the variables that influence stroke volume.
Module 18.16: Cardiac output is regulated by adjustments in heart rate and stroke volume

Factors affecting cardiac output

- Cardiac output varies widely to meet metabolic demands
- Cardiac output can be changed by affecting either heart rate or stroke volume

Heart failure = condition in which the heart cannot meet the demands of peripheral tissues
Module 18.16: Cardiac output adjustment

Factors affecting heart rate (HR)

- Atrial Reflex
  - The atrial reflex involves adjustments in heart rate in response to an increase in the venous return. When the walls of the right atrium are stretched, stretch receptors there stimulate sympathetic activity.

- Body Temperature or Exercise
  - Heart rate rises with increased body temperature and lowers with drop in body temperature.

- Autonomic Nervous System
  - Sympathetic stimulation increases HR, and parasympathetic stimulation decreases HR.

- Hormones
  - Many hormones increase heart rate (catecholamines, epinephrine, and thyroxine).

Factors affecting stroke volume (SV)

- Venous Return
  - In general, when venous return increases, SV increases. When venous return decreases, SV decreases.

- Preload
  - The amount of preload affects the EDV by stretching the myocardium.

- Filling Time
  - Increase in filling time increases the EDV.

- Contractility
  - The greater the contractility, the smaller the ESV.

- Factors affecting contractility:
  - Sympathetic stimulation increases both heart rate and contractility. Parasympathetic action slows the heart rate but has little influence on contractility.

- End-Diastolic Volume (EDV)

- End-Systolic Volume (ESV)

- Afterload
  - The greater the afterload, the lower the pumping efficiency of the heart, and the larger the ESV.

Heart rate (HR) and stroke volume (SV)

Cardiac output (CO) = HR × SV

Stoke volume (SV) = EDV - ESV

Changes in filling time increase the EDV.

Increases both heart rate and contractility.

Sympathetic stimulation increases both heart rate and contractility. Parasympathetic action slows the heart rate but has little influence on contractility.

Many hormones increase contractility in addition to their effects at other target organs.

Changes in peripheral blood flow patterns can increase or decrease venous return.

Large reductions in blood volume due to bleeding or dehydration reduce venous return.

Muscular contractions compress veins and assist valves in directing venous blood toward the right atrium.

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Module 18.15: Review

A. Define *heart failure*.

B. Compute Joe’s stroke volume if his end-systolic volume (ESV) is 40 mL and his end-diastolic volume (EDV) is 125 mL.

*Learning Outcome*: Explain how stroke volume and cardiac output are coordinated.