Al-Farabi Kazakh National University
Higher School of Medicine

# Circulatory System

## -Heart

Cardiac and Smooth Muscle

# ◆ PART I

# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- *Describe the structural and physiological differences between cardiac muscle and skeletal muscle;*
- *explain why these differences are important to cardiac function;*
- *describe the structural and physiological differences between smooth muscle and skeletal muscle;*
- *relate the unique properties of smooth muscle to its locations and functions*

- Any of the three types of muscle cells can be called ***myocytes.***
  - preferable for smooth and cardiac muscle - they do not have the long fibrous shape of skeletal muscle cells.
  - relatively short, and in further contrast
  - have only one or two nuclei.
- Cardiac muscle cells are also called ***cardiomyocytes.***
- *involuntary muscle tissues,*
- cardiac muscle and some smooth muscle receive nerves from the sympathetic and parasympathetic divisions of the autonomic nervous system

# Cardiac Muscle

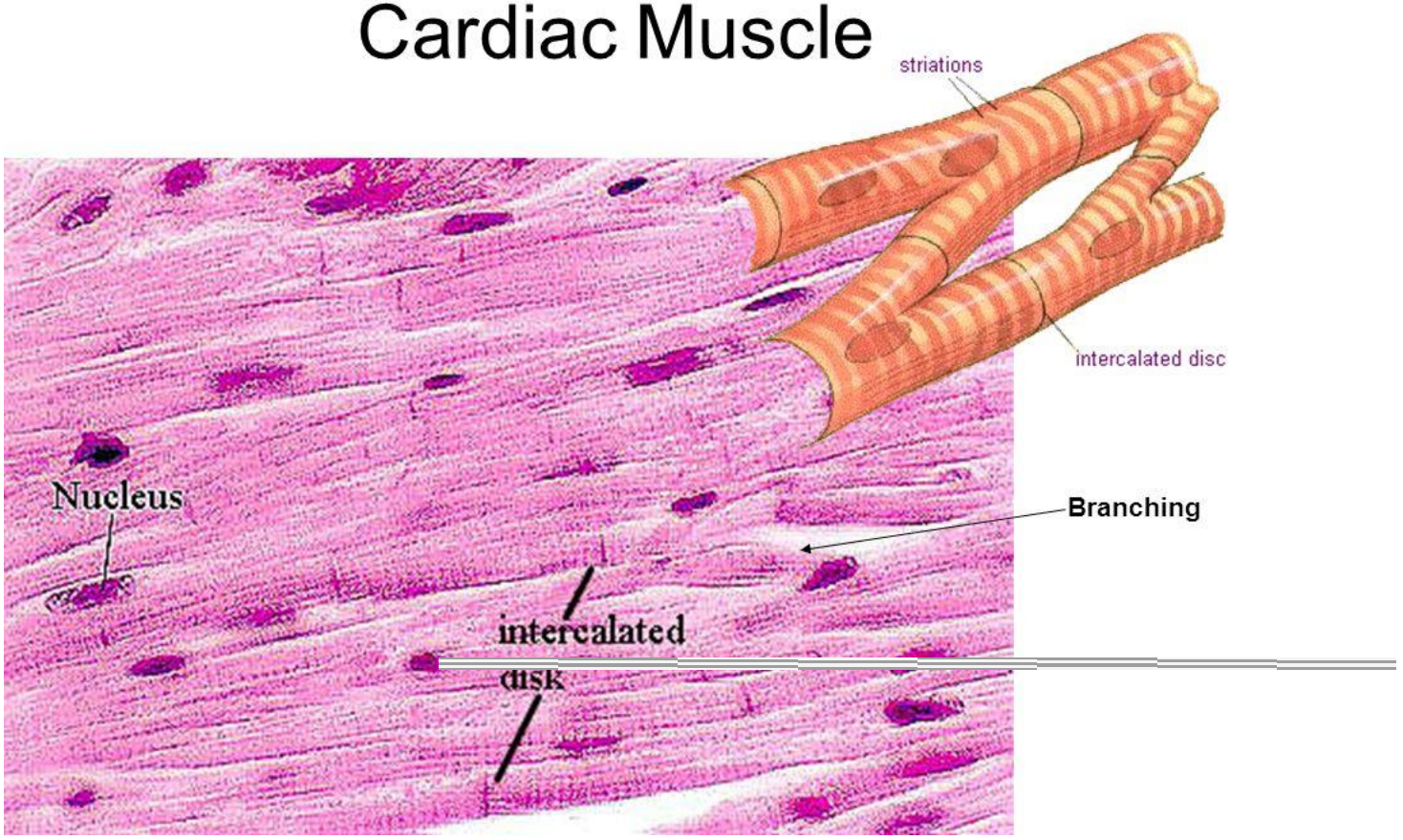
*Cardiac muscle is limited to the heart, where its function is to pump blood.*

**the properties that it must have:**

1. It must contract with a regular rhythm;
2. it must function in sleep and wakefulness, without fail or need of conscious attention;
3. it must be highly resistant to fatigue;
4. the cardiomyocytes of a given heart chamber must contract in unison so that the chamber can effectively expel blood;
5. each contraction must last long enough to expel blood from the chamber.

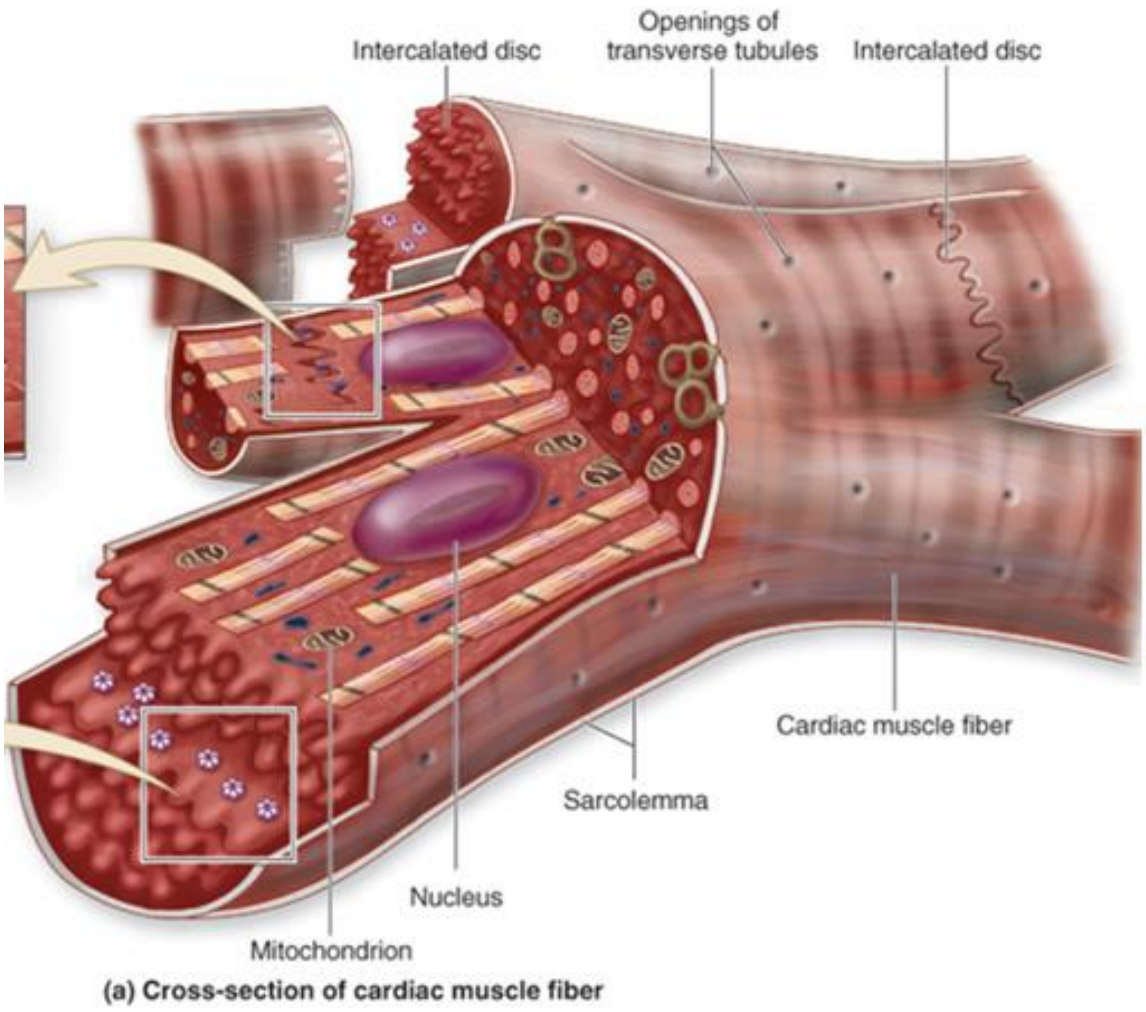
# Cardiac Muscle

- striatede, but cardiomyocytes are shorter and thicker.
- one or two nuclei near the middle of the cell.
- Each cell is enclosed in an ***endomysium***, but there is ***no perimysium or epimysium*** as in skeletal muscle.



# Cardiac Muscle

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition




(a) Cross-section of cardiac muscle fiber

❖ branch slightly so each is joined end to end with several others - intercellular connections, called

**intercalated discs**

• thick dark lines in stained tissue sections.

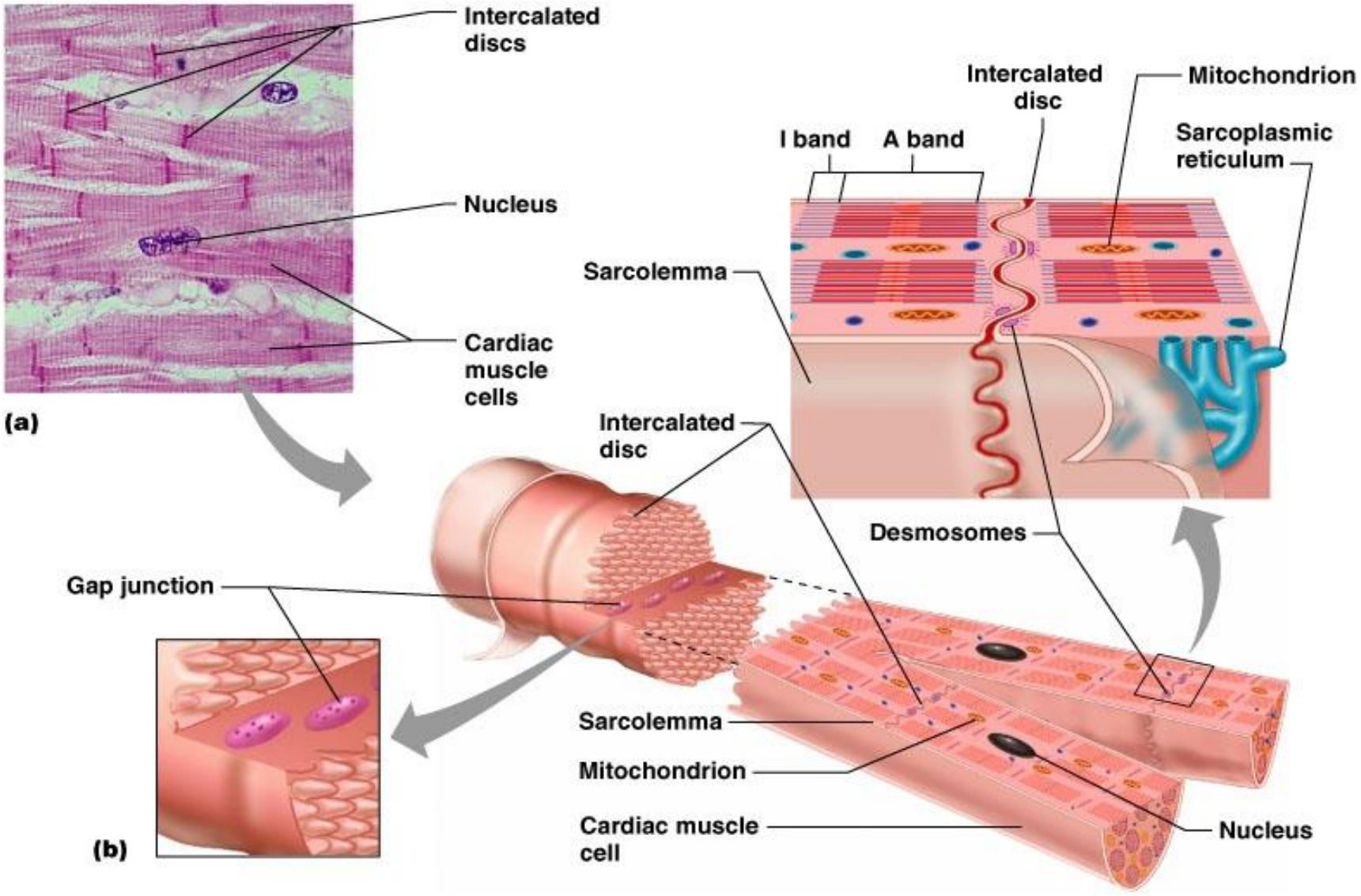
• electrical gap junctions - allow each cardiomyocyte to directly stimulate its neighbors, and mechanical junctions that keep the cardiomyocytes from pulling apart when the heart contracts.

# Cardiac Muscle

- The sarcoplasmic reticulum is less developed than in skeletal muscle
  - T tubules are larger and admit Ca2+ from the extracellular fluid.
- Damaged cardiac muscle is *repaired by fibrosis*.

- *No satellite cells, and even though mitosis has been detected in cardiomyocytes following heart attacks, it does not produce a significant amount of regenerated functional muscle.*

# Cardiac Muscle



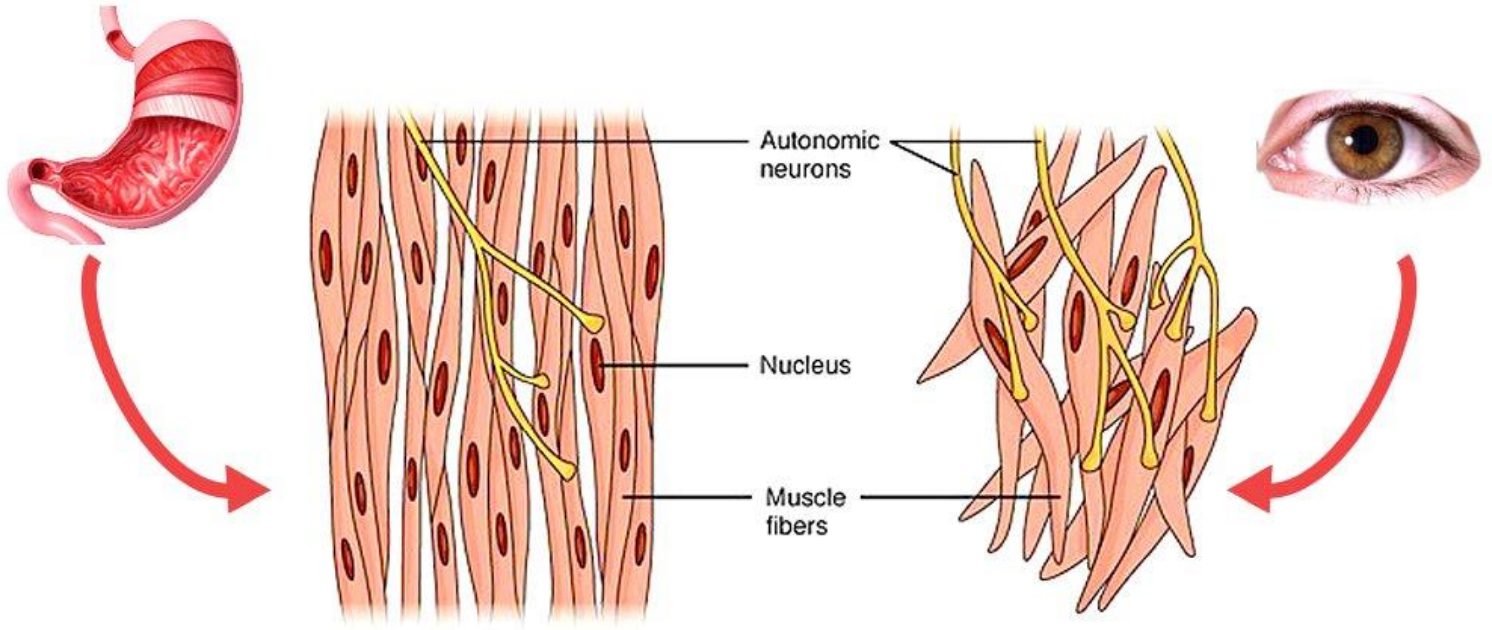
# Cardiac Muscle

- ***Cardiac muscle can contract without the need of nervous stimulation***.
- The heart has a built-in pacemaker that rhythmically sets off a wave of electrical excitation, which travels through the muscle and triggers the contraction of the heart chambers.
- **autorhythmic** - rhythmically and independently.
  - autonomic nervous system - increase or decrease the heart rate and contraction strength.
- does ***not exhibit quick twitches*** like skeletal muscle.
  - Rather, it maintains tension for about 200 to 250 ms, giving the heart time to expel blood.
- **aerobic respiration** almost exclusively.
  - very rich in myoglobin and glycogen
  - it has especially large mitochondria -25% of the cell (2% in a skeletal muscle fiber).
  - very adaptable with respect to the fuel used
  - but very vulnerable to interruptions in oxygen supply.
  - ***Because it makes little use of anaerobic fermentation, cardiac muscle is highly resistant to fatigue.***

# Smooth Muscle

- Smooth muscle -has ***no striations***

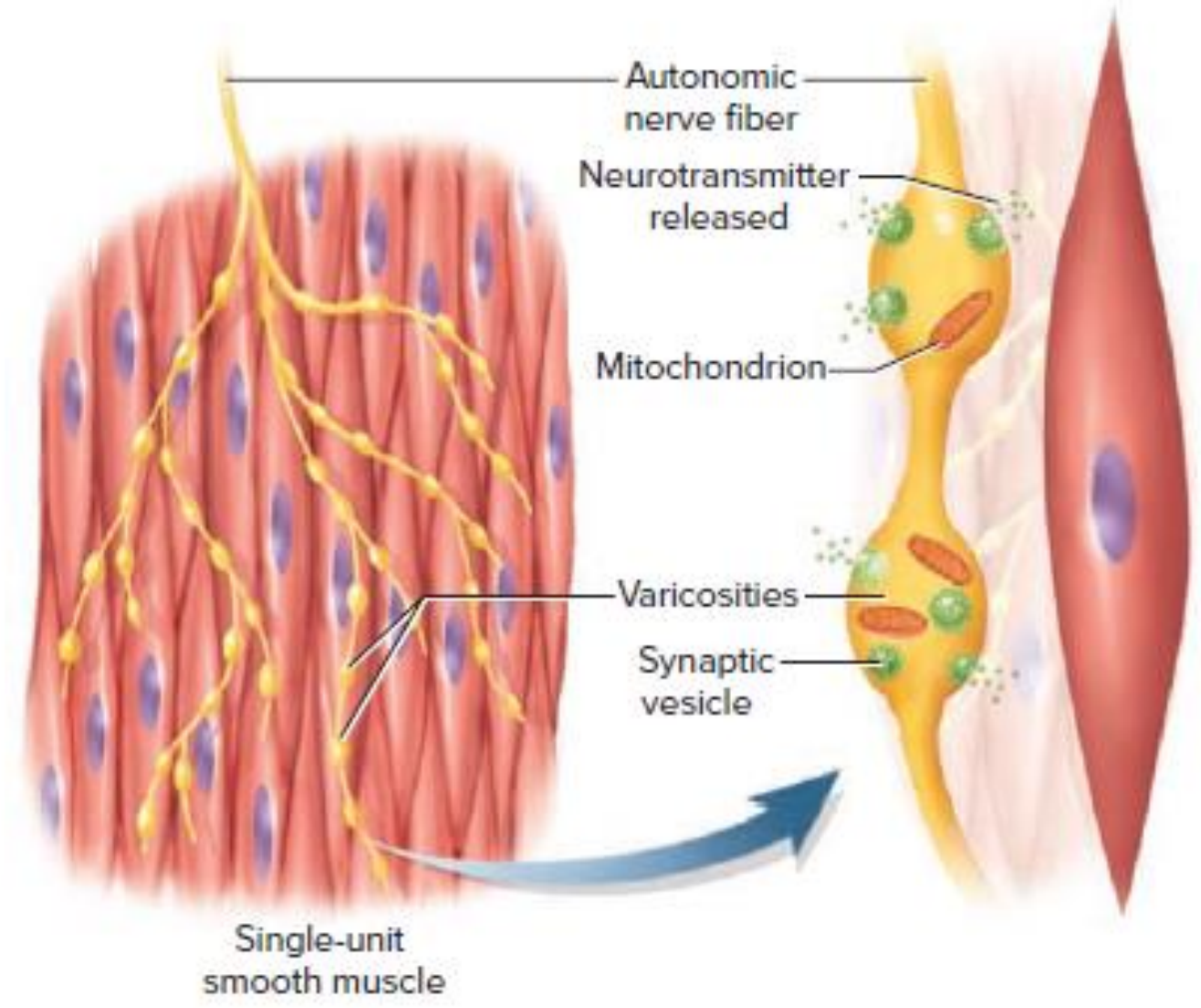
- myocytes are ***relatively small***
  - allowing for fine control of such tissues and organs as a single hair, the iris of the eye, and the tiniest arteries;
  - in the pregnant uterus myocytes become quite large - contribute to the powerful contractions of childbirth.



# Smooth Muscle

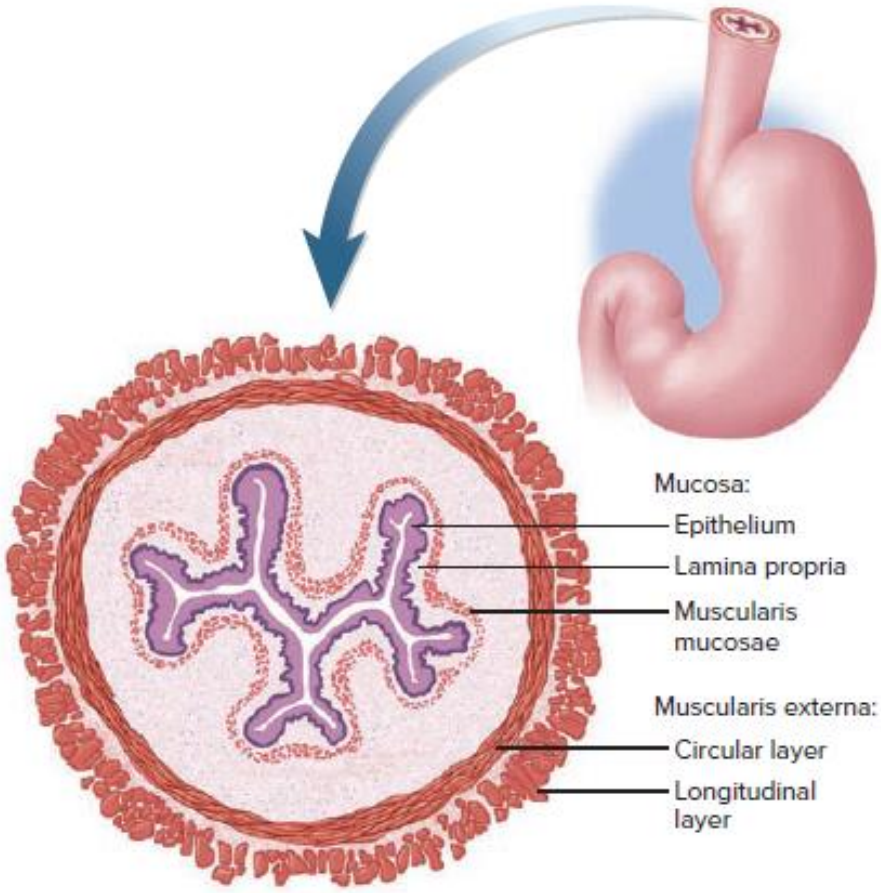
- not innervated or the nerve supply is autonomic
  - Autonomic nerve fibers usually do not form precisely localized neuromuscular junctions with the myocytes.
- nerve fiber has as many as 20,000 periodic swellings called varicosities along its length
  - Each varicosity contains synaptic vesicles that releases neurotransmitters
    - norepinephrine - the sympathetic fibers
    - acetylcholine - the parasympathetic fibers.
  - no motor end plate, but instead has receptors for these neurotransmitters distributed over its surface.
- The varicosities simply release a flood of neurotransmitter into the tissue, and each myocyte may respond to more than one nerve fiber.
  - innervated or not, smooth muscle responds to a wide variety of stimuli and often without any electrical excitation of the sarcolemma.
- It is much slower than skeletal and cardiac muscle to contract and relax
  - but it can remain contracted for a long time ***without fatigue and with minimal energy expenditure.***

# Smooth Muscle



# Smooth Muscle

***Smooth muscle doesn't usually form organs in itself, but forms layers in the walls of larger organs such as the stomach, intestines, uterus, and urinary bladder.***

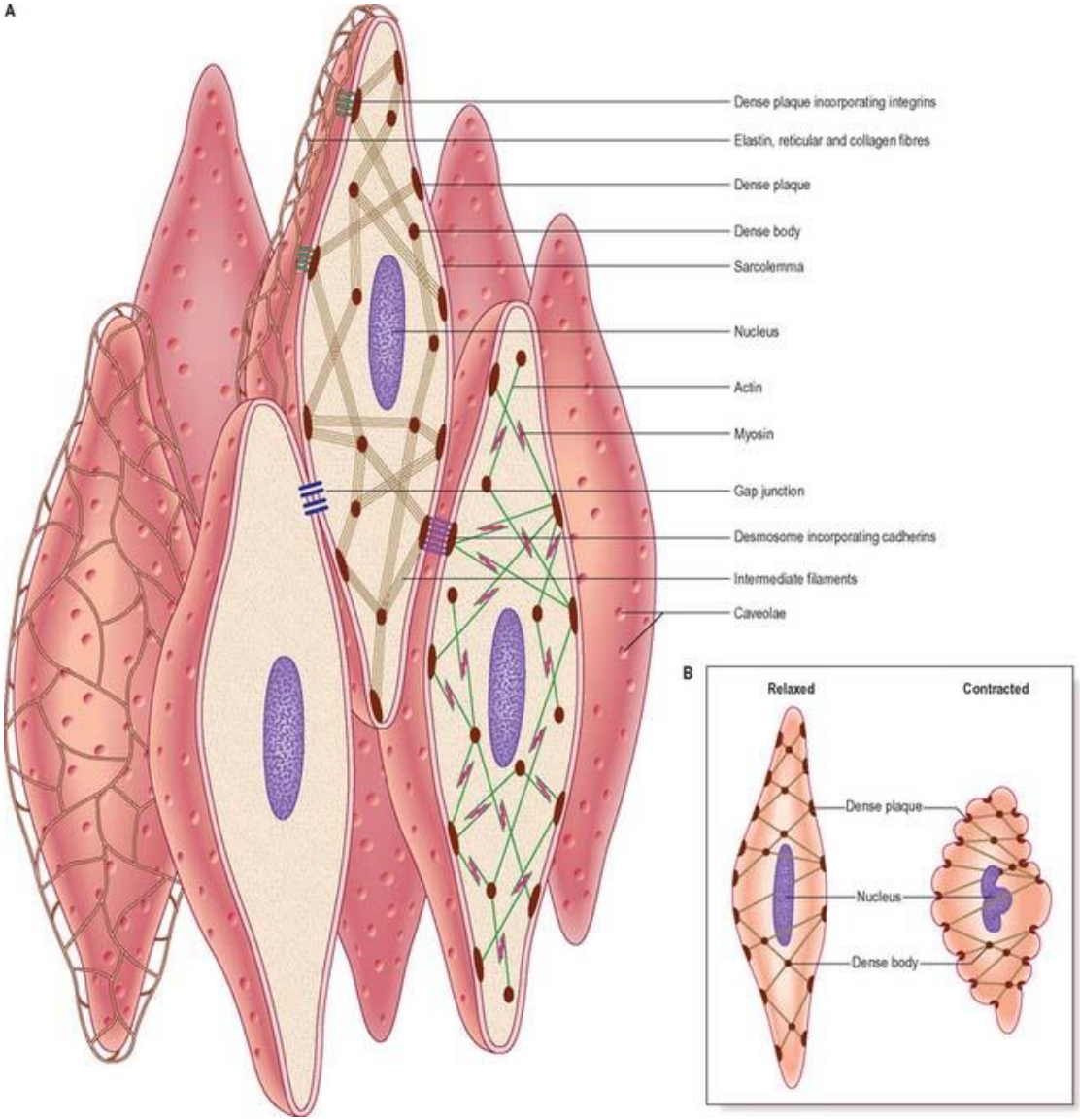


# Smooth Muscle

- can propel the contents of an organ
  - driving food through the digestive tract
  - voiding urine and feces
  - expelling the infant in childbirth.
- By dilating or constricting the blood vessels and airway
  - modify the speed of air and blood flow
  - maintain blood pressure
  - reroute blood from one pathway to another.
- ***capable to hypertrophy (cellular growth), mitosis and hyperplasia (cell division)***
  - pregnant uterus grows by the addition of new myocytes as well as enlargement of existing ones.
  - Injured smooth muscle regenerates well by mitosis.

# Myocyte Structure

- fusiform shape - 5 to 10 μm wide at the middle,
  - ranging from 30 to 200 μm long
  - up to 500 μm long in the pregnant uterus.
- enclosed in endomysium but have no perimysium, fascicles, or epimysium.
- one nucleus, located near the middle of the cell.
- sarcoplasmic reticulum is scanty and there are no T tubules.
- Thick and thin filaments are present



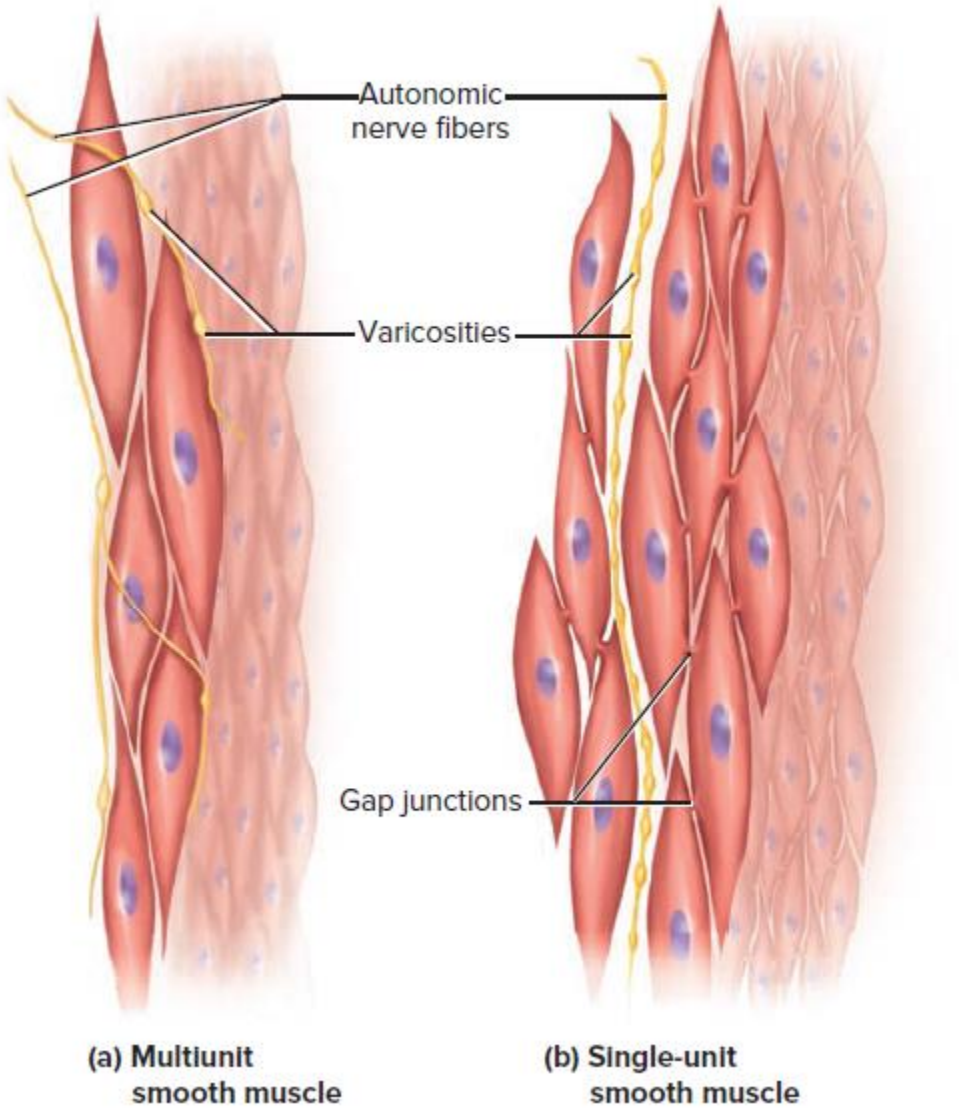
# Myocyte Structure

- no striations, sarcomeres, or myofibrils
  - the myofilaments are not bundled and aligned with each other the way they are in striated muscle.
- Z discs are absent.
  - In their place are protein plaques - **dense bodies**
    - inner face of the plasma membrane or dispersed throughout the sarcoplasm
- dense bodies of one cell are often directly across from those of another - contractile force can be transmitted from cell to cell.
- Associated with the dense bodies is an extensive **cytoskeletal network** of intermediate filaments.
- Actin filaments attach to the intermediate filaments as well as directly to the dense bodies
  - their movement (powered by myosin) is transferred to the sarcolemma and shortens the cell.

# Types of Smooth Muscle

1. **multiunit**
   - in the largest arteries and pulmonary air passages, piloerector muscles, muscles that control the iris and lens.
   - Innervation – autonomic
   - the terminal branches of a nerve fiber synapse with individual myocytes and form a motor unit.
   - 1 varicosity = 1 myocyte= responds independently of all the others—hence the name multiunit.
   - does not, however, generate action potentials.
2. **single-unit (visceral muscle)**
   - most blood vessels and in the digestive, respiratory, urinary, and reproductive tracts
   - myocytes are electrically coupled to each other by gap junctions
   - numerous cells contract as a unit, almost as if they were a single cell
   - nerve varicosities stimulate several of myocytes at once when they release neurotransmitter.

# Types of Smooth Muscle



# Excitation of Smooth Muscle

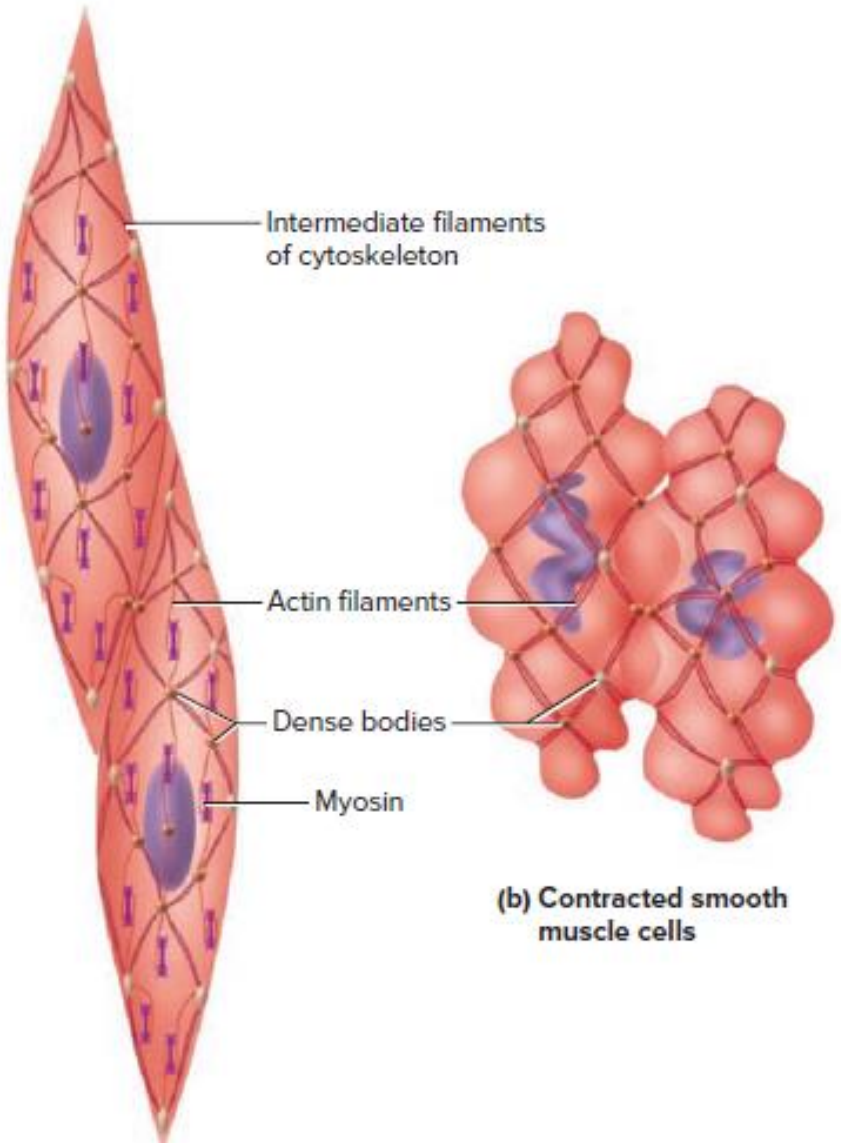
- **Autonomic nerve fibers and neurotransmitters**
- **Chemicals.**
- **Temperature.**
- **Stretch.**
- **Autorhythmicity.**

- Ca2+
  - comes from the sarcoplasmic reticulum (SR),
  - extracellular fluid
- the sarcolemma has numerous little pockets called **caveolae** where its calcium channels are concentrated.
  - Calcium is 10,000 times as concentrated in the ECF as in the cytosol,
  - if these channels are opened, it diffuses quickly into the cell.
- Because smooth muscle cells are relatively small, the incoming Ca2+ quickly reaches all of the myofilaments.

# Contraction and Relaxation

1. Calcium binds with calmodulin
2. Calmodulin - activates an enzyme called myosin light-chain kinase, which adds a phosphate group to a small regulatory protein on the myosin head.
3. This activates the myosin ATPase, enabling it to bind to actin and hydrolyze ATP.
4. The myosin then produces repetitive power and recovery strokes like those of skeletal muscle.
5. As thick filaments pull on the thin ones, the thin filaments pull on the dense bodies and membrane plaques.
6. Through the dense bodies and cytoskeleton, force is transferred to the plasma membrane and the entire cell shortens.
7. When a smooth muscle cell contracts, it puckers and twists somewhat like wringing out a wet towel

# Contraction and Relaxation



# Contraction and Relaxation

- The latent period = 50 to 100 ms long.
- Tension peaks about 500 ms (0.5 second) after the stimulus and then declines over a period of 1 to 2 seconds.
  - smooth muscle is very slow to contract and relax.
    - myosin ATPase is a slow enzyme.
    - slow to relax - the pumps that remove Ca2+ from the cytosol are also slow.
  - As the Ca2+ level falls, myosin releases its phosphate group and is no longer able to hydrolyze ATP and execute power strokes.
  - It has a latch-bridge mechanism that enables it to remain attached to actin for a prolonged time without consuming more ATP.
- ***Smooth muscle often exhibits tetanus and is very resistant to fatigue***

# Response to Stretch

- Distension of the esophagus with food or the colon with feces, for example, evokes a wave of contraction called **peristalsis** that propels the contents along the organ.
- **Stress– relaxation (or receptive-relaxation) response.**

*Smooth muscle can contract powerfully from half to twice its resting length:*

1. There are no Z discs, so thick filaments cannot butt against them and stop the contraction;
2. since the thick and thin filaments are not arranged in orderly sarcomeres, stretching of the muscle does not cause a situation in which there is too little overlap for cross-bridges to form;
3. the thick filaments of smooth muscle have myosin heads along their entire length (there is no bare zone), so cross-bridges can form anywhere, not just at the ends.

**TABLE 11.4** Comparison of Skeletal, Cardiac, and Smooth Muscle

| Feature | Skeletal Muscle | Cardiac Muscle | Smooth Muscle |
|---|---|---|---|
| Location | Associated with skeletal system | Heart | Walls of viscera and blood vessels, iris of eye, piloerector of hair follicles |
| Cell shape | Long threadlike fibers | Short, slightly branched cells | Short fusiform cells |
| Cell length | 100 µm–30 cm | 50–120 µm | 30–200 µm |
| Cell width | 10–500 µm | 10–20 µm | 5–10 µm |
| Striations | Present | Present | Absent |
| Nuclei | Multiple nuclei, adjacent to sarcolemma | Usually one nucleus, near middle of cell | One nucleus, near middle of cell |
| Connective tissues | Endomysium, perimysium, epimysium | Endomysium only | Endomysium only |
| Sarcoplasmic reticulum | Abundant | Present | Scanty |
| T tubules | Present, narrow | Present, wide | Absent |
| Gap junctions | Absent | Present in intercalated discs | Present in single-unit smooth muscle |
| Autorhythmicity | Absent | Present | Present in single-unit smooth muscle |
| Thin filament attachment | Z discs | Z discs | Dense bodies |
| Regulatory proteins | Tropomyosin, troponin | Tropomyosin, troponin | Calmodulin, myosin light-chain kinase |
| $Ca^{2+}$ source | Sarcoplasmic reticulum | Sarcoplasmic reticulum and extracellular fluid | Mainly extracellular fluid |
| $Ca^{2+}$ receptor | Troponin of thin filament | Troponin of thin filament | Calmodulin of thick filament |
| Innervation and control | Somatic motor fibers (voluntary) | Autonomic fibers (involuntary) | Autonomic fibers (involuntary) |
| Nervous stimulation required? | Yes | No | No |
| Effect of nervous stimulation | Excitatory only | Excitatory or inhibitory | Excitatory or inhibitory |
| Mode of tissue repair | Limited regeneration, mostly fibrosis | Limited regeneration, mostly fibrosis | Relatively good capacity for regeneration |

# Circulatory System

# -Heart

Al-Farabi Kazakh National University
Higher School of Medicine

**Overview of the Cardiovascular System.**

**Gross Anatomy of the heart**

# ◆ PART II

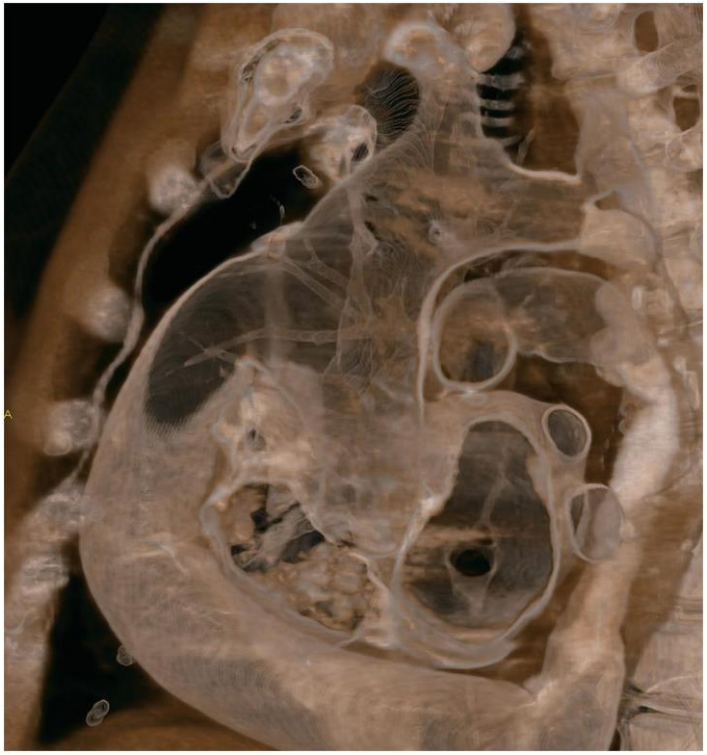
# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- ***Define and distinguish between the pulmonary circuit systemic circuit;***
- ***describe the general location, size, and shape of the heart;***
- ***describe the pericardial sac that encloses the heart.***
- ***describe the three layers of the heart wall;***
- ***identify the four chambers of the heart;***
- ***identify the surface features of the sac heart and correlate them with its internal four-chambered anatomy;***
- ***identify the four valves of the heart;***
- ***trace the flow of blood through the four chambers and valves of the heart and adjacent blood vessels;***
- ***describe the arteries that nourish the myocardium and the veins that drain it***

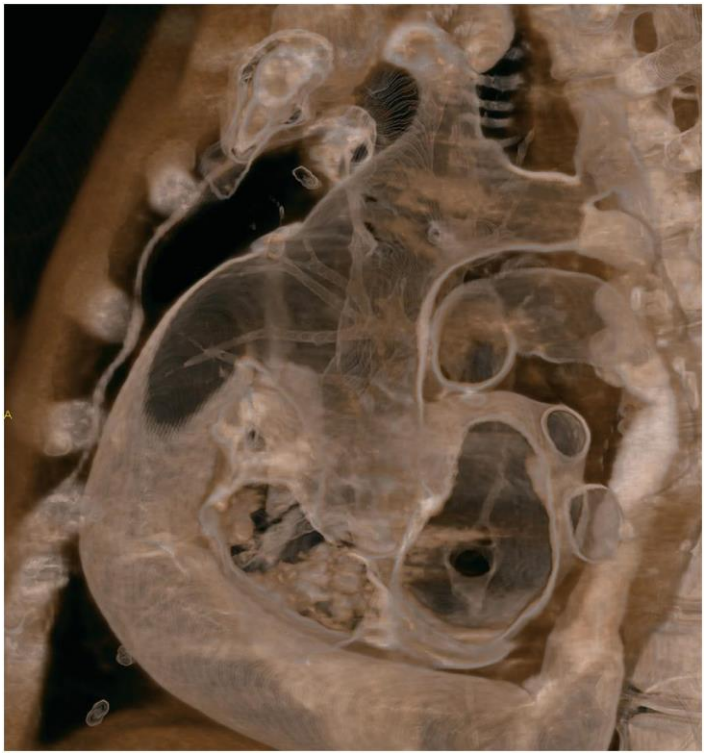
# Circulatory System

- **cardiology** – the scientific study of the heart and the treatment of its disorders
- **cardiovascular system**
  – heart and blood vessels
- **circulatory system**
  – heart, blood vessels, and the blood

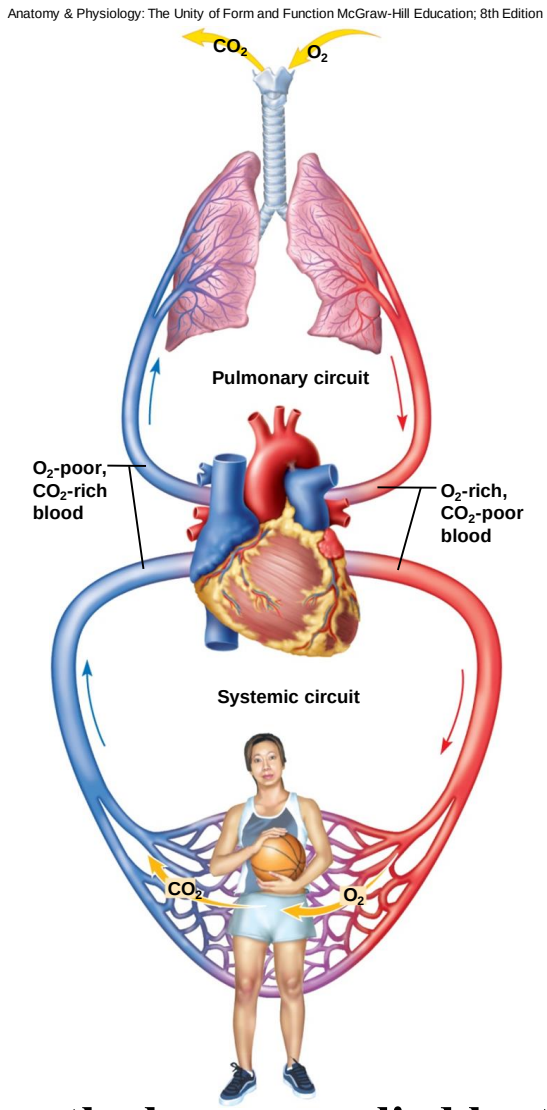


# Circulatory System

- **major divisions of circulatory system**
  - **pulmonary circuit** - right side of heart
    - carries blood to lungs for gas exchange and back to heart
  - **systemic circuit** - left side of heart
    - supplies oxygenated blood to all tissues of the body and returns it to the heart



# Cardiovascular System Circuit




- left side of heart
  - fully oxygenated blood arrives from lungs via pulmonary veins
  - blood sent to all organs of the body via aorta
- right side of heart
  - lesser oxygenated blood arrives from inferior and superior vena cava
  - blood sent to lungs via pulmonary trunk

**Are the lungs supplied by the pulmonary circuit, the systemic circuit, or both? Explain**

# Position, Size, and Shape

- heart located in mediastinum, between lungs
- **base** – wide, superior portion of heart, blood vessels attach here
- **apex** - inferior end, tilts to the left, tapers to point
- 3.5 in.(9cm) wide at base, 5 in.(13cm) from base to apex and 2.5 in(6cm). anterior to posterior; weighs 10 oz.(300g)

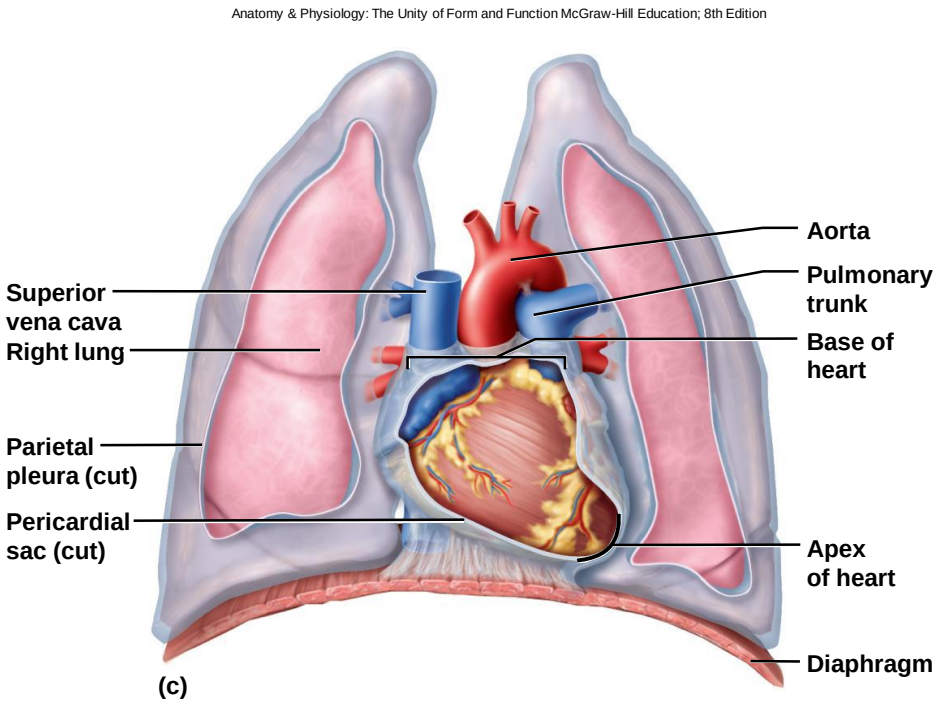
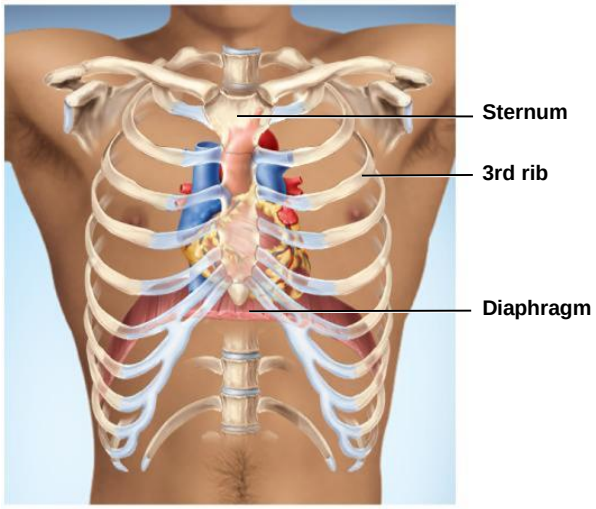



Figure 19.2c

# Heart Position

**Does most of the heart lie to the right or left of the median plane?**

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition



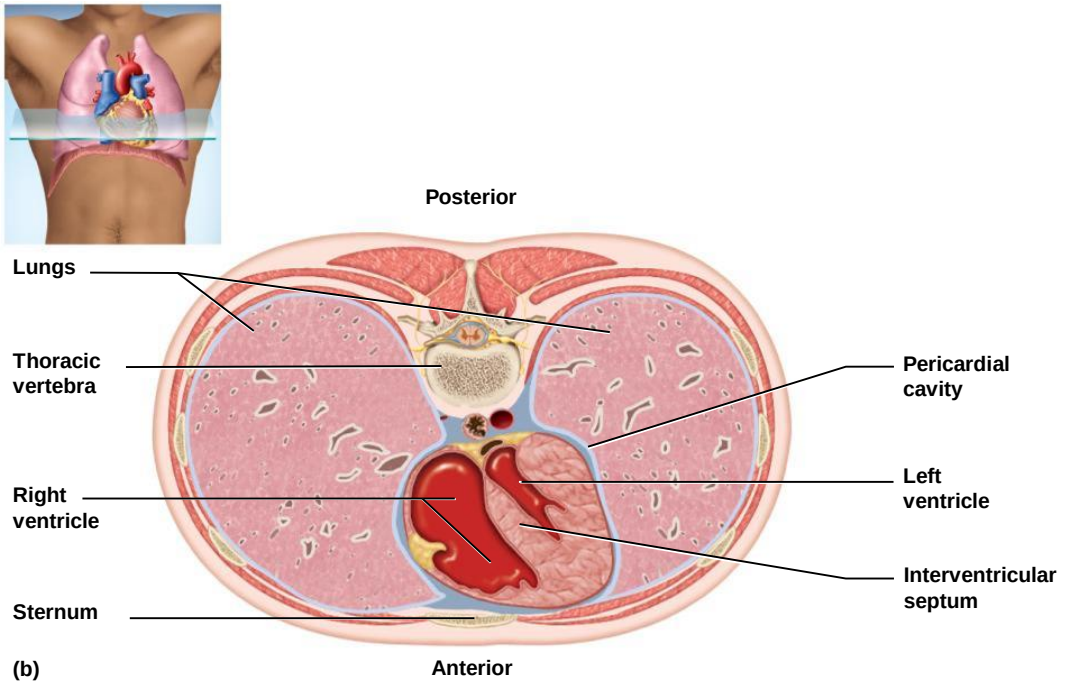




Figure 19.2 a-b

# Pericardium

- **pericardium** - double-walled sac (pericardial sac) that encloses the heart
  - allows heart to beat without friction, provides room to expand, yet resists excessive expansion
  - anchored to diaphragm inferiorly and sternum anteriorly
- **parietal pericardium** – outer wall of sac
  - superficial **fibrous layer** of connective tissue
  - a deep, thin **serous layer**
- **visceral pericardium (epicardium)** – heart covering
  - serous lining of sac turns inward at base of heart to cover the heart surface
- **pericardial cavity** - space inside the pericardial sac filled with 5 - 30 mL of pericardial fluid
- **pericarditis** – inflammation of the membranes
  - painful friction rub with each heartbeat

# Pericardium and Heart Wall

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

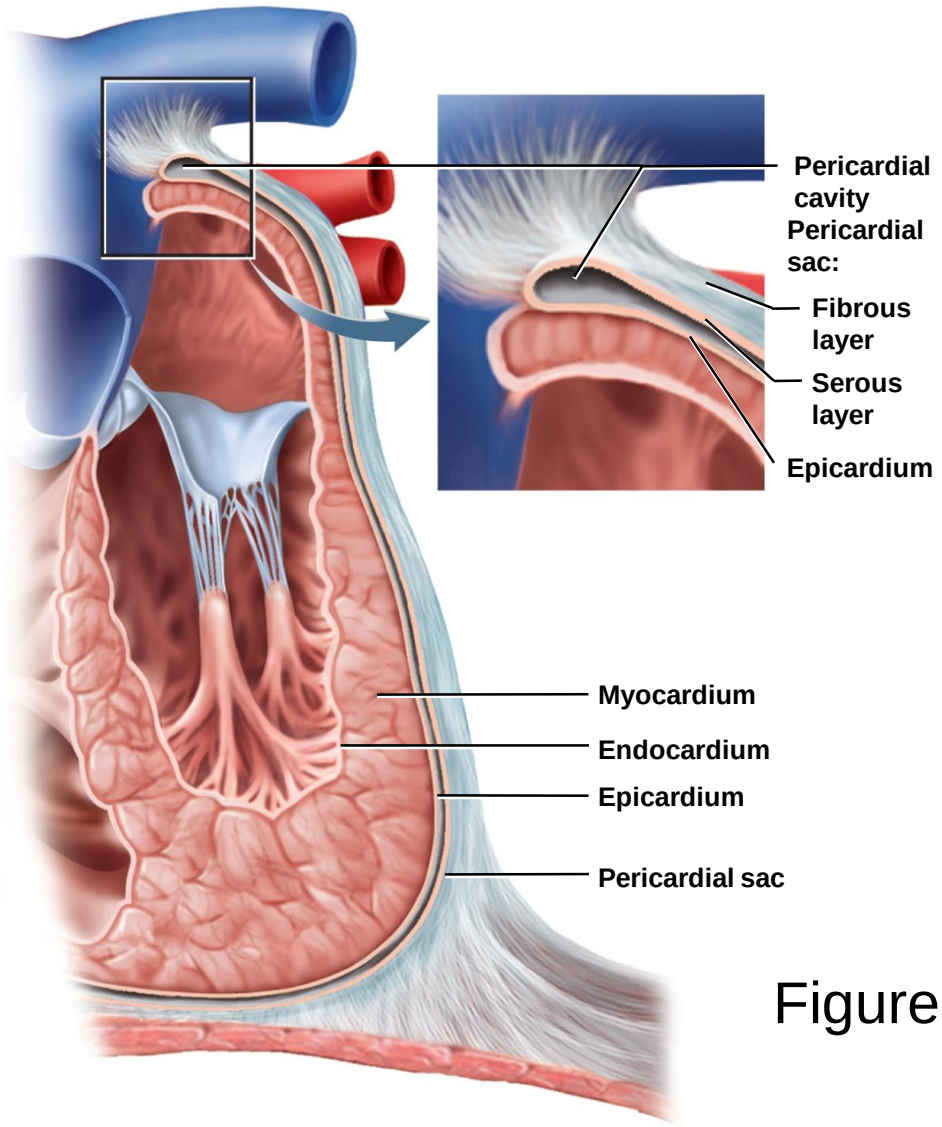



Figure 19.3

# Heart Wall

- **epicardium** (visceral pericardium)
  - serous membrane covering heart
  - adipose in thick layer in some places
  - coronary blood vessels travel through this layer

- **endocardium**
  - smooth inner lining of heart and blood vessels
  - covers the valve surfaces and continuous with endothelium of blood vessels

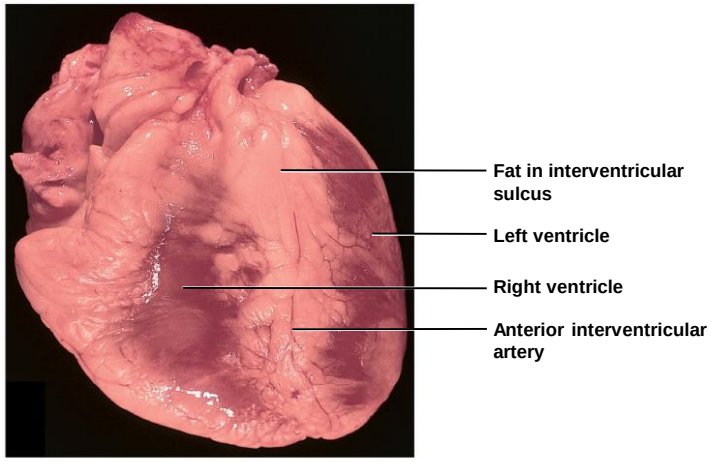
- **myocardium**
  - layer of **cardiac muscle** proportional to work load
    - muscle spirals around heart which produces wringing motion
  - **fibrous skeleton of the heart -** framework of **collagenous and elastic fibers**
    - provides structural support and attachment for cardiac muscle and anchor for valve tissue
    - electrical insulation between atria and ventricles important in timing and coordination of contractile activity

# ▶▶▶APPLY WHAT YOU KNOW

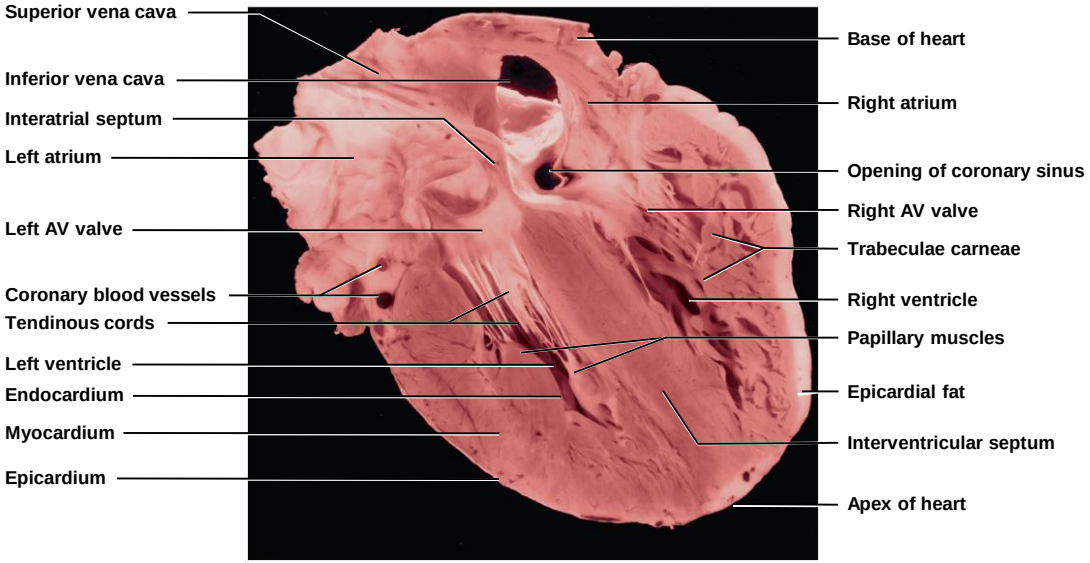
- **Parts of the fibrous skeleton sometimes become calcified in old age. How would you expect this to affect cardiac function**

# Cadaver Heart

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition




(a) Anterior view, external anatomy




(b) Posterior view, internal anatomy

Figure 19.4 a-b

# Heart Chambers

- **four chambers**
  - **right and left atria**
    - two superior chambers
    - receive blood returning to heart
    - auricles (seen on surface) enlarge chamber
  - **right and left ventricles**
    - two inferior chambers
    - pump blood into arteries

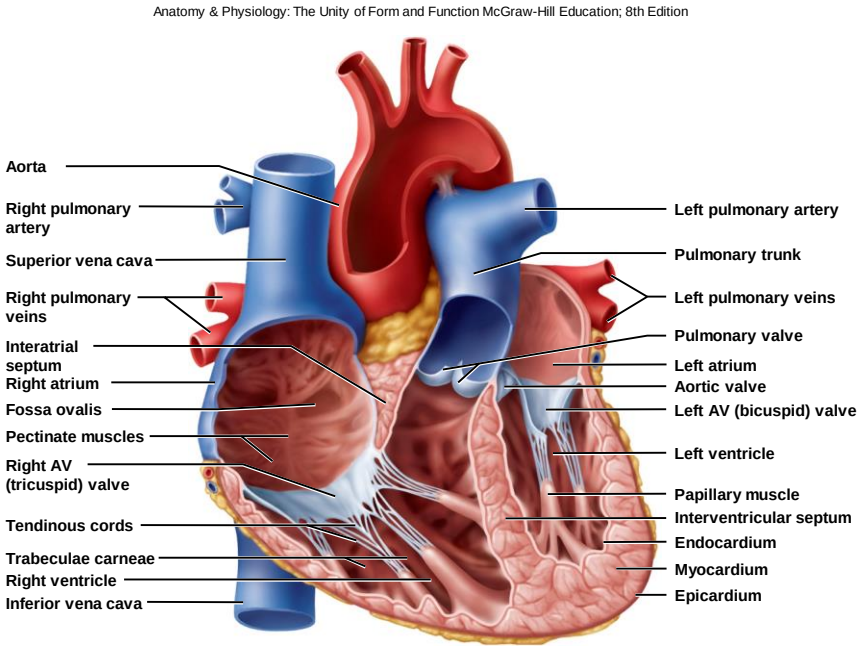



Figure 19.7

# External Anatomy - Anterior

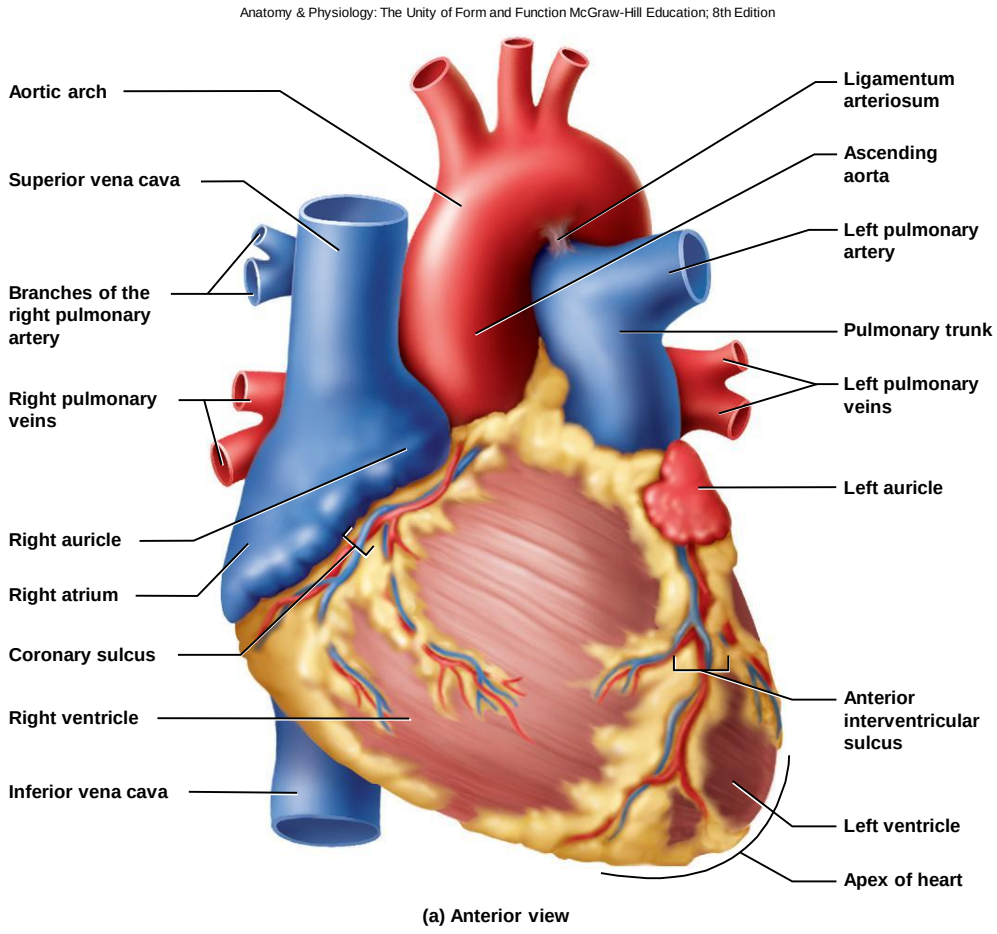
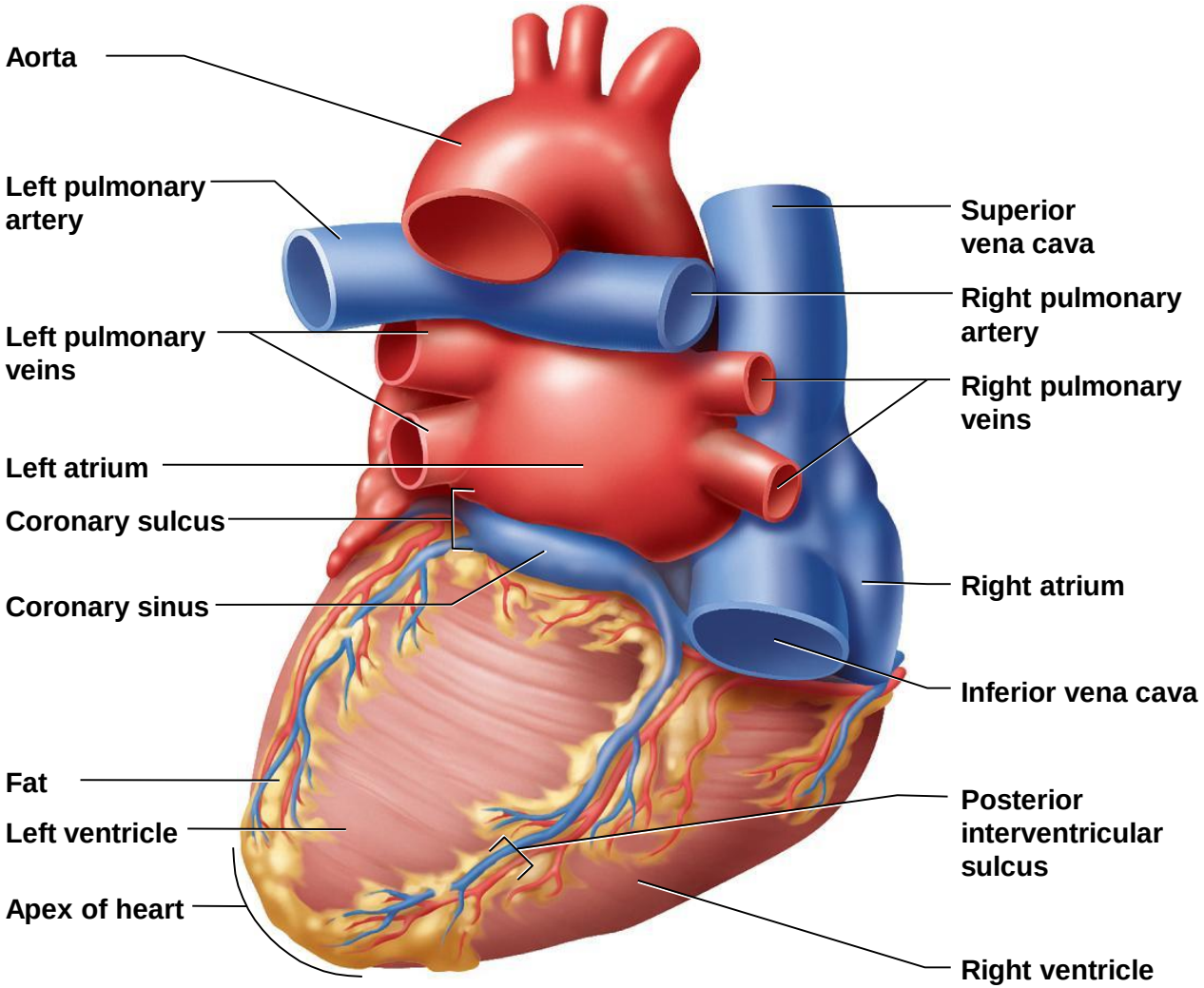



Figure 19.5a

- **atrioventricular sulcus** - separates atria and ventricles
- **interventricular sulcus** - overlies the interventricular septum that divides the right ventricle from the left
- sulci contain coronary arteries

# External Anatomy - Posterior

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition




(b) Posterior view

Figure 19.5b

# Heart Chambers - Internal

- **interatrial septum**
  - wall that separates atria
- **pectinate muscles**
  - internal ridges of myocardium in right atrium and both auricles
- **interventricular septum**
  - muscular wall that separates ventricles
- **trabeculae carneae**
  - internal ridges in both ventricles

# Internal Anatomy - Anterior

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

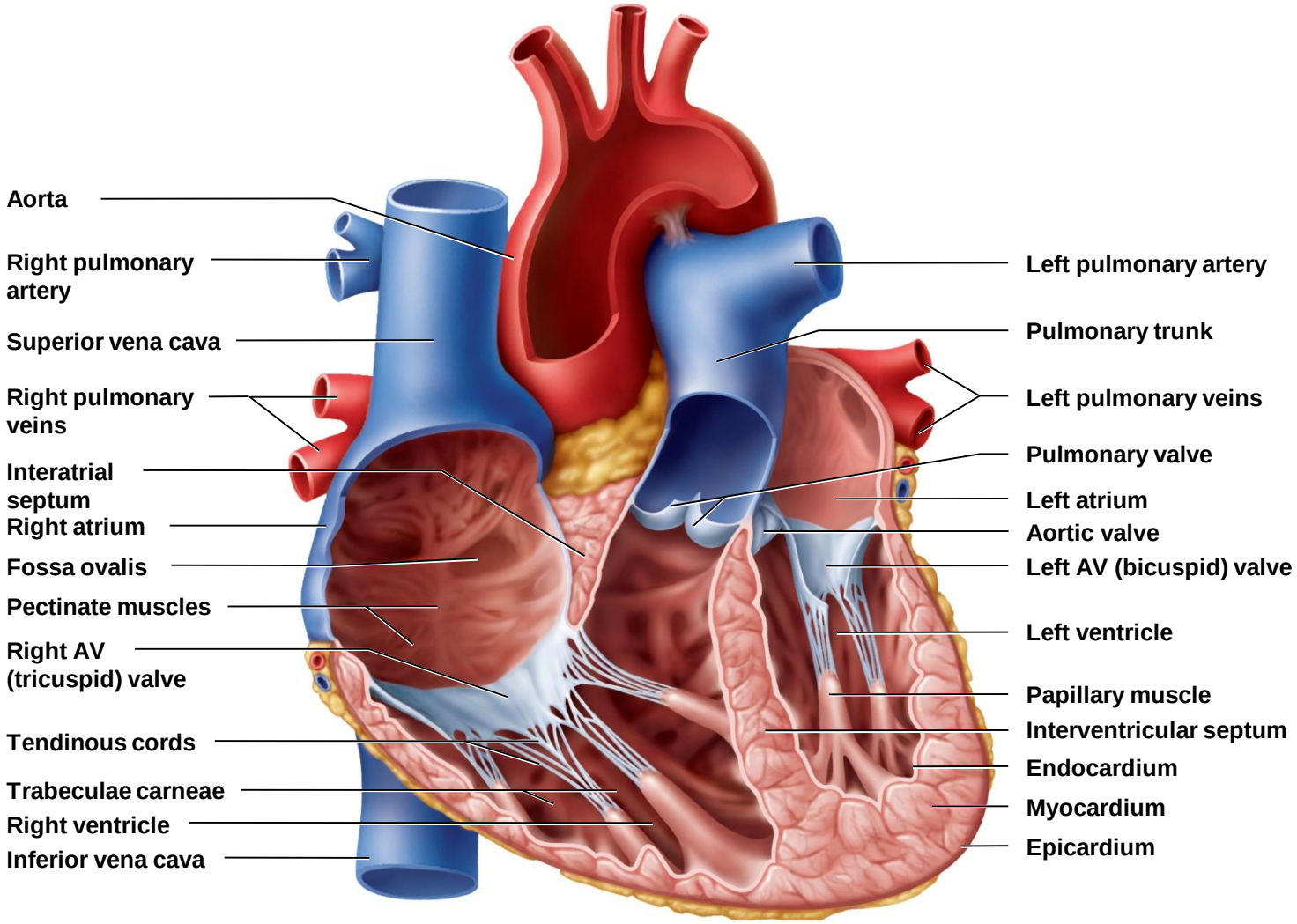



Figure 19.7

# Heart Valves

- valves ensure a one-way flow of blood through the heart

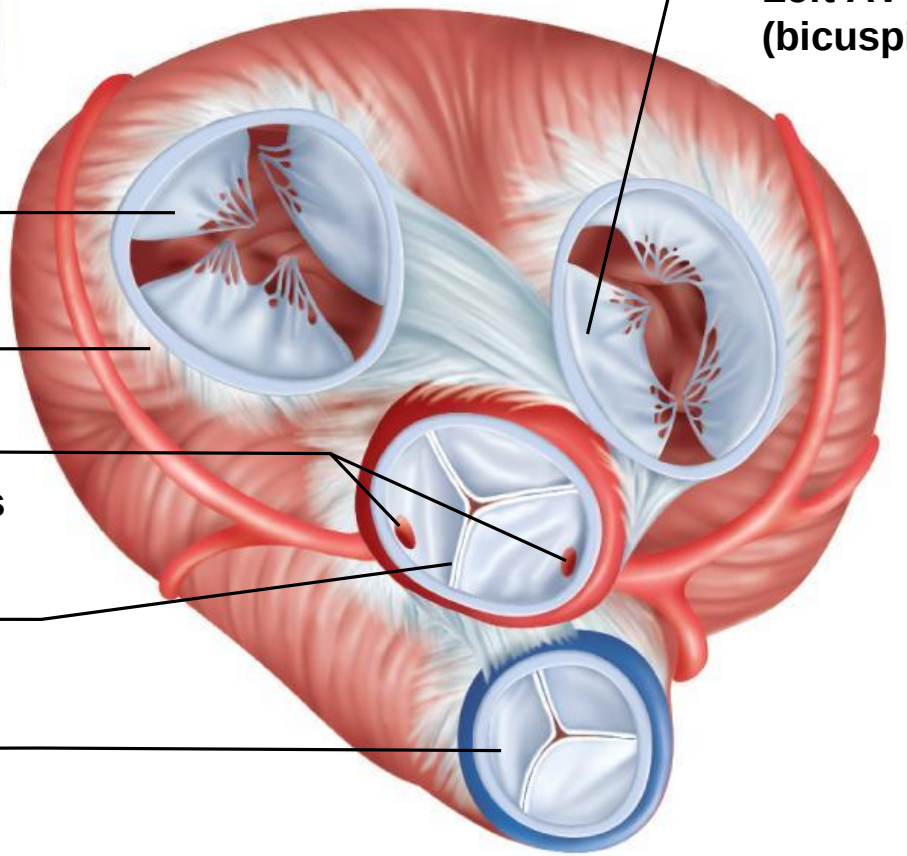
- **atrioventricular (AV) valves** – controls blood flow between atria and ventricles
  - **right AV valve** has 3 cusps (**tricuspid valve**)
  - **left AV valve** has 2 cusps (**mitral or bicuspid valve**)
  - **chordae tendineae** - cords connect AV valves to **papillary muscles** on floor of ventricles
    - prevent AV valves from flipping inside out or bulging into the atria when the ventricles contract

- **semilunar valves** - control flow into great arteries – open and close because of blood flow and pressure

  - **pulmonary semilunar valve** - in opening between right ventricle and pulmonary trunk
  - **aortic semilunar valve** in opening between left ventricle and aorta

# Heart Valves

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

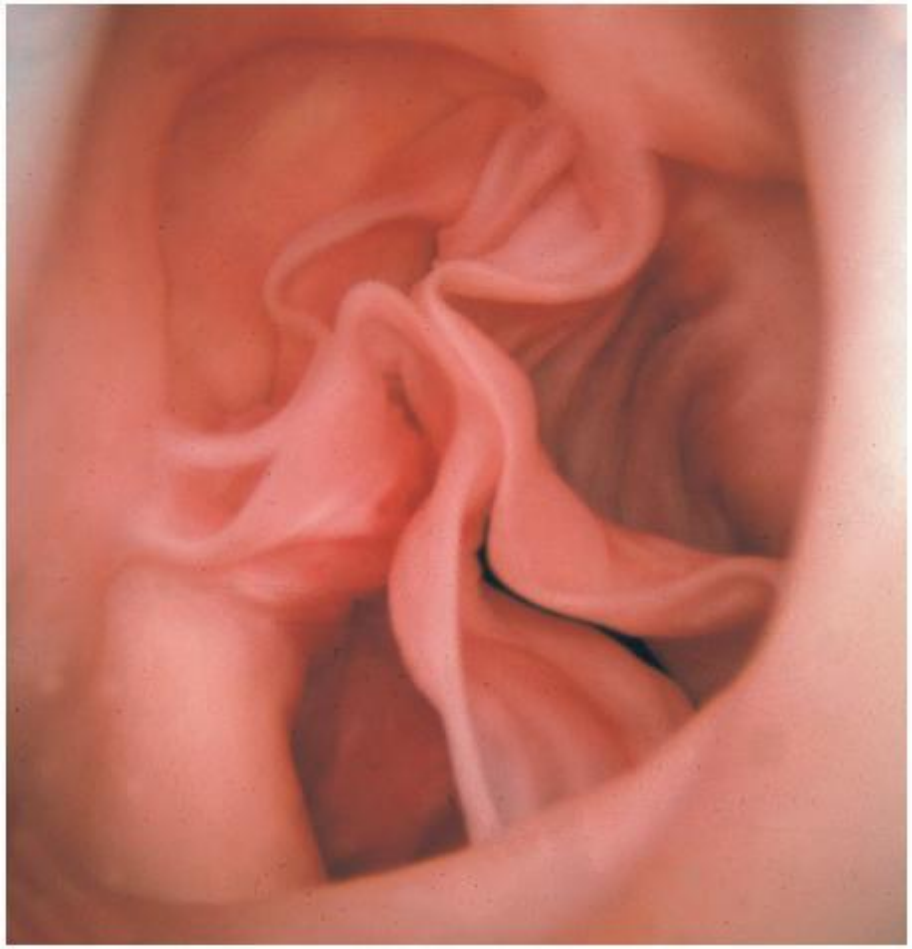



(a)

Figure 19.8a

# Endoscopic View of Heart Valve

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition



**(b)**



Figure 19.8b

# Heart Valves

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

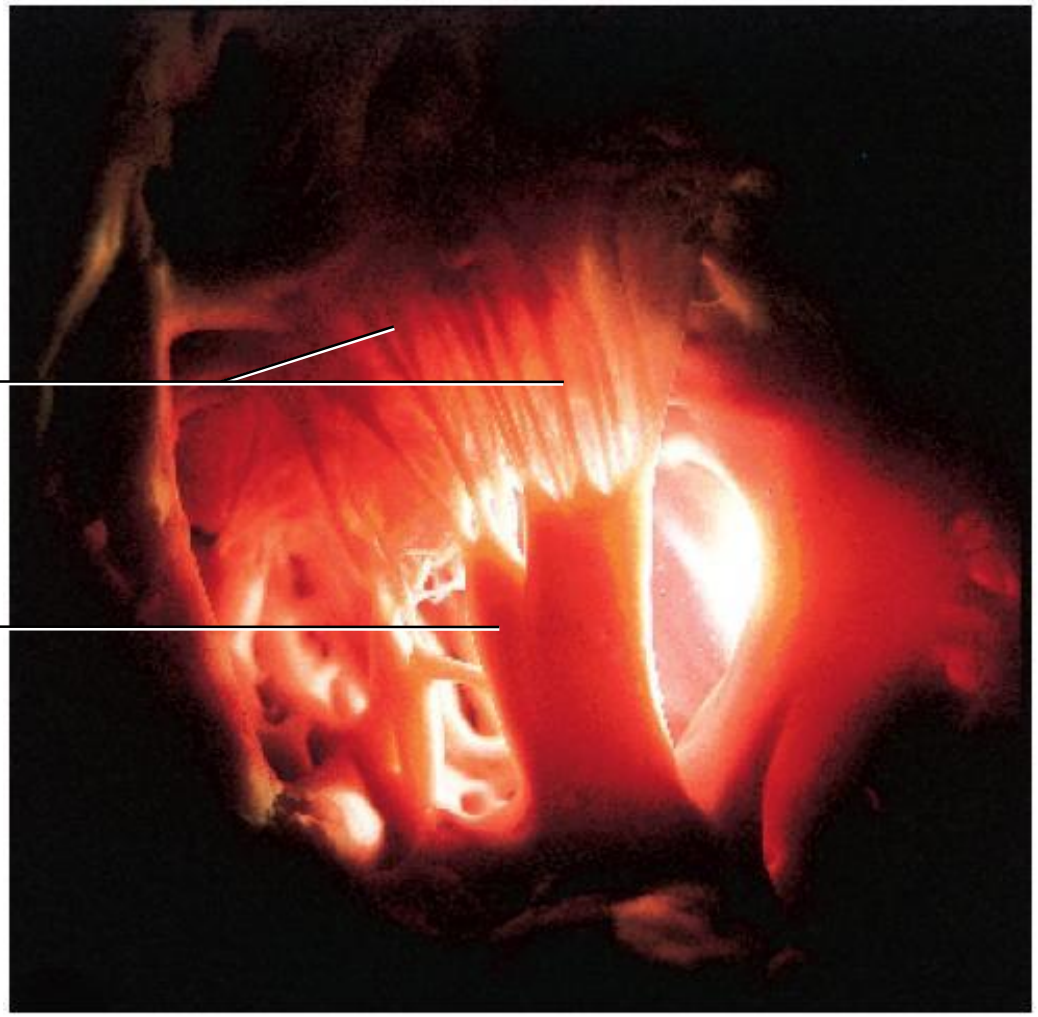



Figure 19.8c

(c)

# AV Valve Mechanics

- ventricles relax
  - pressure drops inside the ventricles
  - semilunar valves close as blood attempts to back up into the ventricles from the vessels
  - AV valves open
  - blood flows from atria to ventricles

- ventricles contract
  - AV valves close as blood attempts to back up into the atria
  - pressure rises inside of the ventricles
  - semilunar valves open and blood flows into great vessels

# Blood Flow Through Heart

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

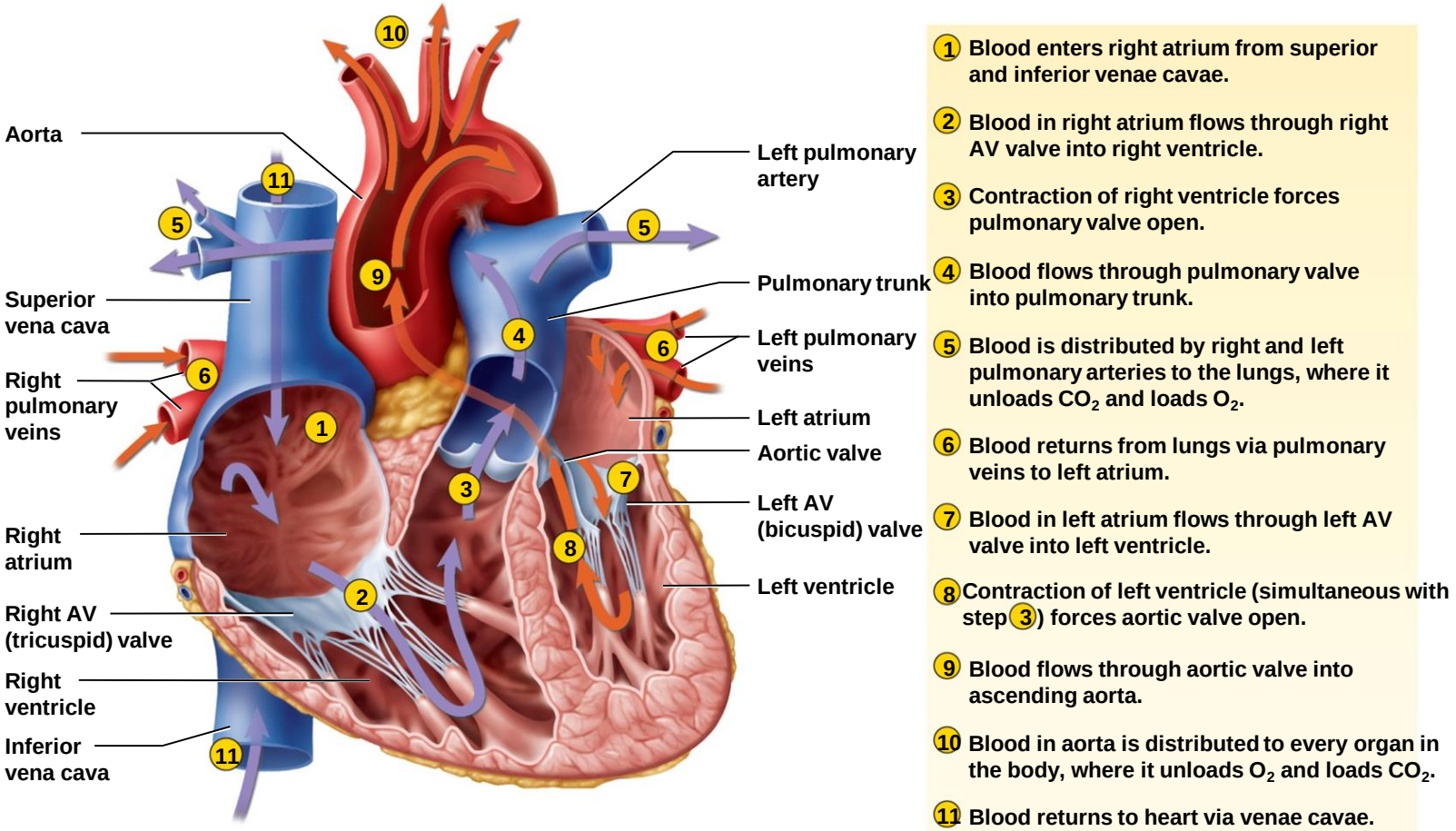



Figure 19.9

blood pathway travels from the right atrium through the body and back to the starting point

# Coronary Circulation

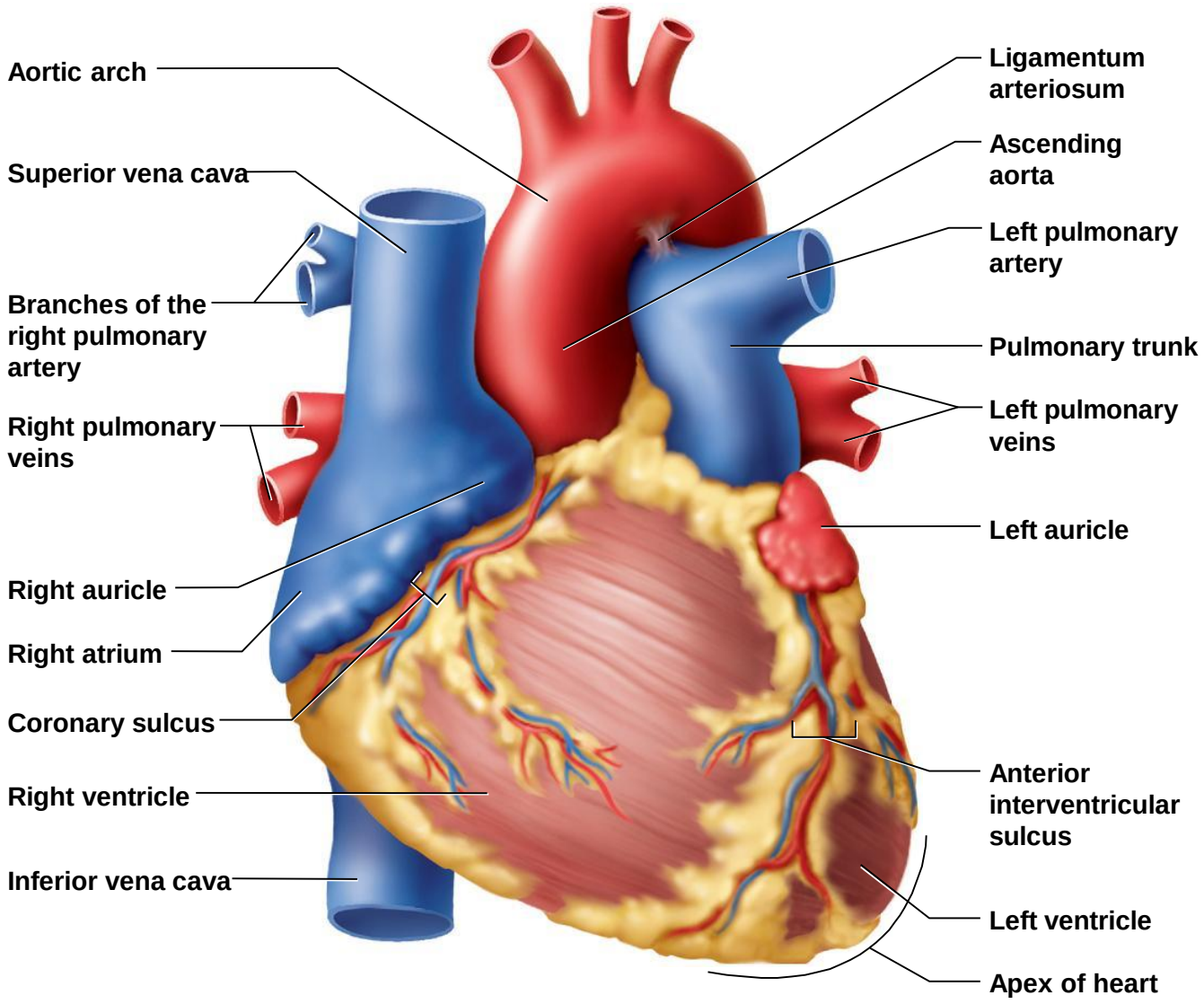
- 5% of blood pumped by heart is pumped to the heart itself through the coronary circulation to sustain its strenuous workload
  - 250 ml of blood per minute
  - needs abundant $O_2$ and nutrients

- **left coronary artery** (LCA) branch off the ascending aorta
  - **anterior interventricular branch**
    - supplies blood both ventricles and anterior two-thirds of the interventricular septum
  - **circumflex branch**
    - passes around left side of heart in coronary sulcus
    - gives off **left marginal branch** and then ends on the posterior side of the heart
    - supplies left atrium and posterior wall of left ventricle

- **right coronary artery** (RCA) branch off the ascending aorta
  - supplies right atrium and sinoatrial node (pacemaker)
  - **right marginal branch**
    - supplies lateral aspect of right atrium and ventricle
  - **posterior interventricular branch**
    - supplies posterior walls of ventricles

# Coronary Vessels - Anterior

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

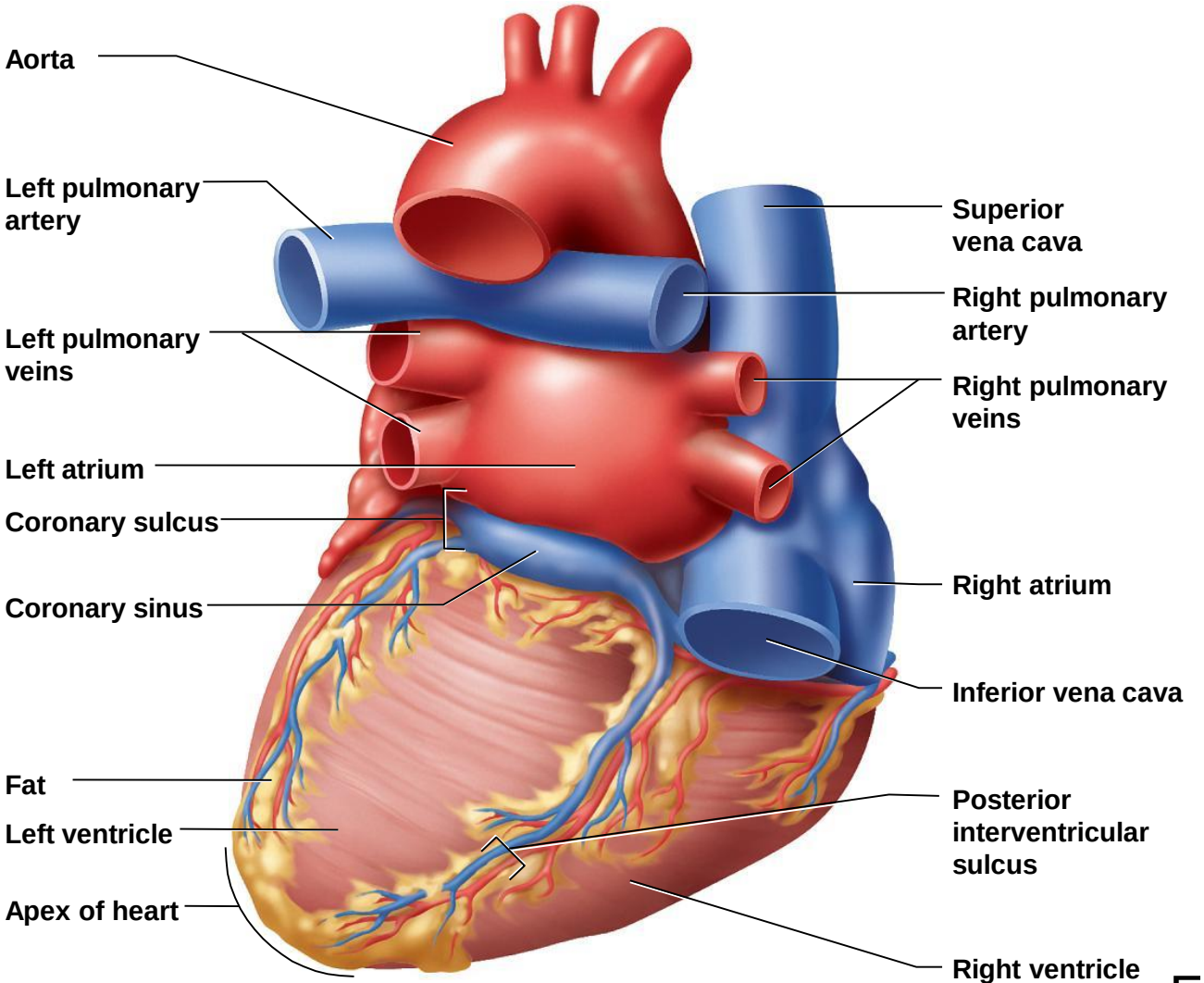



(a) Anterior view

Figure 19.5a

# Coronary Vessels - Posterior

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition




(b) Posterior view

Figure 19.5b

## DEEPER INSIGHT 19.1

## CLINICAL APPLICATION

### *Angina and Heart Attack*

An obstruction of coronary blood flow can cause a chest pain known as *angina pectoris*[16] (an-JY-na PEC-toe-riss) or, more seriously, *myocardial infarction* (heart attack). Angina is a sense of heaviness or pain in the chest resulting from temporary and reversible *ischemia*[17] (iss-KEE-me-ah), or deficiency of blood flow to the cardiac muscle. It typically occurs when a partially blocked coronary artery constricts. The oxygen-deprived myocardium shifts to anaerobic fermentation, producing lactate, which stimulates pain receptors in the heart. The pain abates when the artery relaxes and normal blood flow resumes.

## DEEPER INSIGHT 19.1

## CLINICAL APPLICATION

### *Angina and Heart Attack*

Myocardial infarction (MI), on the other hand, is the sudden death of a patch of myocardium resulting from long-term obstruction of the coronary circulation. Coronary arteries often become obstructed by a blood clot or fatty deposit called an *atheroma* (see Deeper Insight 19.4). As cardiac muscle downstream from the obstruction dies, the indi- vidual commonly feels a sense of heavy pressure or squeezing pain in the chest, often "radiating" to the shoulder and left arm. Some MIs are painless, "silent" heart attacks, especially in elderly or diabetic individu- als. Infarctions weaken the heart wall and disrupt electrical conduction pathways, potentially leading to fibrillation and cardiac arrest (discussed later in this chapter). MI causes about 27% of deaths in the United States.

# Coronary Blood Flow

- **myocardial infarction** (MI) (heart attack)
  - interruption of blood supply to the heart from a blood clot or fatty deposit (atheroma) can cause death of cardiac cells within minutes
  - some protection from MI is provided by **arterial anastomoses** which provides an alternative route of blood flow (**collateral circulation)** within the myocardium

- blood flow to the heart muscle during ventricular contraction is slowed, unlike the rest of the body

- three reasons:
  - contraction of the myocardium compresses the coronary arteries and obstructs blood flow
  - opening of the aortic valve flap during ventricular systole covers the openings to the coronary arteries blocking blood flow into them
  - during ventricular diastole, blood in the aorta surges back toward the heart and into the openings of the coronary arteries
    - blood flow to the myocardium increases during ventricular relaxation

# Angina and Heart Attack

- **angina pectoris** – chest pain from partial obstruction of coronary blood flow
  - pain caused by ischemia of cardiac muscle
  - obstruction partially blocks blood flow
  - myocardium shifts to anaerobic fermentation producing lactic acid stimulating pain

- **myocardial infarction** – sudden death of a patch of myocardium resulting from long-term obstruction of coronary circulation
  - atheroma (blood clot or fatty deposit) often obstruct coronary arteries
  - cardiac muscle downstream of the blockage dies
  - heavy pressure or squeezing pain radiating into the left arm
  - some painless heart attacks may disrupt electrical conduction pathways, lead to fibrillation and cardiac arrest
    - silent heart attacks occur in diabetics & elderly
  - MI responsible for about half of all deaths in the United States

# Venous Drainage of Heart

- 5 -10% drains directly into heart chambers, right atrium and right ventricle, by way of the **thebesian veins**

- the rest returns to right atrium by the following routes:
  - **great cardiac vein**
    - travels along side of anterior interventricular artery
    - collects blood from anterior portion of heart
    - empties into coronary sinus
  - **middle cardiac vein** (posterior interventricular)
    - found in posterior sulcus
    - collects blood from posterior portion of heart
    - drains into coronary sinus
  - **left marginal vein**
    - empties into coronary sinus

- **coronary sinus**
    - large transverse vein in coronary sulcus on posterior side of heart
    - collects blood and empties into right atrium

l-Farabi Kazakh National University
Higher School of Medicine

# Circulatory System

## -Heart

**Cardiac Muscle and the Cardiac Conduction System + Case**

# ◆ PART III

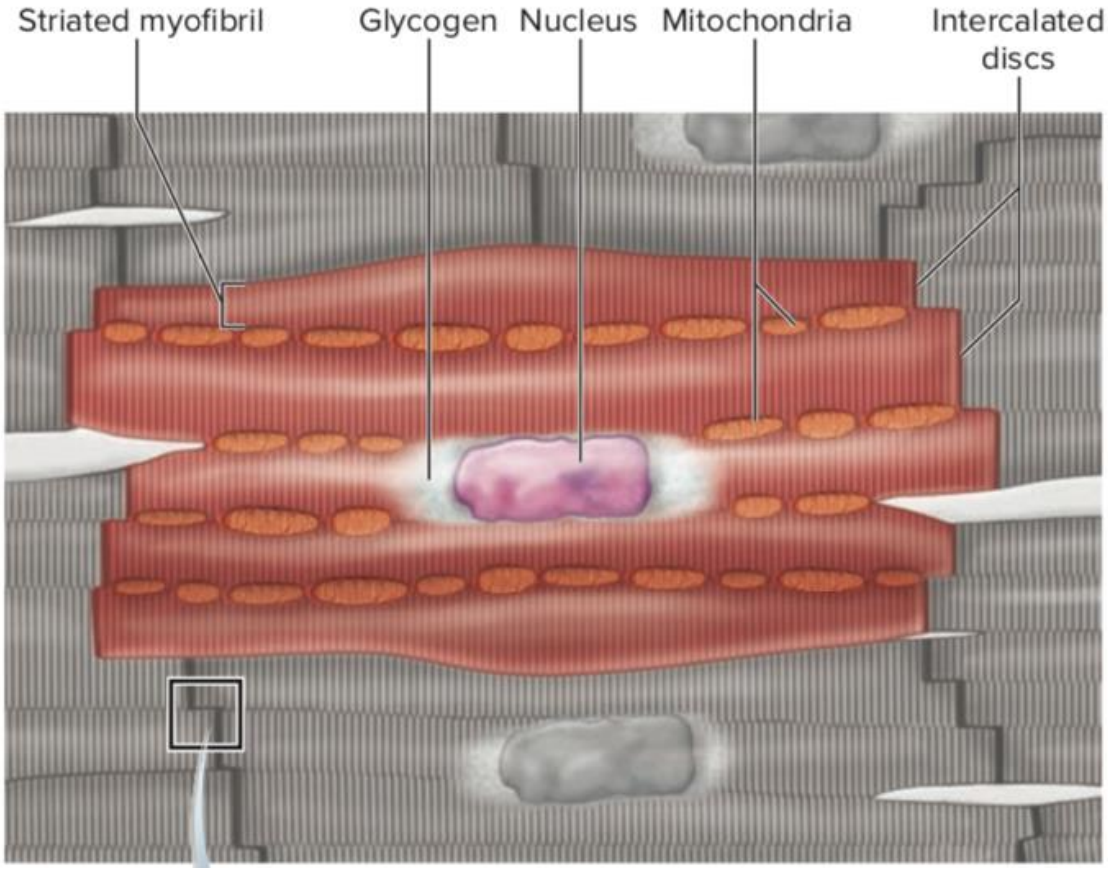
# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- ***Describe the unique structural and metabolic characteristics of cardiac muscle;***
- ***explain the nature and functional significance of the intercellular junctions between cardiac muscle cells;***
- ***describe the heart's pacemaker and internal electrical conduction system;***
- ***describe the nerve supply to the heart and explain its role.***

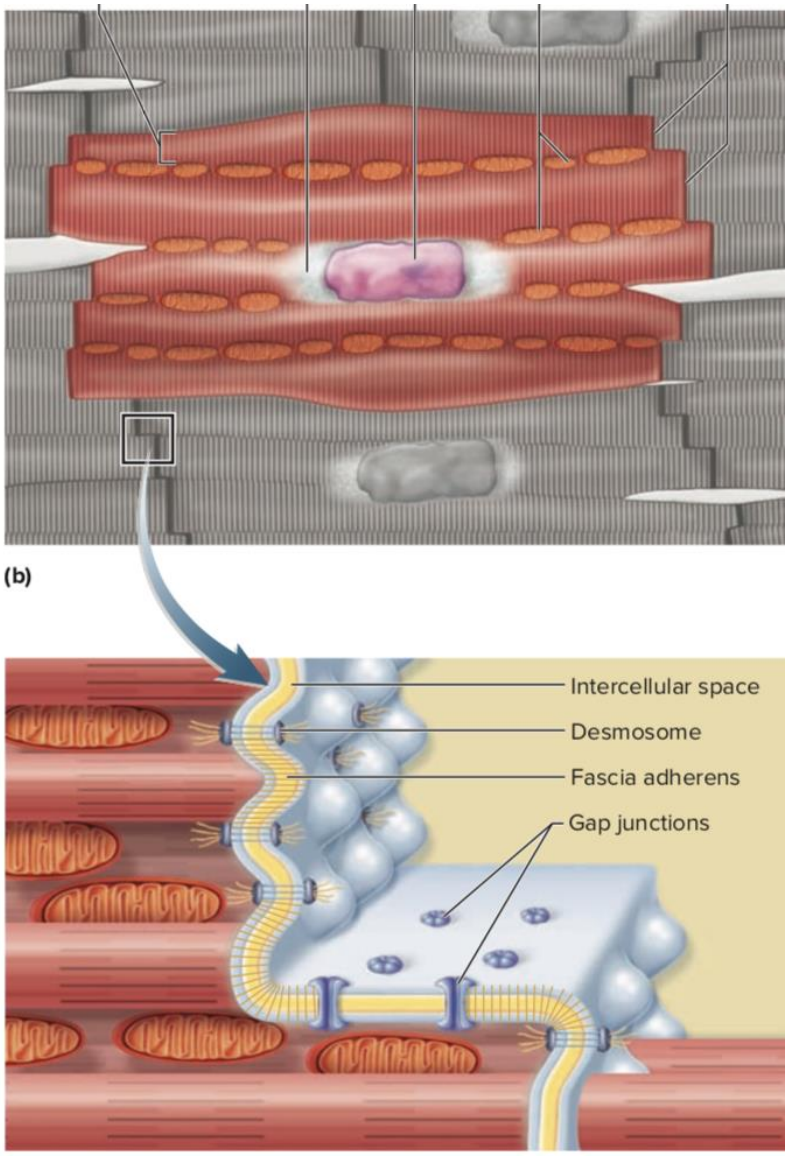
# Structure of Cardiac Muscle

- **cardiocytes** - striated, short, thick, branched cells, one central nucleus surrounded by light staining mass of glycogen



# Structure of Cardiac Muscle

- **intercalated discs -** join cardiocytes end to end
  - **interdigitating folds** – folds interlock with each other, and increase surface area of contact
  - **mechanical junctions** tightly join cardiocytes
    - **fascia adherens** – broad band in which the actin of the thin myofilaments is anchored to the plasma membrane
      - each cell is linked to the next via transmembrane proteins
    - **desmosomes** - weldlike mechanical junctions between cells
      - prevents cardiocytes from being pulled apart
  - **electrical junctions** - **gap junctions** allow ions to flow between cells – can stimulate neighbors
    - entire myocardium of either two atria or two ventricles acts like single unified cell



# Structure of Cardiac Muscle

What organelle(s) is/are less developed in cardiac muscle than in skeletal muscle? What one(s) is/are more developed? What is the functional significance of these differences between muscle types?

- **Myocardial contractile cells** constitute the bulk (99 percent) of the cells in the atria and ventricles.
- - Contractile cells conduct impulses and are responsible for contractions that pump blood through the body.
- **Myocardial conducting cells** (1 percent of the cells) are the autorhythmic cells
- - form the conduction system of the heart.
- repair of damage of cardiac muscle is almost entirely by **fibrosis** (scarring)

# Metabolism of Cardiac Muscle

- **cardiac muscle** depends almost exclusively on **aerobic respiration** used to make ATP
  - rich in myoglobin and glycogen
  - huge mitochondria – fill 25% of cell

- **adaptable to organic fuels** used
  - fatty acids (60%), glucose (35%), ketones, lactic acid and amino acids (5%)
  - more vulnerable to oxygen deficiency than lack of a specific fuel

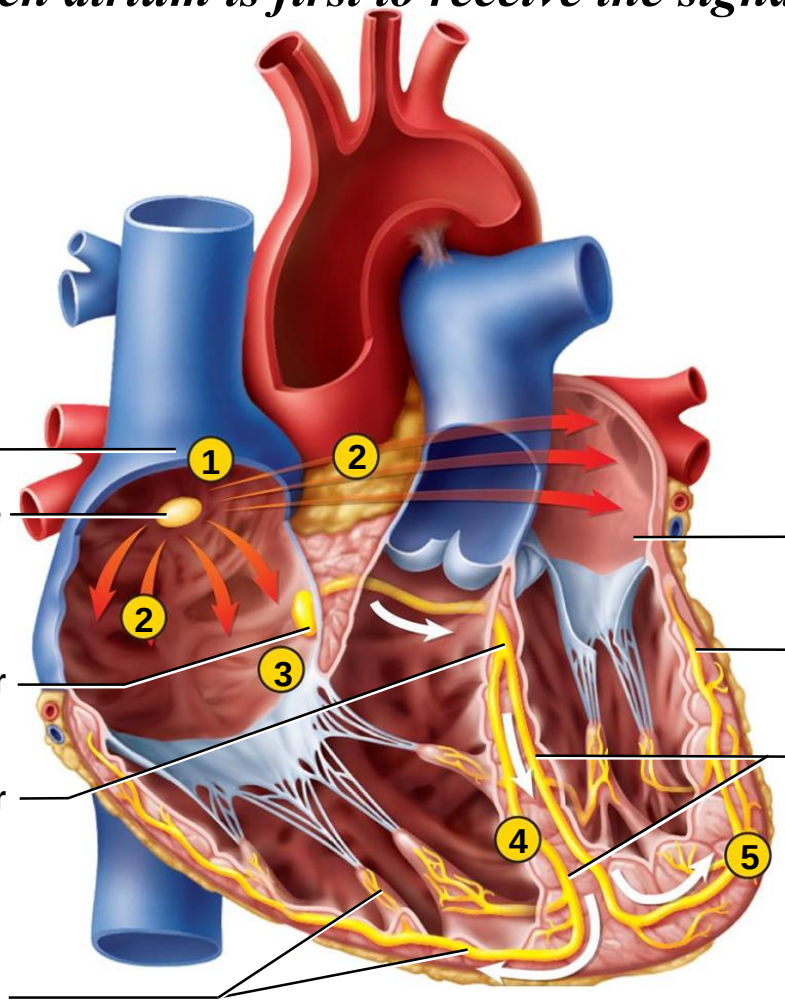
- **fatigue resistant** since makes little use of anaerobic fermentation or oxygen debt mechanisms
  - Does not fatigue for a lifetime

**Cardiac muscle rarely uses anaerobic fermentation to generate ATP. What benefit do we gain from this fact?**

# Cardiac Conduction System

- **Cardiac Conduction System**
  - coordinates the heartbeat
  - composed of an **internal pacemaker** and **nervelike conduction pathways** through myocardium
  - generates and conducts rhythmic electrical signals in the following order:
- **sinoatrial (SA) node**
- **atrioventricular (AV) node**
- **atrioventricular (AV) bundle (bundle of His)**
- **Purkinje fibers**
- signal pass from cell to cell through **gap junctions**

## *Which atrium is first to receive the signal that induces atrial contraction?*



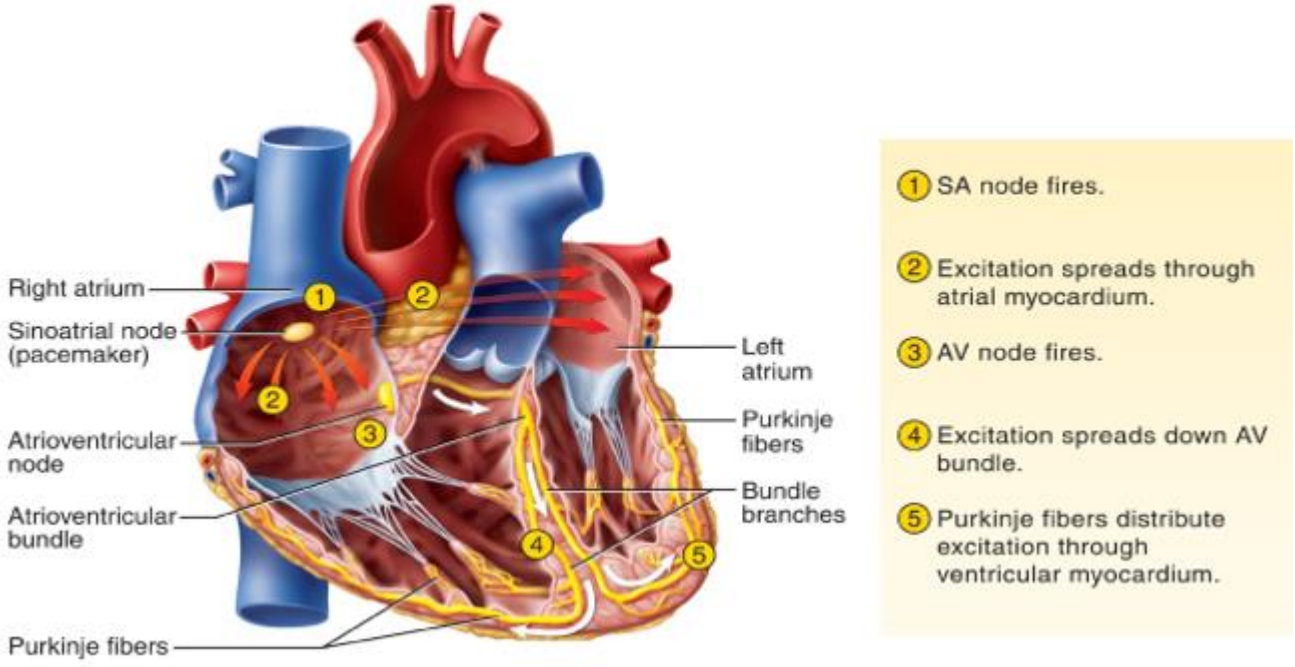

1. SA node fires.
2. Excitation spreads through atrial myocardium.
3. AV node fires.
4. Excitation spreads down AV bundle.
5. Purkinje fibers distribute excitation through ventricular myocardium.

Figure 19.12

# Cardiac Conduction System

- **sinoatrial (SA) node** - modified cardiocytes
  - initiates each heartbeat and determines heart rate
  - signals spread throughout atria
  - **pacemaker** in right atrium near base of superior vena cava

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition



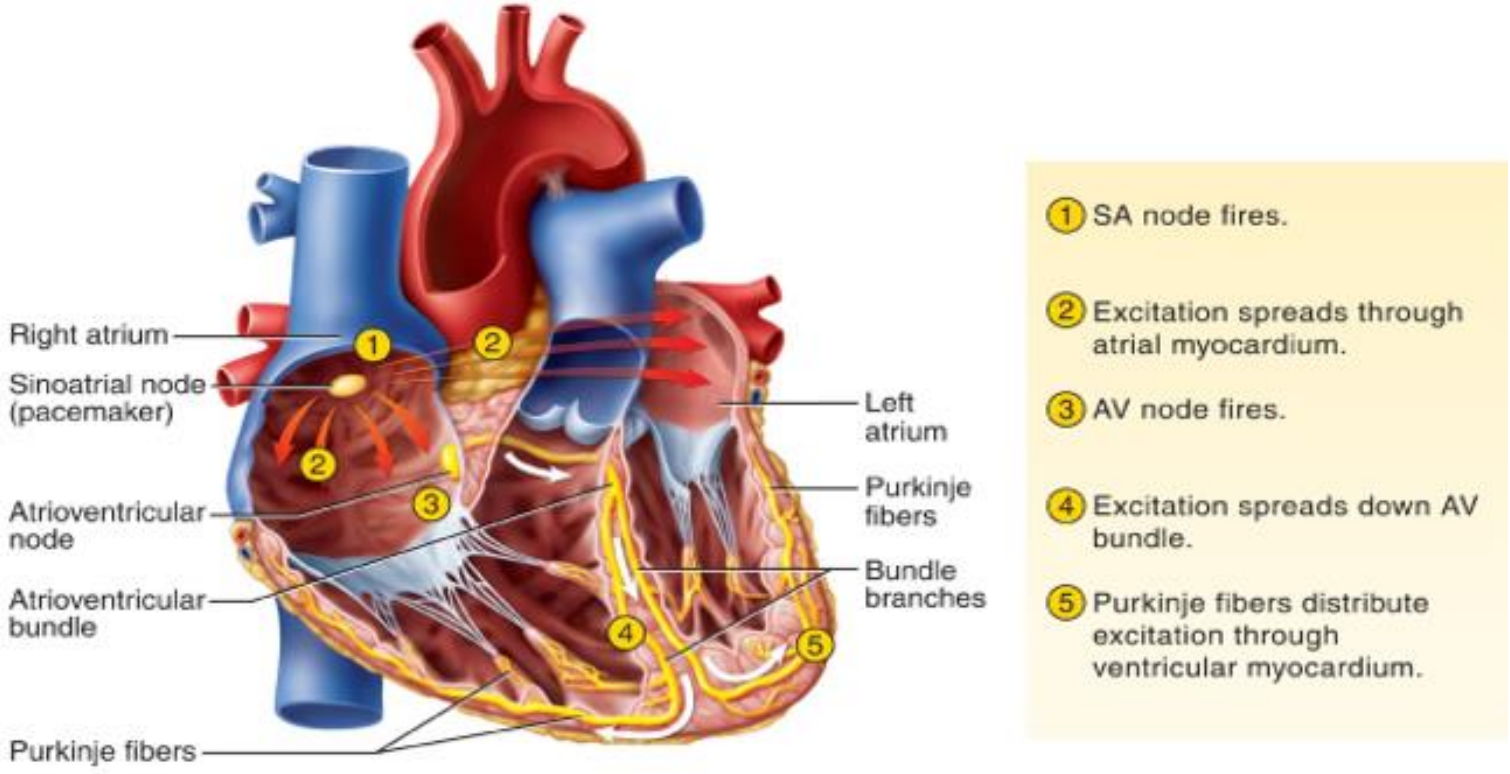
# Cardiac Conduction System

- **atrioventricular (AV) node**
  - located near the right AV valve at lower end of interatrial septum
  - electrical gateway to the ventricles
  - fibrous skeleton acts as an insulator to prevent currents from getting to the ventricles from any other route

How does the delay of the impulse at the atrioventricular node contribute to cardiac function?

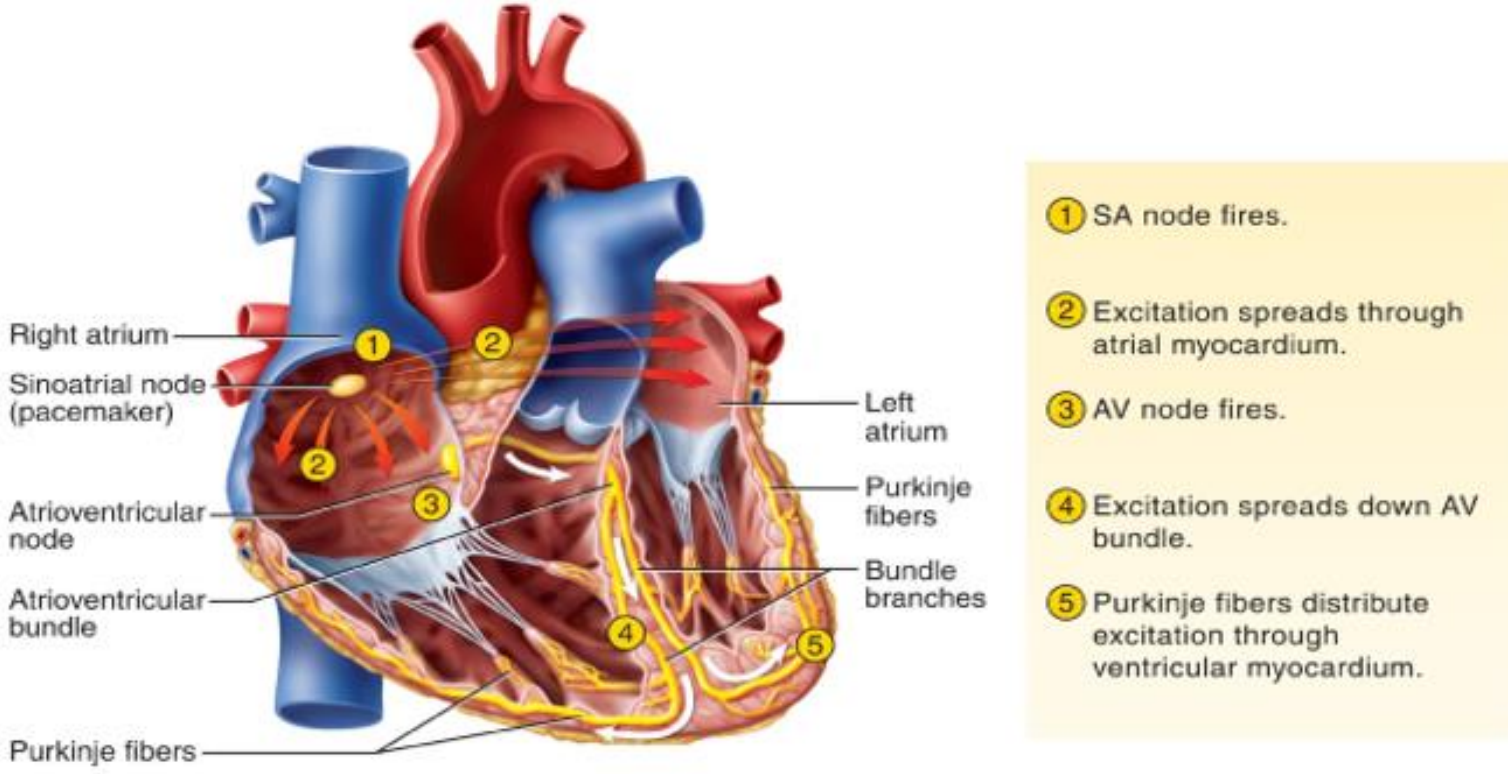
# Cardiac Conduction System

- **atrioventricular (AV) bundle (bundle of His)**
  - bundle forks into right and left bundle branches
  - these branches pass through interventricular septum toward apex



# Cardiac Conduction System

- **Purkinje fibers**
  - nervelike processes spread throughout ventricular myocardium

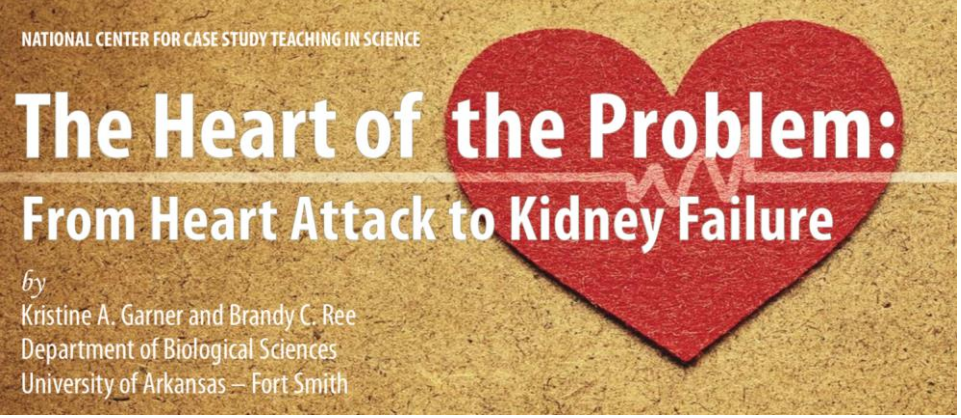


# Nerve Supply to Heart

- **sympathetic nerves (raise heart rate)**
  - sympathetic pathway to the heart originates in the **lower cervical** to **upper thoracic segments** of the spinal cord
  - continues to adjacent **sympathetic chain ganglia**
  - some pass through **cardiac plexus** in mediastinum
  - continue as **cardiac nerves** to the heart
  - fibers terminate in SA and AV nodes, in atrial and ventricular myocardium, as well as the aorta, pulmonary trunk, and coronary arteries
    - increase heart rate and contraction strength
    - dilates coronary arteries to increase myocardial blood flow

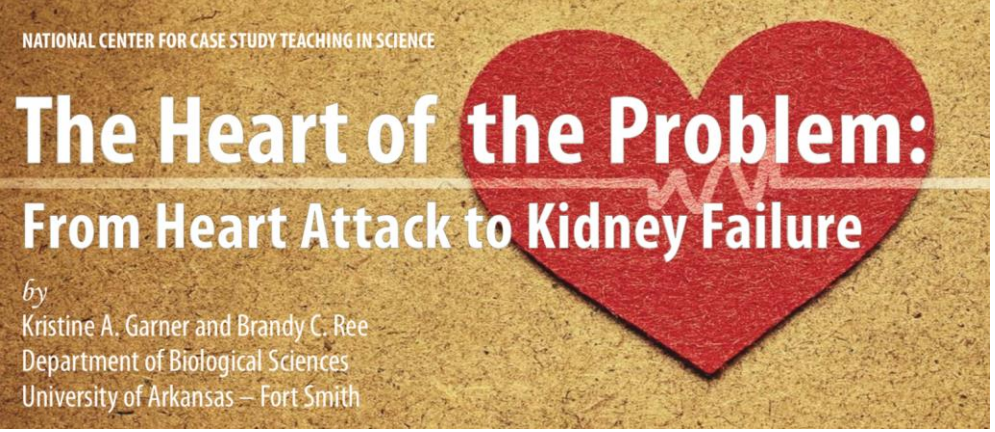
- **parasympathetic nerves (slows heart rate)**
  - pathway begins with **nuclei of the vagus nerves** in the **medulla oblongata**
  - extend to **cardiac plexus** and continue to the heart by way of the **cardiac nerves**
  - fibers of **right vagus nerve** lead to the **SA node**
  - fibers of **left vagus nerve** lead to the **AV node**
  - little or no vagal stimulation of the myocardium
    - parasympathetic stimulation reduces the heart rate

**Why does the heart have a nerve supply, since it continues to beat even without one?**




## Part I – Emergency

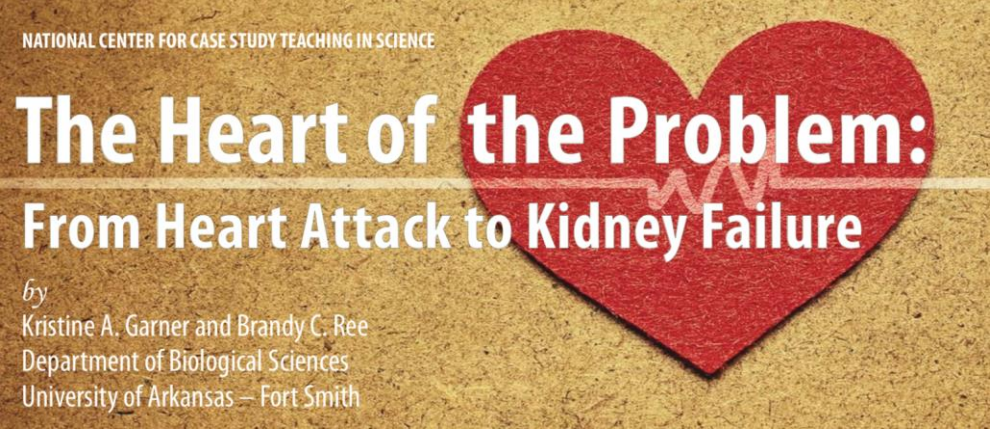
Mrs. Helms came in through the front door of her house with an armful of groceries. She put the bag down on the kitchen counter and called to her husband. "Herb, I'm home! Are you ready for lunch?" She didn't get an answer, so she walked to the living room and found Mr. Helms lying on the floor. "Herb! Are you okay?" she asked as she grabbed his shoulder. Mr. Helms responded weakly while clutching his chest. Mrs. Helms frantically called 911. It only took EMS a few minutes to arrive and the paramedics transported Mr. Helms to the hospital. Upon admission to the hospital, Mr. Helms' vital signs were recorded as follows:




| | *Mr. Helms* | *Normal* |
|---|---|---|
| Systolic blood pressure (mm Hg) | 90 | 120 |
| Diastolic blood pressure (mm Hg) | 52 | 80 |
| Oral temperature (°F) | 98.9 | 97.8 to 99.1 |
| Heart rate (beats per minute) | 120, irregular | 60–80 |
| Respiratory rate (breaths per minute) | 33, labored | 12 to 20 |
| Oxygen saturation | 89% | 95–100% |

***Questions***

**Which of Mr. Helms' vital signs and lab values were abnormal? In other words, what other information would be useful?**

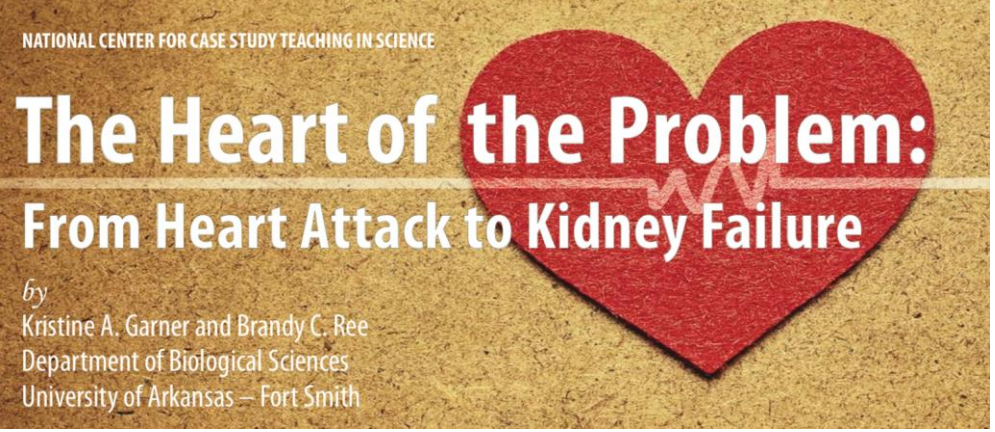



***Part II – Cardiac Involvement***

Mr. Helms was admitted to the hospital with chest pains and shortness of breath. His wife was panicked since her 72 year-old husband had a history of heart disease. After examination and an echocardiogram, Dr. Collins spoke with Mrs. Helms. "I'm very sorry, but your husband has had another heart attack resulting in valve failure. A papillary muscle that controls a valve in his heart has been severely damaged and is no longer working."

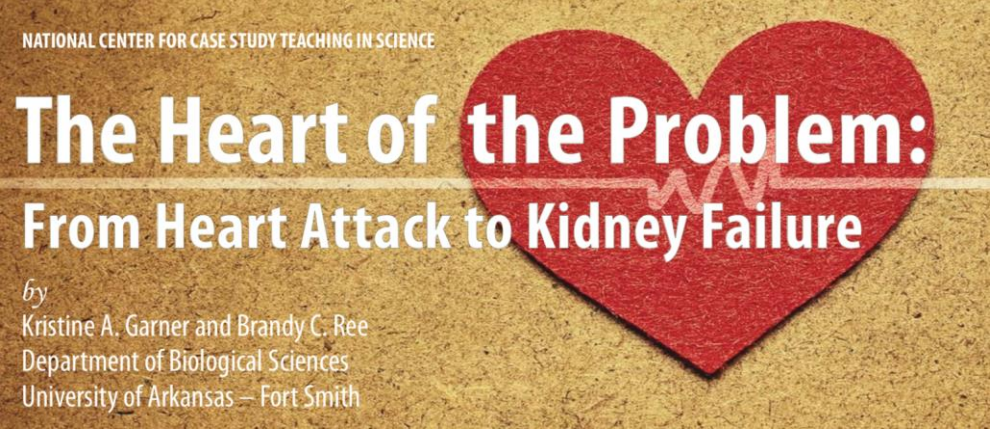
***Questions***

***What is the purpose of blood flow?***



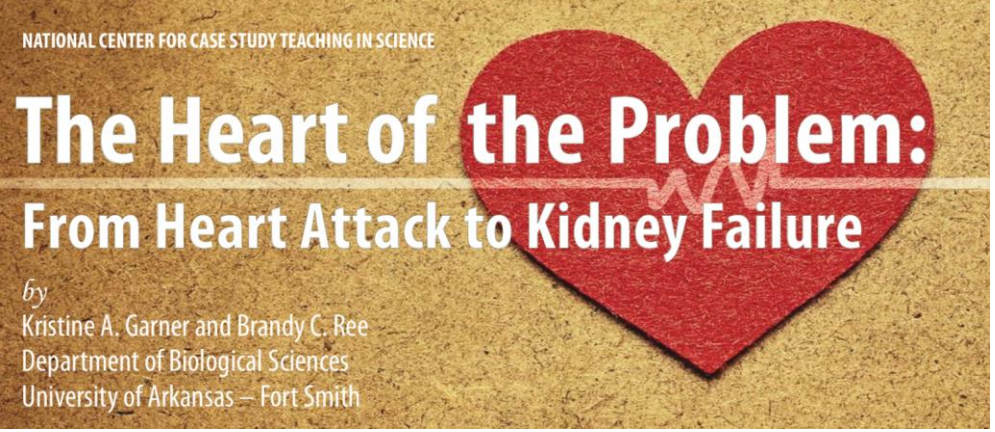

## Questions

***Describe blood flow through the heart starting with blood entering the right side of the heart and including all chambers and valves.***



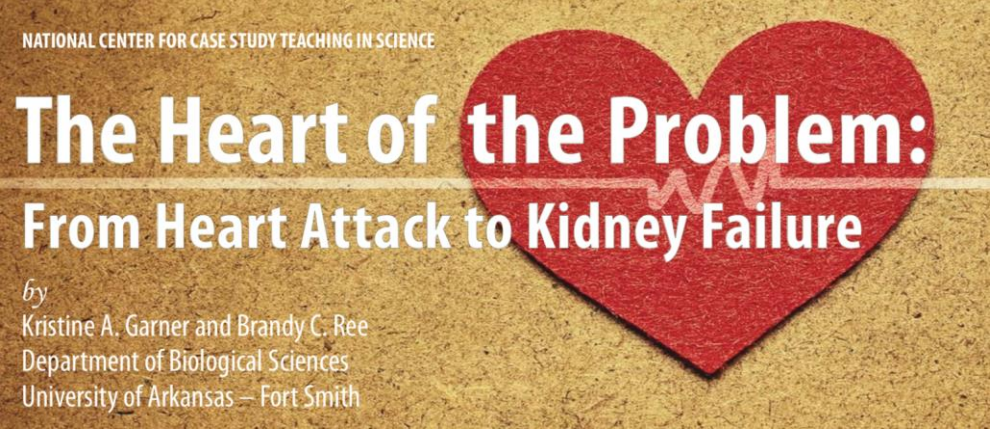

## Questions

***What is the function of heart valves?***




## Questions

***What is the function of papillary muscles?***




# Questions

***Which valve is affected with damage to the papillary muscle in the left ventricle?***

l-Farabi Kazakh National University
Higher School of Medicine

# Circulatory System

# -Heart

## Electrical and Contractile Activity of the Heart

# ◆ PART IV

# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- ***describe explain why the SA node fires spontaneously and rhythmically***
- ***describe the unusual action potentials of cardiac muscle and relate them to the contractile behavior of the heart;***
- ***interpret a normal electrocardiogram***

# Cardiac Rhythm

- cycle of events in heart – special names
  - **systole** – atrial or ventricular contraction
  - **diastole** – atrial or ventricular relaxation
- **sinus rhythm** - normal heartbeat triggered by the SA node
  - set by SA node at 60 – 100 bpm
  - adult at rest is 70 to 80 bpm (vagal tone)
- **ectopic focus** - another parts of heart fires before SA node
  - caused by hypoxia, electrolyte imbalance, or caffeine, nicotine, and other drugs

# Pacemaker Physiology

- **SA node** does not have a stable resting membrane potential
  - starts at -60 mV and drifts upward from a slow inflow of $Na^+$
    - gradual depolarization is called **pacemaker potential**
      - slow inflow of $Na^+$ without a compensating outflow of $K^+$
  - when reaches **threshold** of -40 mV, voltage-gated **fast $Ca^{2+}$ and $Na^+$ channels** open
    - faster **depolarization** occurs peaking at 0 mV
    - **$K^+$ channels** then open and $K^+$ leaves the cell
      - causing **repolarization**
      - once $K^+$ channels close, pacemaker potential starts over

- each depolarization of the SA node sets off one heartbeat
  - at rest, fires every 0.8 seconds or 75 bpm

- SA node is the system's pacemaker

# SA Node Potentials

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

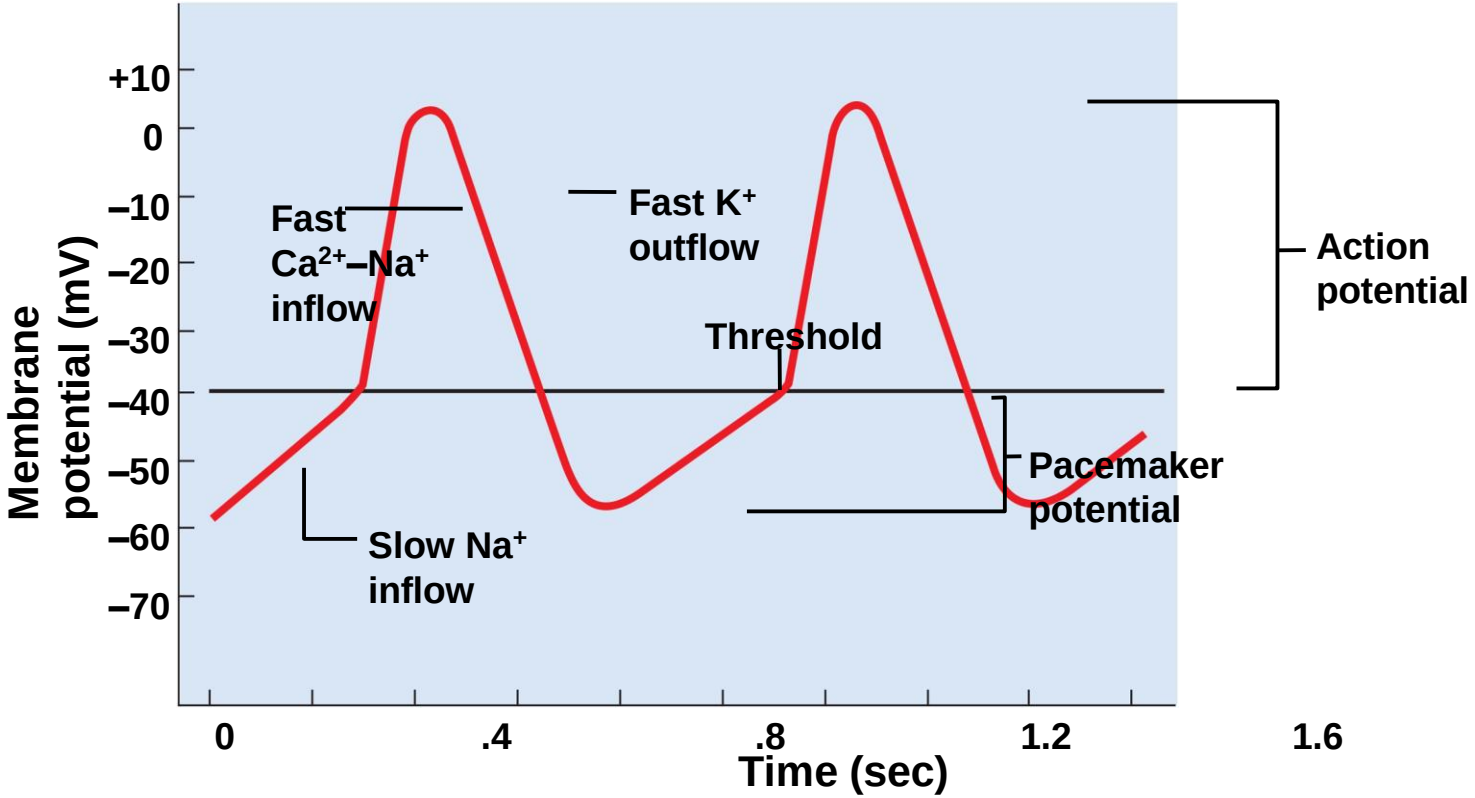



Figure 19.13

# Impulse Conduction to Myocardium

- signal from **SA node** stimulates two atria to contract almost simultaneously
  - reaches AV node in 50 msec
- signal slows down through **AV node**
  - thin cardiocytes have fewer gap junctions
  - delays signal 100 msec which allows the ventricles to fill
- signals travel very quickly through **AV bundle** and **Purkinje fibers**
  - entire ventricular myocardium depolarizes and contracts in near unison
    - papillary muscles contract an instant earlier than the rest, tightening slack in chordae tendineae
- ventricular systole progresses up from the apex of the heart
  - spiral arrangement of cardiocytes twists ventricles slightly
  - like someone wringing out a towel

# Electrical Behavior of Myocardium

- cardiocytes have a stable resting potential of -90 mV
- depolarize only when stimulated
  - **depolarization phase** (very brief)
    - stimulus opens voltage regulated $Na^+$ gates, ($Na^+$ rushes in) membrane **depolarizes rapidly**
    - action potential peaks at +30 mV
    - $Na^+$ gates close quickly
  - **plateau phase** lasts 200 to 250 msec, sustains contraction for expulsion of blood from heart
    - $Ca^{2+}$ channels are slow to close and SR is slow to remove $Ca^{2+}$ from the cytosol
  - **repolarization phase** - $Ca^{2+}$ channels close, $K^+$ channels open, rapid diffusion of $K^+$ out of cell returns it to resting potential
- has a long absolute refractory period of 250 msec compared to 1 – 2 msec in skeletal muscle
  - prevents wave summation and tetanus which would stop the pumping action of the heart

# Action Potential of a Cardiocyte

1) $Na^+$ gates open
2) Rapid depolarization
3) $Na^+$ gates close
4) Slow $Ca^{2+}$ channels open
5) $Ca^{2+}$ channels close, $K^+$ channels open (repolarization)

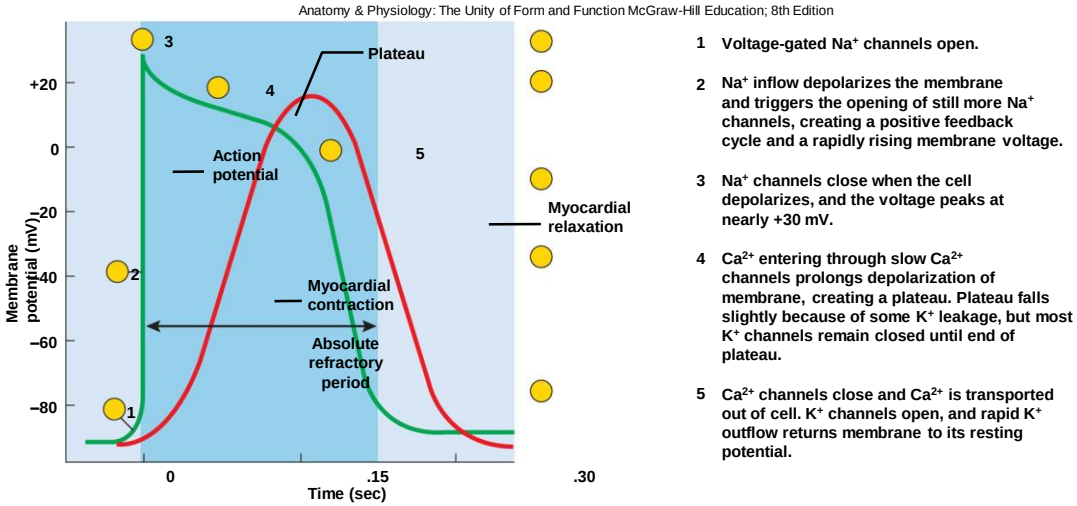



Figure 19.14

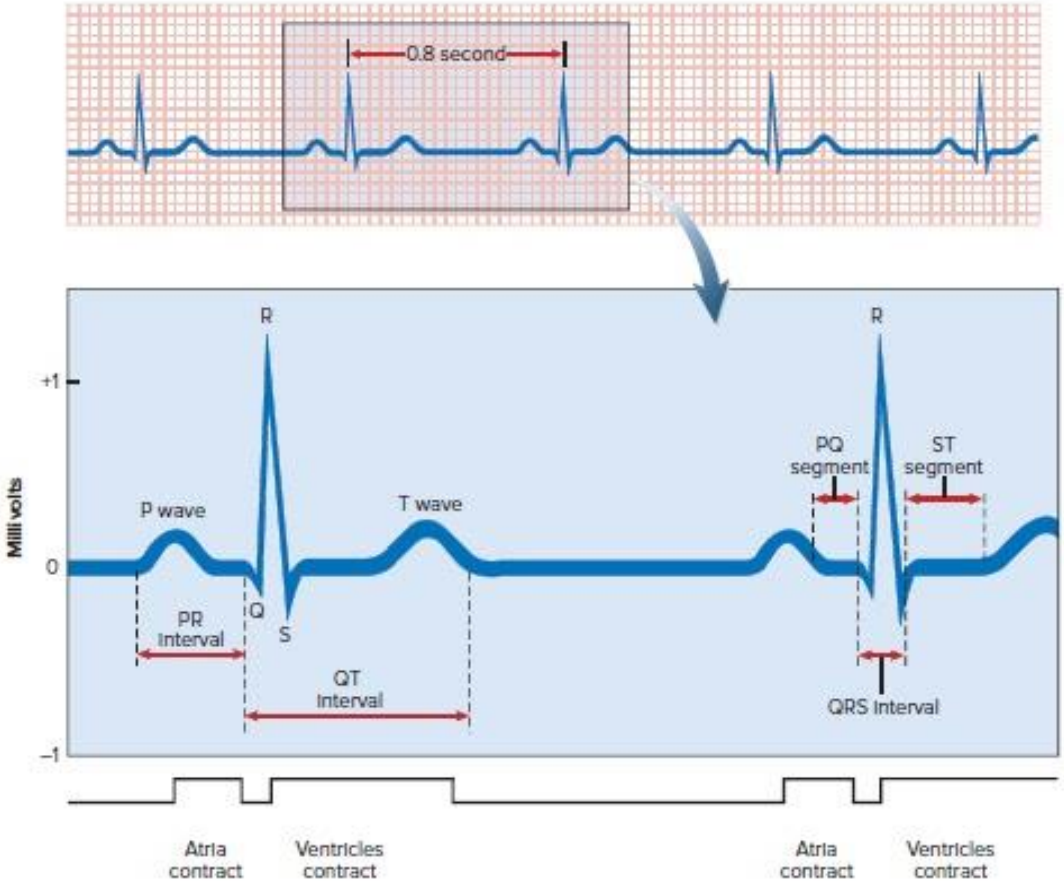
# The Electrocardiogram

record of electrical currents in the heart by means of electrodes (leads) applied to the skin.

- electrodes are attached to the wrists, ankles, and six locations on the chest.
- An ECG is a composite recording of all action potentials produced by the nodal and myocardial cells—it should not be misconstrued as a tracing of a single action potential.

Typical ECG - three principal deflections above and below the baseline:

1. the P wave
2. QRS complex
3. T wave.


0.8 second
R
R
+1
0
–1
Milli volts
P wave
T wave
PQ segment
ST segment
PR Interval
Q
S
QT Interval
QRS Interval
Atria contract
Ventricles contract
Atria contract
Ventricles contract

**The P wave** - is produced when a signal from the SA node spreads through the atria and depolarizes them.

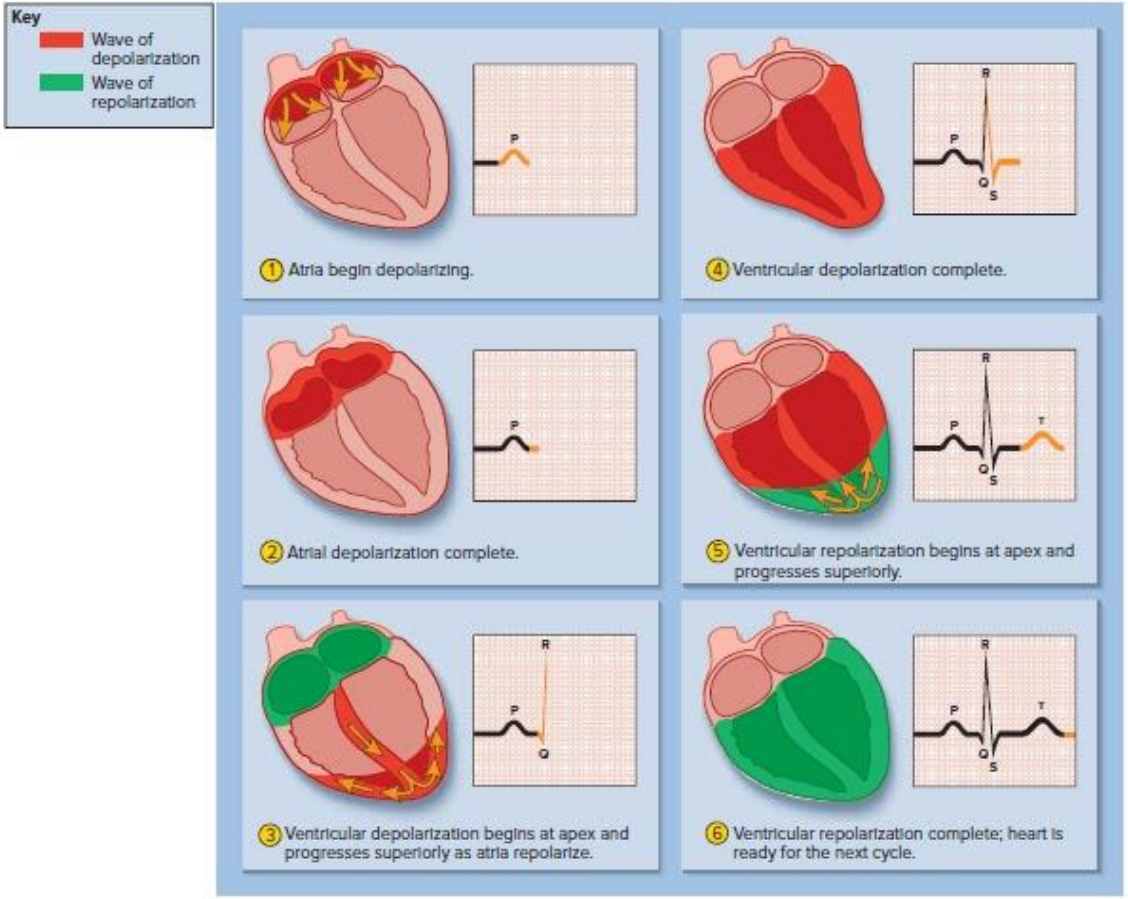
- Atrial systole (100 ms after the P wave begins) - **PQ segment** -160 ms long and represents - impulses from the SA node to the AV node.

**The QRS complex** consists of a small downward deflection (Q), a tall sharp peak (R), and a final downward deflection (S).

- signal from the AV node spreads through the ventricular myocardium and depolarizes the muscle.
- QRS - *depolarization of the ventricles*, which constitute the largest muscle mass of the heart and generate the greatest electrical current.
- Ventricular systole - **the ST segment.**
- *Atrial repolarization and diastole also occur during the QRS interval*, but sends a relatively weak signal that is obscured by the electrical activity of the more muscular ventricles.
- The ST segment corresponds to the plateau in the myocardial action potential and thus represents the time during which the ventricles contract and eject blood.

**The T wave** - ventricular repolarization immediately before diastole.

- T wave - smaller and more spread out than the QRS complex, and it has a rounder peak.


Key
Wave of depolarization
Wave of repolarization
P
R
Q
S
T
1 Atria begin depolarizing.
2 Atrial depolarization complete.
3 Ventricular depolarization begins at apex and progresses superiorly as atria repolarize.
4 Ventricular depolarization complete.
5 Ventricular repolarization begins at apex and progresses superiorly.
6 Ventricular repolarization complete; heart is ready for the next cycle.

- **Arrhythmia** -any deviation from the regular, SA node–driven sinus rhythm of the heartbeat.
- **Ventricular fibrillation** - the hallmark of a heart attack (myocardial infarction) - the ECG shows weak, chaotic ventricular depolarizations as electrical signals travel randomly about the myocardium and return to repeatedly restimulate the same area instead of dying out like a normal ventricular depolarization.

A fibrillating ventricle pumps no blood, so there is no coronary blood flow and myocardial tissue rapidly dies of ischemia, as does cerebral tissue.

- **Cardiac arrest** is the cessation of cardiac output, with the ventricles either motionless or in fibrillation.
- **Defibrillation** is an emergency procedure in which the heart is given a strong electrical shock with a pair of paddle electrodes.

# Cardiac Arrhythmias

**Atrial fibrillation** is a weak rippling contraction in the atria, manifested in the ECG by chaotic, high-frequency depolarizations 400–650/min.).

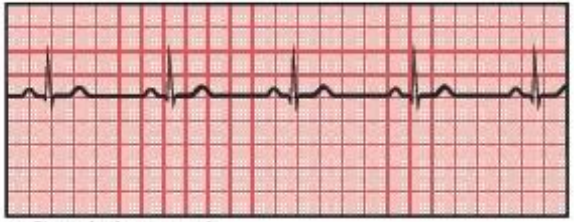
- Fibrillating atria fail to stimulate the ventricles, so we see a dissociation between the random atrial depolarizations and the ventricular QRS and T waves of the ECG.
- This is the most common atrial arrhythmia in the elderly. Result from - valvular disease, thyroid hormone excess, or myocardial inflammation, and is often seen in alcoholism.

**Heart block** is a failure of any part of the cardiac conduction system to conduct signals, usually as the result of disease and degeneration of conduction system fibers.

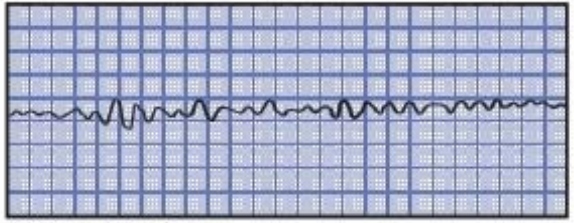
- In the ECG, one sees rhythmic atrial P waves, but the ventricles fail to receive the signal and no QRS wave follows the P. A bundle branch block is a heart block resulting from damage to one or both branches of the AV bundle. Damage to the AV node causes total heart block, in which signals from the atria fail to reach the ventricles at all, and the ventricles beat at their own intrinsic rhythm of 20 to 40 bpm.

**Premature ventricular contraction** (PVC) is the result of a ventricular ectopic focus firing and setting off an extra beat (extrasystole) before the normal signal from the SA node arrives. The P wave is missing and the QRS wave is inverted and misshapen. PVCs can occur singly or in bursts.

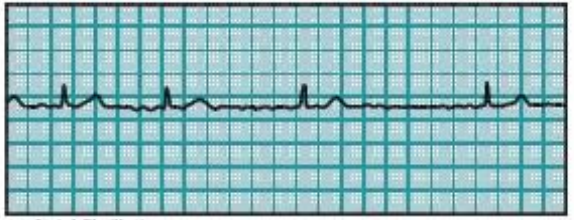
- An occasional extra beat is not serious, and may result from emotional stress, lack of sleep, or irritation of the heart by stimulants (nicotine, caffeine).
- Persistent PVCs - indicate more serious pathology and sometimes lead to ventricular fibrillation and sudden death.



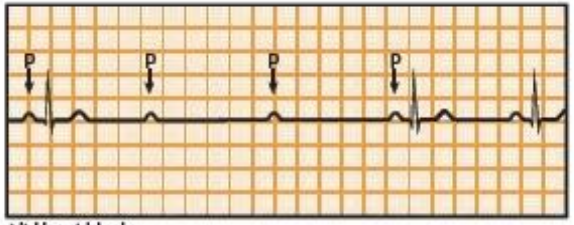
(a) Sinus rhythm (normal)



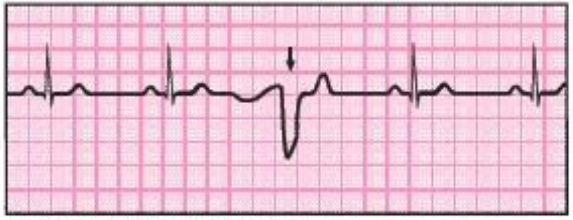
(b) Ventricular fibrillation



(c) Atrial fibrillation




(d) Heart block



(e) Premature ventricular contraction

**FIGURE 19.17 Normal and Pathological Electrocardiograms.** (a) Normal sinus rhythm. (b) Ventricular fibrillation, with grossly irregular waves of depolarization, as seen in a heart attack (myocardial infarction). (c) Atrial fibrillation; between heartbeats, the atria exhibit weak, chaotic, high-frequency depolarizations instead of normal P waves. (d) Heart block, in which some atrial depolarizations (P waves) are not conducted to the ventricles and not followed by ventricular QRS waves. (e) Premature ventricular contraction, or extrasystole (at arrow); note the absence of a P wave, the inverted QRS complex, and the misshapen QRS and elevated T.

l-Farabi Kazakh National University
Higher School of Medicine

# Circulatory System

## -Heart

**Blood Flow, Heart Sounds and the Cardiac Cycle + Case**

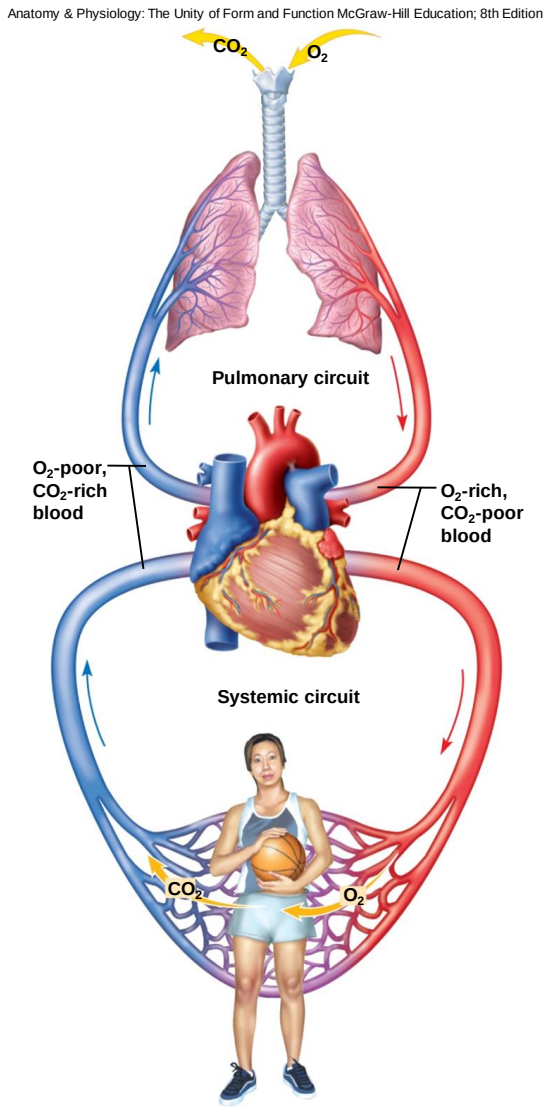
# ◆ PART V

# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- ***Explain why blood pressure is expressed in millimeters of mercury;***
- ***describe how changes in blood pressure operate the heart valves;***
- ***explain what causes the sounds of the heartbeat;***
- ***describe in detail one complete cycle of heart contraction and relaxation;***
- ***relate the events of the cardiac cycle to the volume of blood entering and leaving the heart.***

# Cardiovascular System Circuit




- left side of heart
  - fully oxygenated blood arrives from lungs via pulmonary veins
  - blood sent to all organs of the body via aorta

- right side of heart
  - lesser oxygenated blood arrives from inferior and superior vena cava
  - blood sent to lungs via pulmonary trunk

**Are the lungs supplied by the pulmonary circuit, the systemic circuit, or both? Explain**

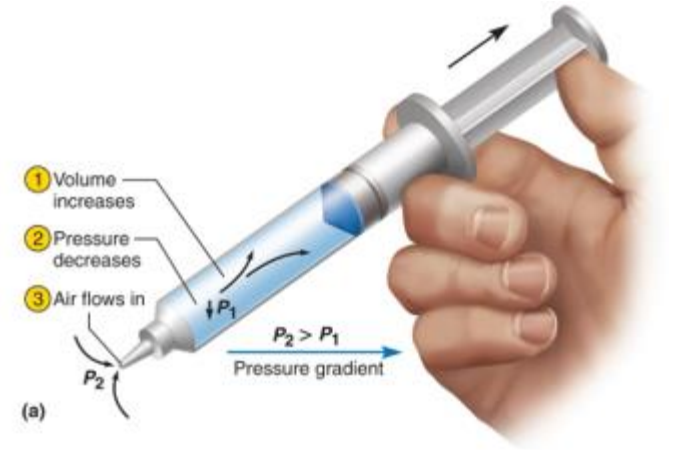
# Cardiac Cycle

- **cardiac cycle** - one complete contraction and relaxation of all four chambers of the heart
- atrial **systole** (contraction) occurs while ventricles are in **diastole** (relaxation)
- atrial **diastole** occurs while ventricles in **systole**
- **quiescent period** all four chambers relaxed at same time

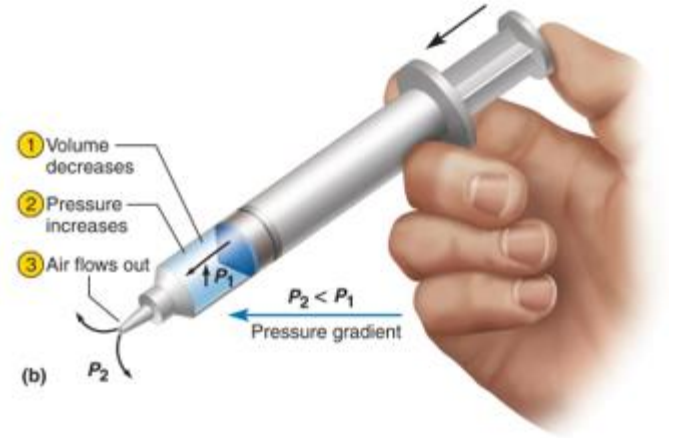
# Principles of Pressure and Flow

- **pressure -** causes a fluid to flow (fluid dynamics)
  - pressure gradient - pressure difference between two points
  - measured in mm Hg with a manometer or sphygmomanometer

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition



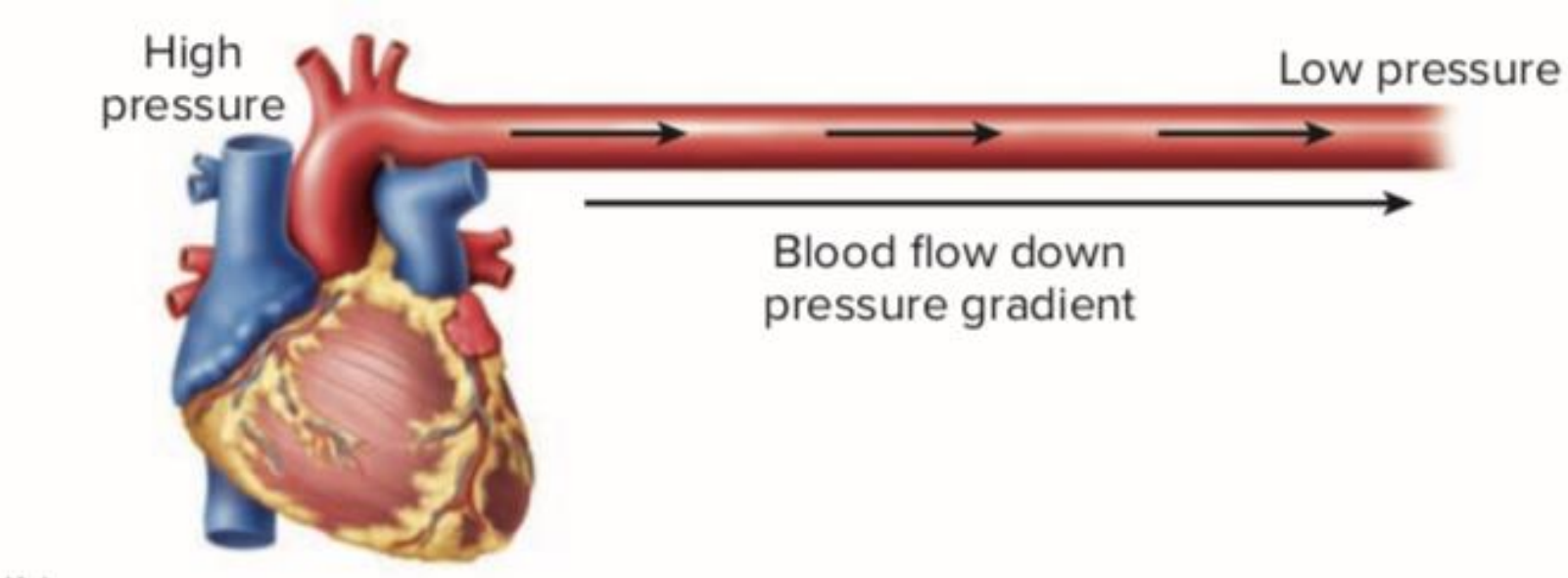

- **resistance -** opposes fluid flow
  - great vessels have positive blood pressure
  - ventricular pressure must rise above this resistance for blood to flow into great vessels



## *how does pressure affect blood flow?*

# Pressure Gradients and Flow

- fluid flows only if it is subjected to more pressure at one point than another which creates a **pressure gradient**
  - fluid flows **down its pressure gradient** from high pressure to low pressure

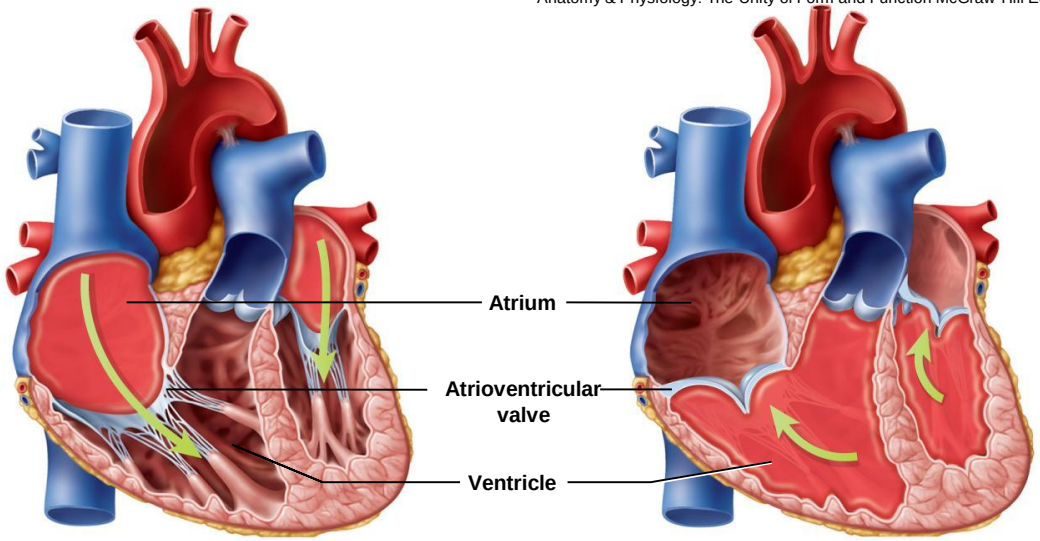


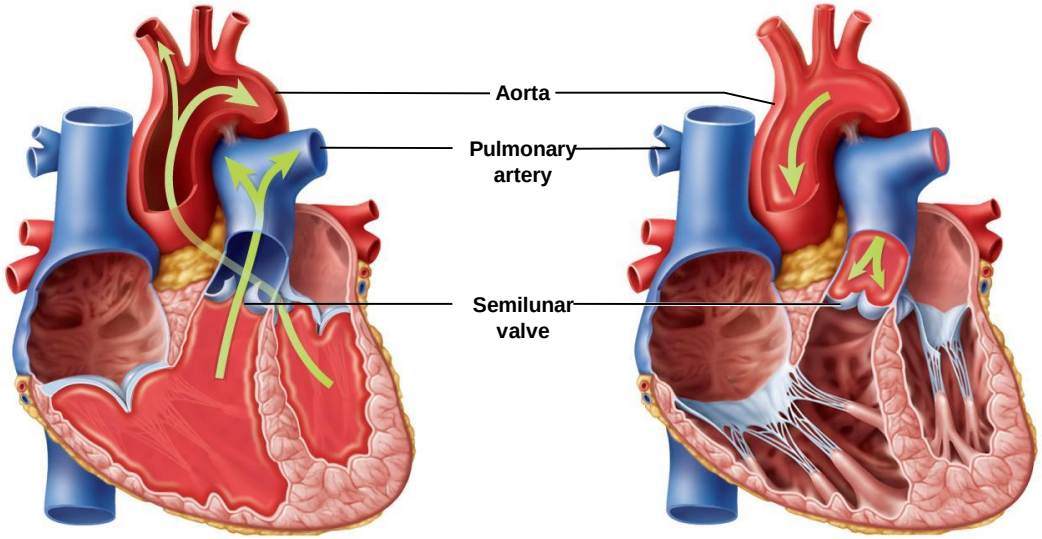
# Pressure Gradients and Flow

- events occurring on left side of heart
  - when ventricle relaxes and expands, its internal pressure falls
  - if bicuspid valve is open, blood flows into left ventricle
  - when ventricle contracts, internal pressure rises
  - AV valves close and the aortic valve is pushed open and blood flows into aorta from left ventricle
- opening and closing of valves are governed by these pressure changes
  - AV valves limp when ventricles relaxed
  - semilunar valves under pressure from blood in vessels when ventricles relaxed

# Operation of Heart Valves

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition




**(a)The atrioventricular valves.** When atrial pressure is greater than ventricular pressure, the valve opens and blood flows through (green arrows). When ventricular pressure rises above atrial pressure, the blood in the ventricle pushes the valve cusps closed.

**(b)The semilunar valves.** When the pressure in the ventricles is greater than the pressure in the great arteries, the semilunar valves are forced open and blood is ejected. When ventricular pressure is lower than arterial pressure, arterial blood holds these valves closed.

## DEEPER INSIGHT 19.3

## CLINICAL APPLICATION

### *Valvular Insufficiency*

*Valvular insufficiency (incompetence)* **refers to any failure of a valve to prevent** *reflux* **(regurgitation)—the backward flow of blood.** *Valvular stenosis*[26] **is a form of insufficiency in which the cusps are stiffened and the opening is constricted by scar tissue. It frequently results from rheumatic fever, an autoimmune disease in which antibodies produced to fight a bacterial infection also attack the mitral and aortic valves. As the valves become scarred and constricted, the heart is overworked by the effort to force blood through the openings and may become enlarged. Regurgitation of blood through the incompetent valves creates turbulence that can be heard with a stethoscope as a** *heart murmur.*

## DEEPER INSIGHT 19.3

## CLINICAL APPLICATION

### *Valvular Insufficiency*

*Mitral valve prolapse (MVP)* **is an insufficiency in which one or both mitral valve cusps bulge into the atrium during ventricular contraction. It is often hereditary and affects about 1 out of 40 people, especially young women. In many cases, it causes no serious dysfunction, but in some people it causes chest pain, fatigue, and shortness of breath.**

**In some cases, an incompetent valve can eventually lead to heart failure. A defective valve can be surgically repaired or replaced with an artificial valve or a valve transplanted from a pig heart.**

▶▶▶APPLY WHAT YOU KNOW **How would aortic valvular stenosis affect the amount of blood pumped into the aorta? How might this affect a person's physical stamina? Explain your reasoning.**

***Valvular stenosis reduces cardiac output because the valvular orifice is narrowed and less blood flows through it. Because of reduced cardiac output, organs on the systemic circuit receive less perfusion. Among other effects, a person's physical stamina is compromised because the muscles are not as well supplied with oxygen.***

## *how are heart sounds produced?*

# Heart Sounds

- **auscultation** - listening to sounds made by body
- **first heart sound** ($S_1$), louder and longer “lubb”, occurs with closure of AV valves, turbulence in the bloodstream, and movements of the heart wall
- **second heart sound** ($S_2$), softer and sharper “dupp” occurs with closure of semilunar valves, turbulence in the bloodstream, and movements of the heart wall
- $S_3$ - rarely heard in people over 30
- exact cause of each sound is not known with certainty

# Phases of Cardiac Cycle

- ventricular filling
- isovolumetric contraction
- ventricular ejection
- isovolumetric relaxation
- all the events in the cardiac cycle are completed in less than one second!

# Ventricular Filling

- during diastole, ventricles expand
  - their pressure drops below that of the atria
  - AV valves open and blood flows into the ventricles

- **ventricular filling** occurs in three phases:
  - **rapid ventricular filling** - first one-third
    - blood enters very quickly
  - **diastasis** - second one-third
    - marked by slower filling
    - P wave occurs at the end of diastasis
  - **atrial systole** - final one-third
    - atria contract

- end-diastolic volume (EDV) – amount of blood contained in each ventricle at the end of ventricular filling
  - **130 mL of blood**

# Isovolumetric Contraction

- **atria repolarize** and relax
  - remain in diastole for the rest of the cardiac cycle
- **ventricles depolarize**, create the QRS complex, and begin to contract
- **AV valves close** as ventricular blood surges back against the cusps
- **heart sound $S_1$** occurs at the beginning of this phase
- '**isovolumetric**' [***iso* = same; *volum* =volume; *metr* = measure** ] because even though the ventricles contract, they do not eject blood
  - because pressure in the aorta (80 mm Hg) and in pulmonary trunk (10 mm Hg) is still greater than in the ventricles
- cardiocytes exert force, but with all four valves closed, the blood cannot go anywhere

# Ventricular Ejection

- ejection of blood begins when the ventricular pressure exceeds arterial pressure and forces semilunar valves open
  - pressure peaks in left ventricle at about **120 mm Hg and 25 mm** Hg in the right
- blood spurts out of each ventricle rapidly at first – **rapid ejection**
- then more slowly under reduced pressure – **reduced ejection**
- ventricular ejections last about 200 – 250 msec
  - corresponds to **the plateau phase of the cardiac action potential**
- **T wave** occurs late in this phase
- **stroke volume (SV)** of about **70 mL** of blood is ejected of the 130 mL in each ventricle
  - **ejection fraction** of about 54%
  - as high as 90% in vigorous exercise
- **end-systolic volume (ESV)** – the **60 mL** of blood left behind

# Isovolumetric Relaxation

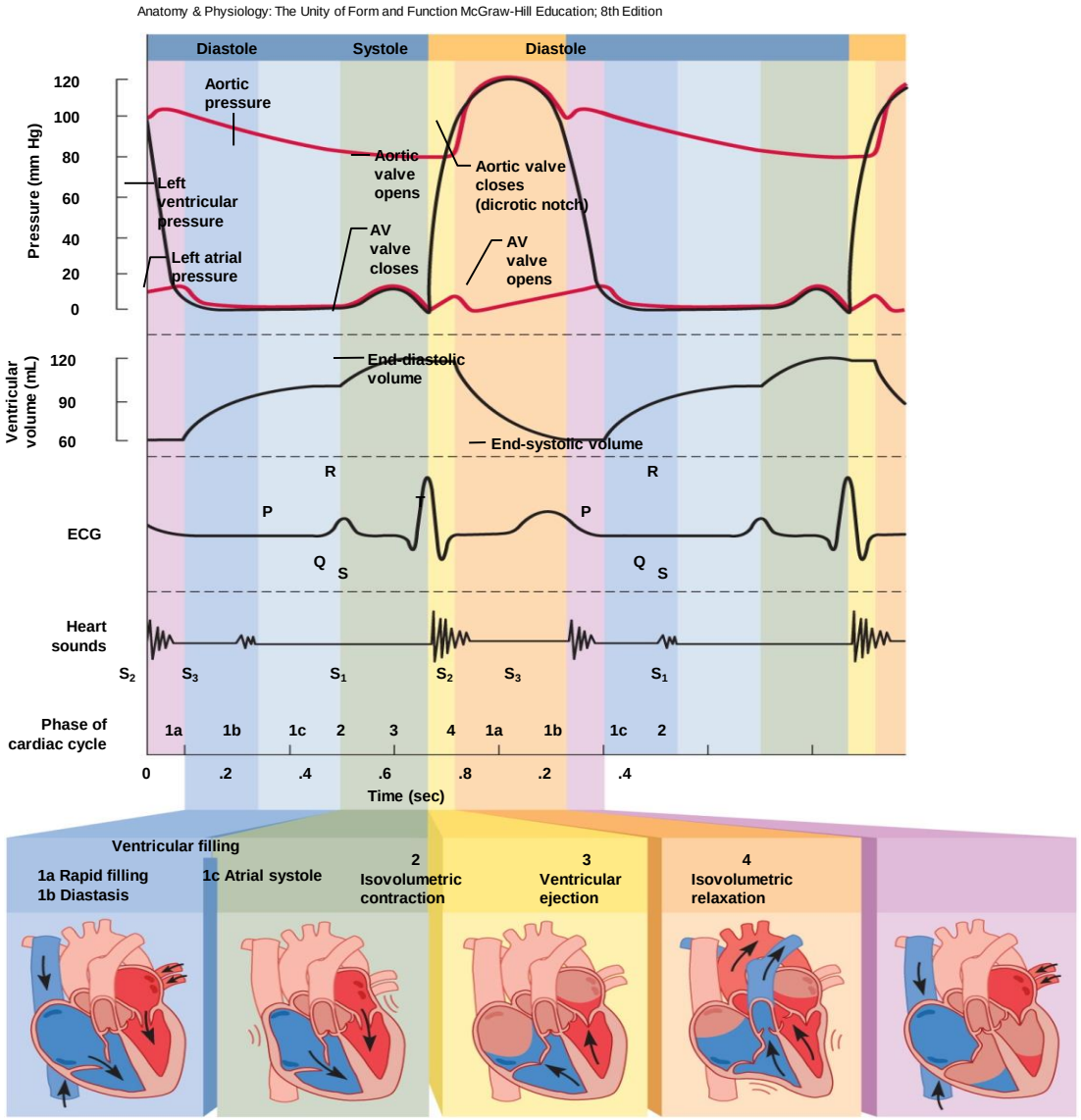
- early **ventricular diastole**
  - when **T wave ends** and **the ventricles begin to expand**
- elastic recoil and expansion would cause pressure to drop rapidly and suck blood into the ventricles
  - blood from the aorta and pulmonary briefly **flows backwards**
  - **filling the semilunar valves** and **closing the cusps**
  - creates a **slight pressure rebound** that appears as the **dicrotic notch** of the aortic pressure curve
  - **heart sound $S_2$** occurs as blood rebounds from the closed semilunar valves and the ventricle expands
  - '**isovolumetric**' because semilunar valves are closed and AV valves have not yet opened
    - ventricles are therefore taking in no blood
  - when AV valves open, ventricular filling begins again

# Timing of Cardiac Cycle

- in a resting person
  - **atrial systole** last about **0.1 sec**
  - **ventricular systole** about **0.3 sec**
  - **quiescent period**, when all four chambers are in diastole, **0.4 sec**

- **total duration of the cardiac cycle** is therefore **0.8 sec** in a heart beating **75 bpm**

# Major Events of Cardiac Cycle




- ventricular filling
- isovolumetric contraction
- ventricular ejection
- isovolumetric relaxation

Figure 19.20

# Overview of Volume Changes

**end-systolic volume (ESV)** **60 ml**

-passively added to ventricle during atrial diastole +30 ml

-added by atrial systole +40 ml

total: **end-diastolic volume (EDV)** **130 ml**

---

**stroke volume (SV)** ejected by ventricular systole **-70 ml**

leaves: **end-systolic volume (ESV)** **60 ml**

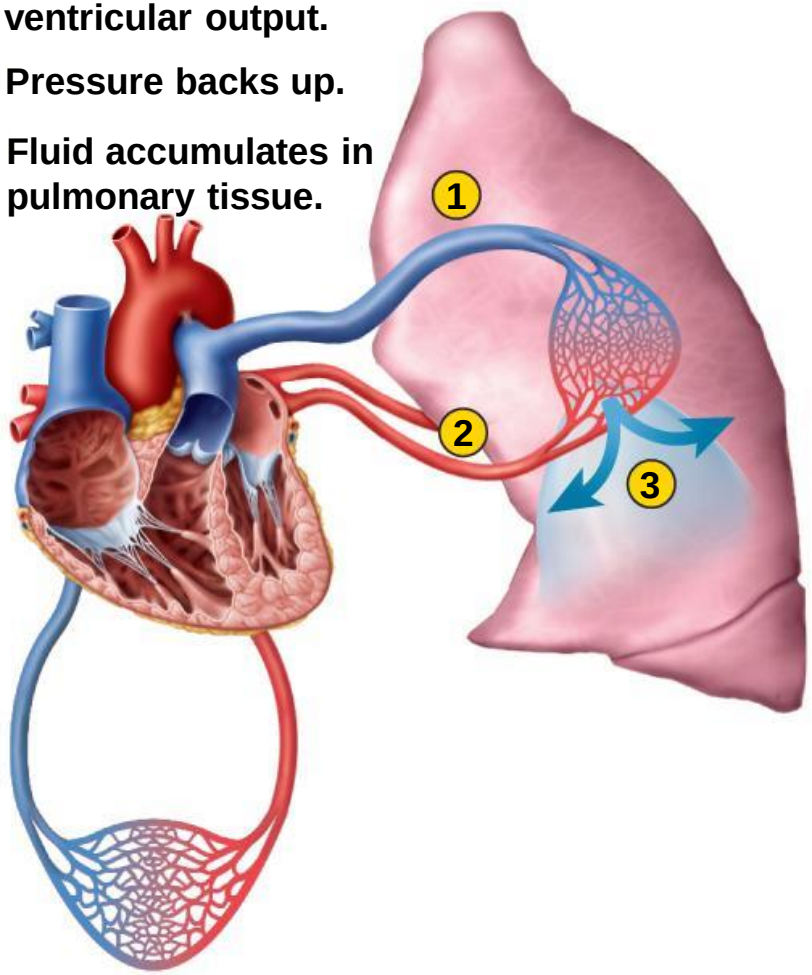
both ventricles must eject same amount of blood

---

# Unbalanced Ventricular Output

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

1. Right ventricular output exceeds left ventricular output.
2. Pressure backs up.
3. Fluid accumulates in pulmonary tissue.




(a) Pulmonary edema

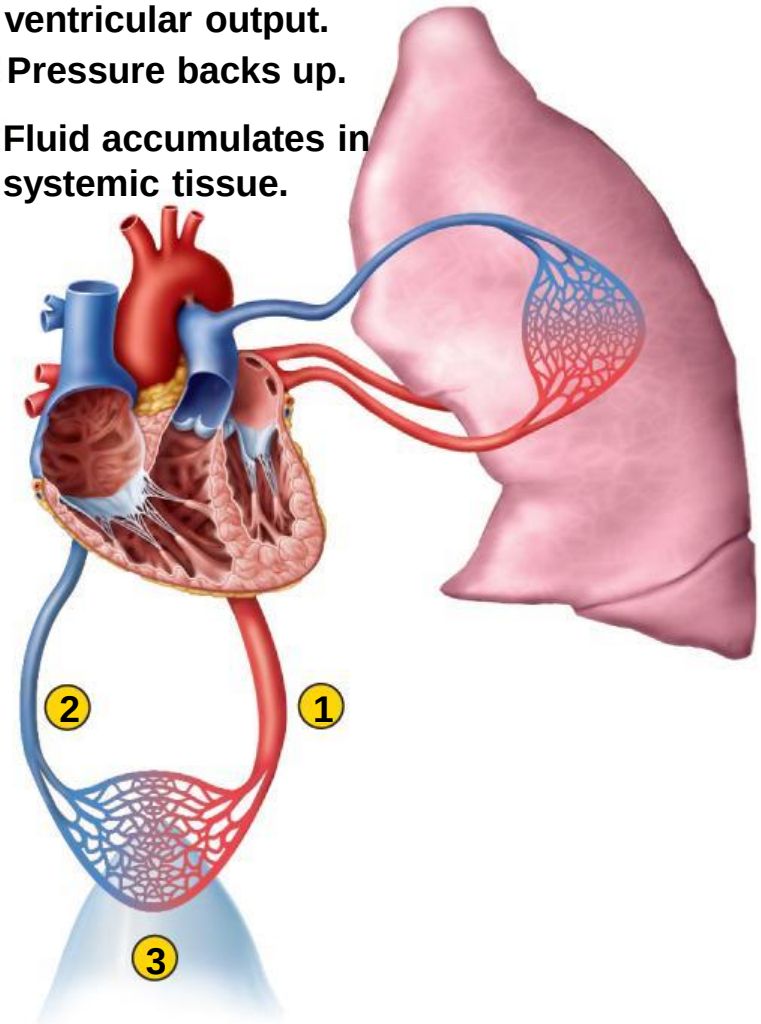
pulmonary edema

Figure 19.21a

# Unbalanced Ventricular Output

Anatomy & Physiology: The Unity of Form and Function McGraw-Hill Education; 8th Edition

1. Left ventricular output exceeds right ventricular output.
2. Pressure backs up.
3. Fluid accumulates in systemic tissue.



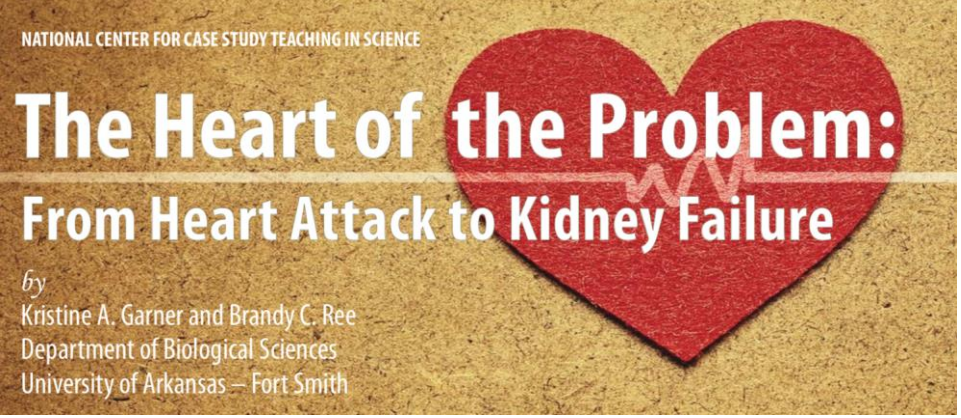

(b) Systemic edema

peripheral edema

Figure 19.21b

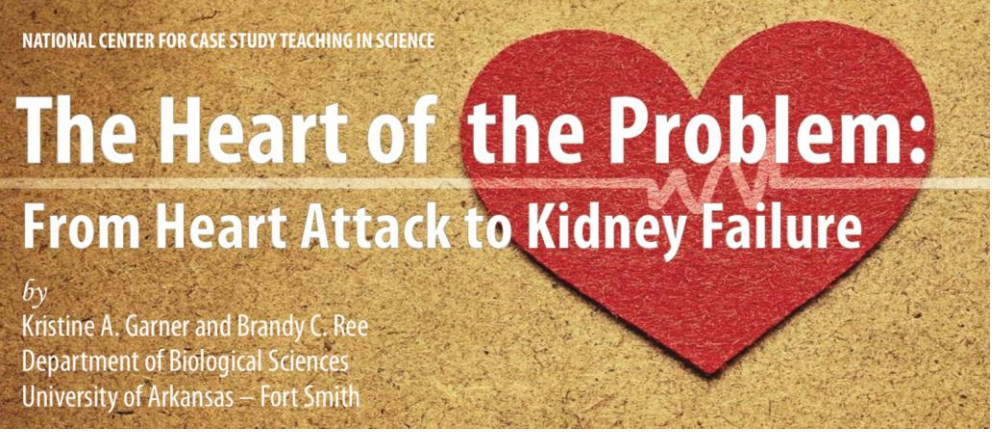
# Congestive Heart Failure

- **congestive heart failure** (CHF) - results from the failure of either ventricle to eject blood effectively
  - usually due to a heart weakened by myocardial infarction, chronic hypertension, valvular insufficiency, or congenital defects in heart structure.
- **left ventricular failure** – blood backs up into the lungs causing pulmonary edema
  - shortness of breath or sense of suffocation
- **right ventricular failure** – blood backs up in the vena cava causing systemic or generalized edema
  - enlargement of the liver, ascites (pooling of fluid in abdominal cavity), distension of jugular veins, swelling of the fingers, ankles, and feet
- eventually leads to total heart failure




## Part I – Emergency

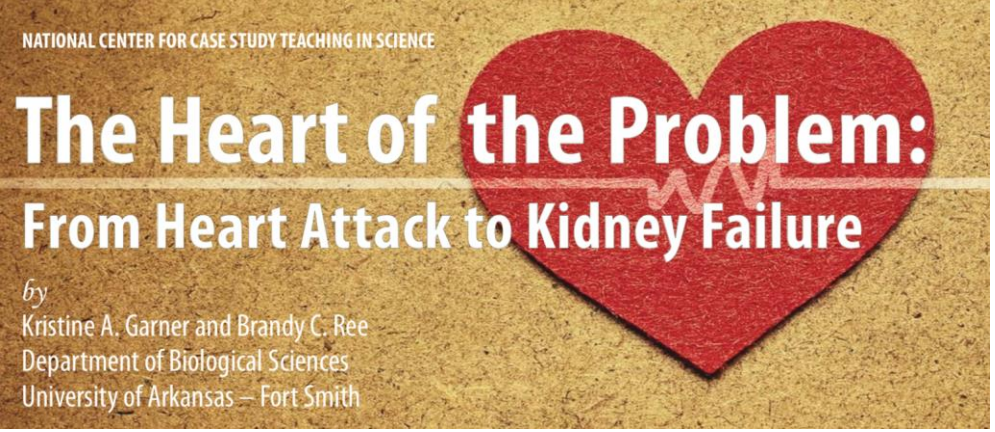
Mrs. Helms came in through the front door of her house with an armful of groceries. She put the bag down on the kitchen counter and called to her husband. "Herb, I'm home! Are you ready for lunch?" She didn't get an answer, so she walked to the living room and found Mr. Helms lying on the floor. "Herb! Are you okay?" she asked as she grabbed his shoulder. Mr. Helms responded weakly while clutching his chest. Mrs. Helms frantically called 911. It only took EMS a few minutes to arrive and the paramedics transported Mr. Helms to the hospital. Upon admission to the hospital, Mr. Helms' vital signs were recorded as follows:




| | *Mr. Helms* | *Normal* |
|---|---|---|
| Systolic blood pressure (mm Hg) | 90 | 120 |
| Diastolic blood pressure (mm Hg) | 52 | 80 |
| Oral temperature (°F) | 98.9 | 97.8 to 99.1 |
| Heart rate (beats per minute) | 120, irregular | 60–80 |
| Respiratory rate (breaths per minute) | 33, labored | 12 to 20 |
| Oxygen saturation | 89% | 95–100% |

## *Questions*

**Which of Mr. Helms' vital signs and lab values were abnormal? In other words, what other information would be useful?**

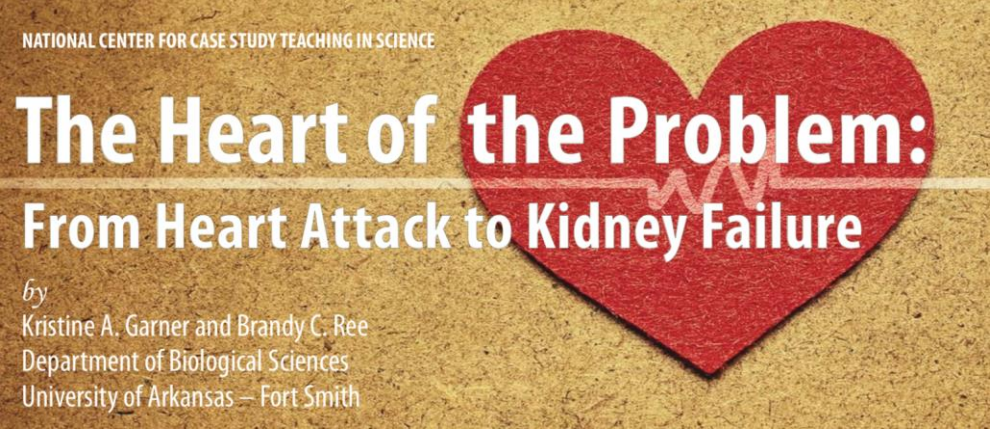



***Part II – Cardiac Involvement***

Mr. Helms was admitted to the hospital with chest pains and shortness of breath. His wife was panicked since her 72 year-old husband had a history of heart disease. After examination and an echocardiogram, Dr. Collins spoke with Mrs. Helms. "I'm very sorry, but your husband has had another heart attack resulting in valve failure. A papillary muscle that controls a valve in his heart has been severely damaged and is no longer working."

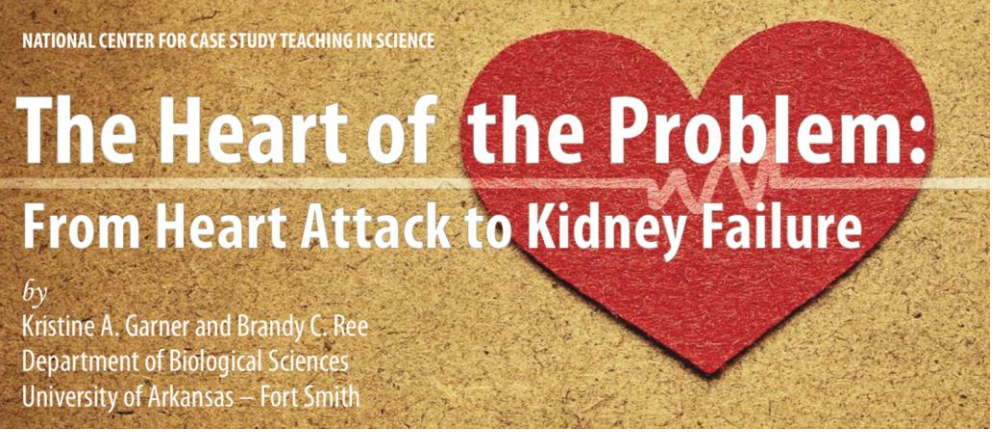
***Questions***

***What is the purpose of blood flow?***



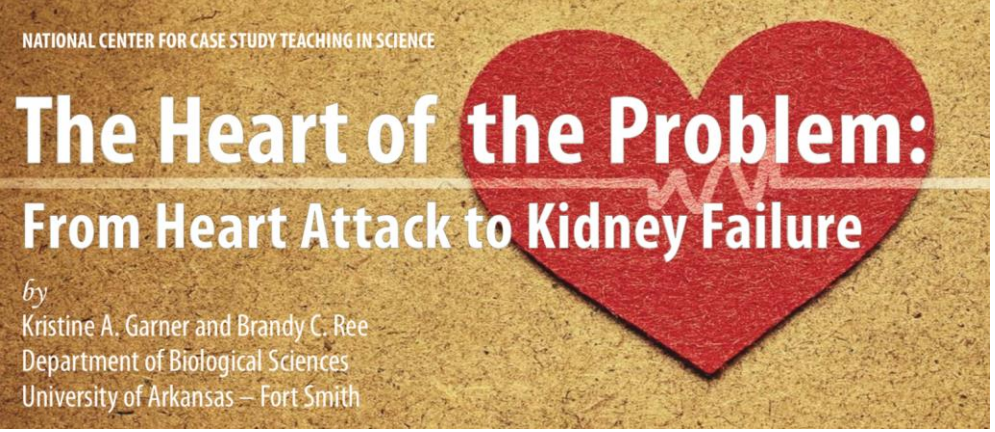

***Questions***

***Describe blood flow through the heart starting with blood entering the right side of the heart and including all chambers and valves.***



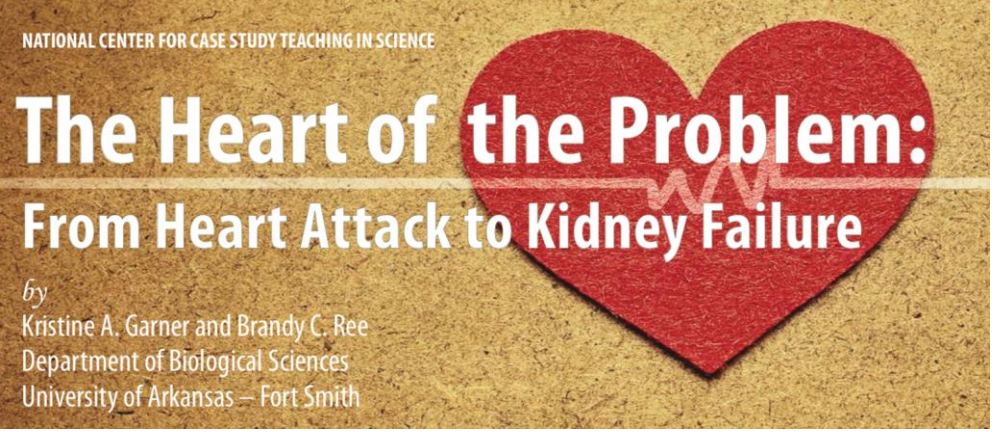

***Questions***

***What is the function of heart valves?***



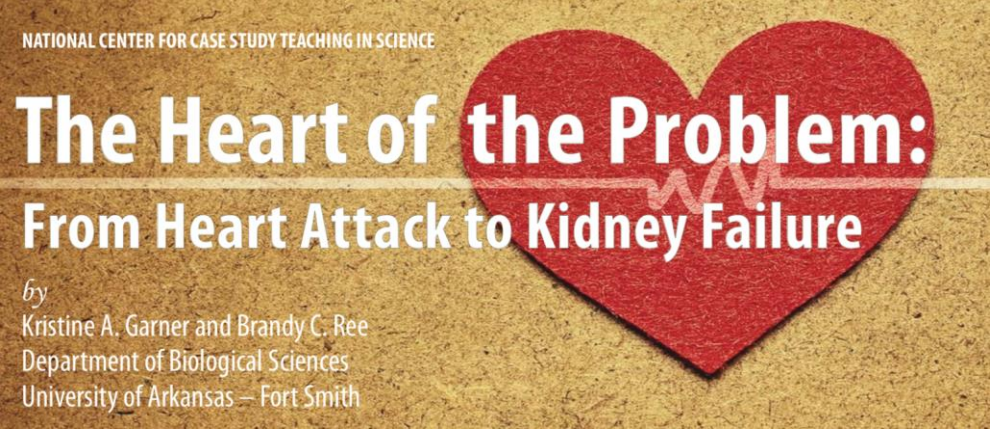

***Questions***

***What is the function of papillary muscles***



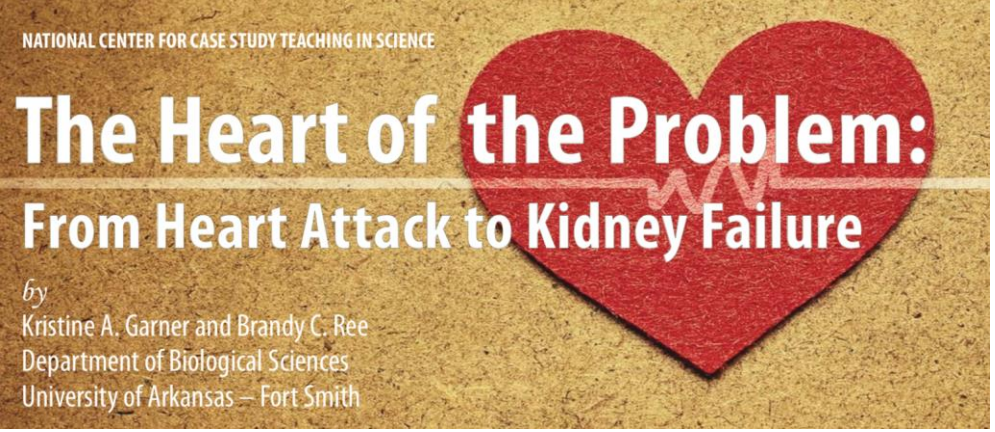

## Part III – Cardiovascular Involvement

Dr. Collins called Nurse Nan from the patient's room and confided, "Mr. Helms is in bad shape. His left posteromedial papillary muscle was damaged from his heart attack. The papillary muscle is no longer able to maintain closure of the valve, and this has resulted in mitral valve prolapse. With decreasing cardiac output, this patient is in for a fight for his life." Nurse Nan knew that maintaining cardiac output was necessary for adequate blood flow through the body. As Dr. Collins walked away, Nurse Nan composed herself to tell Mrs. Helms the bad news and returned to the patient's room. Nurse Nan explained to Mrs. Helms that her husband had left-sided heart failure and that his blood pressure was slowly and steadily decreasing.



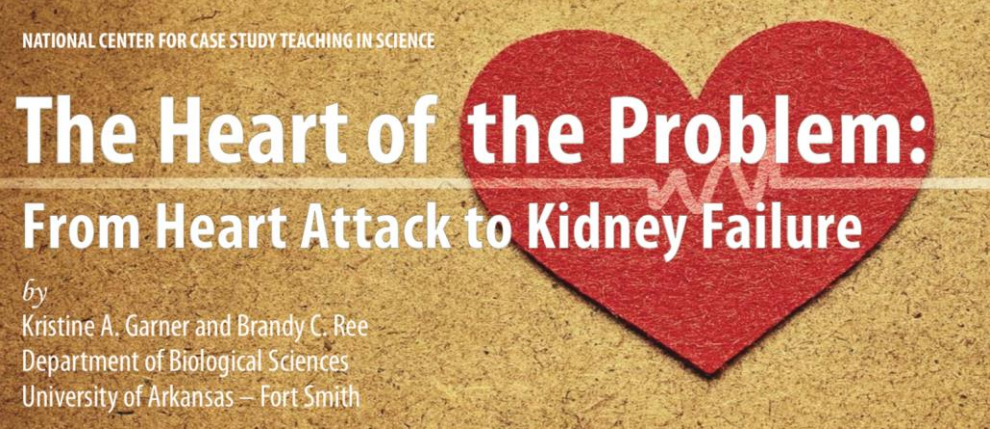

***Questions***

***In general, how is the direction of blood flow disrupted because of mitral valve prolapse?***



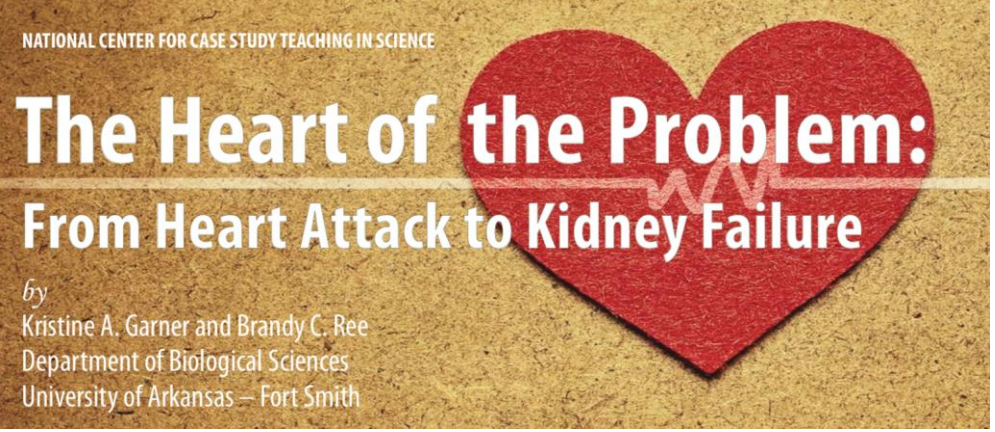

## *Questions*

***Does the mitral valve prolapse increase, decrease, or not change stroke volume (the amount of blood exiting the ventricle with each ventricular contraction)?***



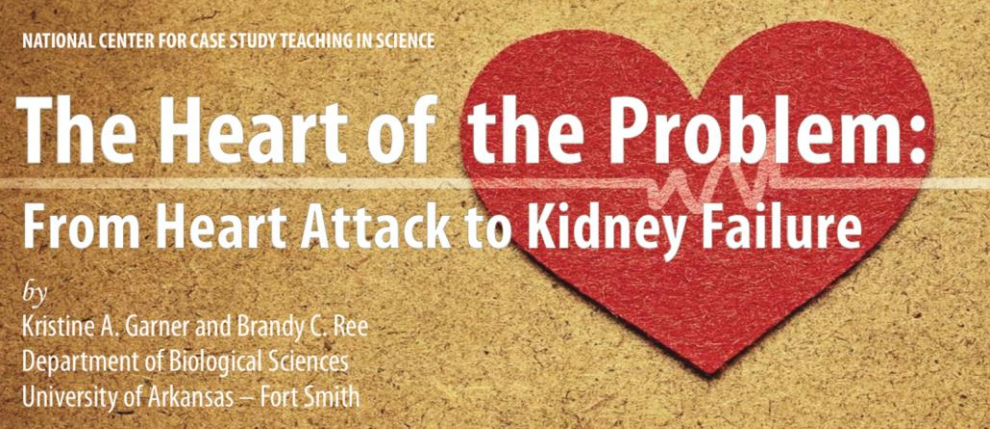

## *Questions*

***How does mitral valve prolapse decrease cardiac output (the amount of blood exiting the ventricle per minute)***




## *Questions*

***Explain how cardiac output determines blood pressure***




## *Questions*

***Why is Mr. Helms' heart rate higher than normal?***

Al-Farabi Kazakh National University
Higher School of Medicine

# Circulatory System

## -Heart

**Cardiac Output**

# ◆ PART VI

# LEARNING OUTCOMES

**As a result of the lesson you will be able to:**

- ***Define cardiac output and explain its importance;***
- ***Identify the factors that govern cardiac output;***
- ***Discuss some of the nervous and chemical factors that alter heart rate, stroke volume, and cardiac output;***
- ***Explain how the right and left ventricles achieve balanced output;***
- ***Describe some effects of exercise on cardiac output.***

# Cardiac Output (CO)

- **cardiac output** (CO) – the amount ejected by ventricle in 1 minute
- cardiac output is related to the quantity of blood delivered to various parts of the body, it is an important indicator of how efficiently the heart can meet the body's demands for perfusion.
- The function of the heart is to drive blood through the circulatory system in a cycle that delivers oxygen, nutrients and chemicals to the body's cells and removes cellular waste.
- **cardiac output = heart rate x stroke volume**
  - about **4 to 6 L/min at rest**

# Cardiac Output (CO)

- **cardiac reserve** – the difference between a person's maximum and resting CO
  - increases with fitness, decreases with disease

- to keep cardiac output constant as we increase in age, the heart rate increases as the stroke volume decreases

  - a RBC leaving the left ventricle will arrive back at the left ventricle in abo 1 minute
  - vigorous exercise increases CO to 21 L/min for fit person and up to 35 L/min for world class athlete

# Heart Rate

- **pulse** – surge of pressure produced by each heart beat that can be felt by palpating a superficial artery with the fingertips
- **the radial artery in the wrist or carotid artery in the neck.**

  - infants have HR of 120 bpm or more
  - young adult females avg. 72 - 80 bpm
  - young adult males avg. 64 to 72 bpm
  - heart rate rises again in the elderly

# Heart Rate

- **tachycardia** [*tachy* **= speed, fast;** *card* **= heart;** *ia* **= condition** ]- resting adult heart rate above 100 bpm
  - stress, anxiety, drugs, heart disease, or fever
  - loss of blood or damage to myocardium

- **bradycardia[***brady* **= slow;** *card* **= heart;** *ia* **= condition ]** - resting adult heart rate of less than 60 bpm
  - in sleep, low body temperature, and endurance trained athletes

- **positive chronotropic[***chrono* **= time;** *trop* **= to change, to influence ] agents** – factors that raise the heart rate

- **negative chronotropic agents** – factors that lower heart rate

**Why does the heart have a nerve supply, since it continues to beat even without one?**

# Chronotropic Effects of the Autonomic Nervous System

- The **autonomic[***auto* = self; *nom* = rule (self-governed) **] nervous system (ANS)** can be defined as a motor nervous system that controls glands, cardiac muscle, and smooth muscle.
- autonomic nervous system does not initiate the heartbeat, it modulates rhythm and force.

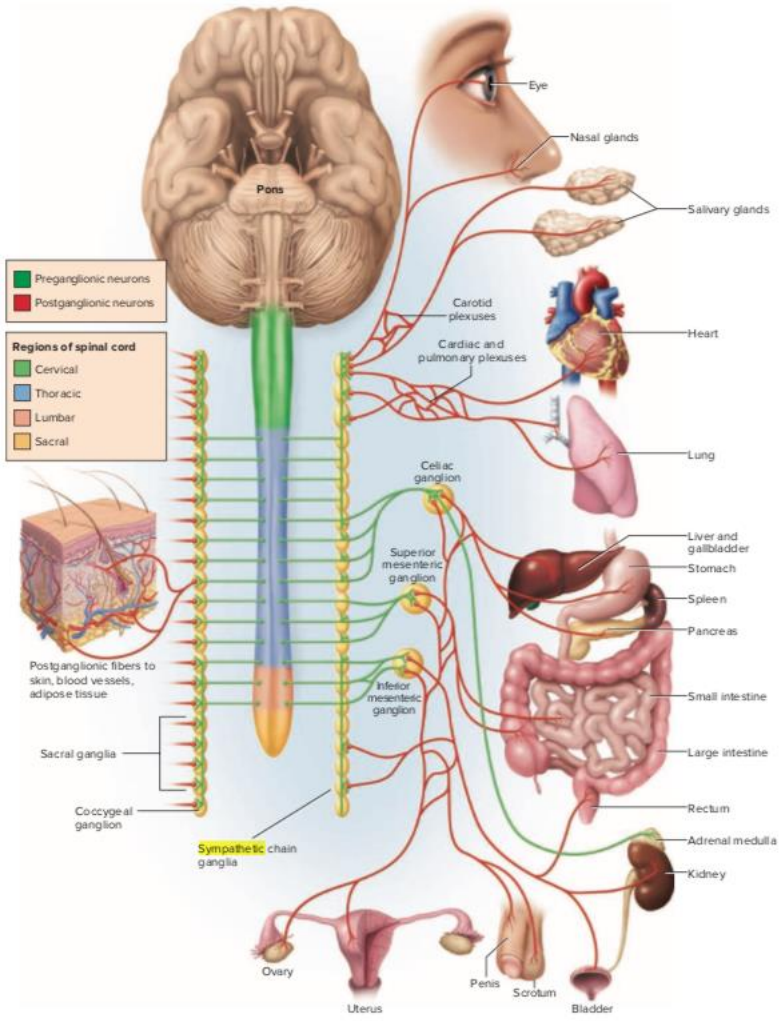
- **cardiac centers** in the **reticular formation of the medulla oblongata** initiate autonomic output to the heart

- **cardiostimulatory effect** – some neurons of the cardiac center transmit signals to the heart by way of **sympathetic** pathways

- **cardioinhibitory effect** – others transmit **parasympathetic** signals by way of the **vagus nerve**

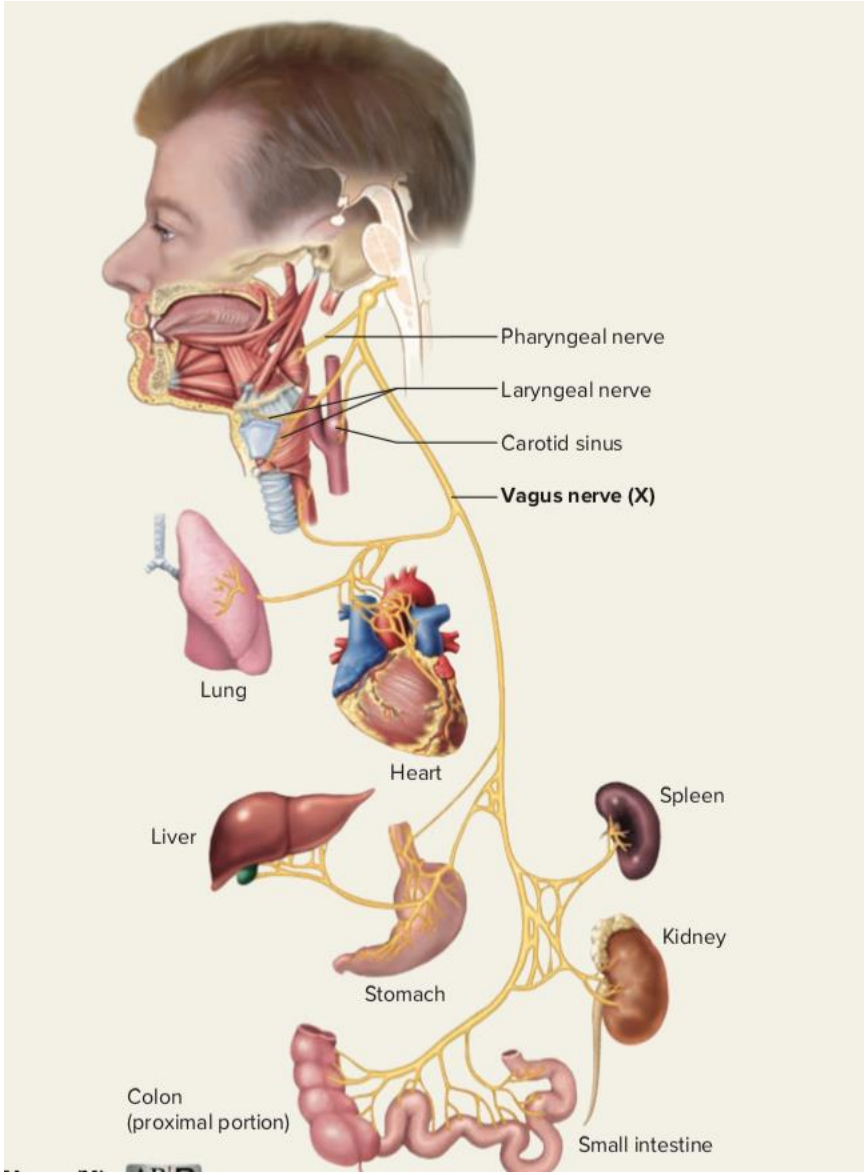
# Nerve Supply to Heart

- **sympathetic nerves (raise heart rate)**
  - sympathetic pathway to the heart originates in the **lower cervical** to **upper thoracic segments** of the spinal cord
  - continues to adjacent **sympathetic chain ganglia**
  - some pass through **cardiac plexus** in mediastinum
  - A **plexus** (from the Latin for "braid") is a branching network of the vessels or nerves.
  - continue as **cardiac nerves** to the heart
  - fibers terminate in SA and AV nodes, in atrial and ventricular myocardium, as well as the aorta, pulmonary trunk, and coronary arteries
    - increase heart rate and contraction strength
    - dilates coronary arteries to increase myocardial blood flow



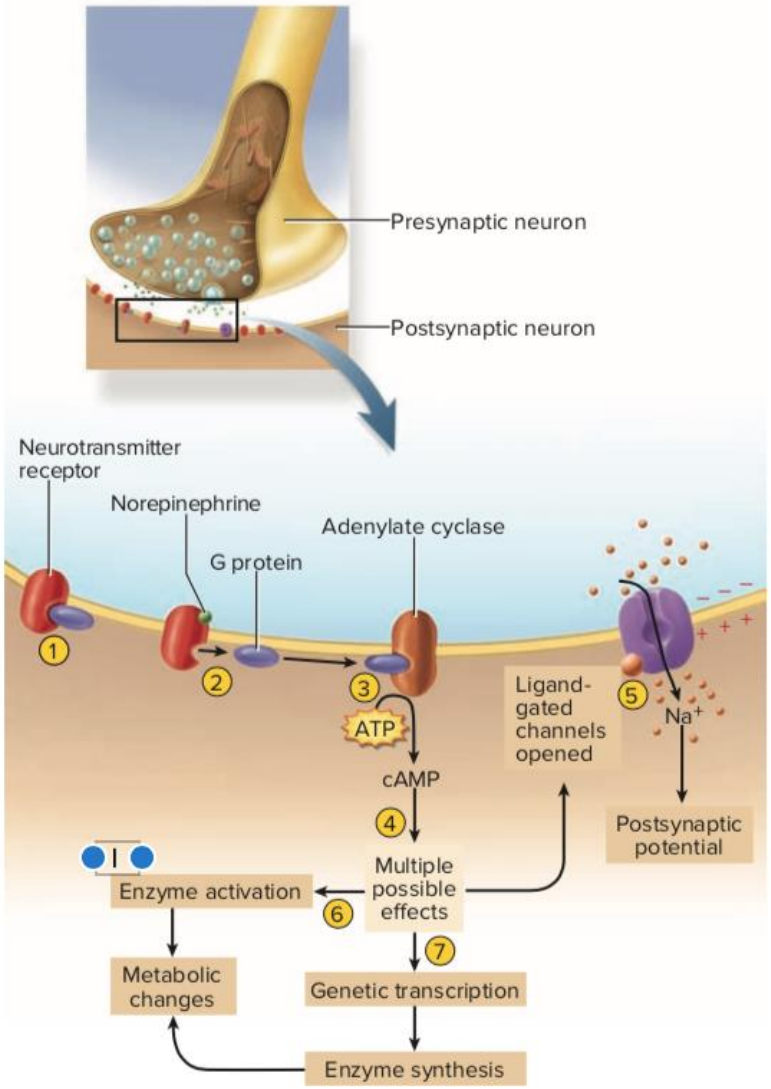
# Nerve Supply to Heart

- **parasympathetic nerves (slows heart rate)**
  - pathway begins with **nuclei of the vagus nerves** in the **medulla oblongata**
  - extend to **cardiac plexus** and continue to the heart by way of the **cardiac nerves**
  - fibers of **right vagus nerve** lead to the **SA node**
  - fibers of **left vagus nerve** lead to the **AV node**
  - little or no vagal stimulation of the myocardium
    - parasympathetic stimulation reduces the heart rate



# Chronotropic Effects of the Autonomic Nervous System

- **sympathetic** postganglionic fibers are adrenergic
  - they release **norepinephrine**
  - binds to **β-adrenergic fibers** in the heart
  - activates **c-AMP second-messenger system** in cardiocytes and nodal cells
  - leads to **opening of $Ca^{2+}$ channels** in plasma membrane
  - increased $Ca^{2+}$ inflow **accelerated depolarization of SA node**
  - **cAMP accelerates the uptake of $Ca^{2+}$ by the sarcoplasmic reticulum** allowing the cardiocytes to **relax more quickly**
  - by accelerating both contraction and relaxation, norepinephrine and cAMP increase the heart rate as high as 230 bpm
  - diastole becomes too brief for adequate filling
  - both stroke volume and cardiac output are reduced




**adrenergic synapses** do have an advantage—**signal amplification.**

An **adrenergic synapse** employs the neurotransmitter norepineph- rine (NE), also called noradrenaline. NE, other monoamines, and neuropeptides act through second-messenger systems such as cyclic AMP (cAMP).

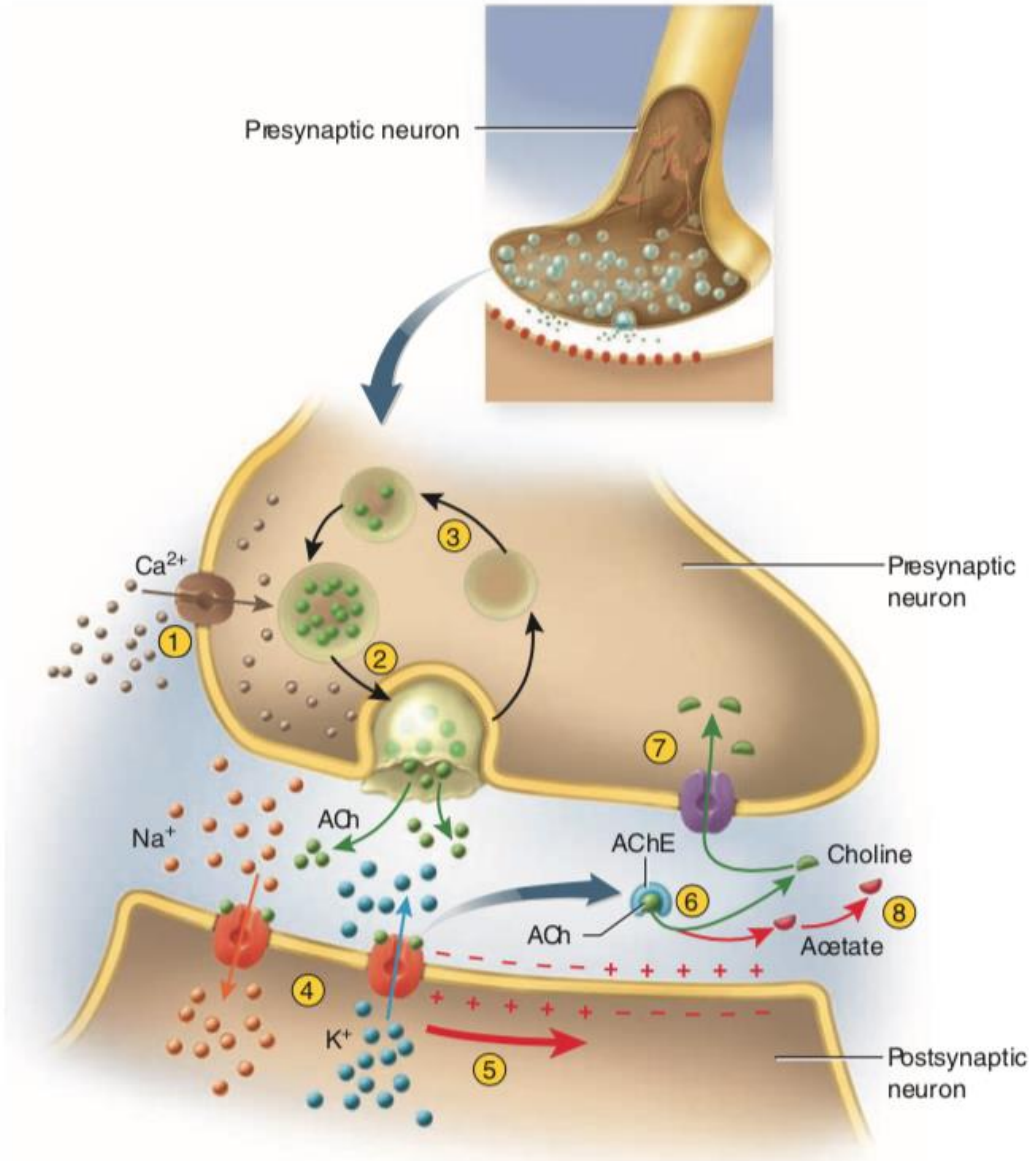
A single NE molecule binding to a receptor can induce the formation of many cAMPs, each of those can activate many enzyme molecules or induce the transcription of a gene to generate numerous mRNA molecules, and each of those can result in the production of a vast number of enzyme molecules and metabolic products such as glucose molecules.

# Chronotropic Effects of the Autonomic Nervous System

- **parasympathetic vagus nerves** have cholinergic, inhibitory effects on the SA and AV nodes
  - **acetylcholine (ACh)** binds to **muscarinic receptors**
  - **opens $K^+$ gates** in the nodal cells
  - as $K^+$ leaves the cells, they become **hyperpolarized** and fire less frequently
  - **heart slows down**
  - parasympathetics work on the heart faster than sympathetics
    - parasympathetics do not need **a second messenger system**

- without influence from the cardiac centers, the heart has **a intrinsic "natural" firing rate of 100 bpm**

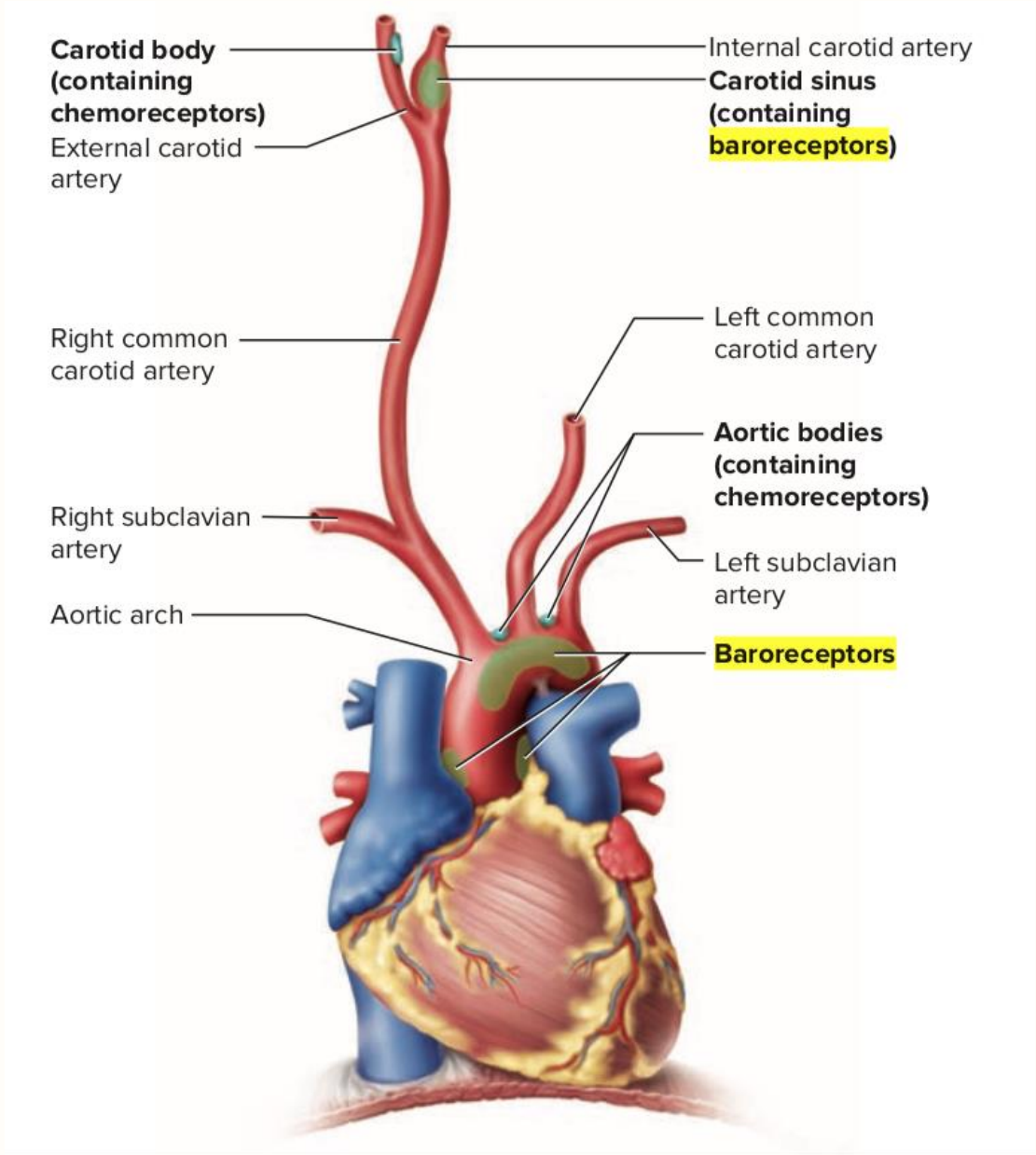
- **vagal tone** – holds down this heart rate to 70 – 80 bpm at rest
  - steady background firing rate of the **vagus nerves**



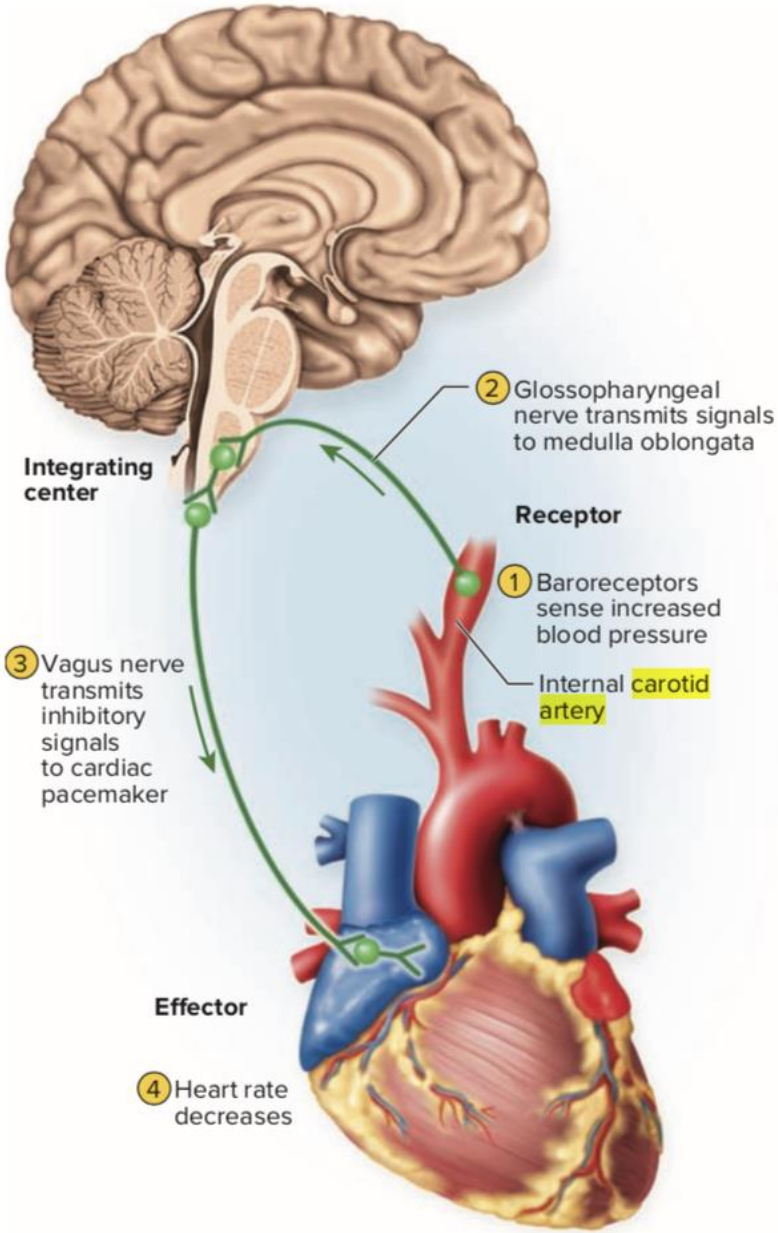

A **cholinergic**(CO-lin-UR-jic) synapse employs acetylcholine (ACh) as its neurotransmitter. ACh excites some postsynaptic cells (such as skeletal muscle) and inhibits others (such as cardiac muscle).

# Inputs to Cardiac Center

- **cardiac centers in the medulla** receive input from many sources and integrate it into the 'decision' to speed or slow the heart
- **higher brain centers** affect heart rate
  - **cerebral cortex**, **limbic system**, **hypothalamus**
    - sensory or emotional stimuli
- medulla also receives input from muscles, joints, arteries, and brainstem
  - **proprioceptors** in the muscles and joints
    - inform cardiac center about changes in activity, HR increases before metabolic demands of muscle arise
  - **baroreceptors** [*baro* **= pressure** ]signal cardiac center
    - pressure sensors in **aorta and internal carotid arteries**
    - blood pressure decreases, signal rate drops, cardiac center increases heart rate
    - if blood pressure increases, signal rate rises, cardiac center decreases heart rate


Carotid body (containing chemoreceptors)
External carotid artery
Internal carotid artery
Carotid sinus (containing baroreceptors)
Right common carotid artery
Left common carotid artery
Aortic bodies (containing chemoreceptors)
Right subclavian artery
Left subclavian artery
Aortic arch
Baroreceptors

To temporarily treat tachycardia and restore the normal resting sinus rhythm, a physician may massage a patient's carotid artery near the angle of the mandible. Propose a mechanism by which this treatment would have the desired effect.




high blood pressure activates a visceral *baro reflex*. It stimulates stretch receptors called *baroreceptors* in the internal carotid arteries and aorta, and they transmit signals via the glossopharyngeal nerves to the brainstem. The medulla integrates this with other information and transmits signals back to the heart by way of the vagus nerves. The vagus nerves slow down the heart and reduce blood pressure, thus completing a homeostatic negative feedback loop.

# Inputs to Cardiac Center

- **chemoreceptors**
  - in aortic arch, carotid arteries and medulla oblongata
  - sensitive to blood pH, $CO_2$ and $O_2$ levels
  - more important in respiratory control than cardiac control
    - if $CO_2$ accumulates in blood or CSF (hypercapnia), reacts with water and causes increase in $H^+$ levels
    - $H^+$ lowers the pH of the blood possibly creating acidosis (pH < 7.35)
  - hypercapnia and acidosis stimulate the cardiac center to increase heart rate
  - also respond to hypoxemia – oxygen deficiency in the blood
    - usually slows down the heart

- chemoreflexes and baroreflexes, responses to fluctuation in blood chemistry, are both negative feedback loops

# Chronotropic Chemicals

- chemicals affect heart rate as well as neurotransmitters from cardiac nerves
  - blood born adrenal catecholamines (NE and epinephrine) are potent cardiac stimulants

- **drugs** that stimulate heart
  - **nicotine** stimulates catecholamine secretion
  - **thyroid hormone** increases number adrenergic receptors on heart so more responsive to sympathetic stimulation
  - **caffeine** inhibits cAMP breakdown prolonging adrenergic effect

# Chronotropic Chemicals

- electrolytes
  - $K^+$ has greatest chronotropic effect
    - **hyperkalemia** – excess $K^+$ in cardiocytes
      - myocardium less excitable, heart rate slows and becomes irregular
    - **hypokalemia** – deficiency $K^+$ in cardiocytes
      - cells hyperpolarized, require increased stimulation
  - calcium
    - **hypercalcemia** – excess of $Ca^{2+}$
      - decreases heart rate and contraction strength
    - **hypocalcemia** – deficiency of $Ca^{2+}$
      - increases heart rate and contraction strength

# Stroke Volume (SV)

- the other factor that in cardiac output, besides heart rate, is **stroke volume**

- three variables govern stroke volume:
  1. preload
  2. contractility
  3. afterload
- example
  - increased preload or contractility causes increases stroke volume
  - increased afterload causes decrease stroke volume

**Suppose a person has a heart rate of 70 bpm and a stroke volume of 70 mL. A negative inotropic agent then reduces the stroke volume to 50 mL. What would the new heart rate have to be to maintain the same cardiac output?**

In the initial state, the cardiac output is (70 mL/beat)(70 beats/min) = 4,900 mL/min.

To maintain this output with a stroke volume of 50 mL would require a heart rate of (4,900 mL/min)/(50 mL/beat) = 98 beats/min.

# Preload

- **preload** – the amount of tension in ventricular myocardium immediately before it begins to contract
  - increased preload causes increased force of contraction
  - exercise increases venous return and stretches myocardium
  - cardiocytes generate more tension during contraction
  - increased cardiac output matches increased venous return

# Preload

- **Frank-Starling law of heart** - SV∝ EDV
  - stroke volume is proportional to the end diastolic volume
  - ventricles eject as much blood as they receive
  - the more they are stretched, the harder they contract

# Contractility

- **contractility** refers to how hard the myocardium contracts for a given preload.
- **It does not describe the increase in tension produced by stretching the muscle, but rather an increase caused by factors that make the cardiomyocytes more responsive to stimulation.**

# Contractility

- **positive inotropic[***ino* = fiber; *trop* = to change, to influence **]**
- **agents** increase contractility
- Calcium has a strong, positive inotropic effect—it increases the strength of each contraction of the heart.
- Calcium imbalances affect not only heart rate, but also contraction strength.
  - **hypercalcemia** can cause strong, prolonged contractions and even cardiac arrest in systole
- **Agents that affect calcium availability**
  - **catecholamines** increase calcium levels
  - **glucagon** (**The pancreatic hormone** )stimulates cAMP production
  - **digitalis (from the foxglove plant)**raises intracellular calcium levels and contraction strength

# Contractility

- **negative inotropic agents reduce contractility**
  - **hypocalcemia** can cause weak, irregular heartbeat and cardiac arrest in diastole
  - **hyperkalemia** reduces strength of myocardial action potentials and the release of $Ca^{2+}$ into the sarcoplasm
  - **vagus nerves** have effect on atria but too few nerves to ventricles for a significant effect

# Afterload

- **afterload** – the blood pressure in the aorta and pulmonary trunk immediately distal to the semilunar valves
  - opposes the opening of these valves
  - limits stroke volume
- **hypertension** increases afterload and opposes ventricular ejection
- anything that impedes arterial circulation can also increase afterload
  - lung diseases that restrict pulmonary circulation
  - **cor pulmonale** – right ventricular failure due to obstructed pulmonary circulation
    - in emphysema, chronic bronchitis, and black lung disease

# Exercise and Cardiac Output

- exercise makes the heart work harder and increases cardiac output
- **proprioceptors** signal cardiac center
  - at beginning of exercise, signals from joints and muscles reach the cardiac center of brain
  - sympathetic output from cardiac center increases cardiac output
- increased muscular activity increases venous return
  - increases preload and ultimately cardiac output
- increase in heart rate and stroke volume cause an increase in cardiac output
- exercise produces ventricular hypertrophy
  - increased stroke volume allows heart to beat more slowly at rest
  - athletes with increased cardiac reserve can tolerate more exertion than a sedentary person

# Coronary Artery Disease

- **coronary artery disease** (CAD) – a constriction of the coronary arteries
  - usually the result of **atherosclerosis** – accumulation of lipid deposits that degrade the arterial wall and obstruct the lumen
  - endothelium damaged by hypertension, virus, diabetes or other causes
  - monocytes penetrate walls of damaged vessels and transform into macrophages
    - absorb cholesterol and fats to be called **foam cells**
      - look like fatty streak on vessel wall
      - can grow into atherosclerotic plaques (**atheromas**)
  - platelets adhere to damaged areas and secrete platelet-derived growth factor
    - attracting immune cells and promoting mitosis of muscle and fibroblasts, and the deposition of collagen
- bulging mass grows to obstruct arterial lumen

# Affects of Atheromas

- causes **angina pectoris**, intermittent chest pain, by obstructing 75% or more of the blood flow
- immune cells of atheroma stimulate inflammation – may rupture – traveling clots or fatty emboli may result
- cause coronary artery spasms due to lack of secretion of nitric oxide (vasodilator)
- inflammation transforms atheroma into a hardened complicated plaque called **arteriosclerosis**

# Risk

- major risk factor for atherosclerosis is excess of low-density lipoprotein (LDL) in the blood combined with defective LDL receptors in the arterial walls
  - protein-coated droplets of cholesterol, neutral fats, free fatty acids and phospholipids
- most cells have LDL receptors that take up these droplets from blood by receptor-mediated endocytosis
  - dysfunctional receptors in arterial cells accumulate excess cholesterol
- familial hypercholesterolemia
  - dominant gene makes no receptors for LDL
    - heterozygous individual suffer heart attacks by 35
    - homozygous individuals suffer heart attacks by 2
- unavoidable risk factors - heredity, aging, being male
- avoidable risk factors – obesity, smoking, lack of exercise, anxious personality, stress, aggression, and diet

# Prevention and Treatment

- treatment
  - coronary bypass surgery
    - great saphenous vein
  - balloon angioplasty
  - laser angioplasty

# Overview of Volume Changes

**end-systolic volume (ESV)** **60 ml**

-passively added to ventricle during atrial diastole +30 ml

-added by atrial systole +40 ml

total: **end-diastolic volume (EDV)** **130 ml**

---

**stroke volume (SV)** ejected by ventricular systole **-70 ml**

leaves: **end-systolic volume (ESV)** **60 ml**

both ventricles must eject same amount of blood

---

Al-Farabi Kazakh National University
Higher School of Medicine

# CARDIOVASCULAR SYSTEM

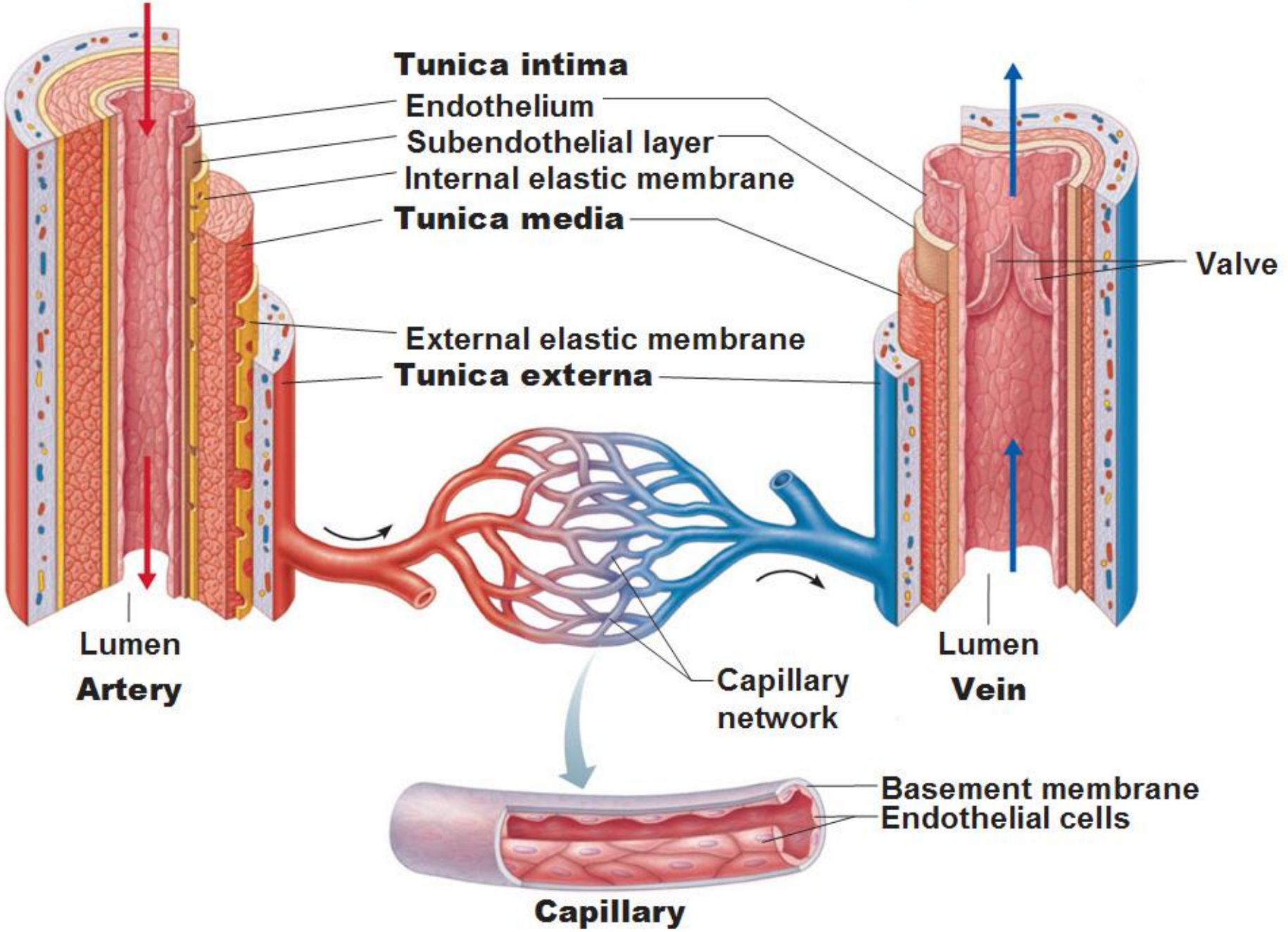
# Learning outcomes:
# As a result of the lesson you will be able to:

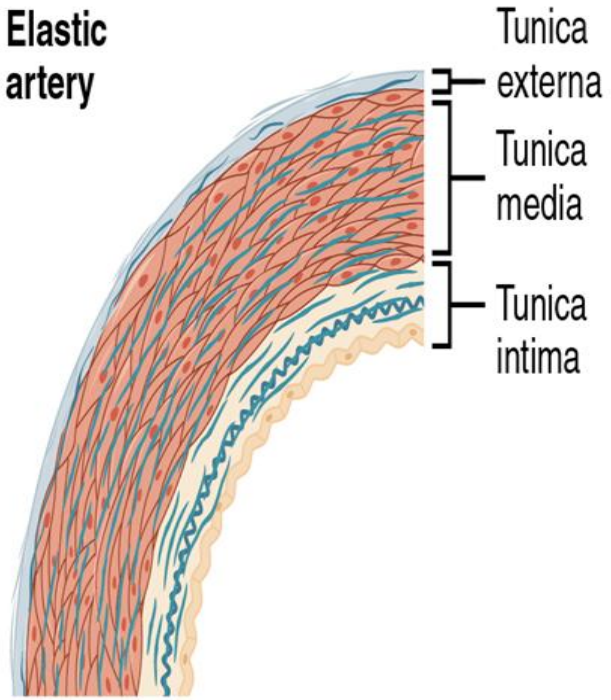
- microscopic and ultramicroscopic structural features of arteries and veins different in types, structural features of blood capillaries, heart, lymph vessels. structural components of aorta, muscular artery, arterioles, capillaries, muscular and fibrous veins, cardiac wall under the microscope and on the photomicrographs.
- Morphologic and functional description of the cardiovascular system
- Classification and development
- Structural features of arteries
- Microcirculation vessels
- The main types of blood capillaries
- Classification and structural features of veins
- Lymph vessel
- Structural features of cardiac wall

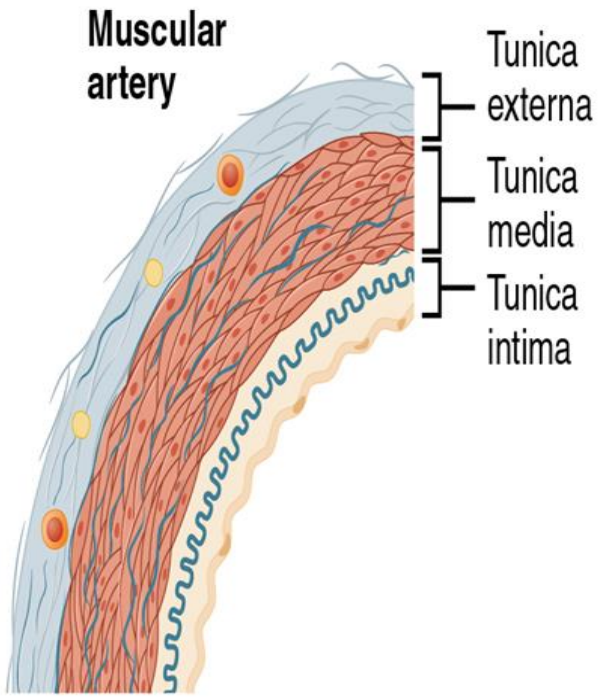
# Types of Blood Vessels

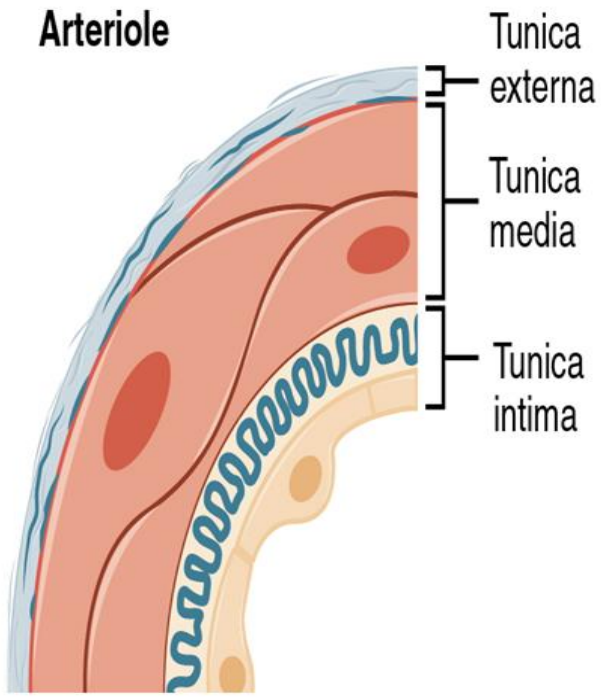
- **Arteries:** *Large or Elastic Artery*
  *Medium sized or Muscular Artery*
- **Arterioles**
- **Capillaries**: *Continuous capillary*
  *Fenestrated capillary*
  *Sinusoidal capillary*
- **Venules**
- **Veins:** *Medium sized Vein*
  *Large Vein*

# Structure of Blood Vessels – 3 Layers "Tunics"




Elastic artery
Tunica externa
Tunica media
Tunica intima

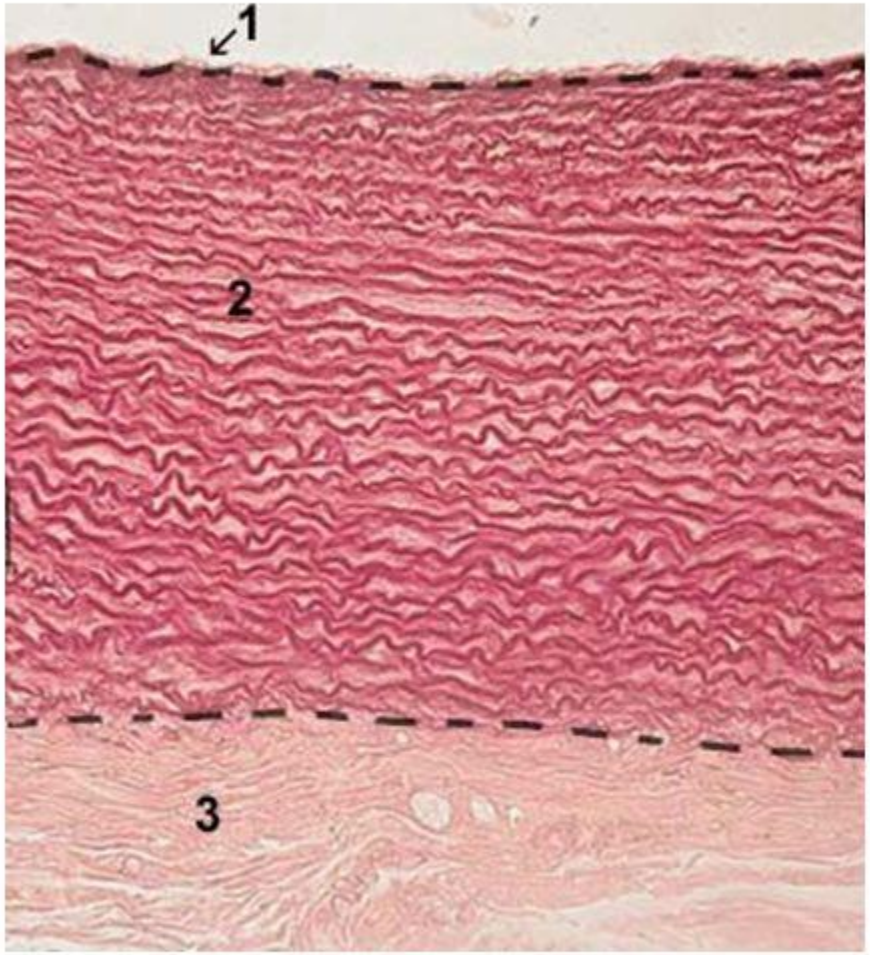

Muscular artery
Tunica externa
Tunica media
Tunica intima


Arteriole
Tunica externa
Tunica media
Tunica intima

# Elastic Artery(stained with orcein)

**Tunica Intima:**

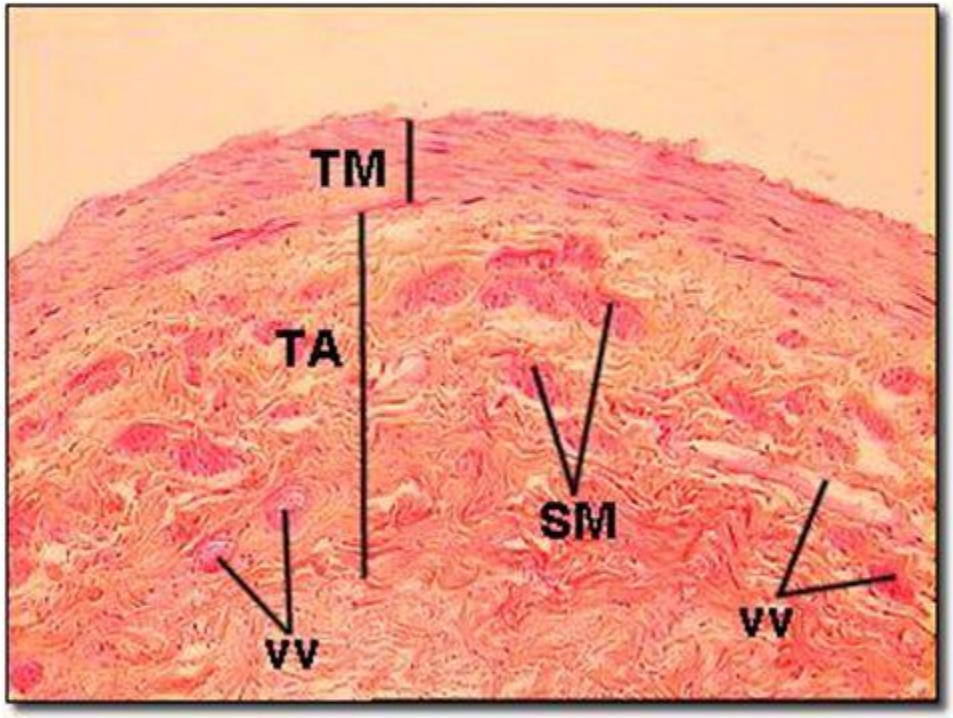
- ***Endothelium***
- ***Basal lamina*** **:A thin layer of glycoprotein**
- ***Subendothelial C T*** **which contains fibrocytes, macrophages and smooth muscle like-cells called Myointimal Cells.**
- **The collagen and elastic fibres are longitudinally arranged.**
- ***Internal Elastic Lamina*** **forms the boundry between T Intima and T media. Consists of elastic fibres is poorly defined and is fenestrated**

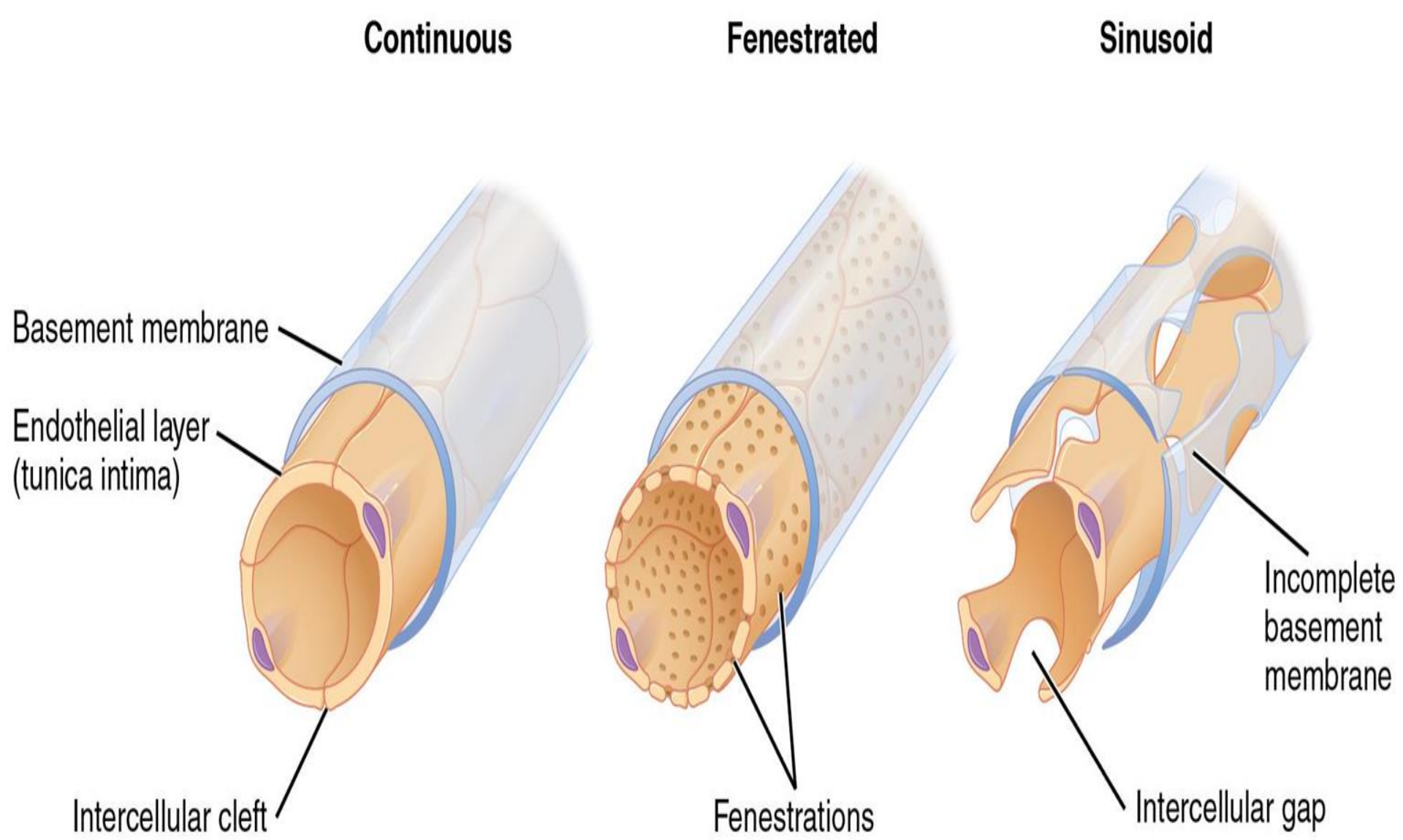


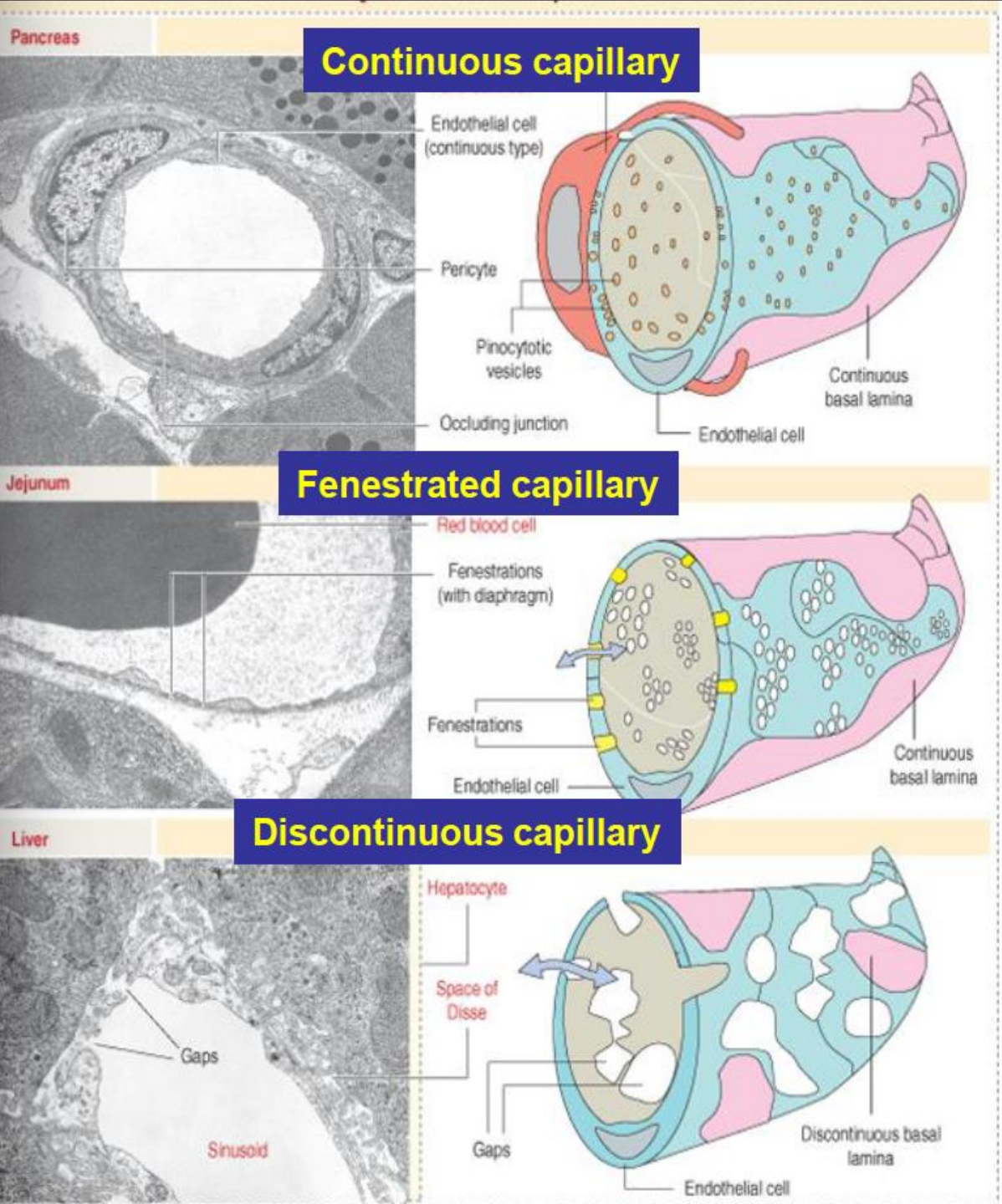
# Histological features of Large vein

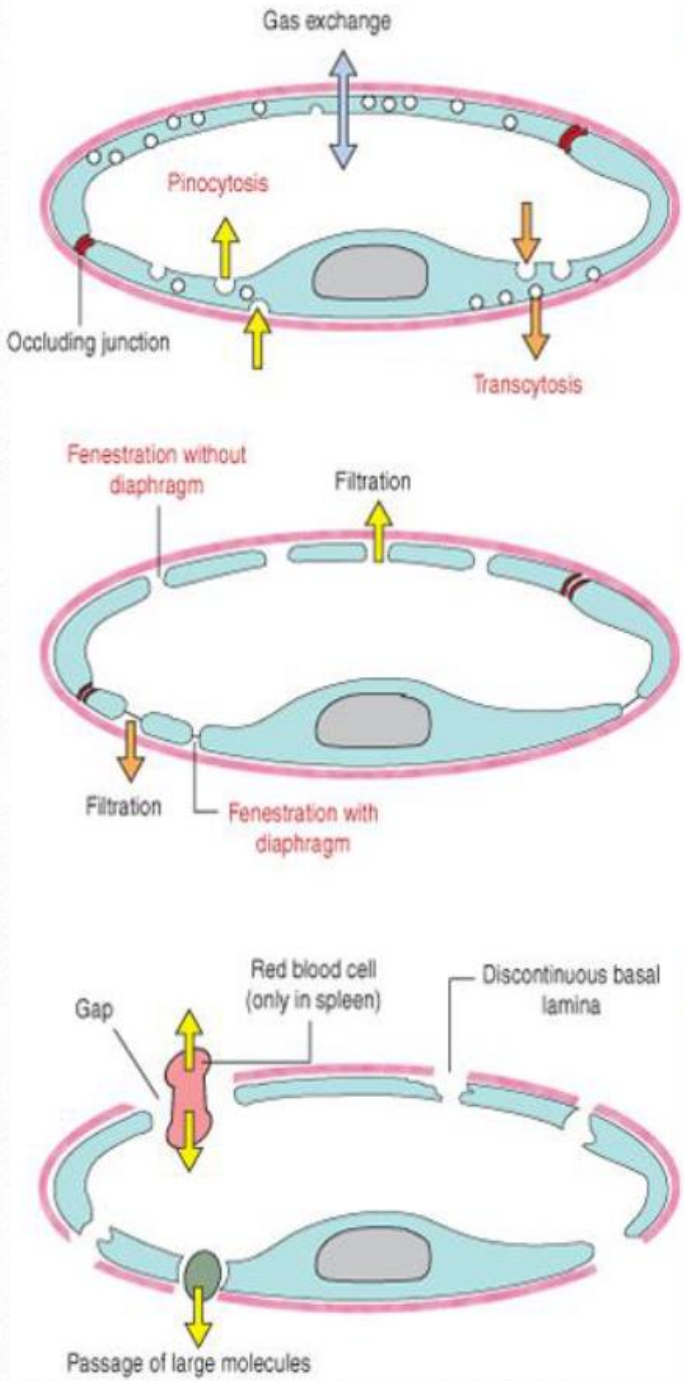
**Examples: SVC and IVC**

- T intima: well developed, endothelium and subendothelial C T +
- T media: thin or –
- **T adventitia: well developed, thickest coat, many longitudinal bundles of smooth muscle fibers are embedded in C T.**
- Longitudinal bundles facilitate ***shortening and elongation of the vena cava*** with respiration.



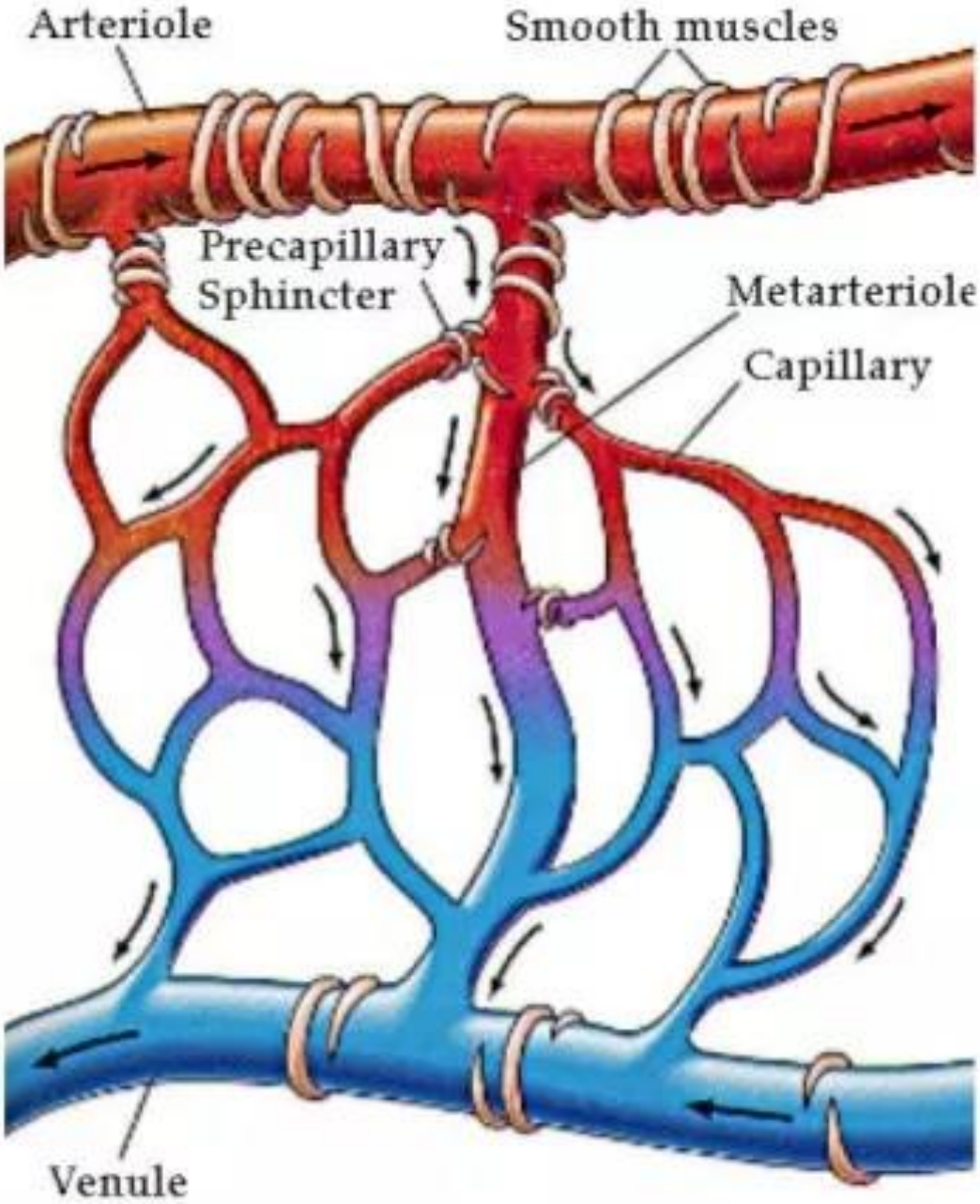

Continuous
Fenestrated
Sinusoid
Basement membrane
Endothelial layer
(tunica intima)
Intercellular cleft
Fenestrations
Incomplete
basement
membrane
Intercellular gap


Pancreas
Continuous capillary
Endothelial cell (continuous type)
Pericyte
Pinocytotic vesicles
Occluding junction
Endothelial cell
Continuous basal lamina
Jejunum
Fenestrated capillary
Red blood cell
Fenestrations (with diaphragm)
Fenestrations
Endothelial cell
Continuous basal lamina
Liver
Discontinuous capillary
Hepatocyte
Space of Disse
Gaps
Gaps
Sinusoid
Discontinuous basal lamina
Endothelial cell


Gas exchange
Pinocytosis
Occluding junction
Transcytosis
Fenestration without diaphragm
Filtration
Filtration
Fenestration with diaphragm
Red blood cell (only in spleen)
Gap
Discontinuous basal lamina
Passage of large molecules

# Metarterioles

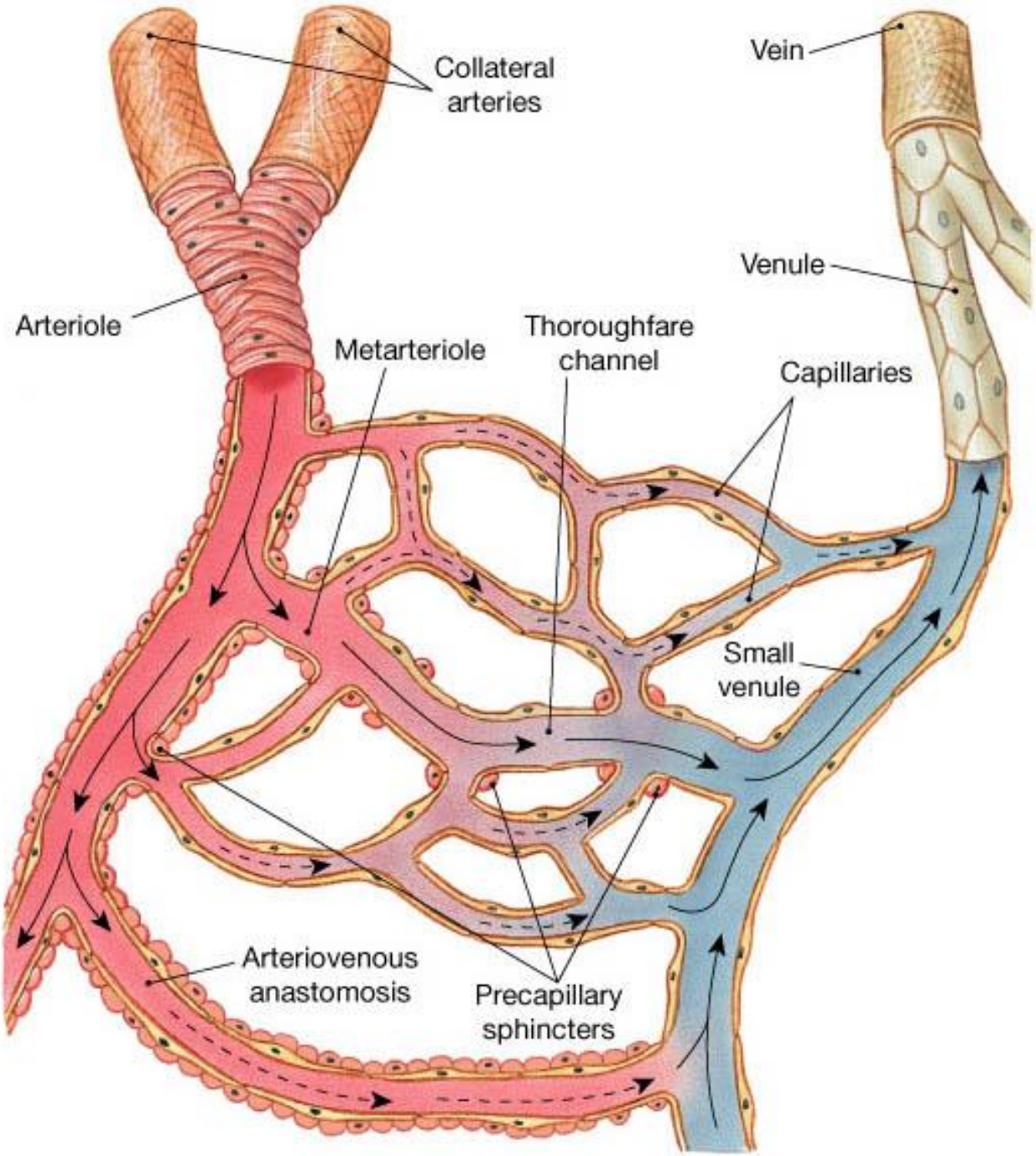
- Regulate blood flow into *capillaries*
- **Precapillary sphincters**
- **Thoroughfare channel**
- **Vasomotion**: fills bed 5-10x/min



# Arterioles:

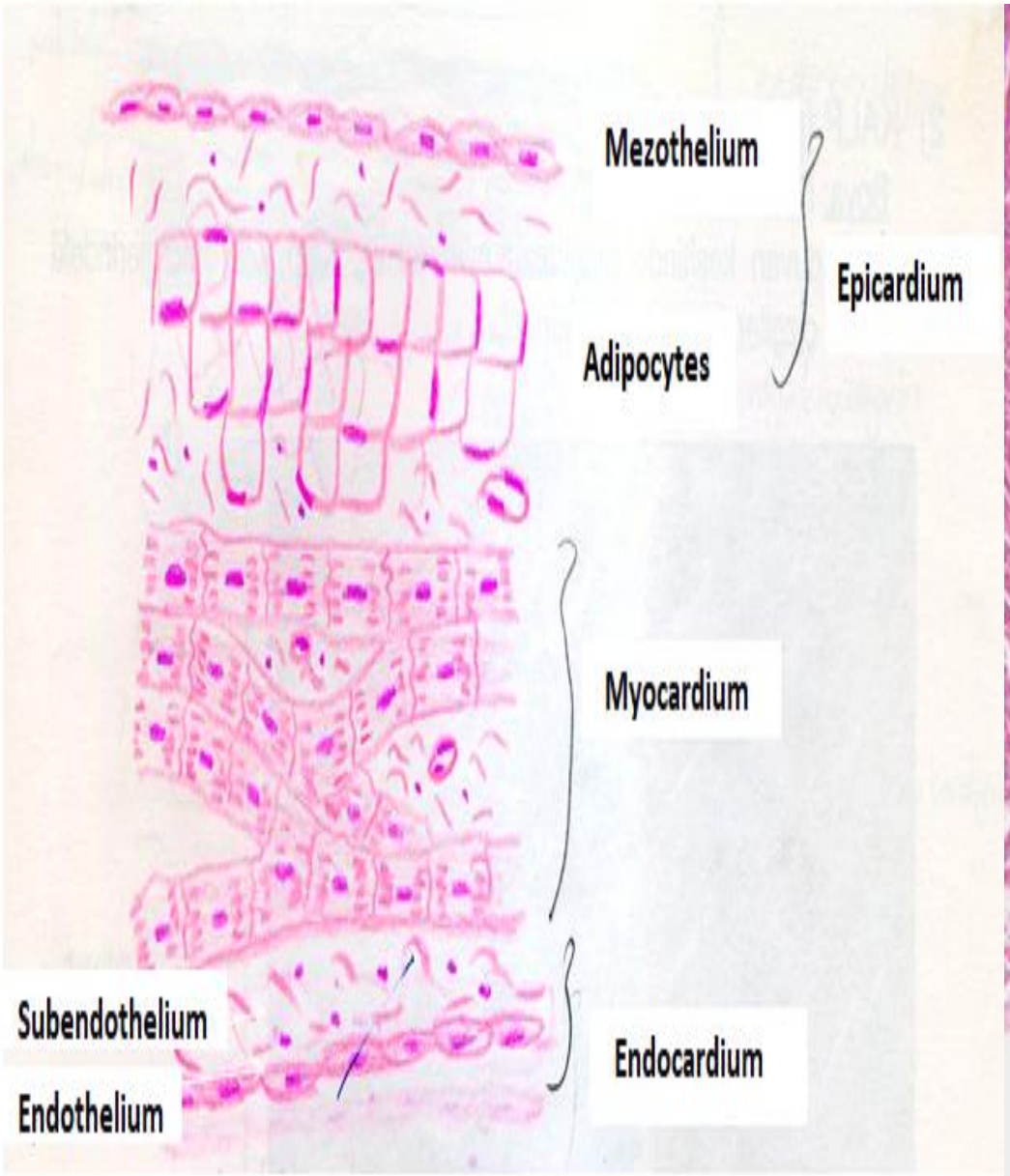
- 1-3 layers of Smooth Muscle
- Greatest effect on blood pressure
- Endo Cells have Weibel-Palade bodies
- Nuclei bulge into lumen
- Round appearance of vessel
- No internal elastic membrane

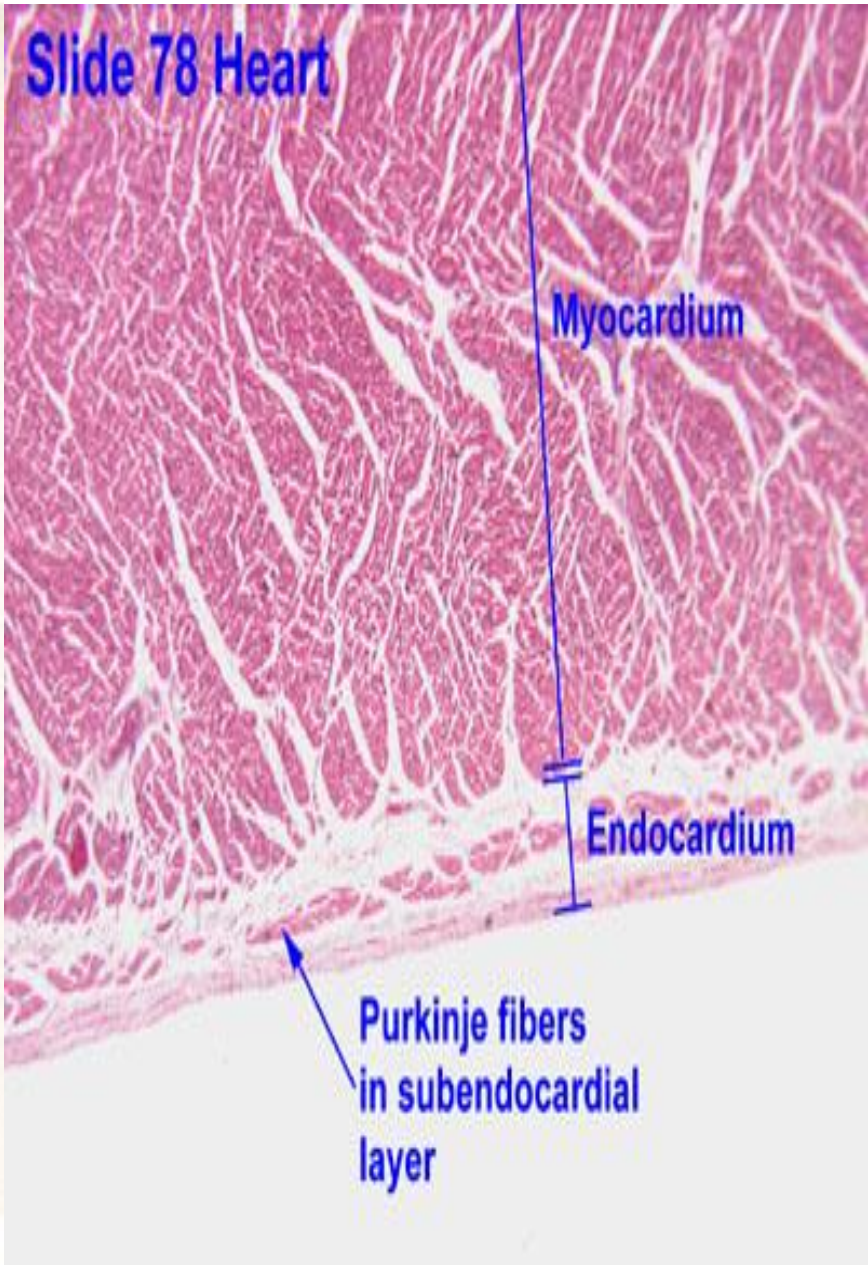
Metarteriole is terminal vessel, has precapillary sphincter. Can regulate flow to capillary bed




**(a) Capillary bed**

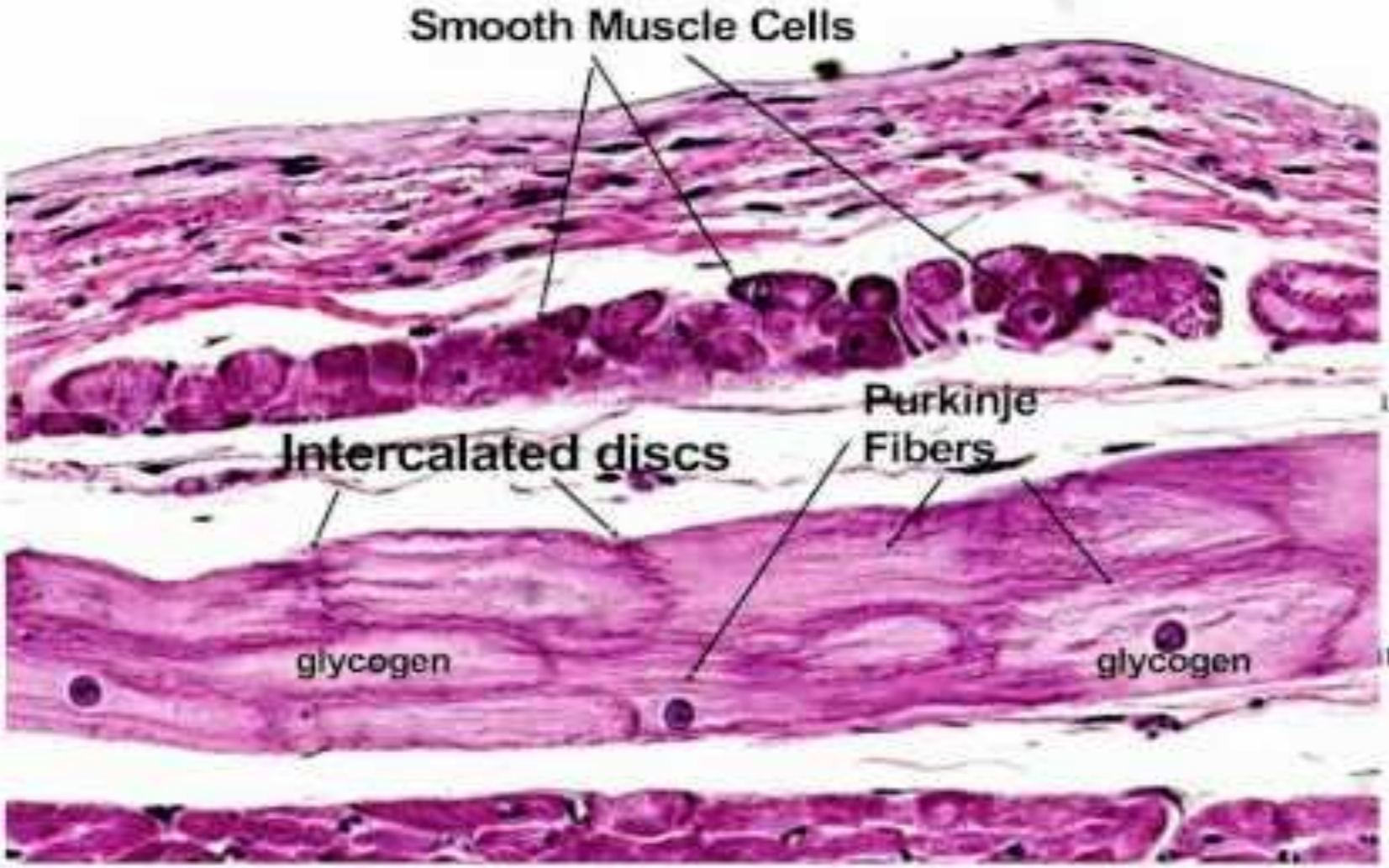
# Heart


Mezothelium
Epicardium
Adipocytes
Myocardium
Subendothelium
Endothelium
Endocardium


Slide 78 Heart
Myocardium
Endocardium
Purkinje fibers
in subendocardial
layer

# Layers of Heart

## Purkinje Fibers



**Recommended literature:**


- **Leslie P.Gartner James L.Hiatt Color Textbook Histology 3 ed. 2007, p251-270**
- Inderbir Singh. Textbook of human histology.- New Delhi.- 2002.- pp.201-224