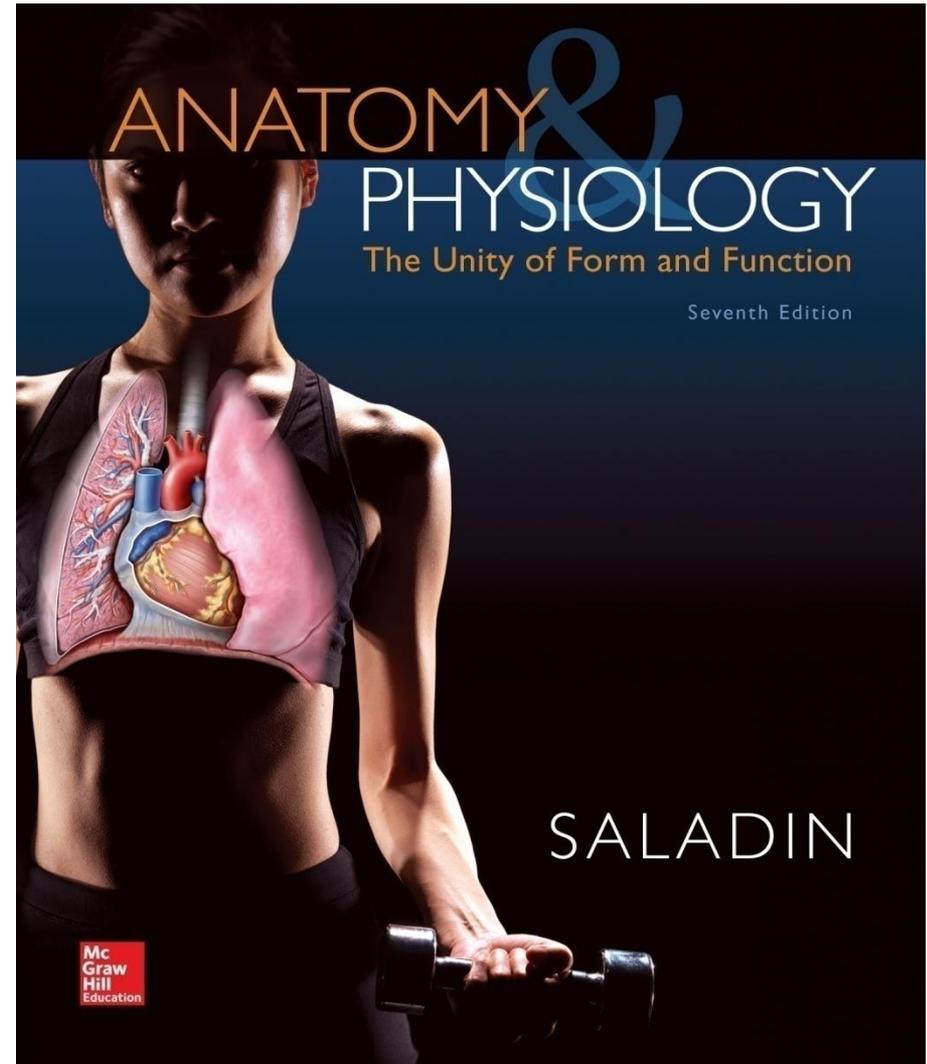


Chapter 19

Lecture Outline

See separate PowerPoint slides for all figures and tables pre-inserted into PowerPoint without notes.



Introduction

- **Some ancient scholars surmised that the heart was a pump for blood, but Aristotle saw it as the seat of emotion**
- **Thirteenth-century physician—Ibn an-Nafis**
 - Described the role of the coronary blood vessels in nourishing the heart
- **Sixteen-century—Vesalius**
 - Greatly improved knowledge of cardiovascular anatomy, the study of the heart, and treatment of its disorders—cardiology

Introduction

(Continued)

- **Twentieth century**
 - Discoveries of nitroglycerin, digitalis; coronary bypass, valve replacement, and others
- **Cardiology is one of today's most dramatic attention-getting fields in medicine!**

Overview of the Cardiovascular System

- **Expected Learning Outcomes**
 - Define and distinguish between the pulmonary and systemic circuits.
 - Describe the general location, size, and shape of the heart.
 - Describe the pericardial sac that encloses the heart.

Overview of the Cardiovascular System

- **Cardiovascular system**
 - Heart and blood vessels
- **Circulatory system**
 - Heart, blood vessels, and the blood

The Pulmonary and Systemic Circuits

- **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

The Pulmonary and Systemic Circuits

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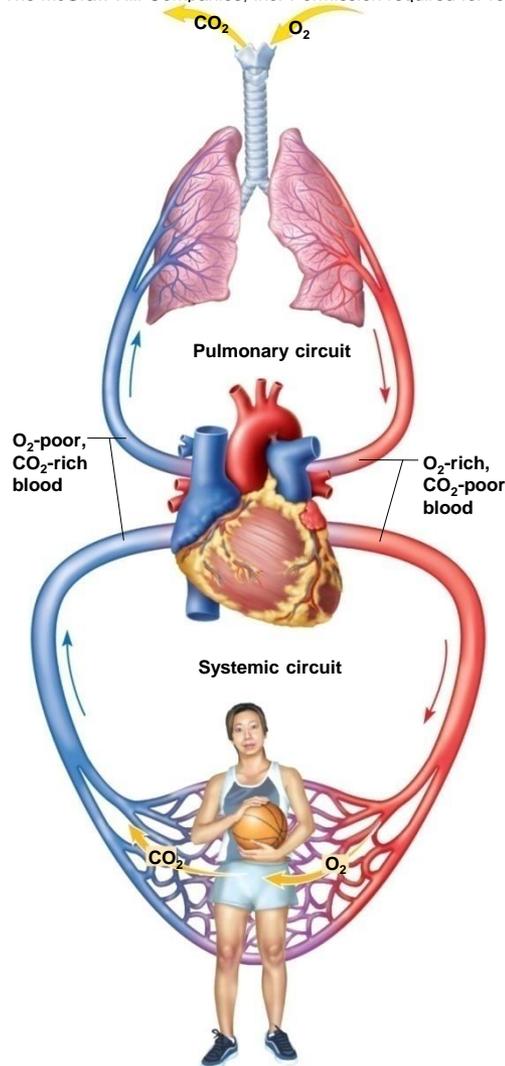


Figure 19.1

- **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**
 - Oxygen-poor blood arrives from inferior and superior venae cavae
 - Blood sent to lungs via pulmonary trunk

Position, Size, and Shape of the Heart

- **Heart located in mediastinum, between lungs**
- **Base**—wide, superior portion of heart, large vessels attach here
- **Apex**—tapered inferior end, tilts to the left
- **In adult: weighs 10 ounces, 3.5 in. wide at base, 5 in. from base to apex**
- **At any age, heart is size of fist**

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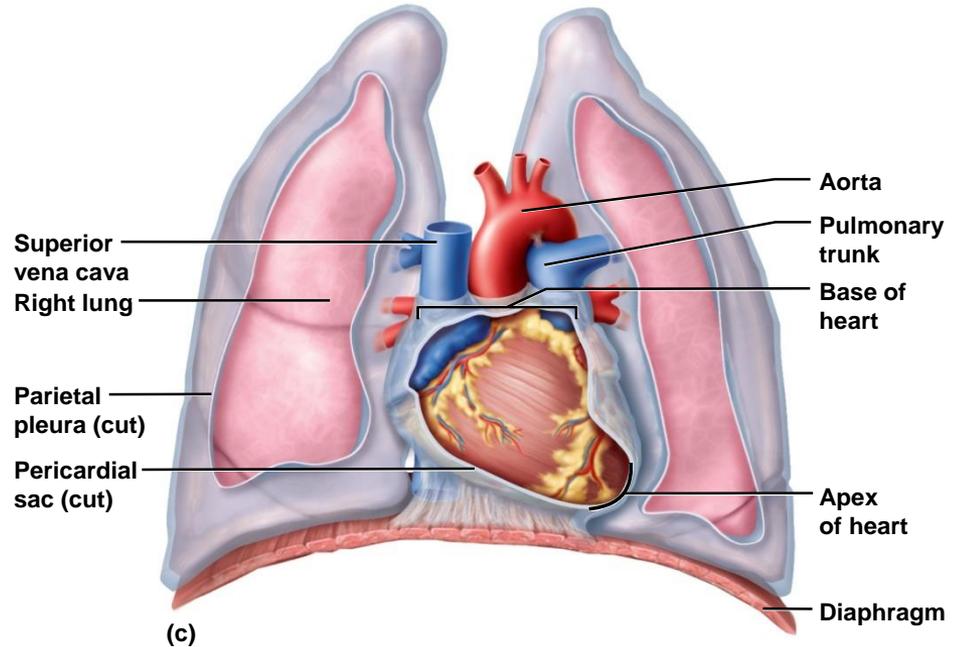
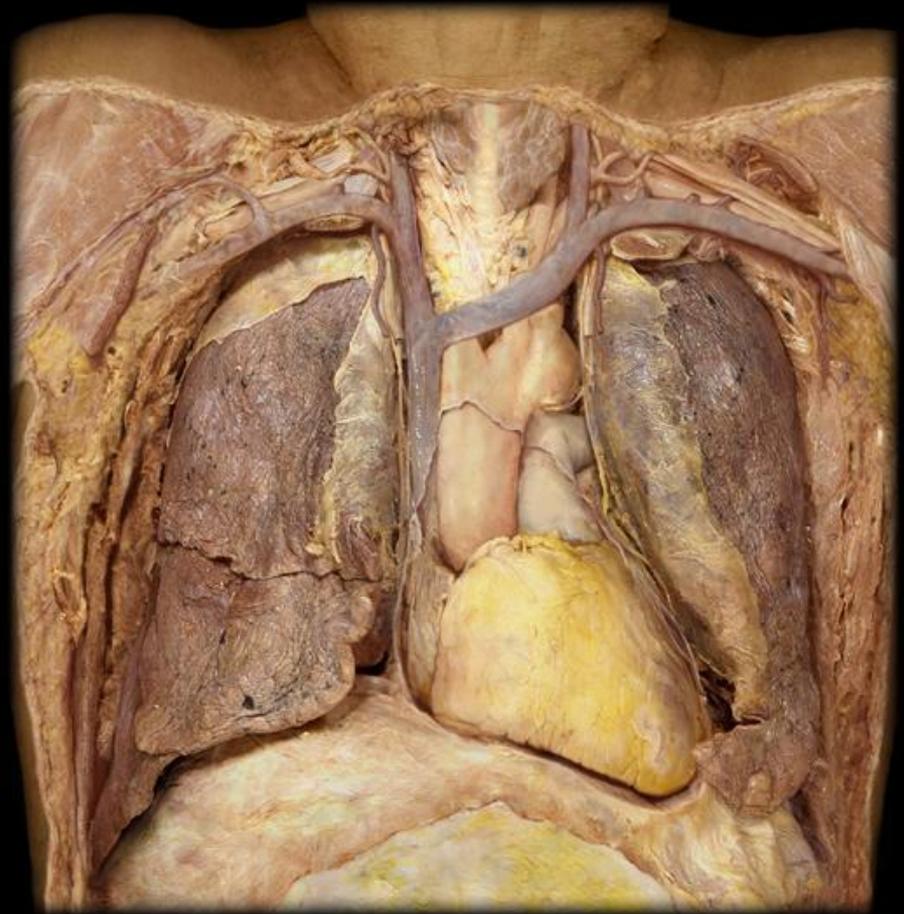
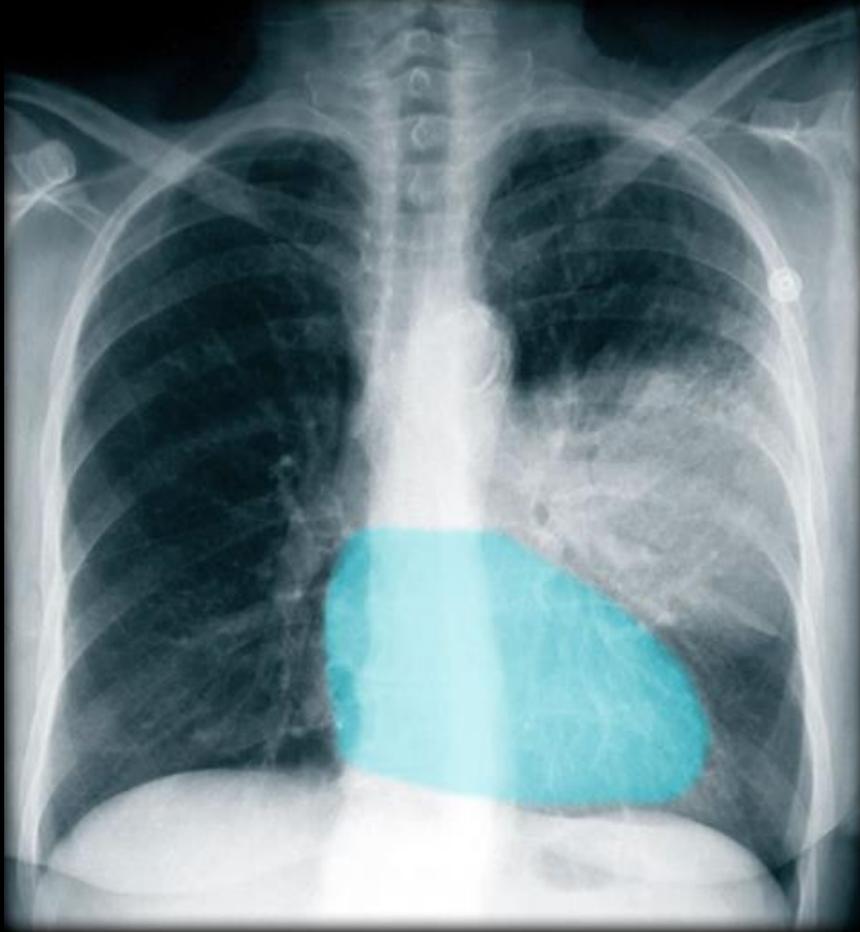
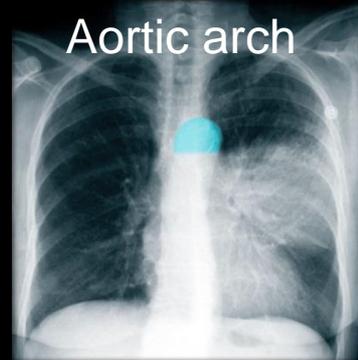
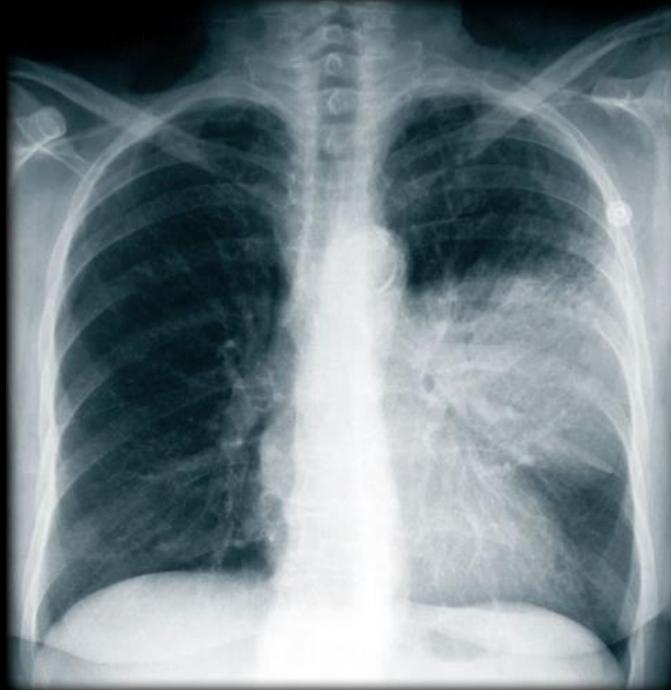
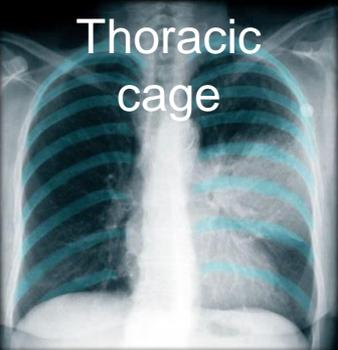


Figure 19.2c

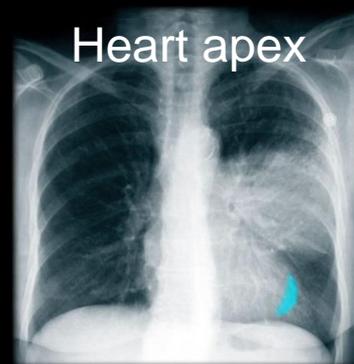
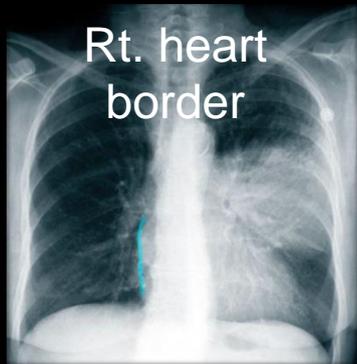
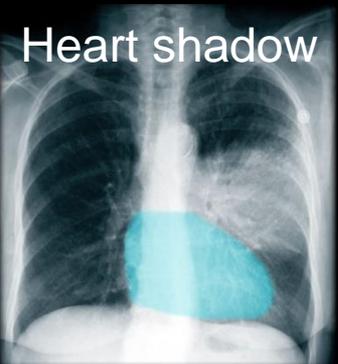
Heart Location



Cardiovascular System Xray: Posterior-Anterior View

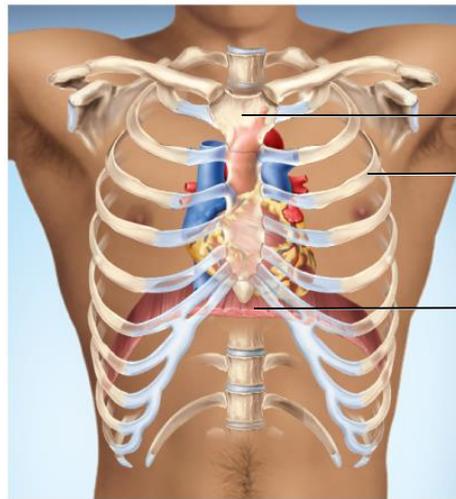


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Position, Size, and Shape of the Heart

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(a)



Lungs

Thoracic
vertebra

Right
ventricle

Sternum

(b)

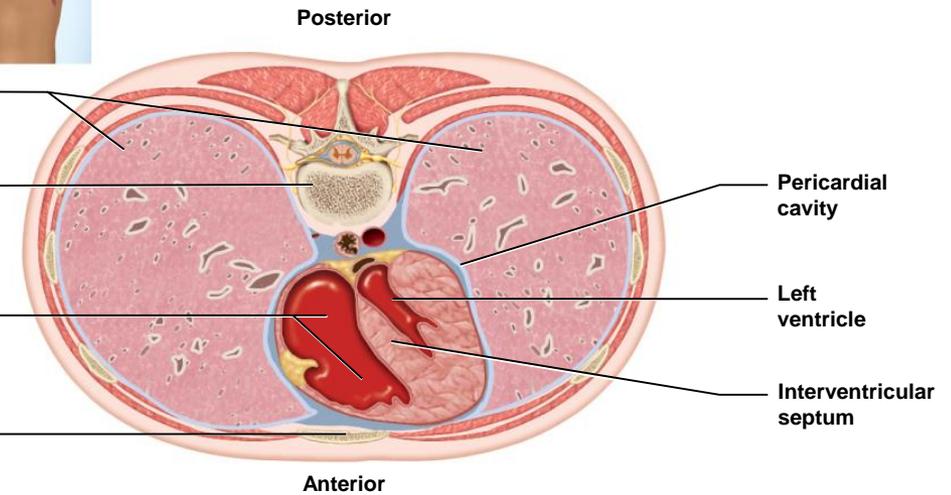


Figure 19.2a,b

Thoracic CT Scans



Axial



Sagittal

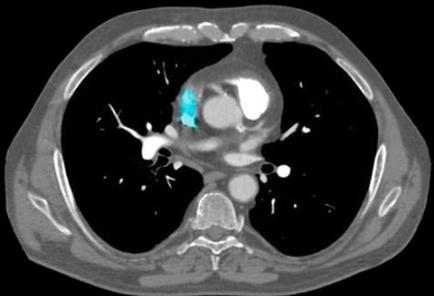
Heart: Right Ventricle CT scan: Axial



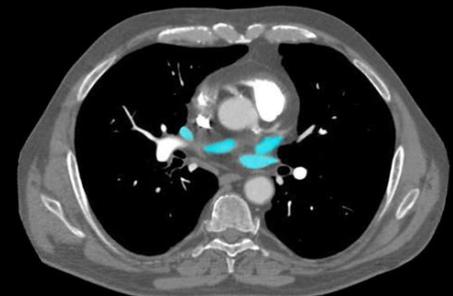
Heart



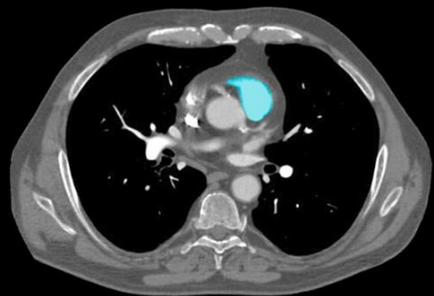
Pulmonary aa.



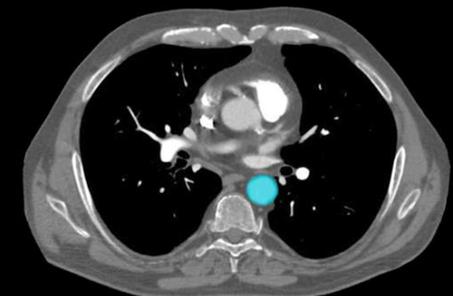
Rt. atrium



Pulmonary vv.



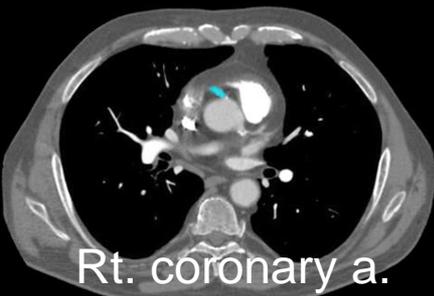
Rt. ventricle



Descending aorta



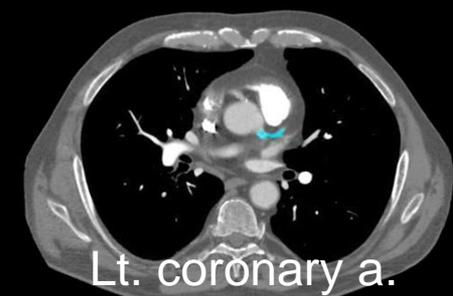
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Rt. coronary a.



Ascending aorta



Lt. coronary a.

The Pericardium

- **Pericardium**—double-walled 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**—pericardial sac
 - Superficial **fibrous layer** of connective tissue
 - Deep, thin **serous layer**
- **Visceral pericardium (epicardium)**
 - Serous membrane covering heart
- **Pericardial cavity**—space inside the pericardial sac filled with 5 to 30 mL of pericardial fluid
- **Pericarditis**—painful inflammation of the membranes

The Pericardium

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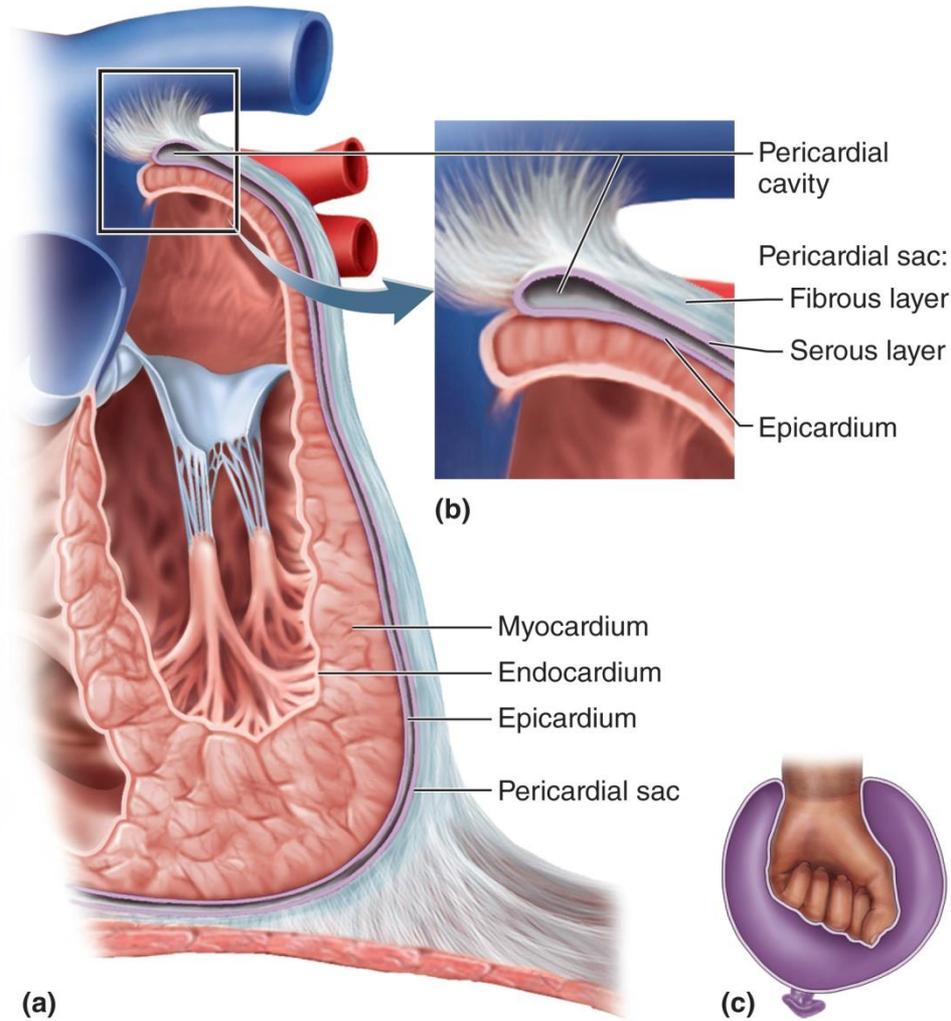
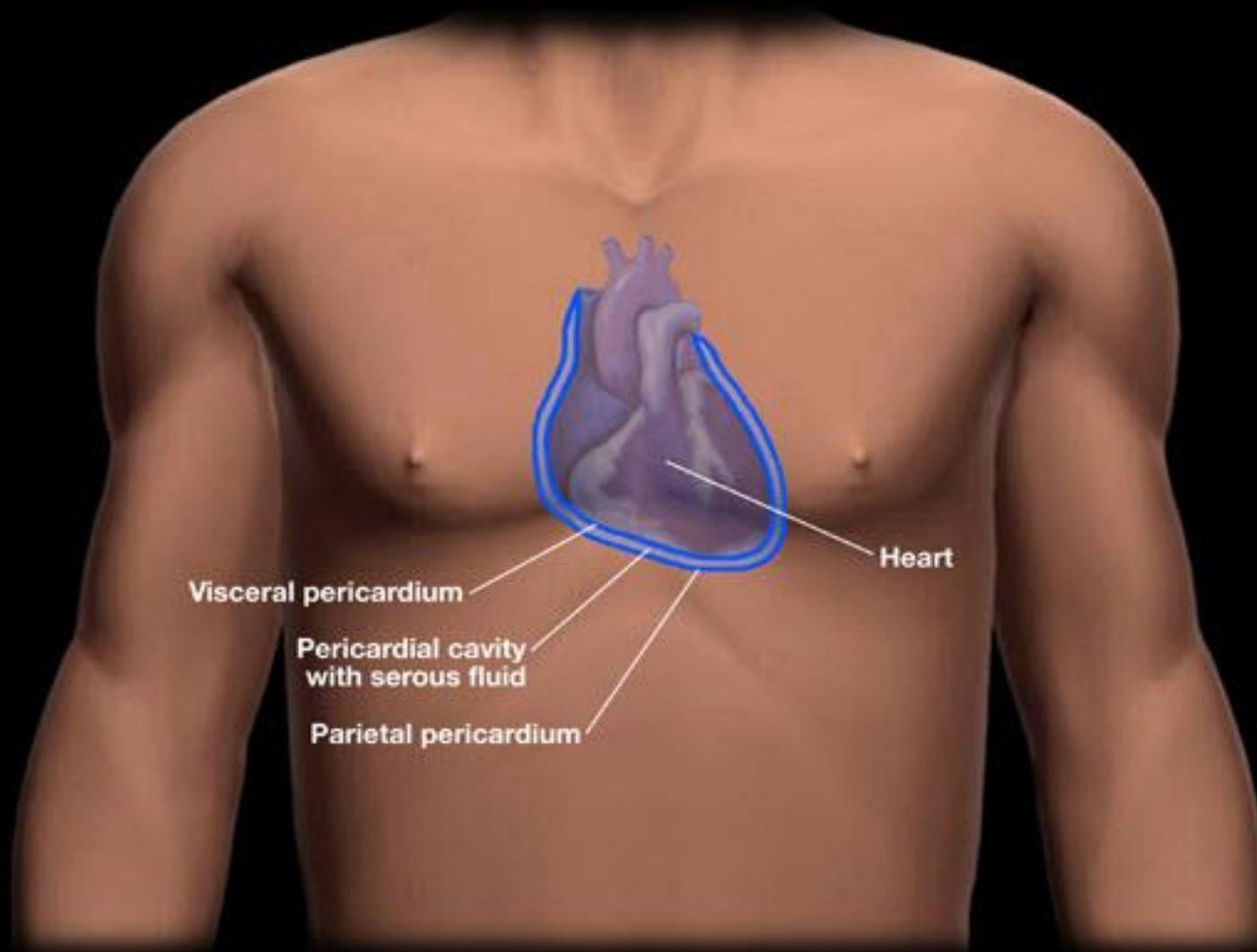
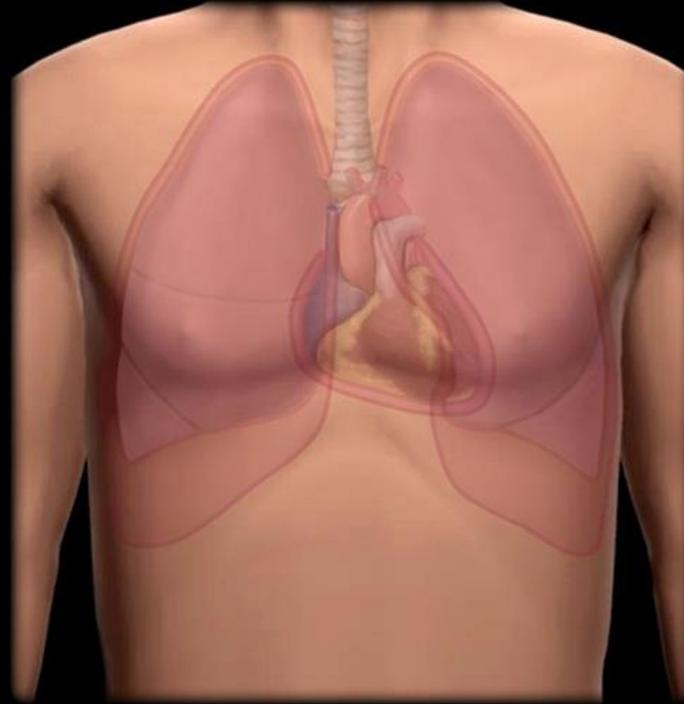


Figure 19.3

Pericardium

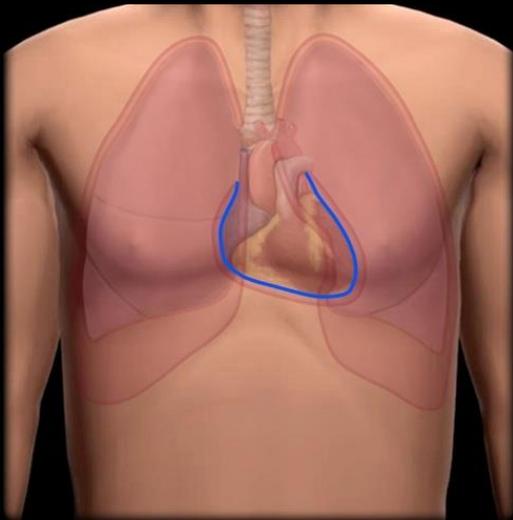


Pericardium

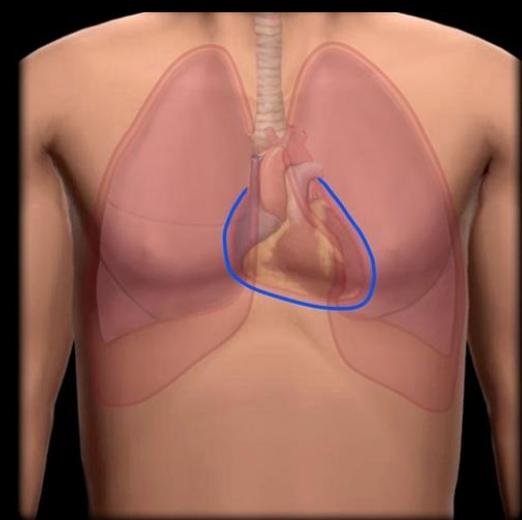


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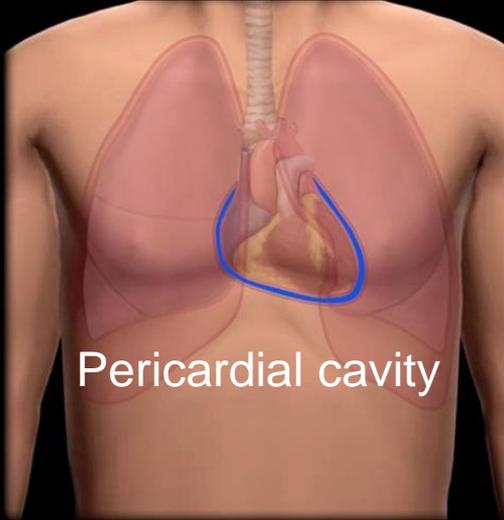
Visceral pericardium



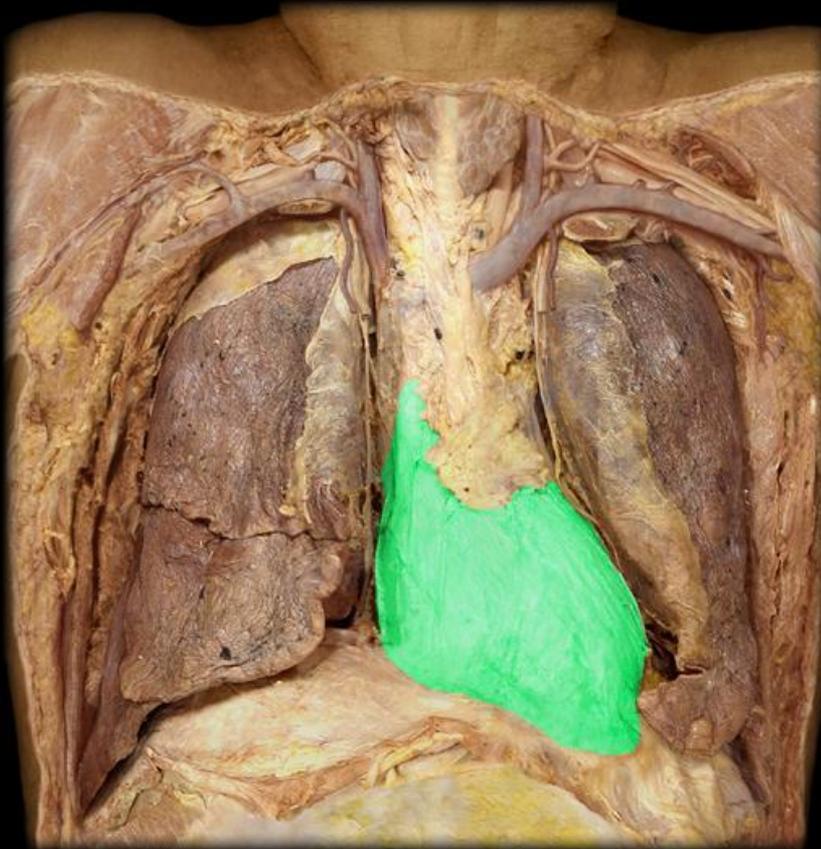
Parietal pericardium



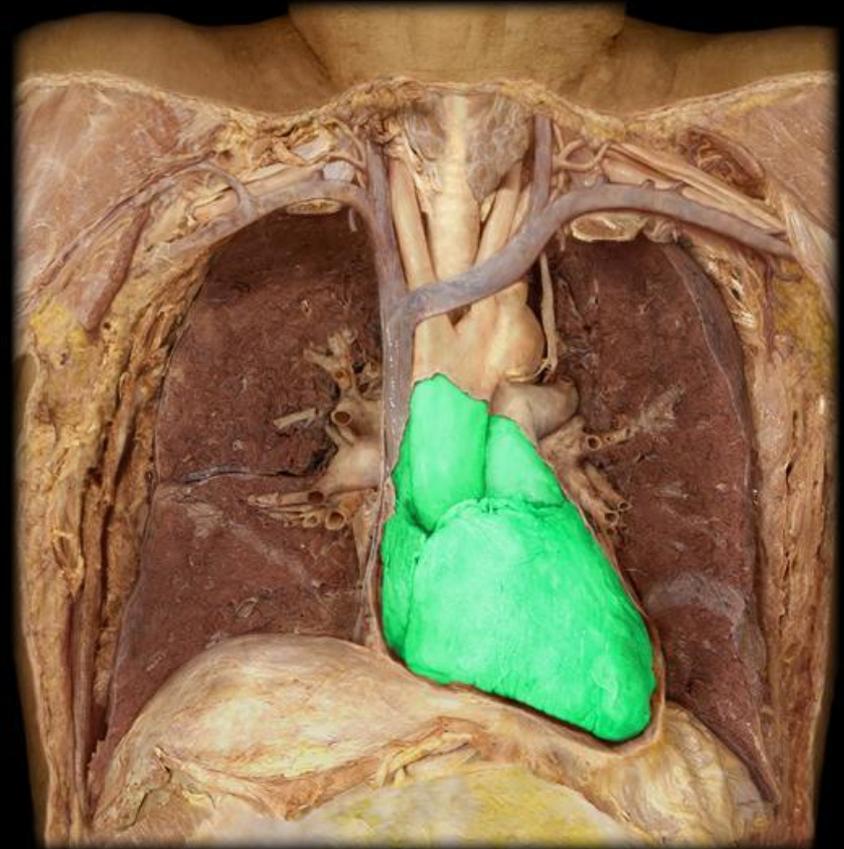
Pericardial cavity



Pericardium



Fibrous Pericardium



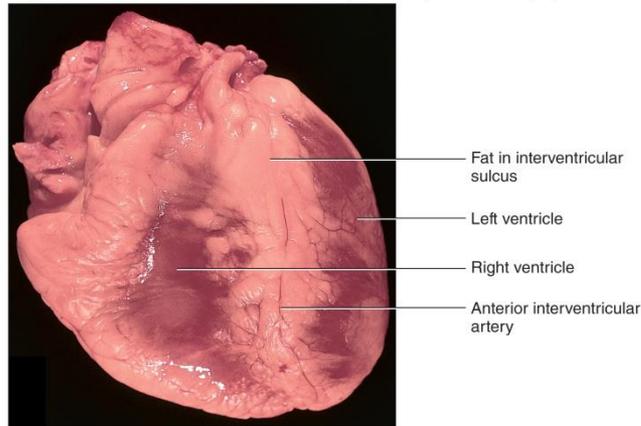
Serous (Visceral) Pericardium

Gross Anatomy of the Heart

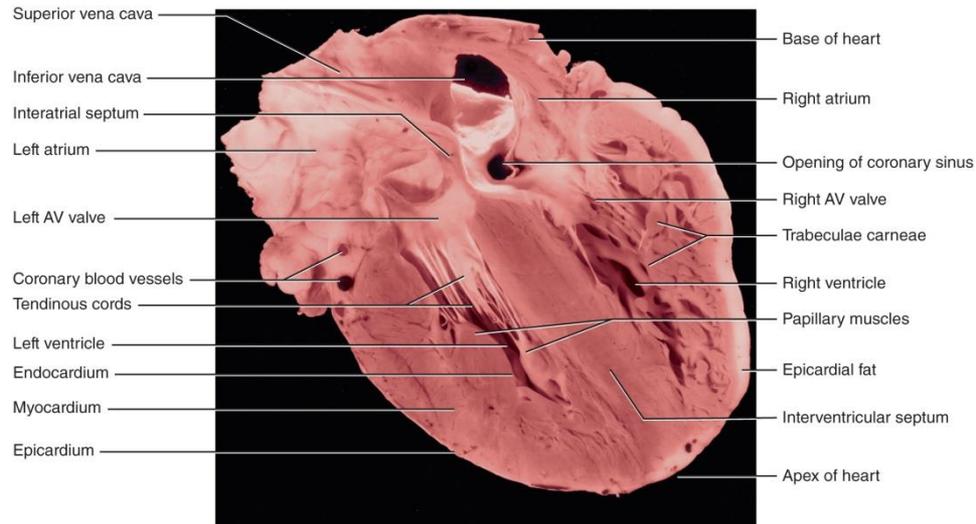
- **Expected Learning Outcomes**
 - Describe the three layers of the heart wall.
 - Identify the four chambers of the heart.
 - Identify the surface features of the heart and correlate them with its internal four-chambered anatomy.
 - Identify the four valves of the heart.
 - Trace the flow of blood 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.

Cadaver Heart

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(a) Anterior view, external anatomy



(b) Posterior view, internal anatomy

a,b: ©The McGraw-Hill Companies, Inc.

Figure 19.4a,b

The Heart Wall

- **Heart wall** has three layers: epicardium, myocardium and endocardium
- **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 is continuous with endothelium of blood vessels

The Heart Wall

- **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

The Pericardium

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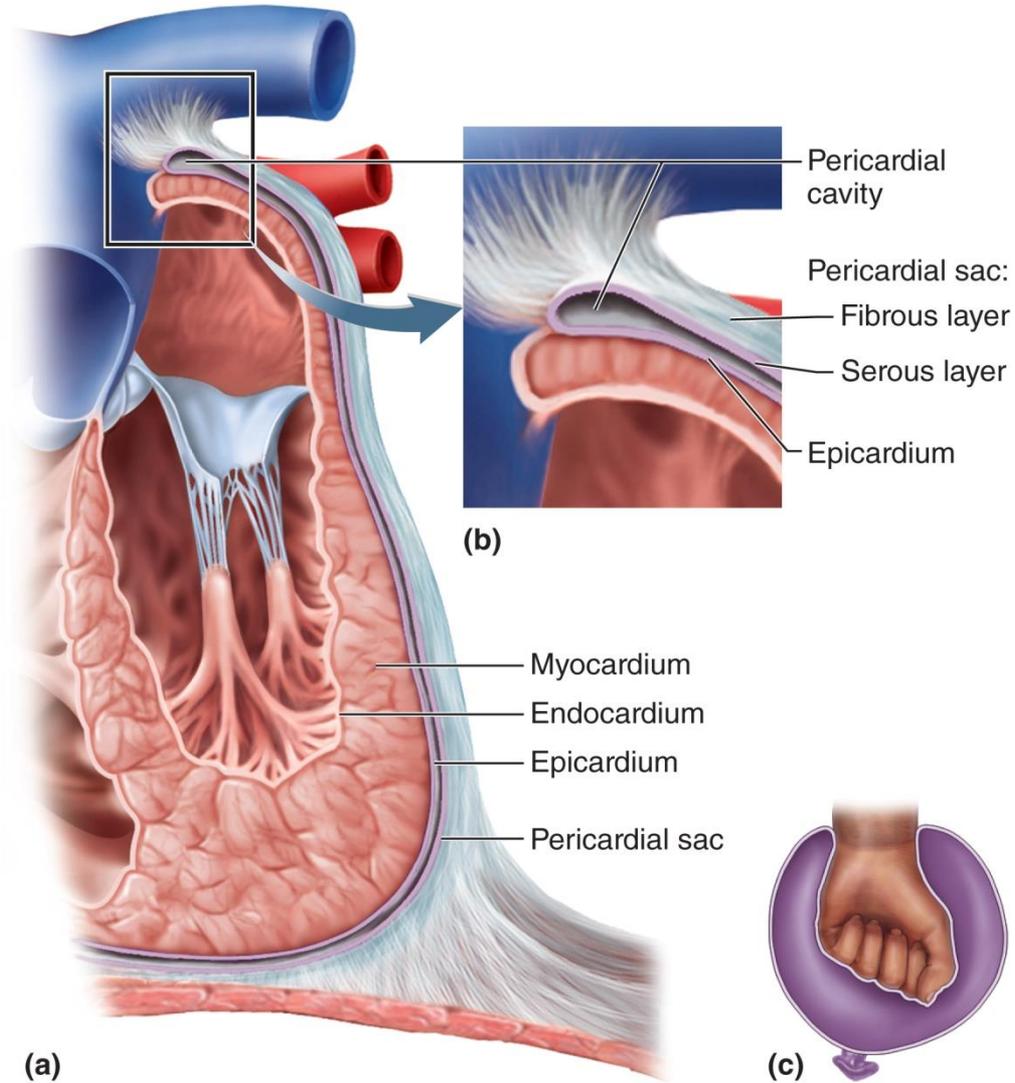
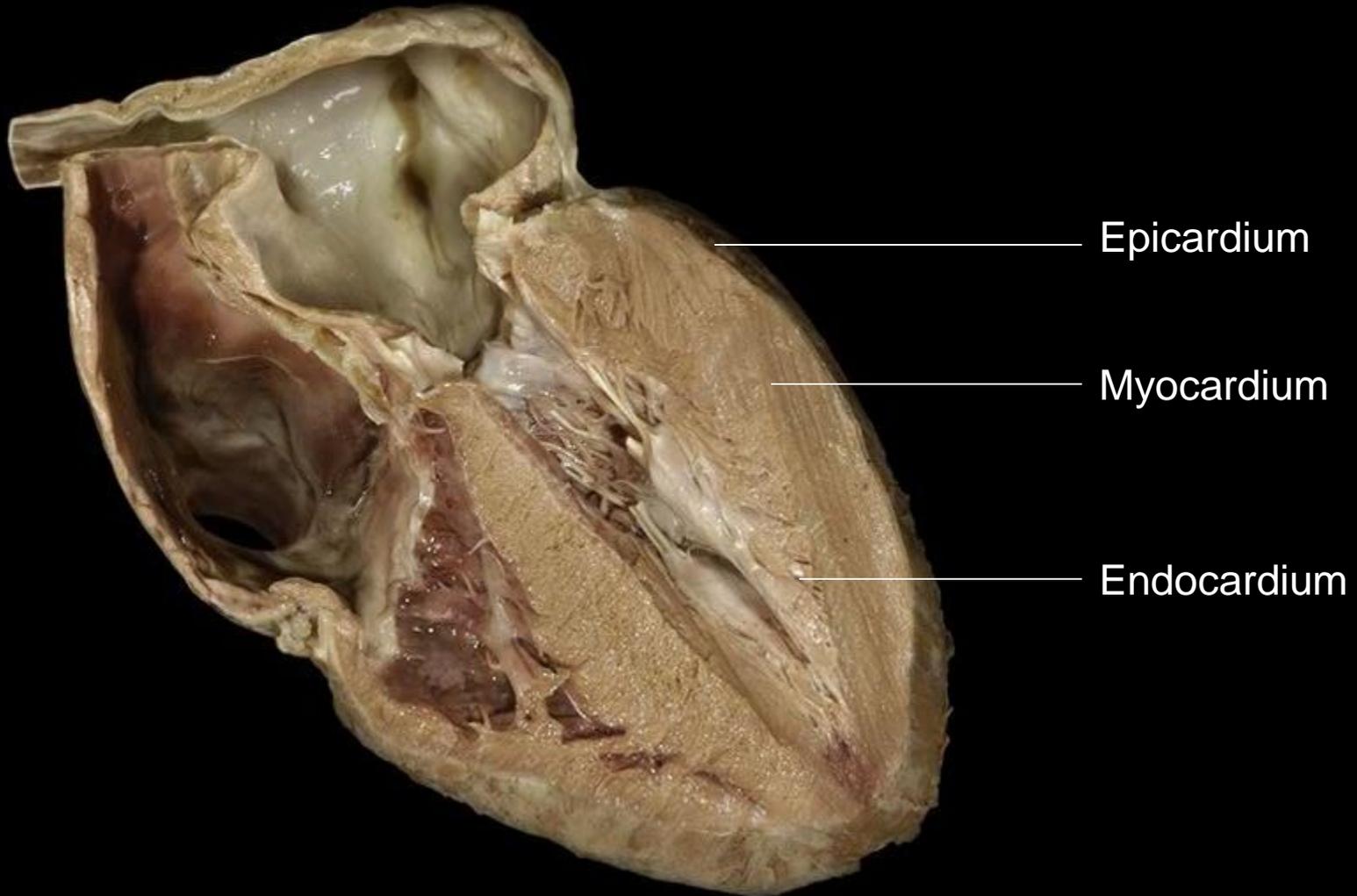


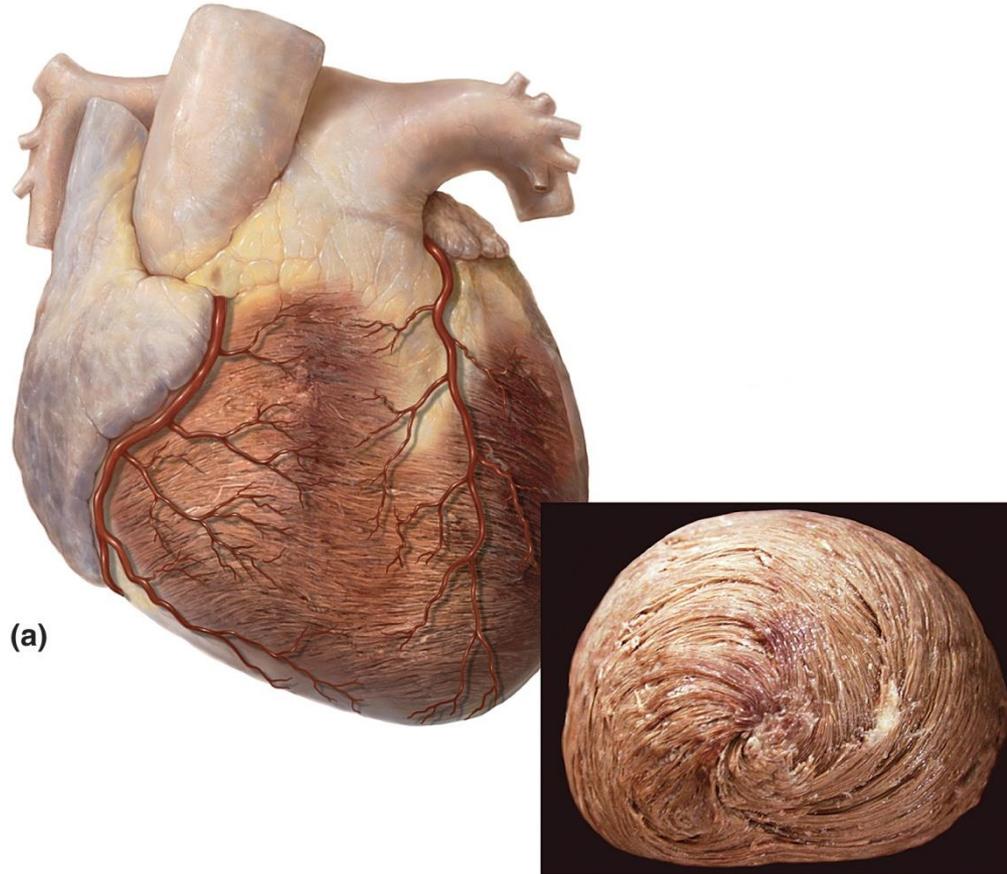
Figure 19.3

Heart Wall



Spiral Orientation of Myocardial Muscle

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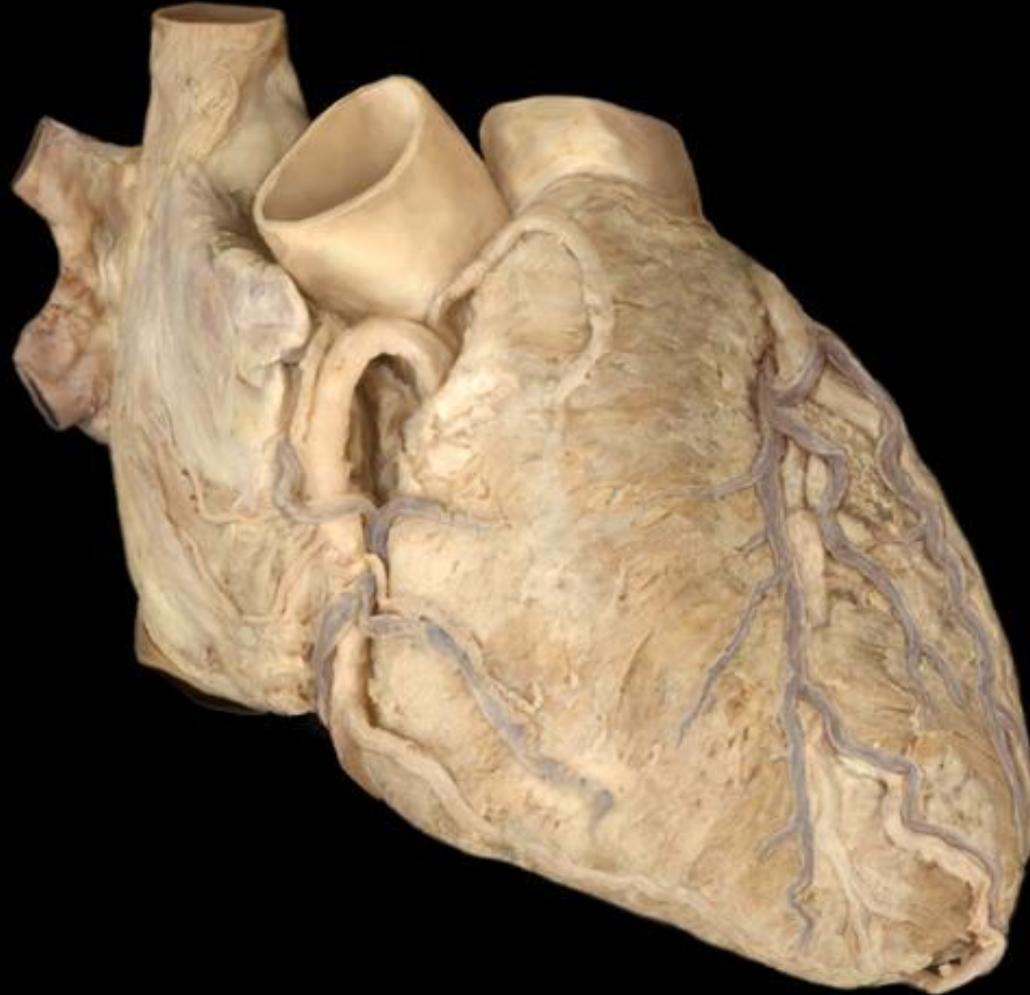
(a)

(b)

(a, b): Photo and illustration by Roy Schneider, University of Toledo. Plastinated heart model for illustration courtesy of Dr. Carlos Baptista, University of Toledo

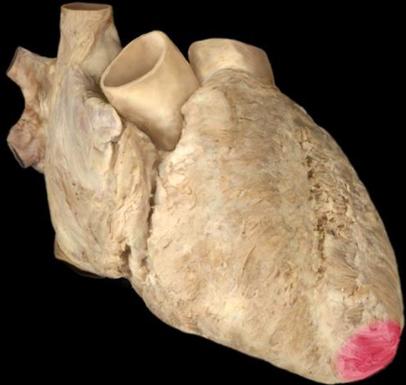
Figure 19.6

Gross Heart Anatomy

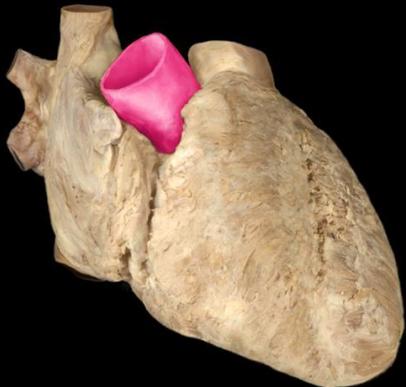
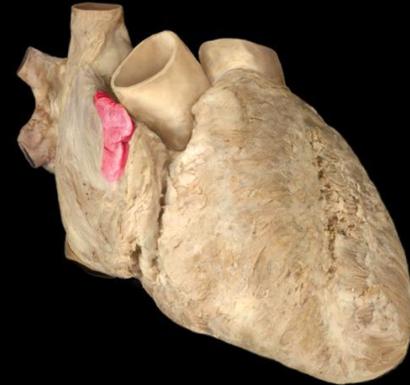


Gross Heart Anatomy

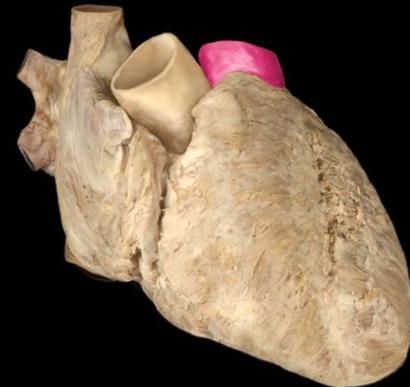
Apex



Rt. auricle



Ascending aorta

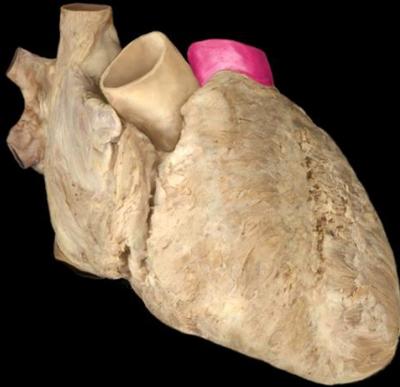


Pulmonary trunk

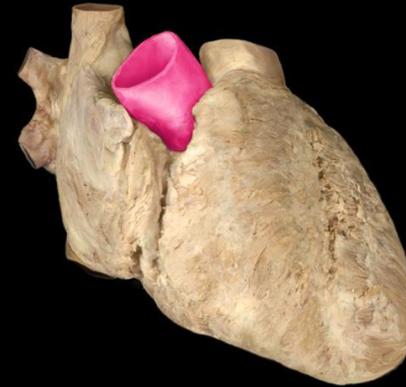
Heart External Anatomy

Great Vessels

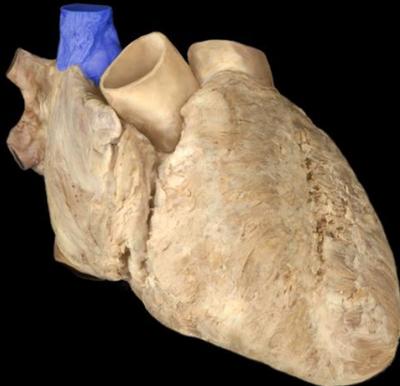
Pulmonary trunk



Ascending aorta



Superior vena cava



Inferior vena cava



The 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

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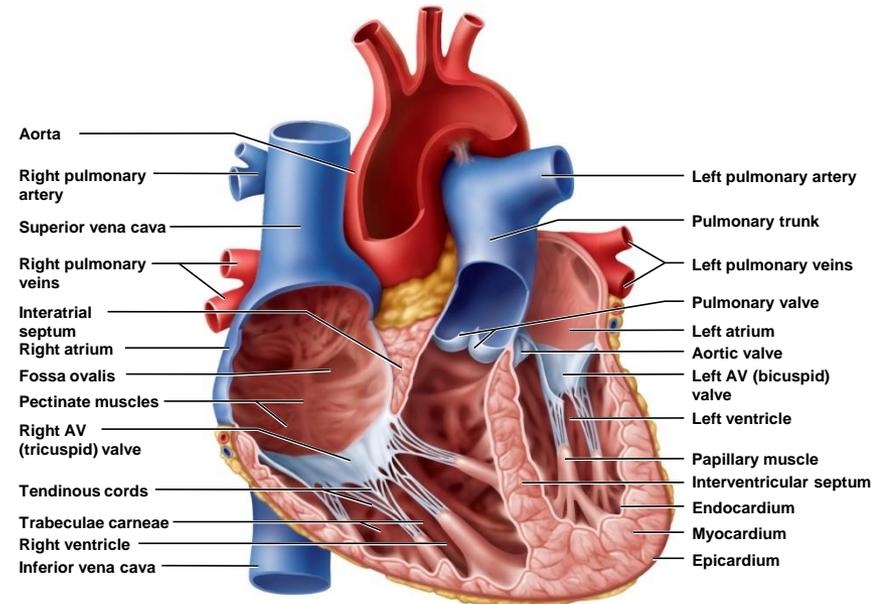
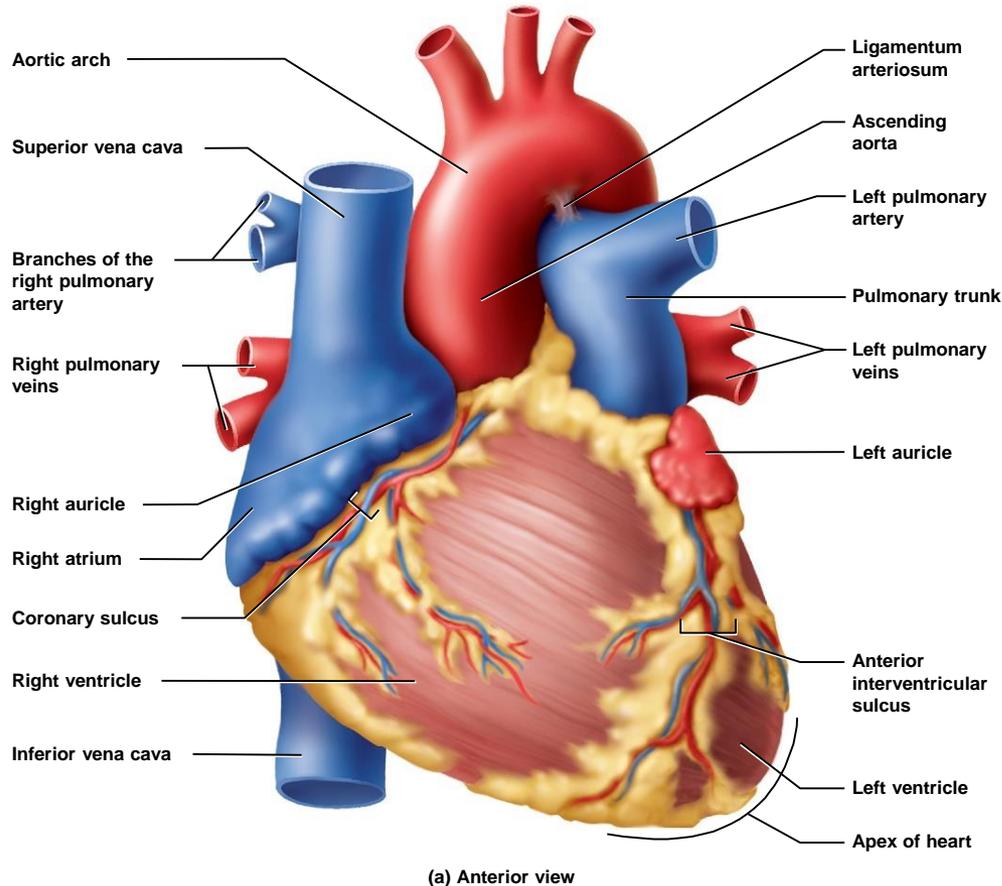


Figure 19.7

The Chambers

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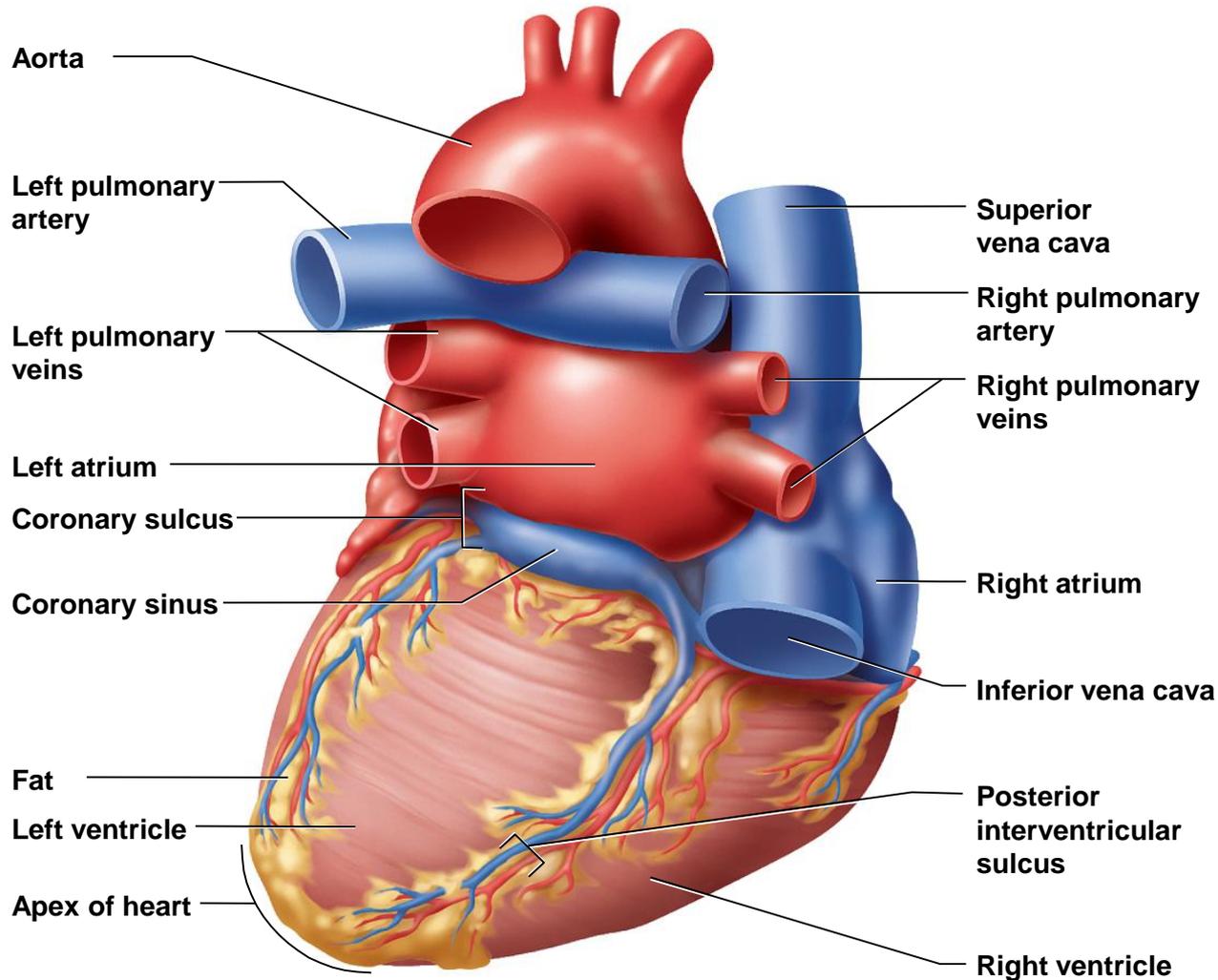
(a) Anterior view

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**

The Chambers

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(b) Posterior view

Figure 19.5b

The Chambers

- **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
 - May prevent ventricle walls from sticking together after contraction

The Chambers

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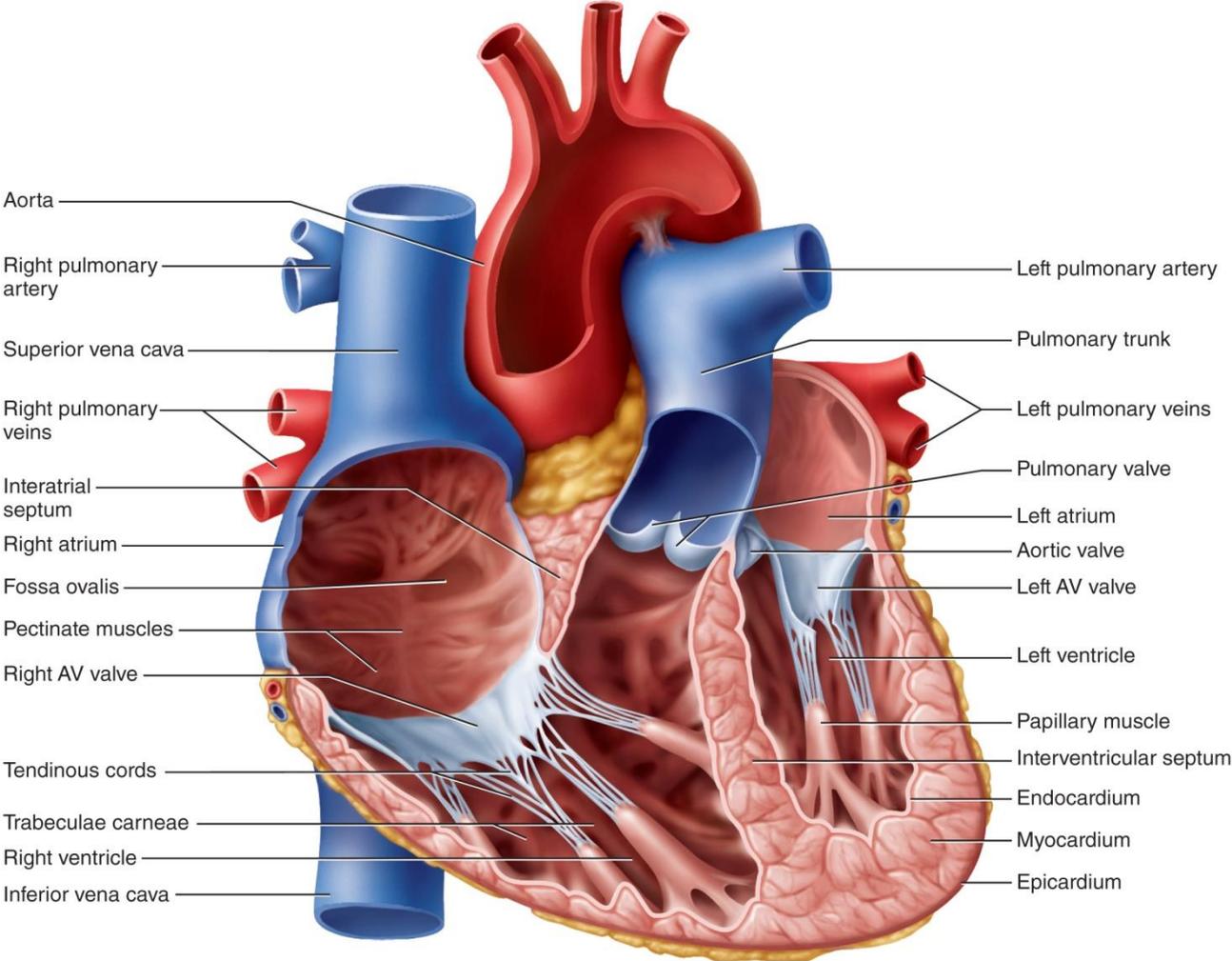
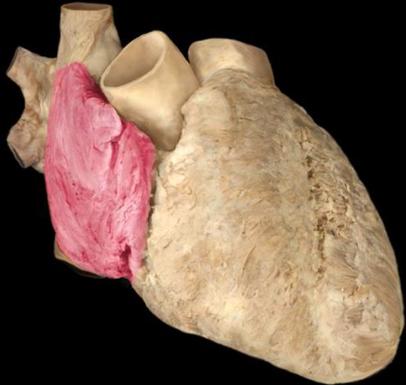


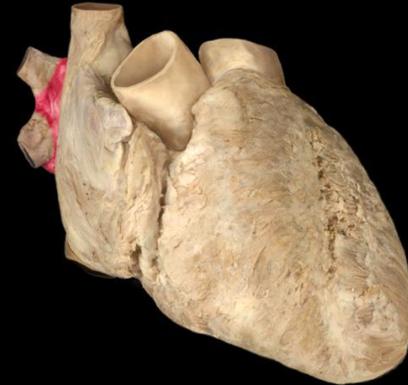
Figure 19.7

The Chambers – Anterior View

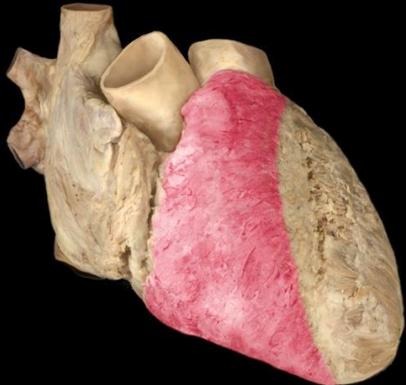
Rt. atrium



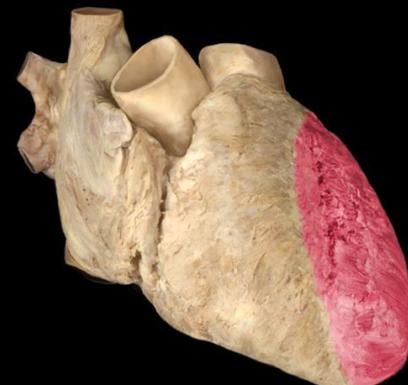
Lt. atrium



Rt. ventricle

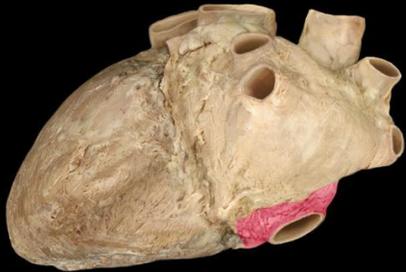


Lt. ventricle

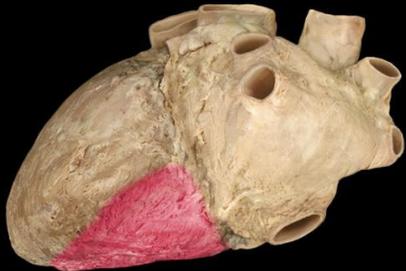
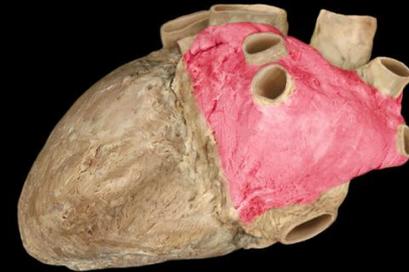


The Chambers – Posterior View

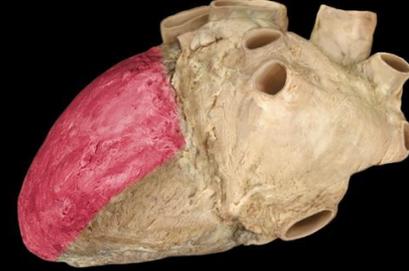
Rt. atrium



Lt. atrium



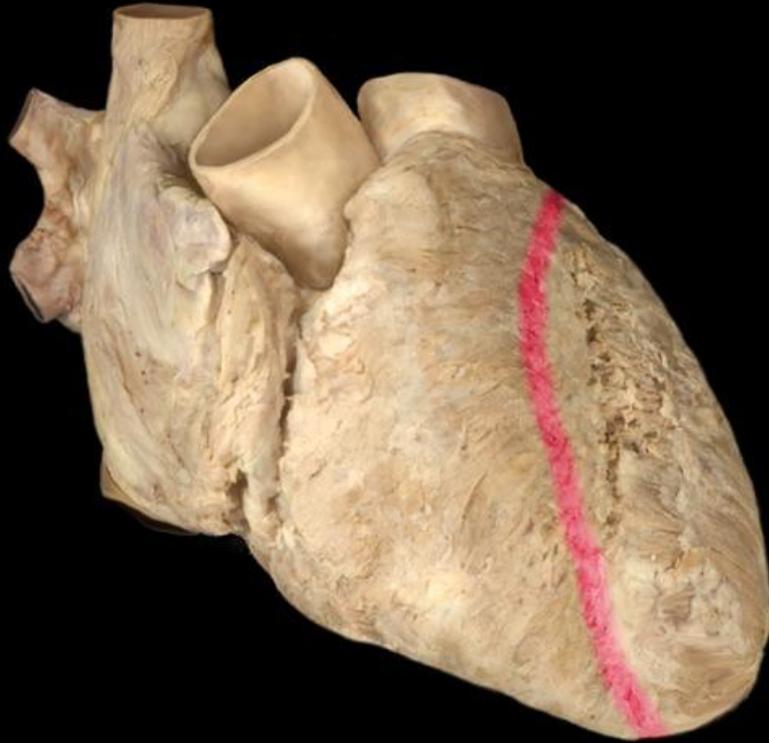
Rt. ventricle



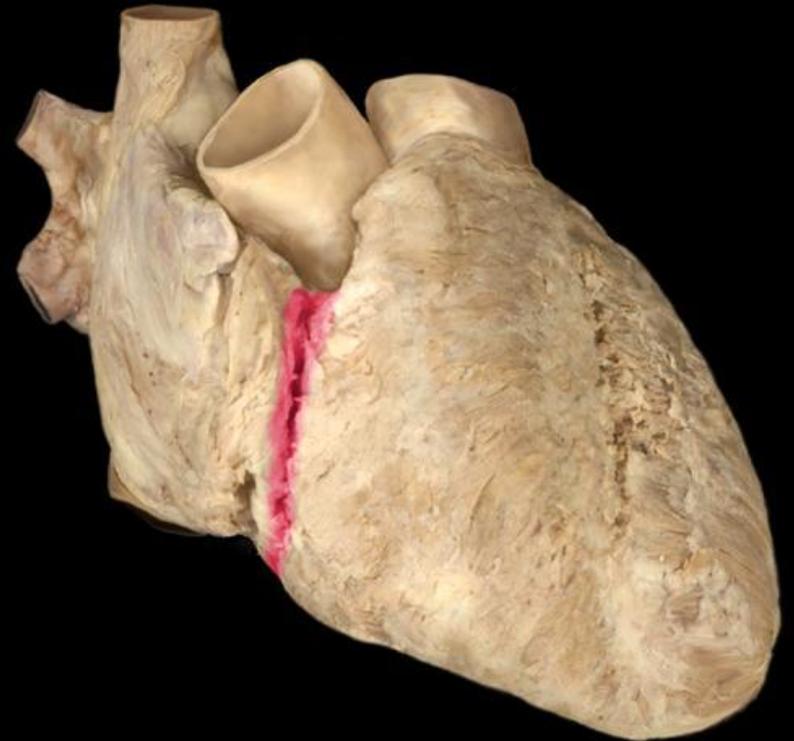
Lt. ventricle

Heart External Anatomy Sulci

Anterior interventricular sulcus

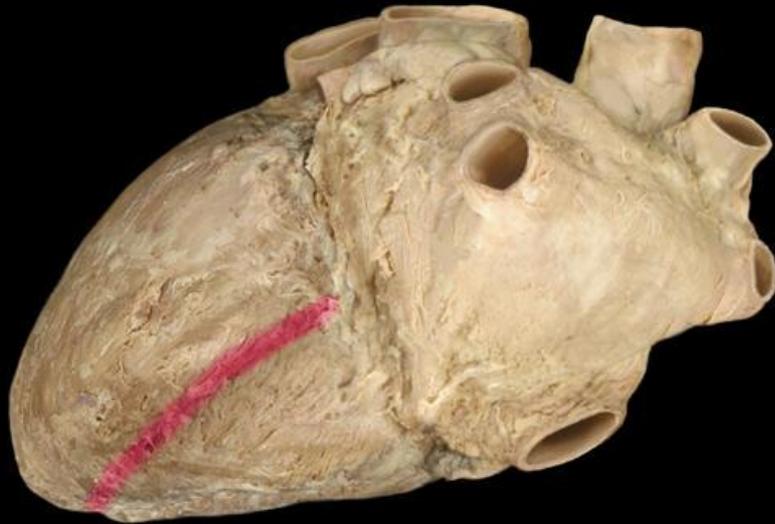


Coronary sulcus

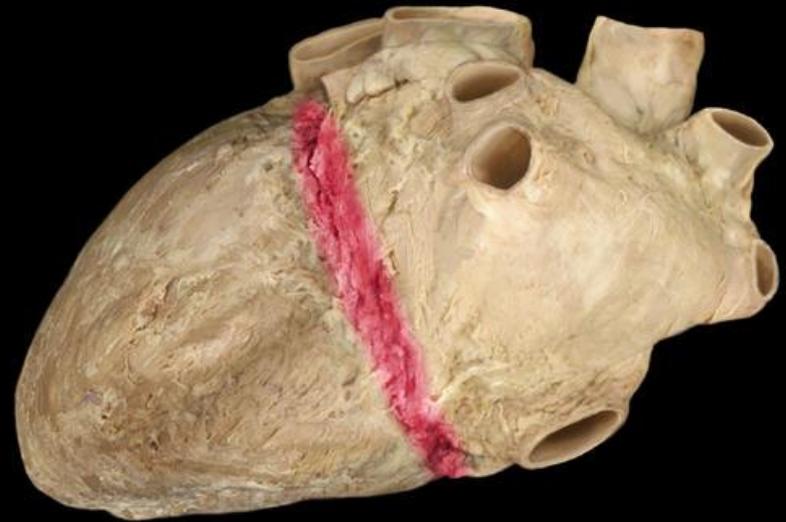


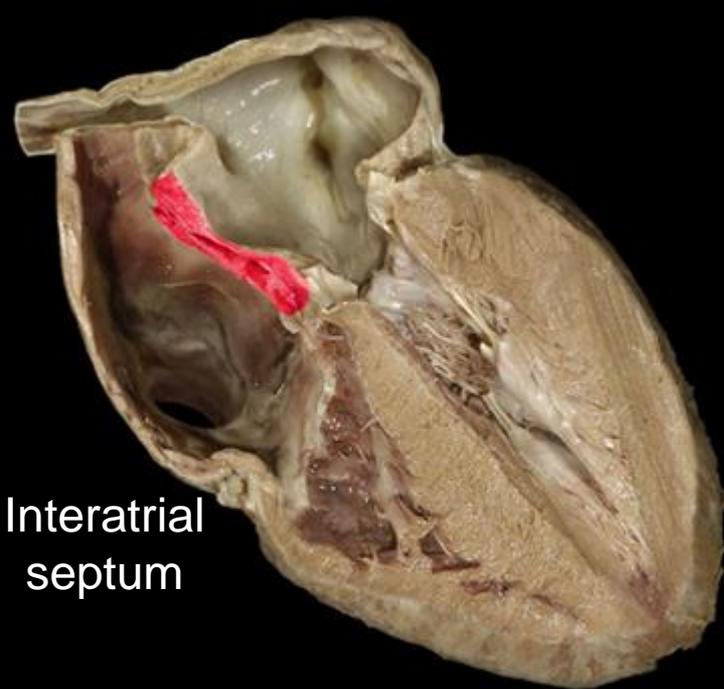
Heart External Anatomy Sulci

Posterior interventricular sulcus

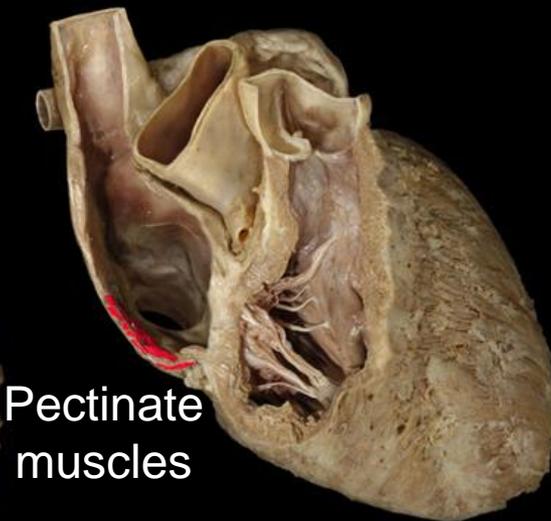


Posterior coronary sulcus

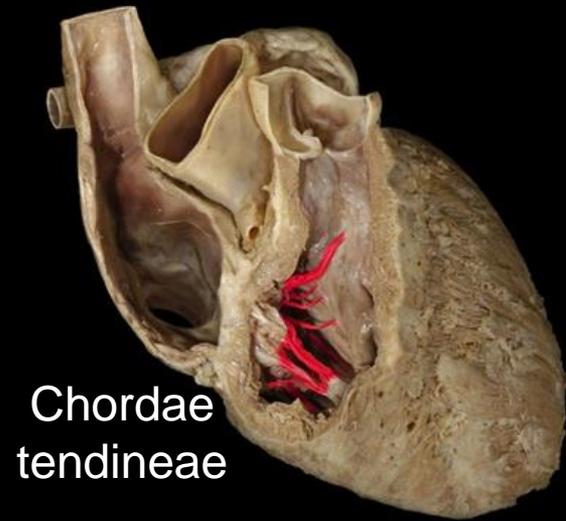




Interatrial
septum

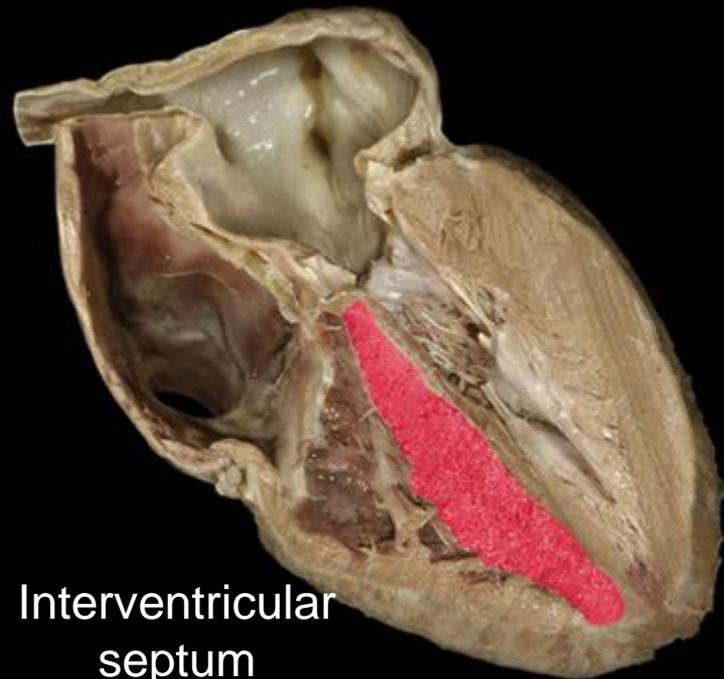


Pectinate
muscles

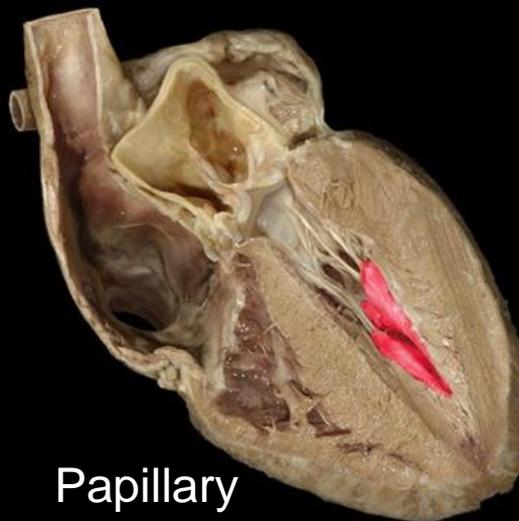


Chordae
tendineae

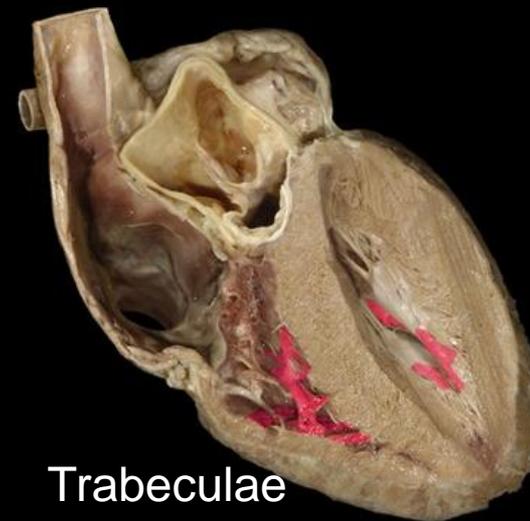
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Interventricular
septum



Papillary
muscles



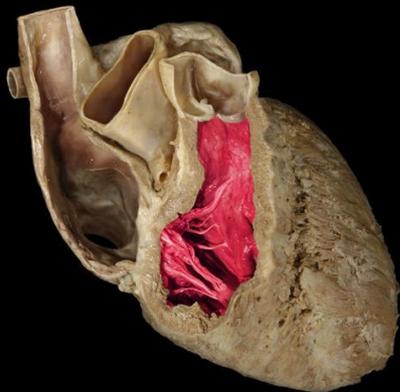
Trabeculae
carneae

The Heart Chambers Internal

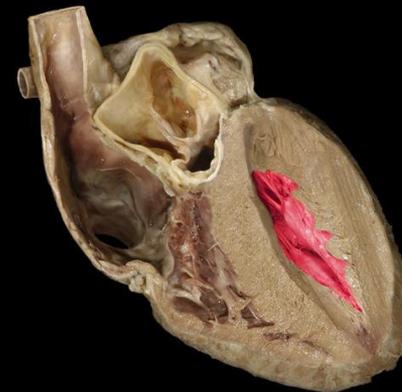
Rt. atrium



Lt. atrium

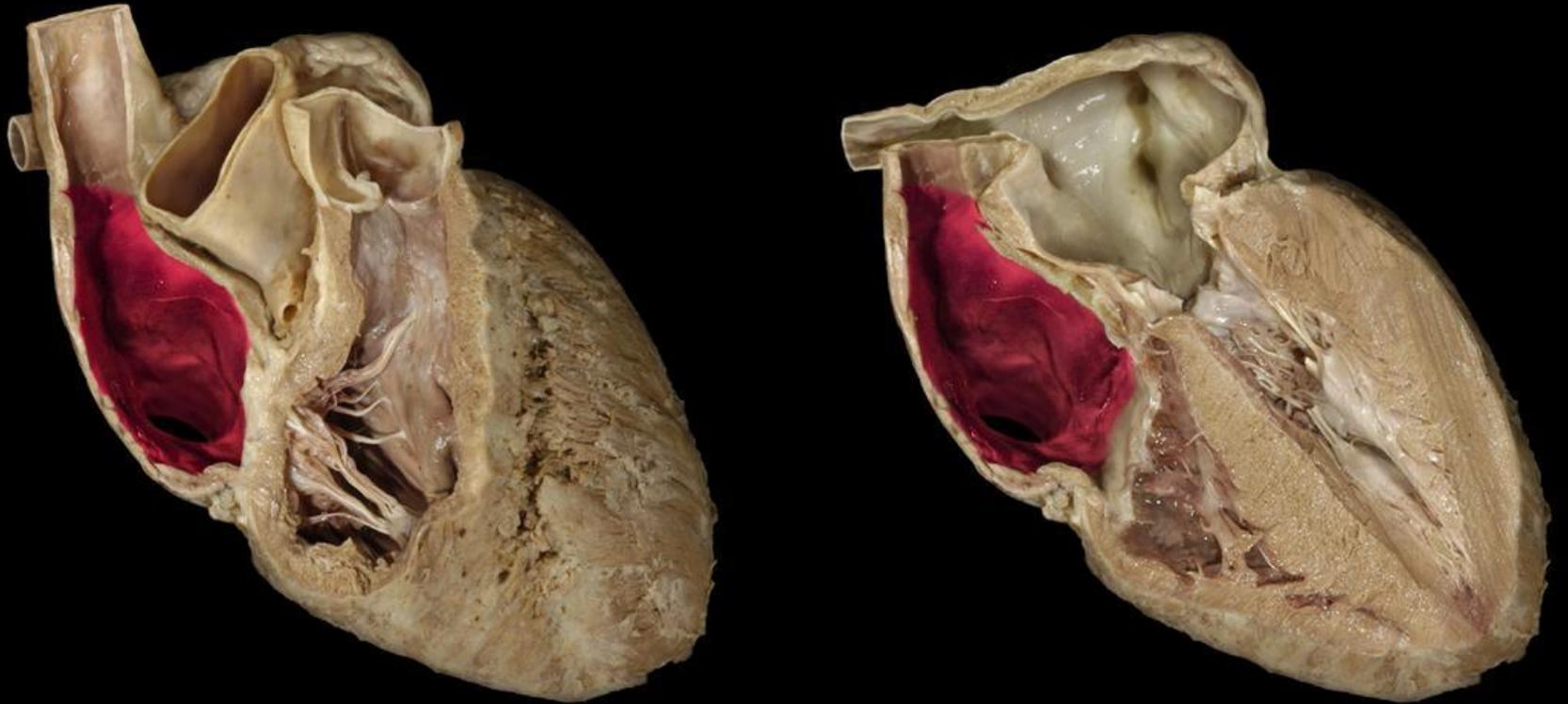


Rt. ventricle



Lt. ventricle

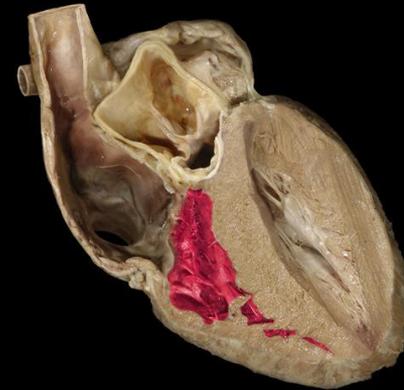
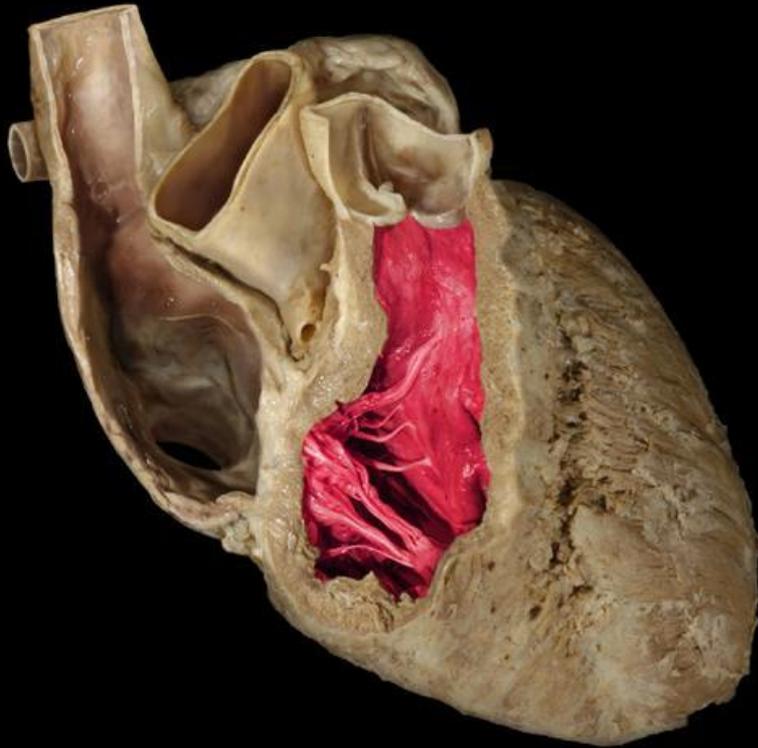
Rt. Atrium



Pectinate muscles and Interatrial septum

Rt. Ventricle

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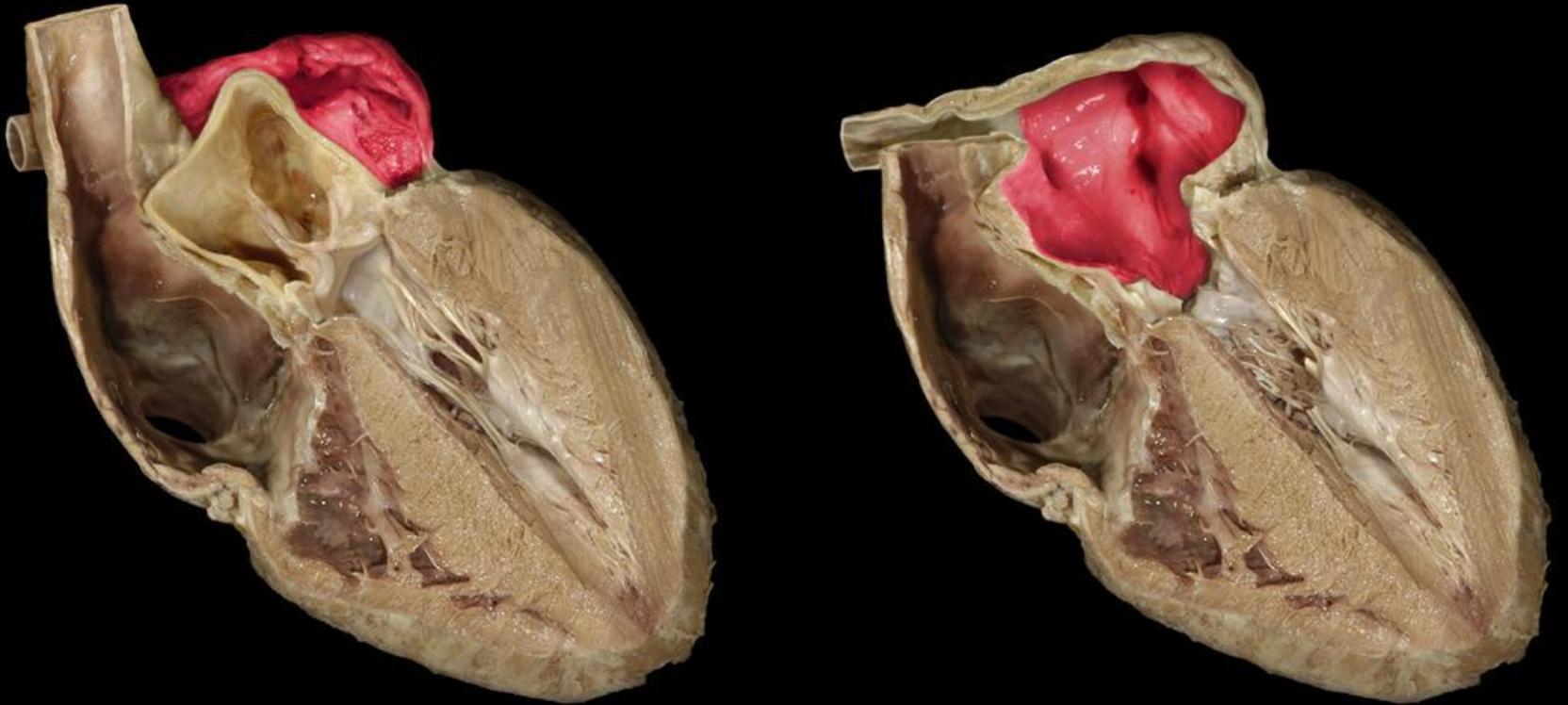


Papillary muscles

Interventricular septum

Trabeculae carne

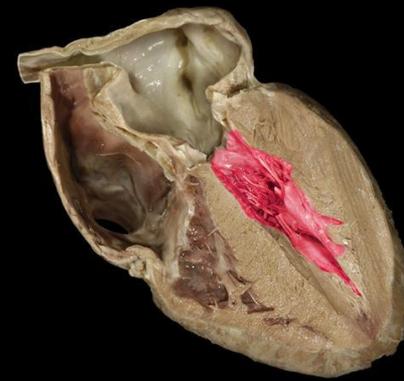
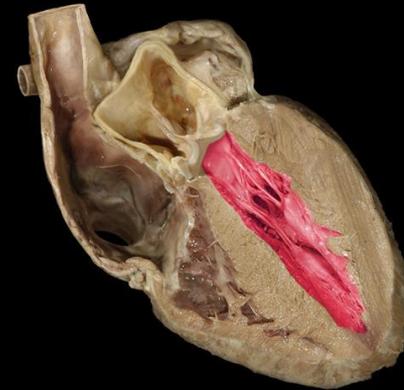
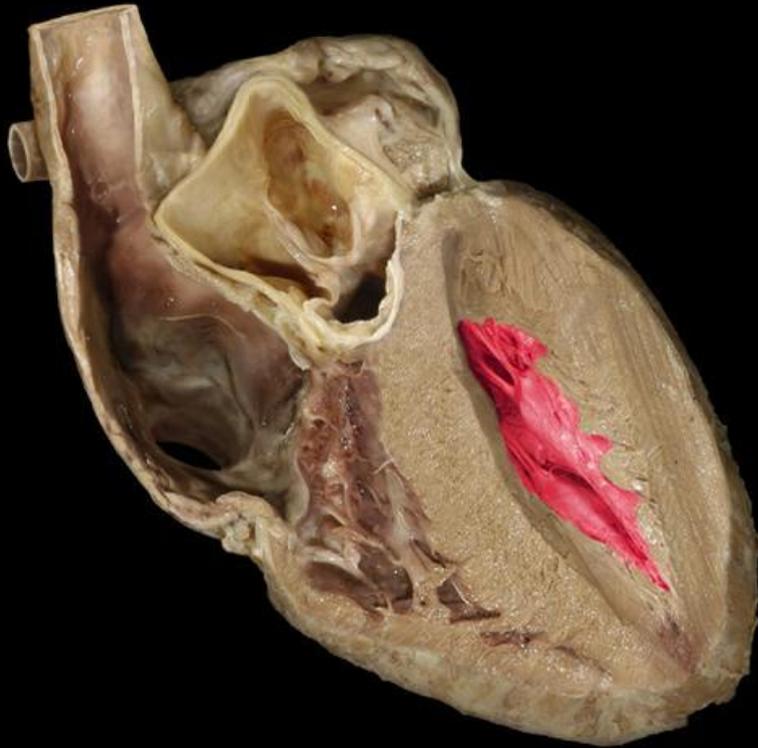
Lt. Atrium



Pectinate muscles and Interatrial septum

Lt. Ventricle

Photos © McGraw-Hill Education



Papillary muscles

Interventricular septum

Trabeculae carne

The Valves

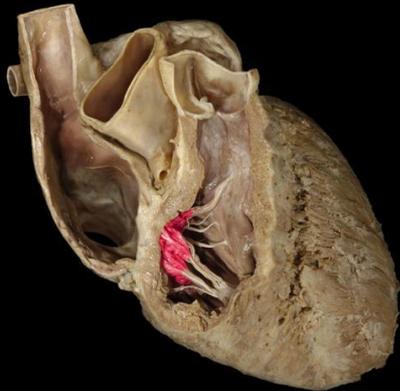
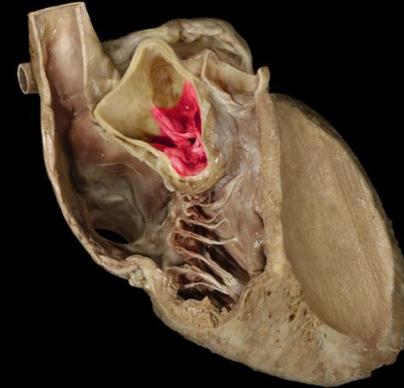
- **Valves ensure one-way flow of blood through heart**
- **Atrioventricular (AV) valves**—control blood flow between atria and ventricles
 - **Right AV valve** has three cusps (**tricuspid valve**)
 - **Left AV valve** has two cusps (**mitral valve, formerly ‘bicuspid’**)
 - **Chordae tendineae:** cords connect AV valves to **papillary muscles** on floor of ventricles
 - Prevent AV valves from flipping or bulging into atria when ventricles contract
 - Each papillary muscle has 2-3 attachments to heart floor (like Eiffel Tower) to distribute physical stress, coordinate timing of electrical conduction, and provide redundancy

The Heart Valves

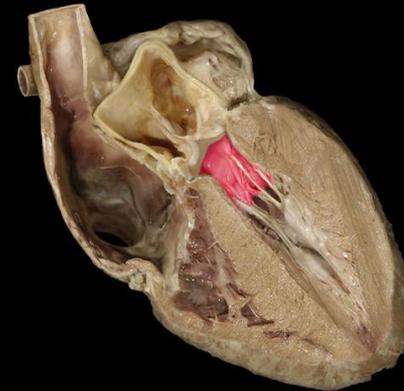
Pulmonary semilunar



Aortic semilunar

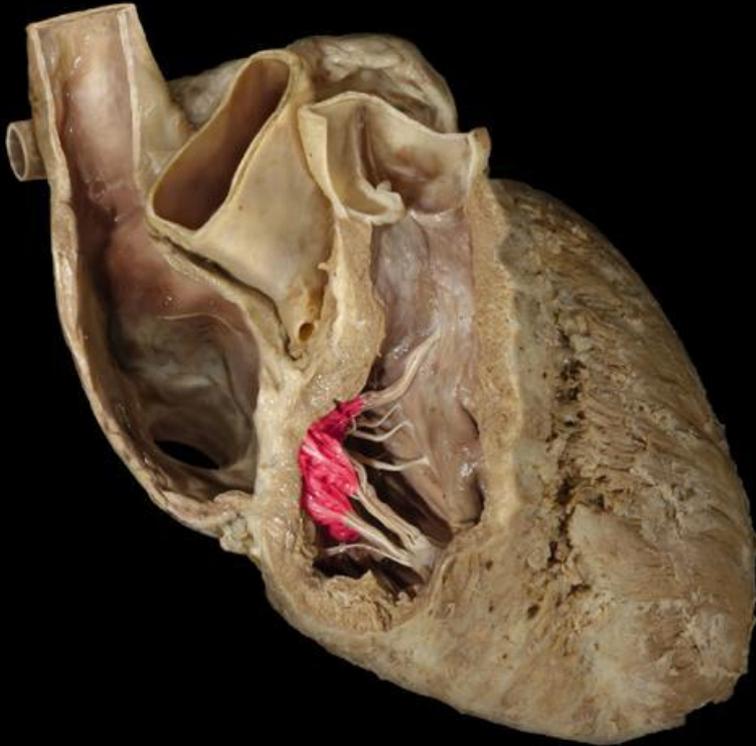


Tricuspid

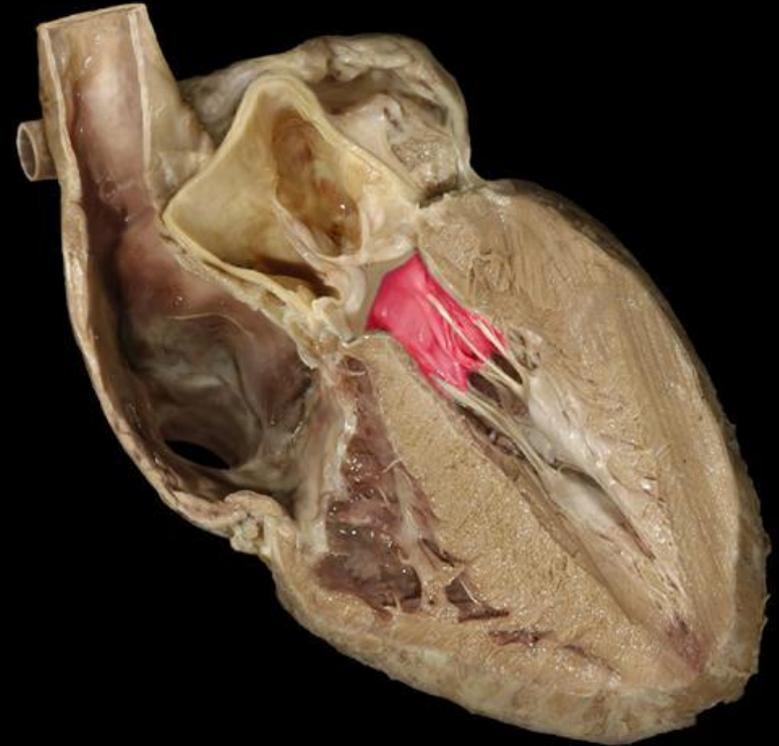


Bicuspid

Atrioventricular Valves

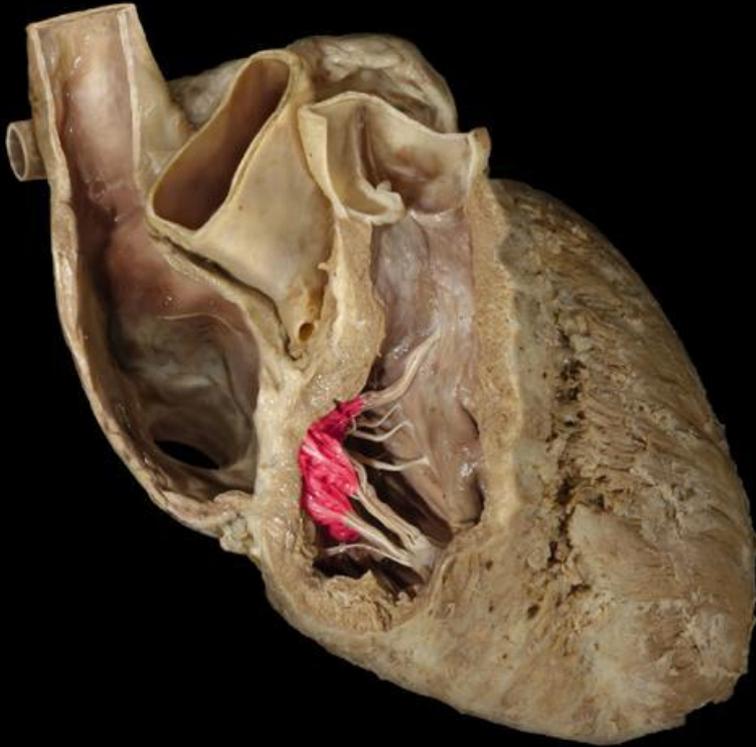


Tricuspid

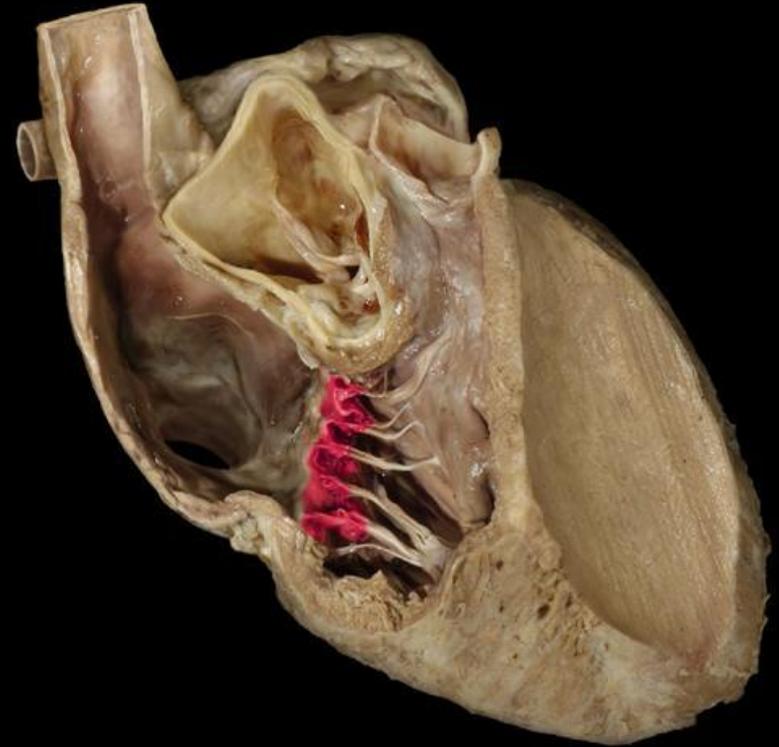


Bicuspid (Mitral)

Tricuspid Valve

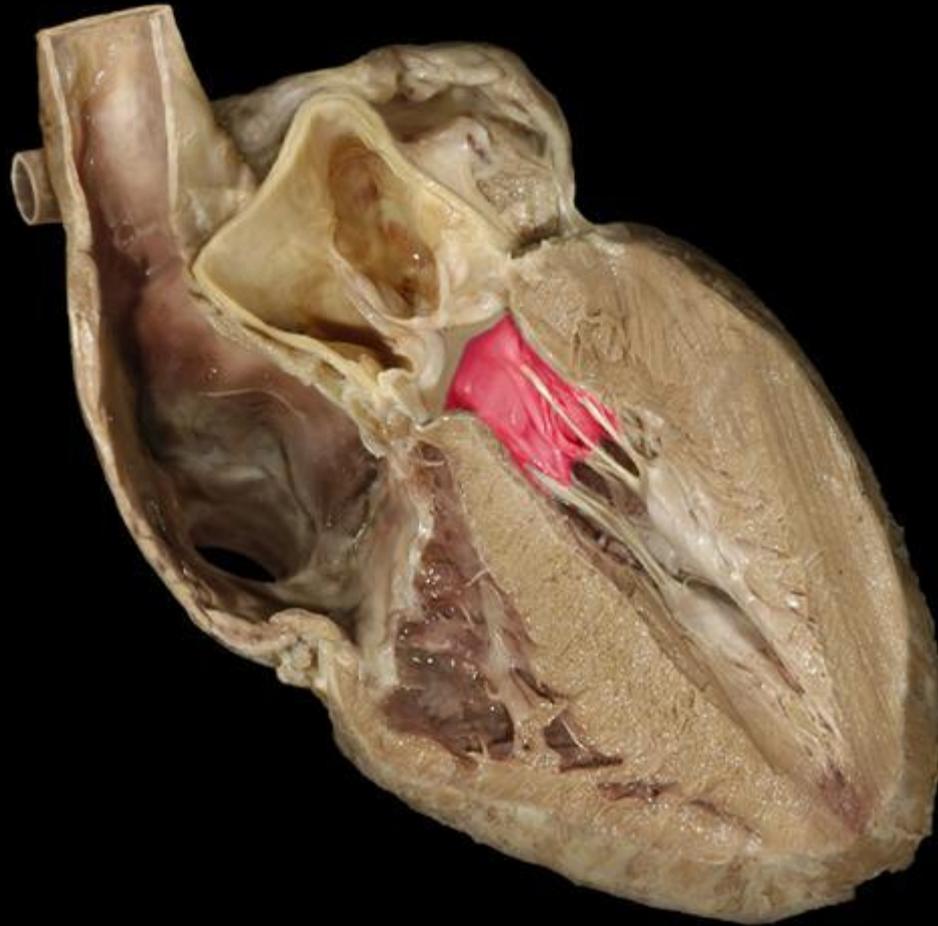


Chordae tendineae



Papillary muscles

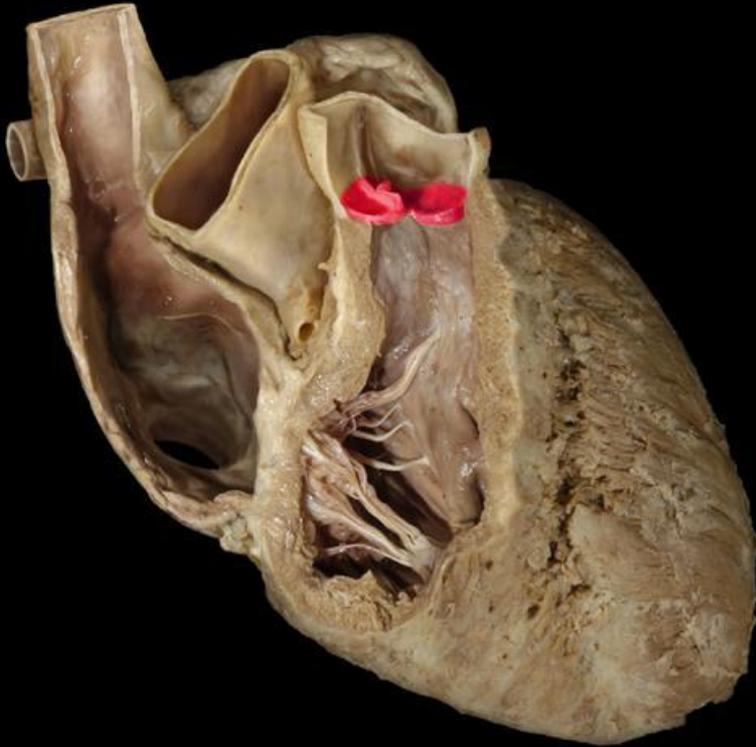
Mitral Valve



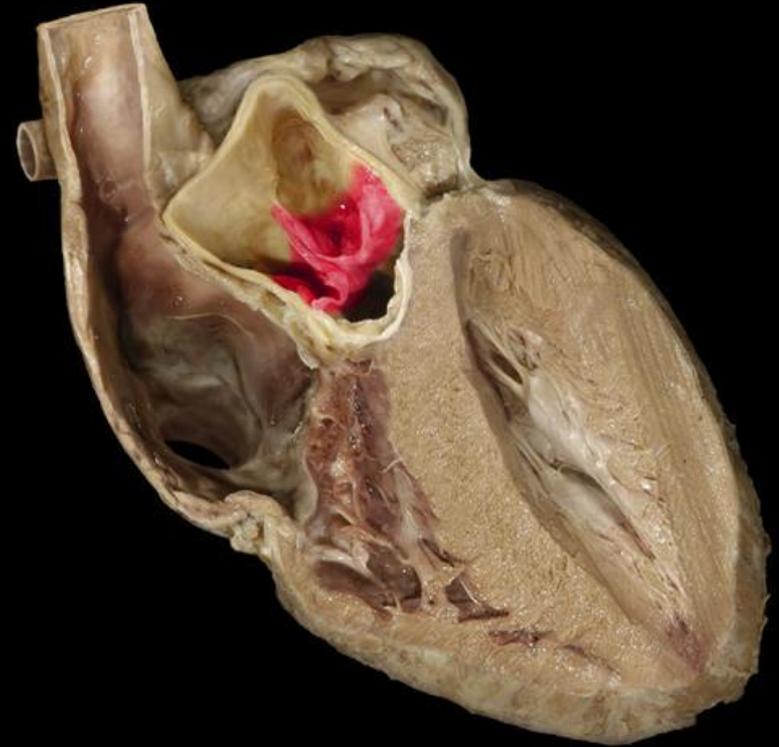
The Valves

- **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

Semilunar Valves

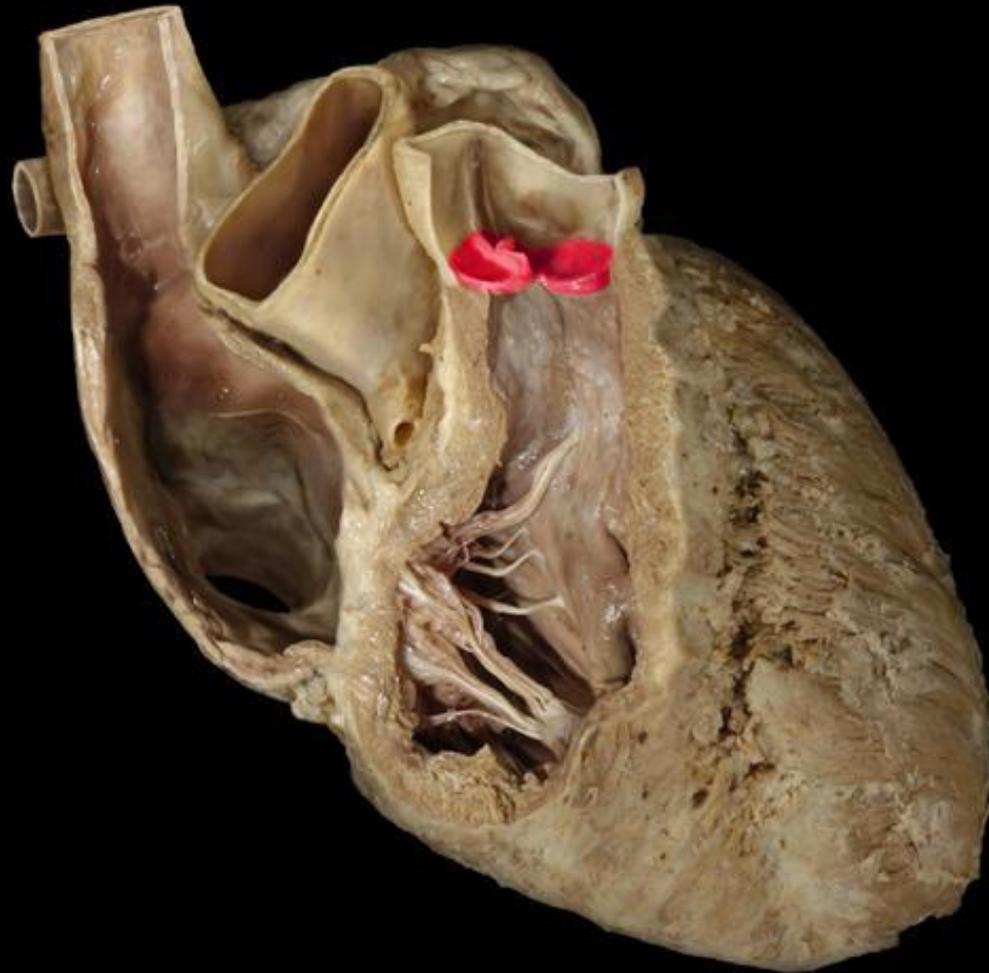


Pulmonary

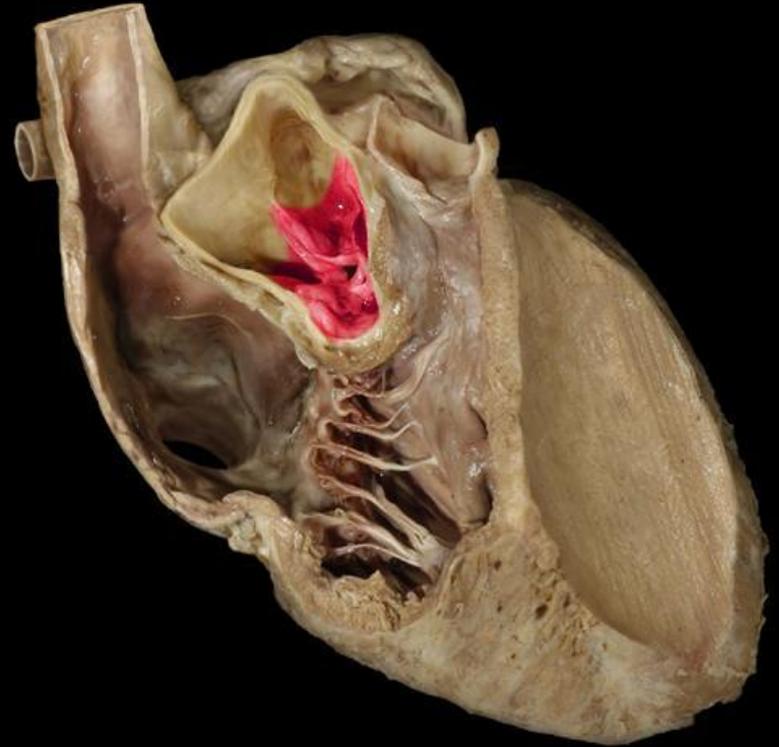
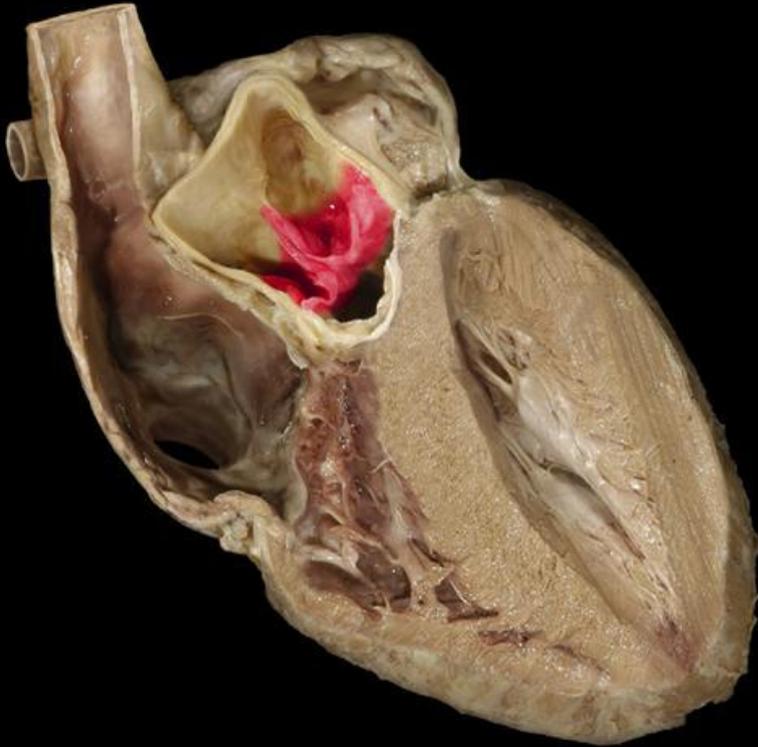


Aortic

Pulmonary Semilunar Valve

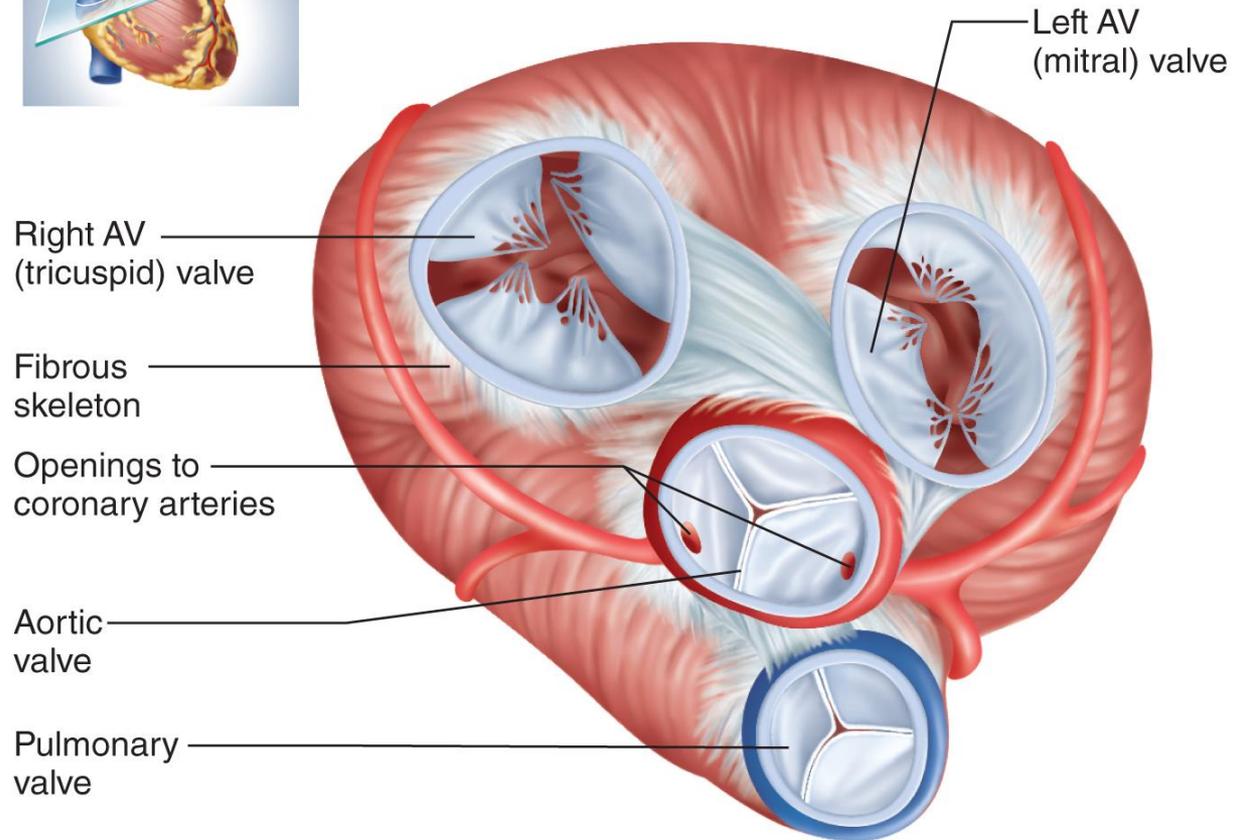
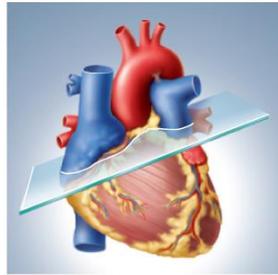


Aortic Semilunar Valve



The Valves

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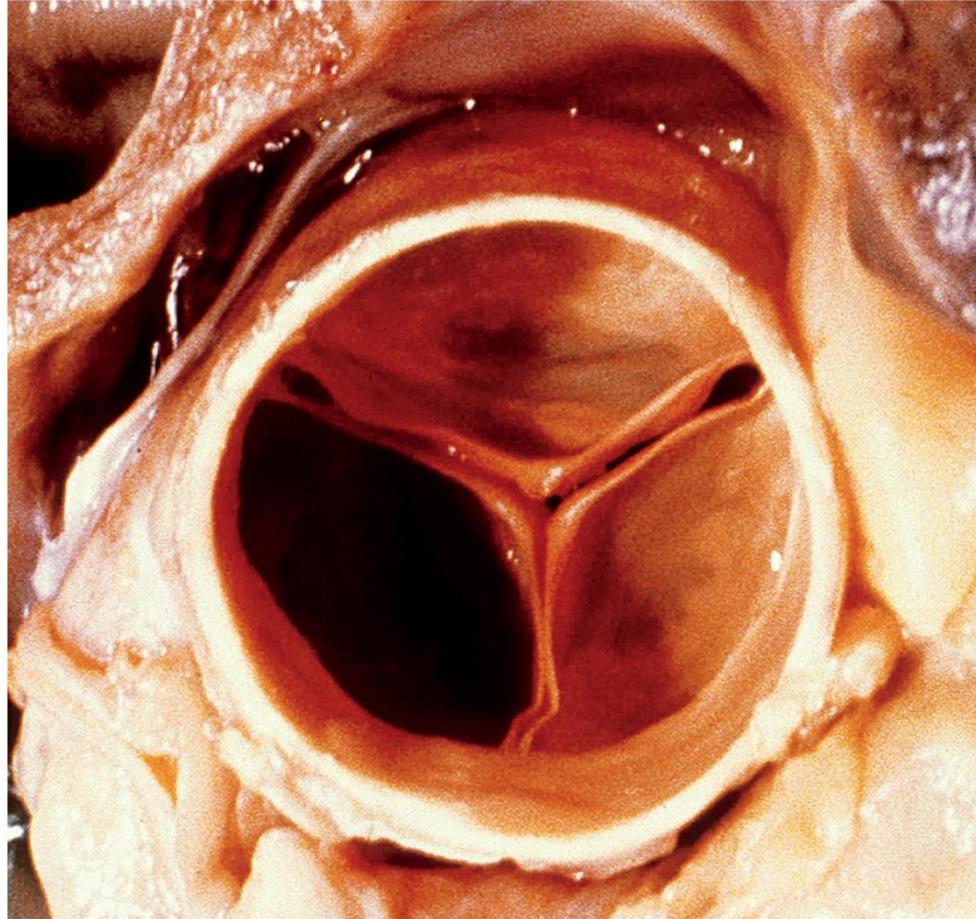


(a)

Figure 19.8a

Aortic Valve

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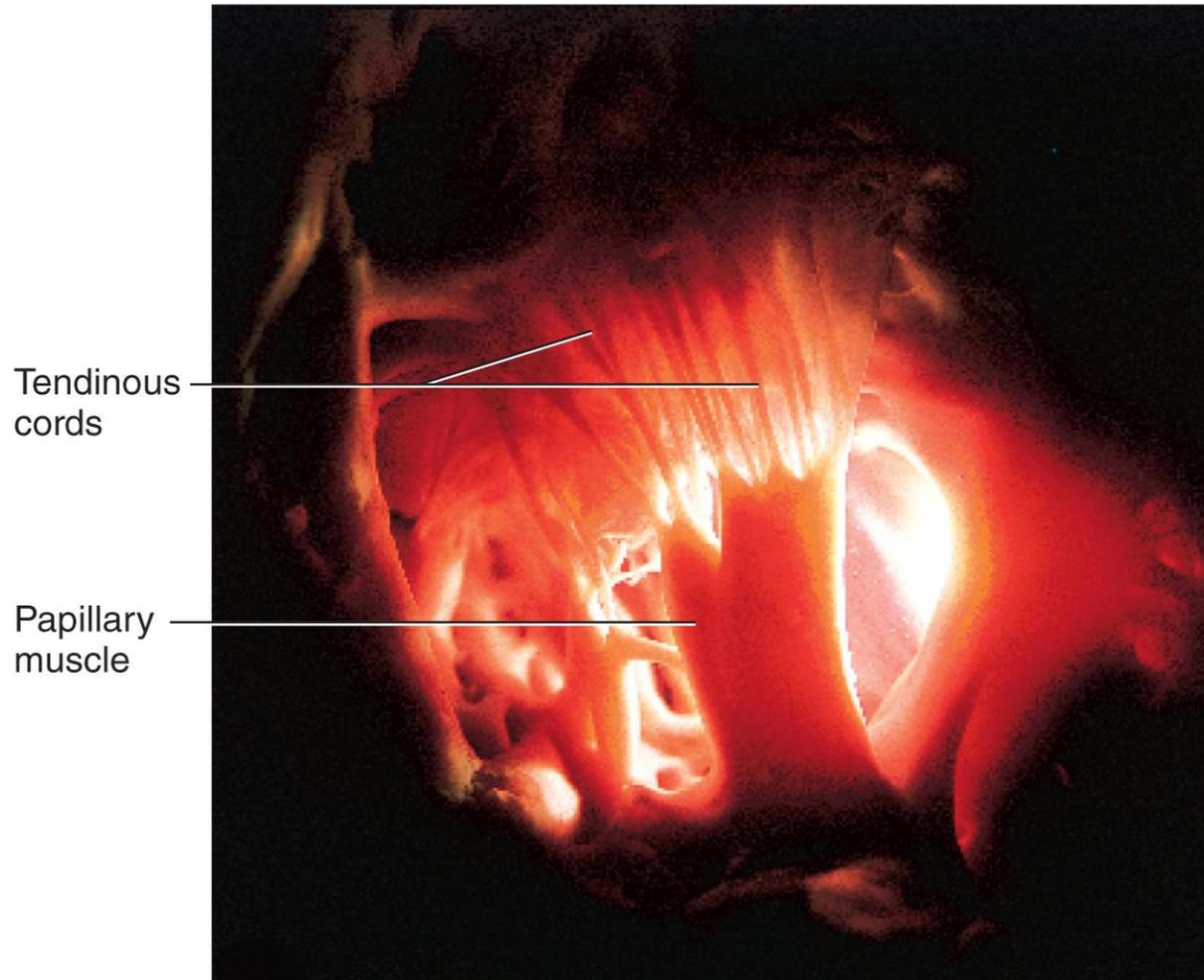
(b)

©Photo Researchers/Biophoto Associates/Getty Images

Figure 19.8b

The Tricuspid Valve

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(c)

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Figure 19.8c

Blood Flow Through the Chambers

- **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

Blood Flow Through the Chambers

- **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 the Chambers

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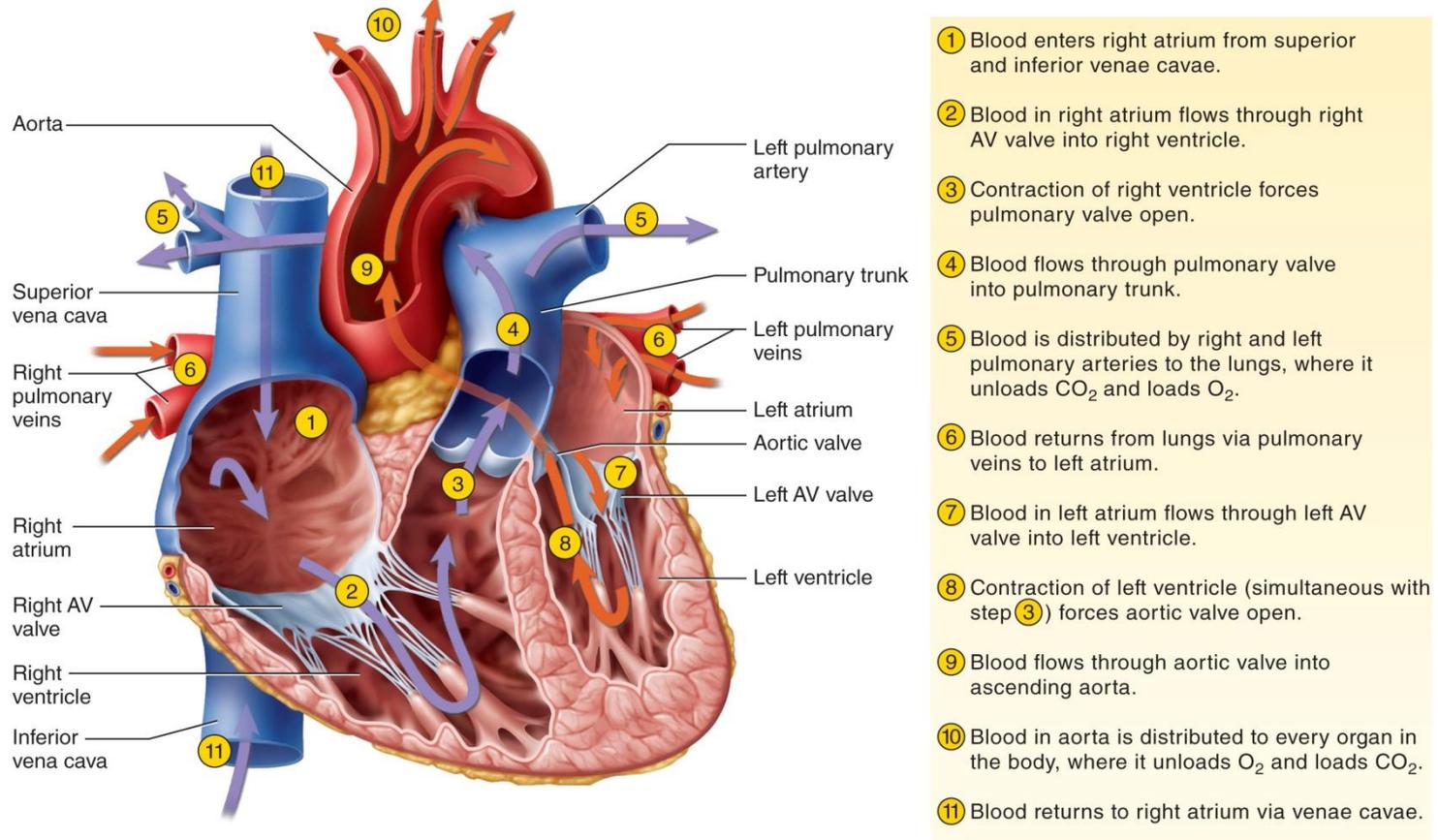


Figure 19.9

- Blood pathway travels from the right atrium through the pulmonary circuit, then systemic circuit (through the body) and back to the starting point

The 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 O₂ and nutrients

The Coronary Circulation

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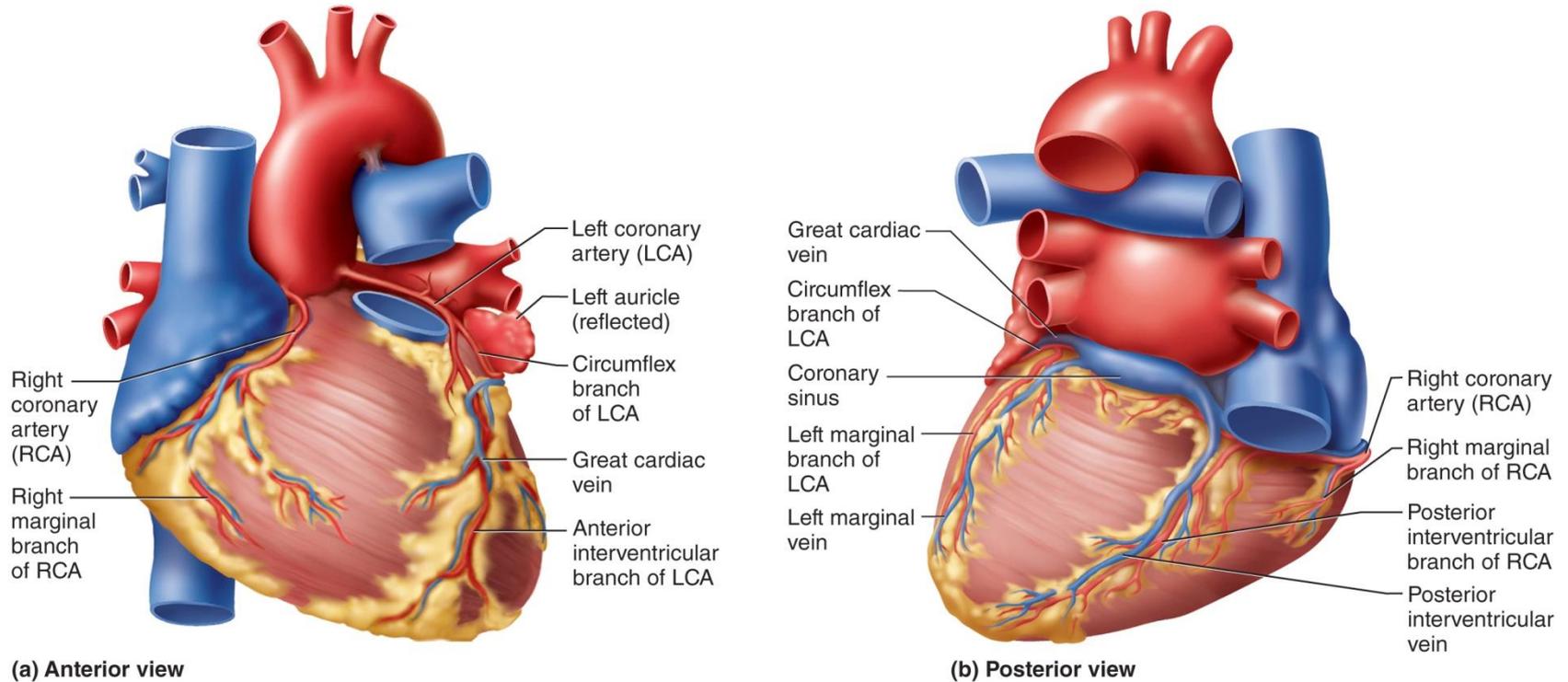


Figure 19.10a,b

Rt. & Lt Coronary Arteries

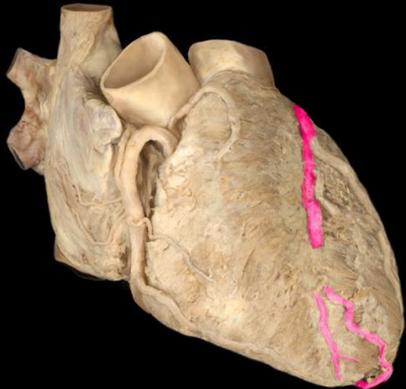
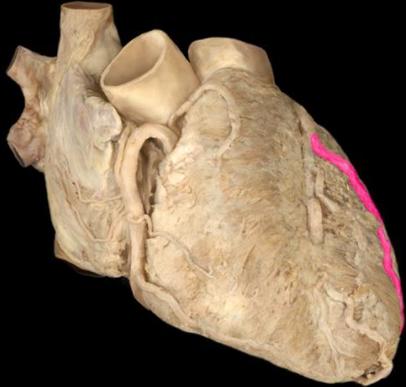


Arterial Supply

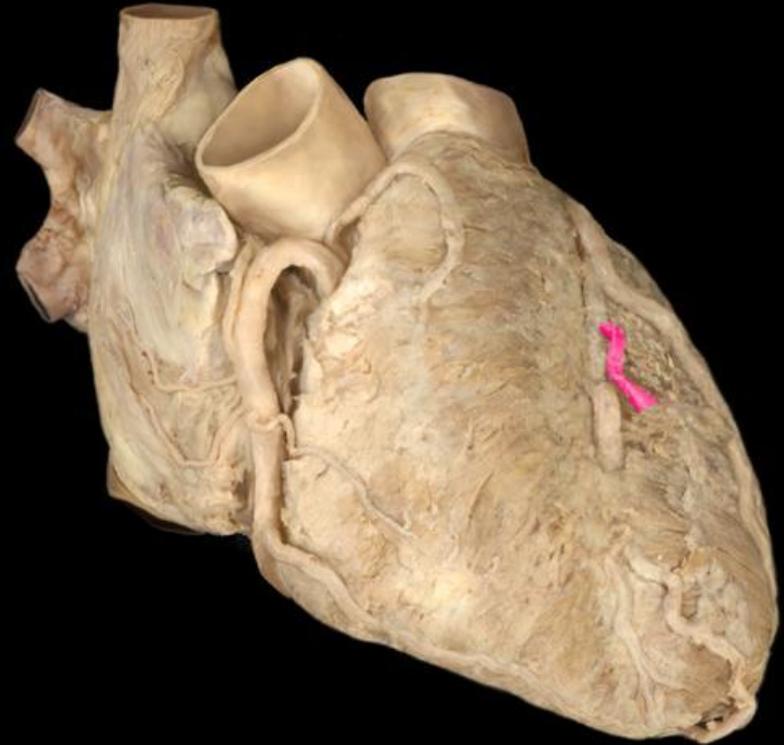
- **Left coronary artery (LCA)** branches off the ascending aorta
 - **Anterior interventricular branch**
 - Supplies blood to both ventricles and anterior two-thirds of the interventricular septum
 - **Circumflex branch**
 - Passes around left side of heart in coronary sulcus
 - Gives off **left marginal branch** and then ends on the posterior side of the heart
 - Supplies left atrium and posterior wall of left ventricle

Lt. Coronary Artery Branches Anterior

Lt. marginal a.



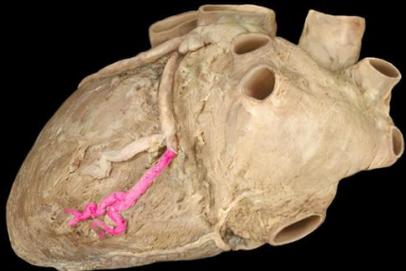
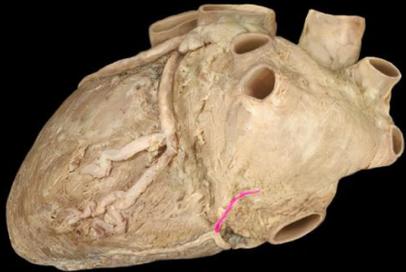
Lt. interventricular a.



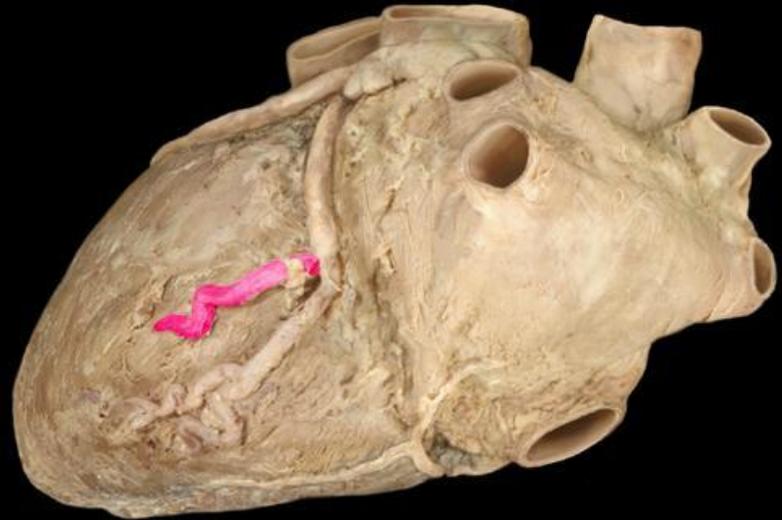
Lt. ant. ventricular a.

Lt. Coronary Artery Branches Posterior

Lt. atrial a.



Lt. post. ventricular a.

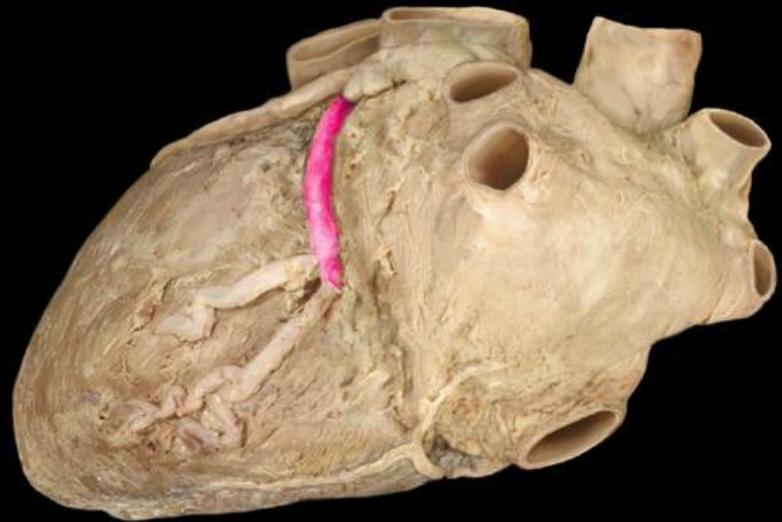


Post. interventricular a.

Lt. Coronary Artery Branches Posterior



Lt. marginal a.



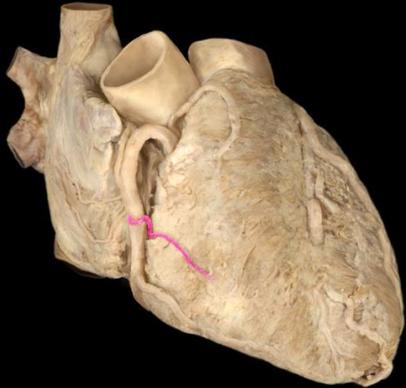
Posterior circumflex a.

Arterial Supply

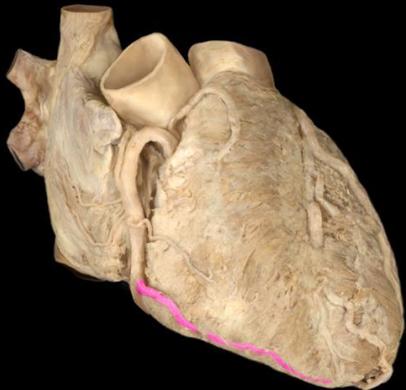
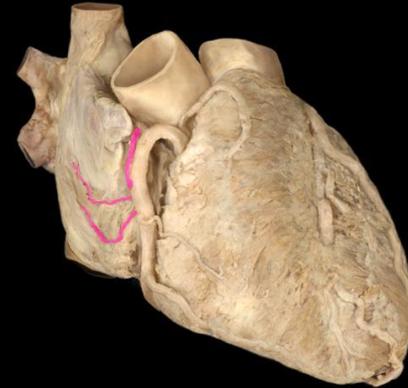
- **Right coronary artery (RCA)** branches off the ascending aorta
 - Supplies right atrium and sinoatrial node (pacemaker)
 - **Right marginal branch**
 - Supplies lateral aspect of right atrium and ventricle
 - **Posterior interventricular branch**
 - Supplies posterior walls of ventricles

Rt. Coronary Artery Branches Anterior

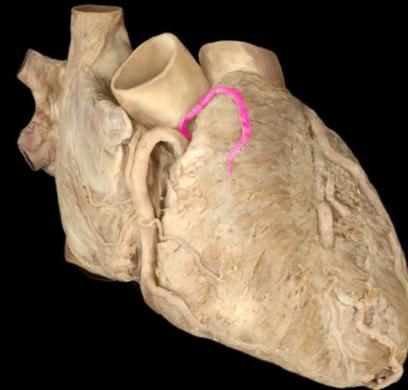
Rt. ant. ventricular a.



Rt. atrial a.

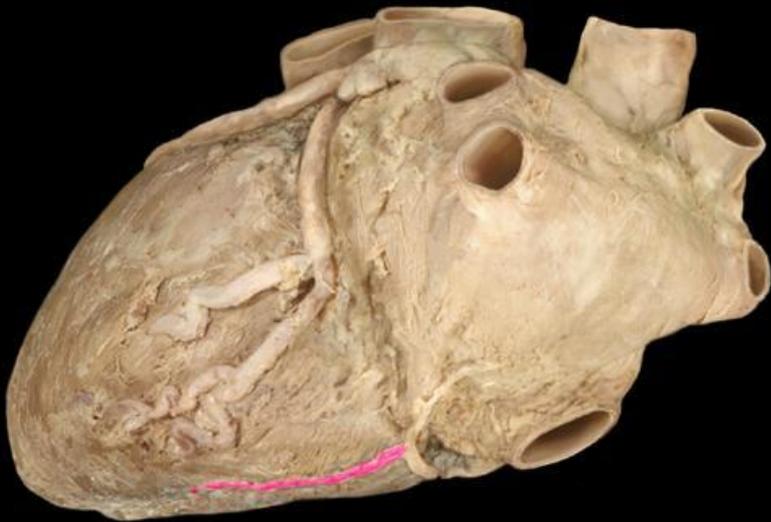


Rt. marginal a.

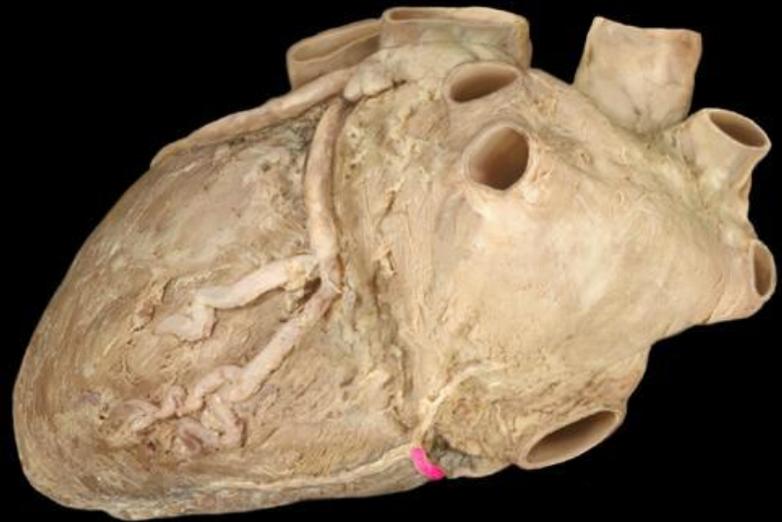


Rt. conus a.

Rt. Coronary Artery Branches Posterior



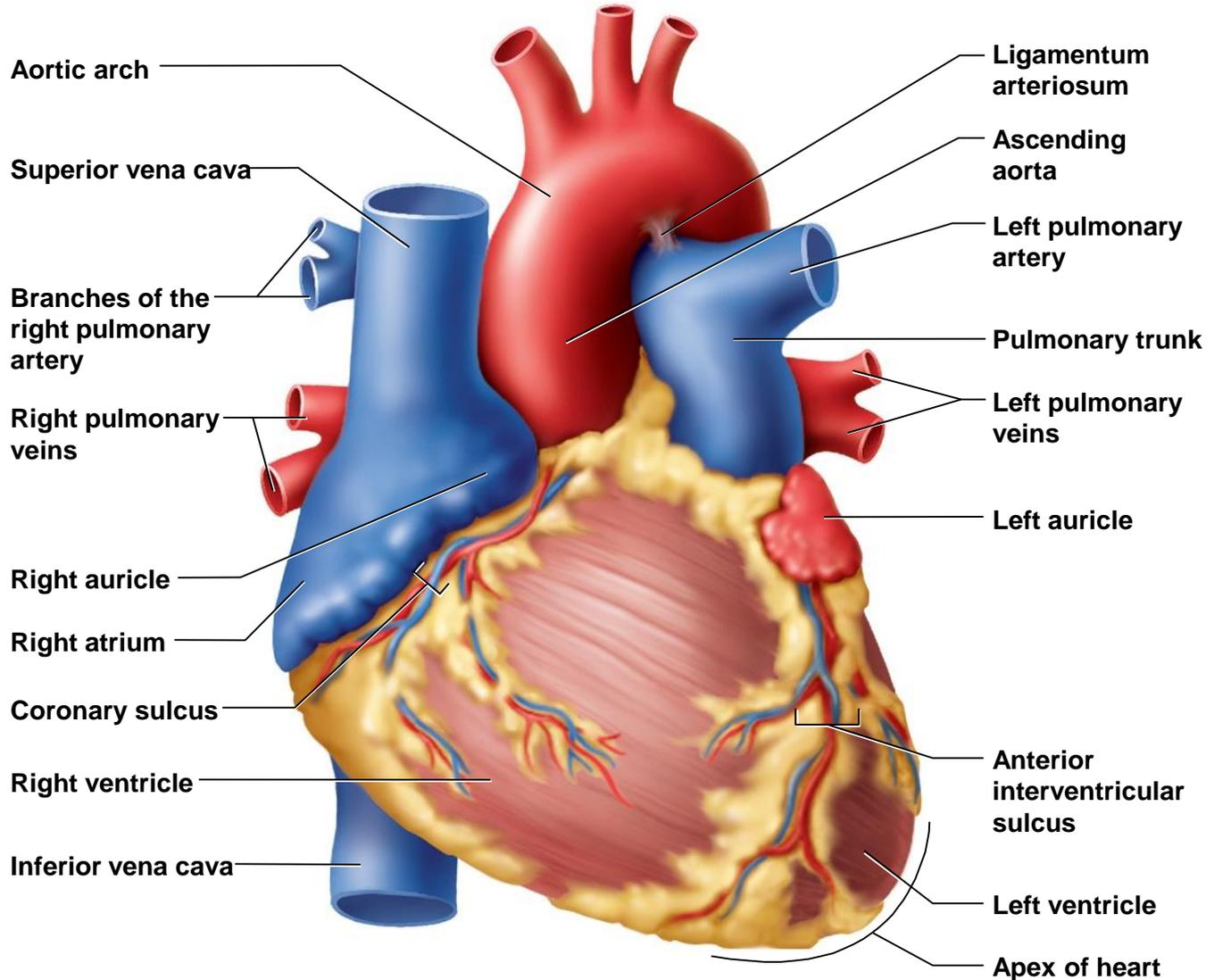
Rt. post. ventricular a.



Rt. coronary a.

Arterial Supply

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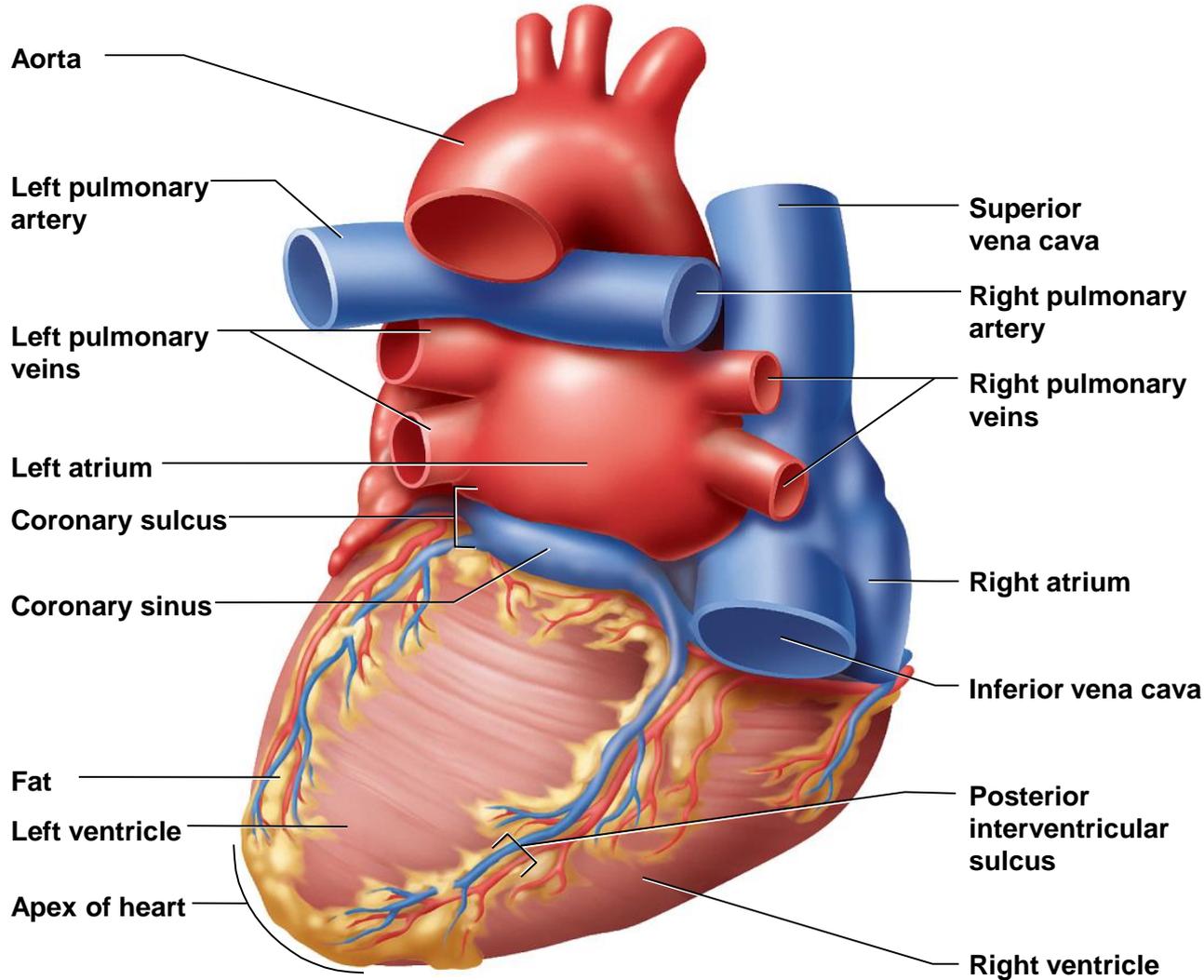


(a) Anterior view

Figure 19.5a

Arterial Supply

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(b) Posterior view

Figure 19.5b

Arterial Supply

- **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 provide alternative routes of blood flow (**collateral circulation**) within the myocardium

Arterial Supply

- **Flow through coronary arteries is greatest when heart relaxes**
 - 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

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 and thus stimulating pain

Angina and Heart Attack

- **Myocardial infarction (MI)**—sudden death of a patch of myocardium resulting from long-term obstruction of coronary circulation
 - Atheroma (blood clot or fatty deposit) often obstructs 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, leading to fibrillation and cardiac arrest
 - Silent heart attacks occur in diabetics and the elderly
 - MI responsible for about 27% of all deaths in the U.S.

Venous Drainage

- 5% to 10% of coronary blood drains directly into heart chambers (mostly right ventricle) by way of the **thebesian veins**
- Most coronary blood returns to right atrium by way of the **coronary sinus** which has three main inputs: great cardiac, posterior interventricular, and left marginal veins
 - **Great cardiac vein**
 - Travels alongside anterior interventricular artery
 - Collects blood from anterior portion of heart
 - Empties into coronary sinus

Venous Drainage

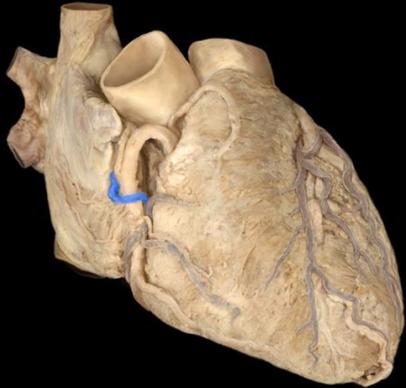
(Continued)

- **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

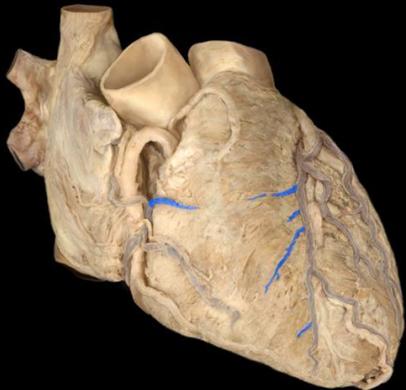
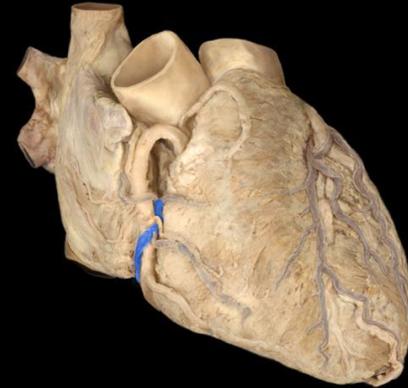
Coronary Vein Branches

Right Anterior

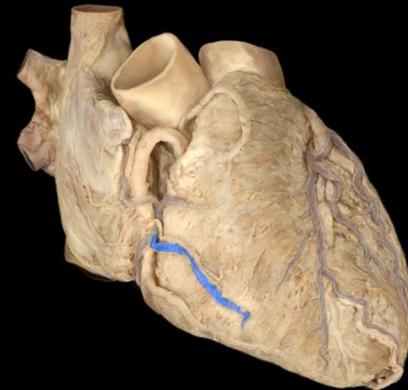
Rt. atrial v.



Small cardiac vein



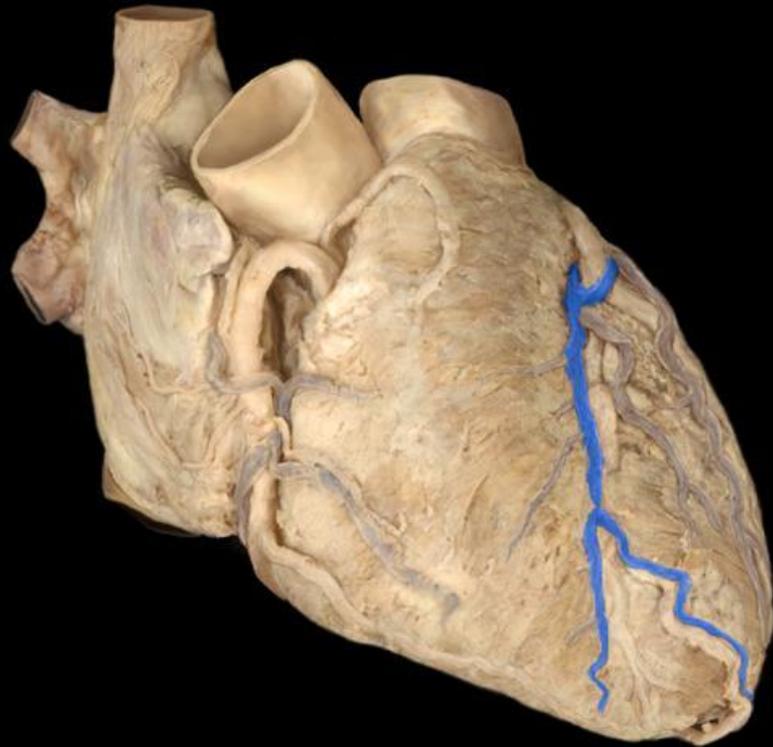
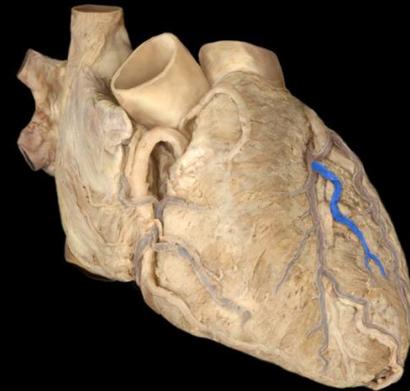
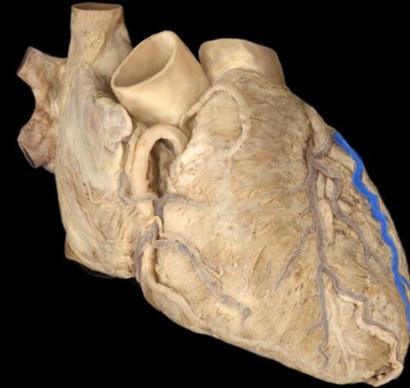
Rt. ant. ventricular v.



Rt. marginal v.

Coronary Vein Branches Left Anterior

Lt. marginal v.

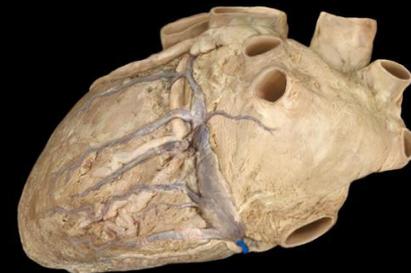
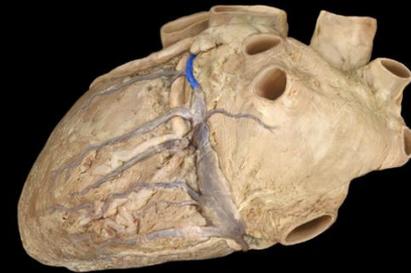


Great cardiac v.

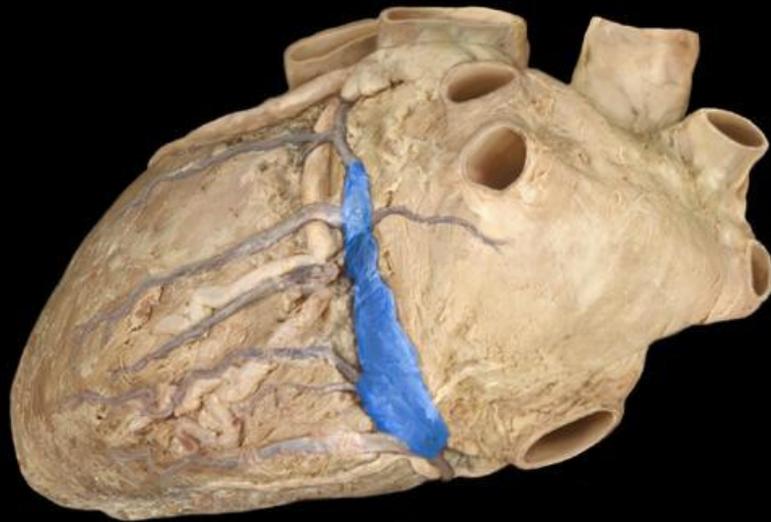
Lt. ant. ventricular vein

Coronary Vein Branches Posterior

Great cardiac vein

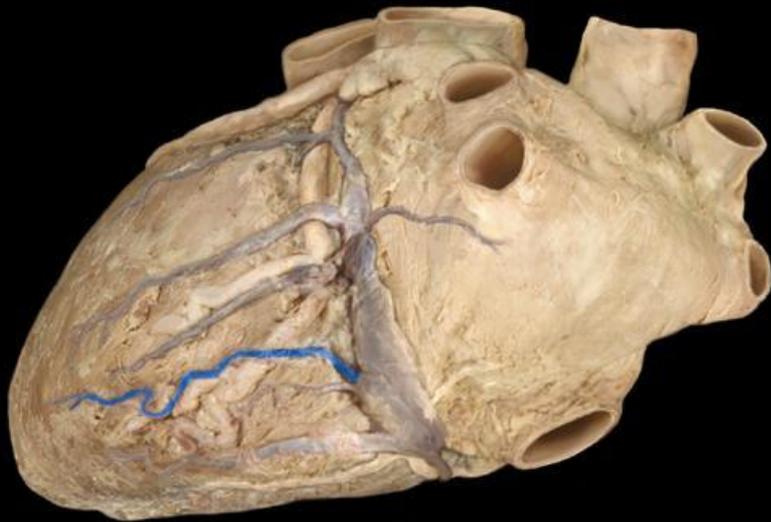


Small cardiac vein

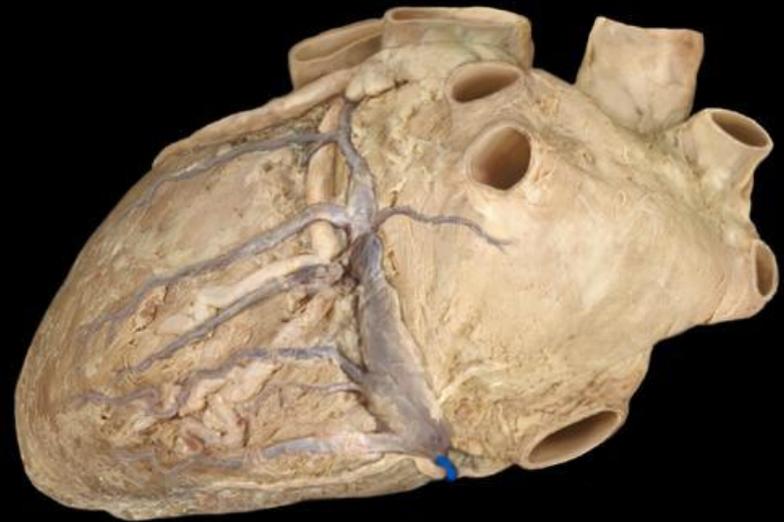


Coronary sinus

Coronary Vein Branches Posterior

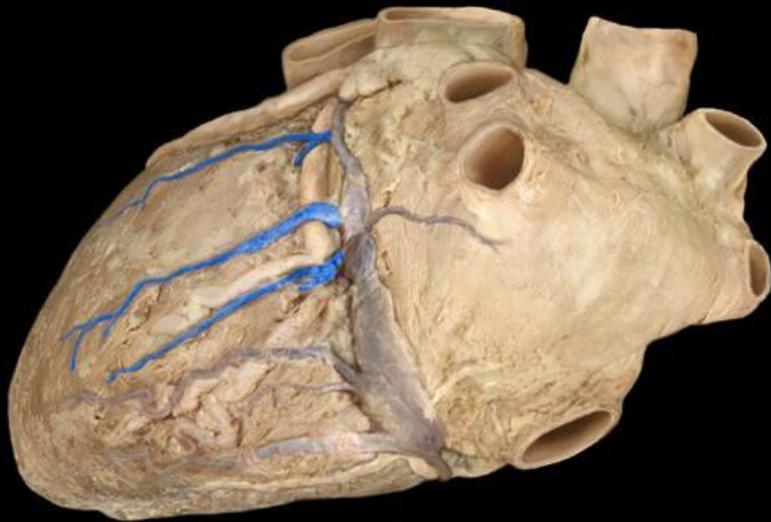


Middle cardiac vein

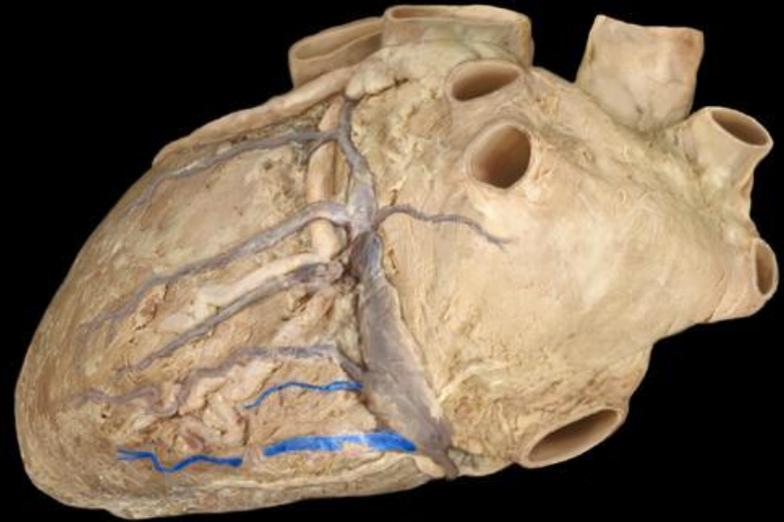


Small cardiac vein

Coronary Vein Branches Posterior

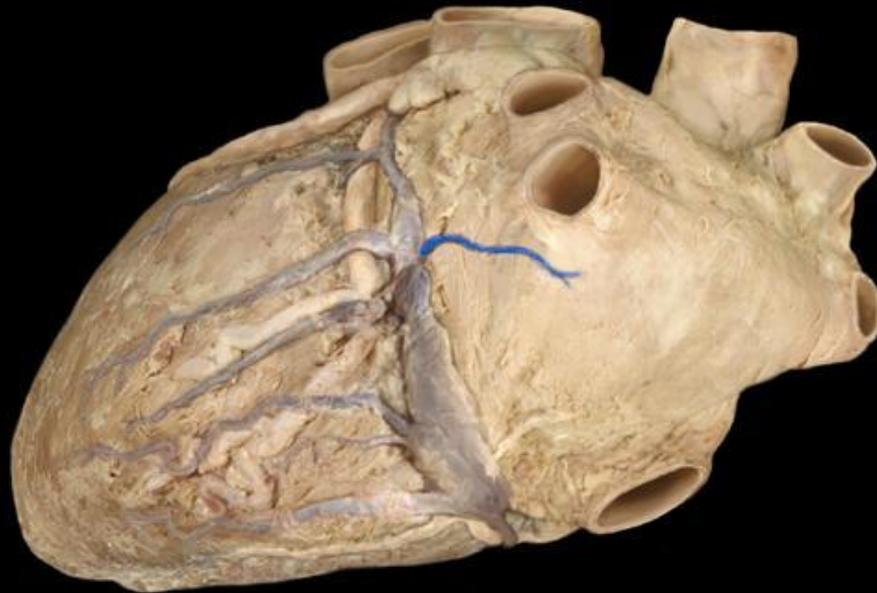


Lt. posterior ventricular v.



Rt. posterior ventricular v.

Coronary Vein Branches Posterior



Oblique vein of Lt. atrium

Cardiac Muscle and the Cardiac Conduction System

- **Expected Learning Outcomes**
 - 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
- Repair of damage of cardiac muscle is almost entirely by **fibrosis (scarring)**
- **Intercalated discs**—join cardiocytes end to end with three features: interdigitating folds, mechanical junctions, and electrical junctions
 - **Interdigitating folds:** folds interlock with each other, and increase surface area of contact

Structure of Cardiac Muscle

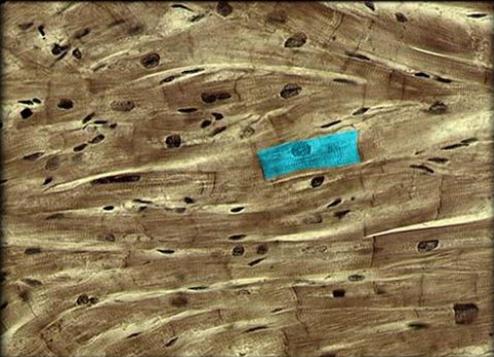
(Continued)

- **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**—mechanical linkages that prevent contracting cardiocytes from being pulled apart from each other
- **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

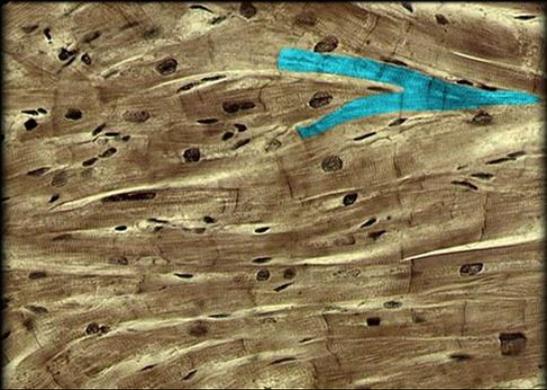
Cardiac Muscle

Photos © McGraw-Hill Education

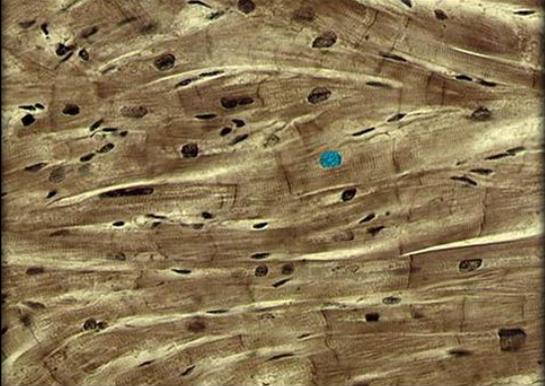
Cardiac muscle fiber



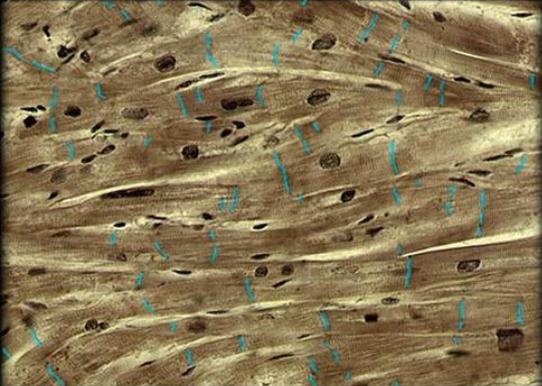
Branched cardiac muscle fiber



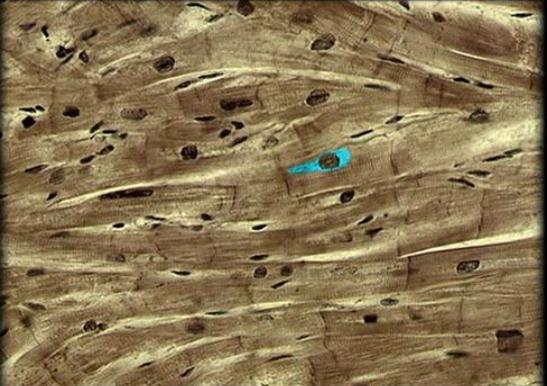
Nucleus of cardiac muscle fiber



Intercalated discs

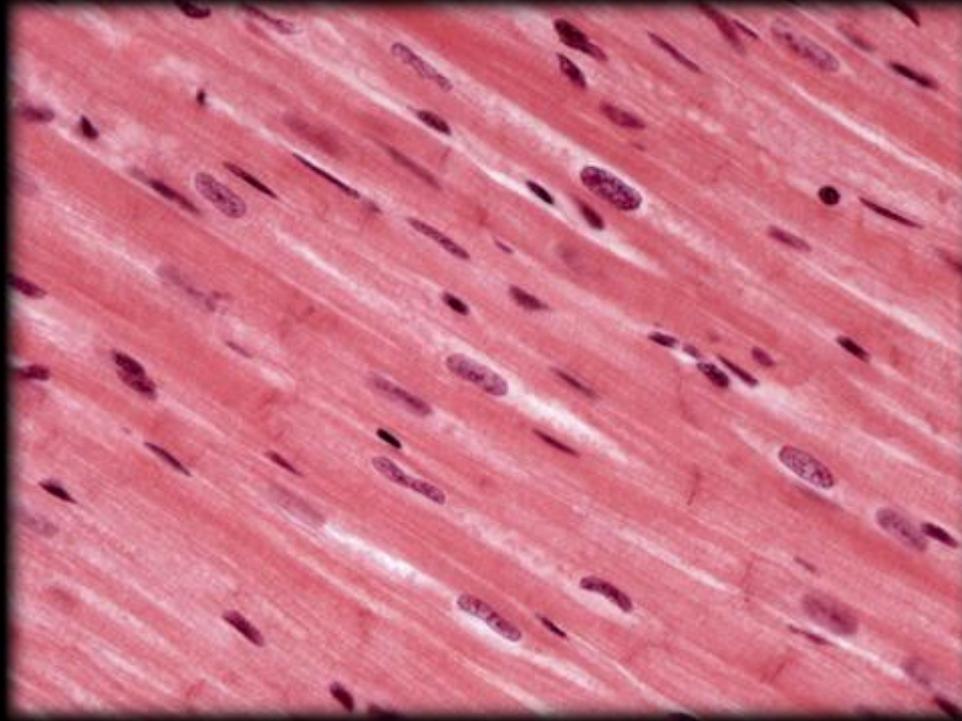


Perinuclear cytoplasm

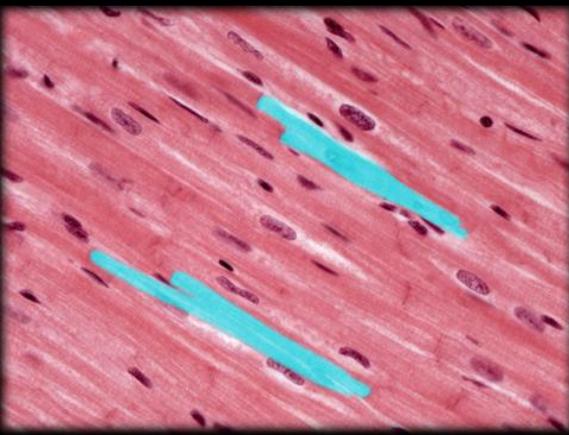


Cardiac Muscle

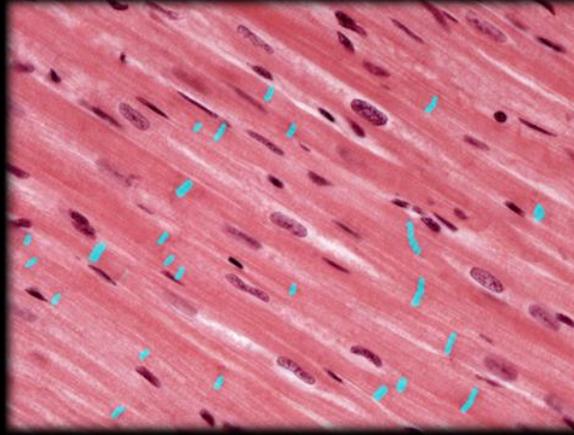
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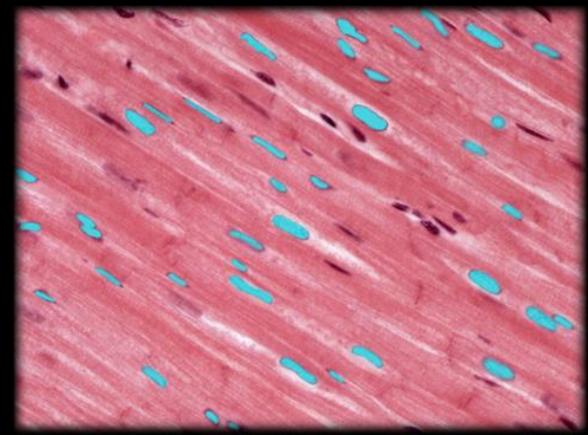
Branched cardiac cell



Intercalated disc

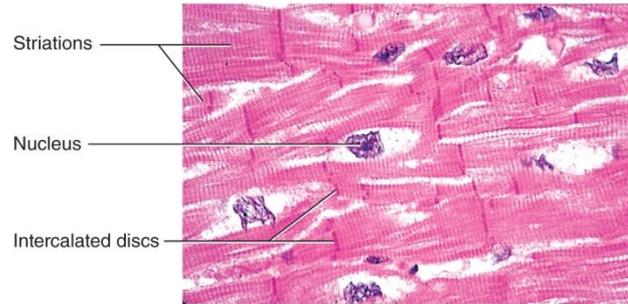


Nuclei of cardiac cell

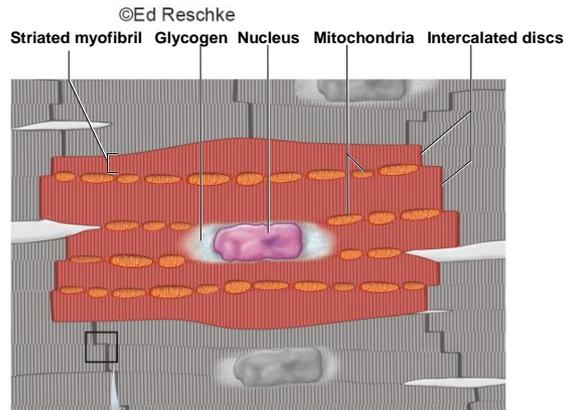


Structure of Cardiac Muscle

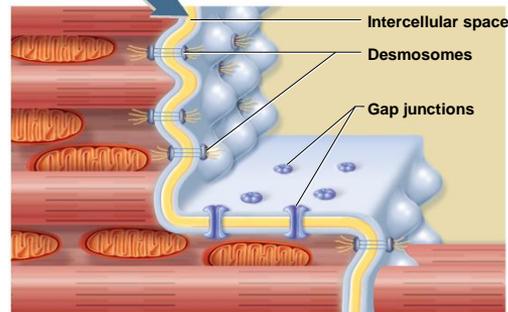
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(a)



(b)



(c)

(a): © Ed Reschke

Figure 19.11a–c

Metabolism of Cardiac Muscle

- **Cardiac muscle** depends almost exclusively on **aerobic respiration** to make ATP
 - Rich in myoglobin and glycogen
 - Huge mitochondria: fill 25% of cell
- **Adaptable to different organic fuels**
 - 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** because it makes little use of anaerobic fermentation or oxygen debt mechanisms
 - Does not fatigue for a lifetime

The Conduction System

- **Coordinates the heartbeat**
 - Composed of an **internal pacemaker** and **nerve-like conduction pathways** through myocardium
- **Generates and conducts rhythmic electrical signals in the following order:**
 - **Sinoatrial (SA) node:** modified cardiocytes
 - Pacemaker initiates each heartbeat and determines heart rate
 - Pacemaker in right atrium near base of superior vena cava
 - Signals spread throughout atria

The Conduction System

(Continued)

– **Atrioventricular (AV) node**

- Located near the right AV valve at lower end of interatrial septum
- Electrical gateway to the ventricles
- Fibrous skeleton—insulator prevents currents from getting to ventricles by any other route

– **Atrioventricular (AV) bundle (bundle of His)**

- Bundle forks into right and left bundle branches
- Branches pass through interventricular septum toward apex

– **Purkinje fibers**

- Nerve-like processes spread throughout ventricular myocardium
- Cardiocytes then pass signal from cell to cell through **gap junctions**

The Conduction System

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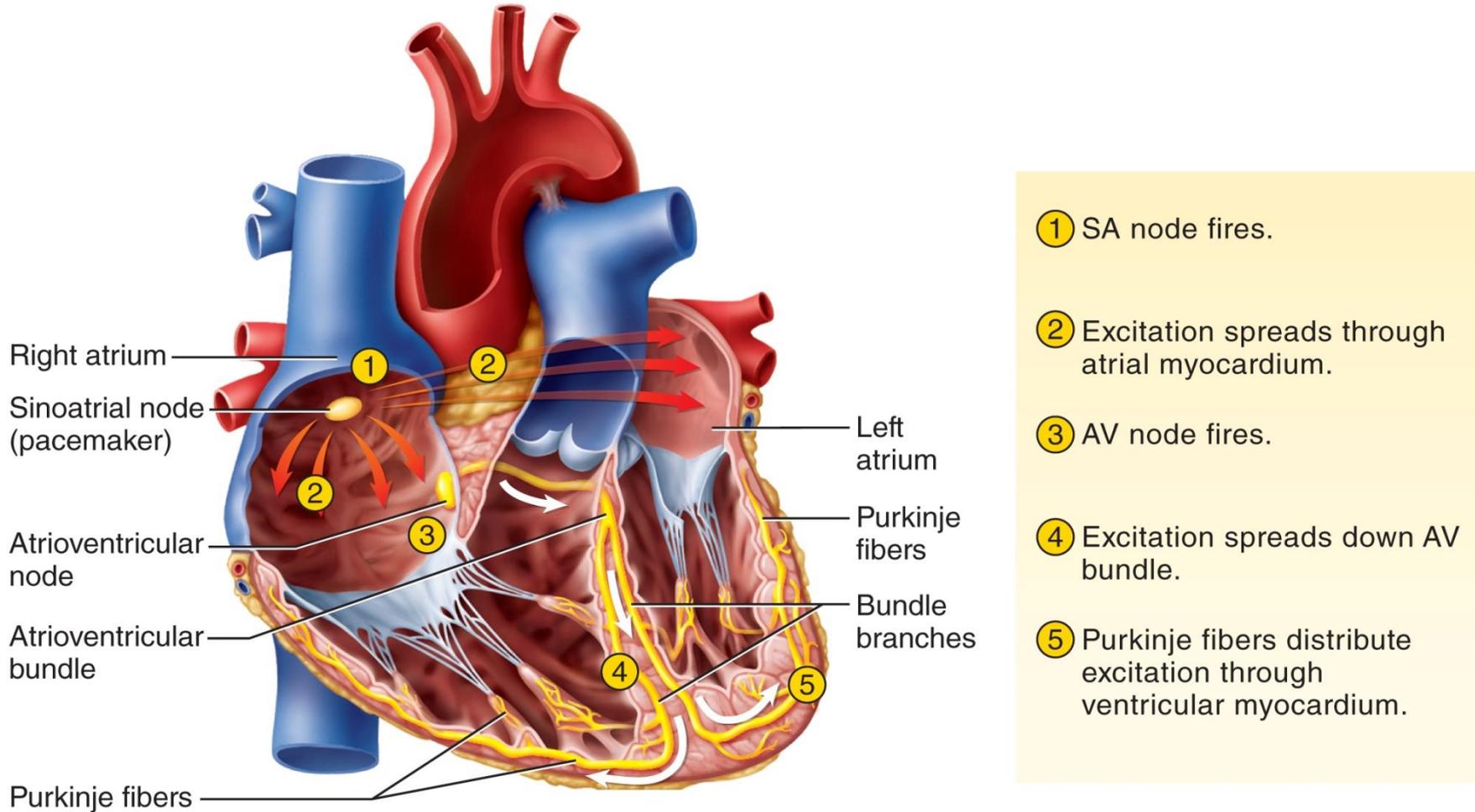


Figure 19.12

Nerve Supply to the Heart

- **Sympathetic** nerves increase heart rate and contraction strength
 - Sympathetic pathway to heart originates in the **lower cervical to upper thoracic segments** of the spinal cord
 - Continues to adjacent **sympathetic chain ganglia** and some ascend to **cervical ganglia**
 - Postganglionic fibers pass through **cardiac plexus** in mediastinum and continue as **cardiac nerves** to the heart
 - Fibers terminate in SA and AV nodes, in atrial and ventricular myocardium (also aorta, pulmonary trunk, and coronary arteries)

Nerve Supply to the Heart

- **Parasympathetic** nerves slow 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

Electrical and Contractile Activity of the Heart

- **Expected Learning Outcomes**
 - Explain why the SA node fires spontaneously and rhythmically.
 - Explain how the SA node excites the myocardium.
 - Describe the unusual action potentials of cardiac muscle and relate them to the contractile behavior of the heart.
 - Interpret a normal electrocardiogram.

Electrical and Contractile Activity of the Heart

- **Cycle of events in heart**
 - **Systole:** contraction
 - **Diastole:** relaxation
- **Although “systole” and “diastole” can refer to contraction and relaxation of either type of chamber, they usually refer to the action of the ventricles**

The Cardiac Rhythm

- **Sinus rhythm**—normal heartbeat triggered by the SA node
 - Adult at rest is typically 70 to 80 bpm (vagal tone)
- **Ectopic focus**—a region of spontaneous firing other than the SA node
 - May govern heart rhythm if SA node is damaged
 - **Nodal rhythm**—if SA node is damaged, heart rate is set by **AV node**, 40 to 50 bpm
 - Other ectopic focal rhythms are 20 to 40 bpm and too slow to sustain life

Pacemaker Physiology

- **SA node** does not have a stable resting membrane potential
 - Starts at -60 mV and drifts upward due to **slow Na^+ inflow**
 - Gradual depolarization is called **pacemaker potential**
 - When it 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
- **When SA node fires it sets off heartbeat**
 - As the internal pacemaker, it typically fires every 0.8 seconds, setting the resting rate at 75 bpm

Pacemaker Physiology

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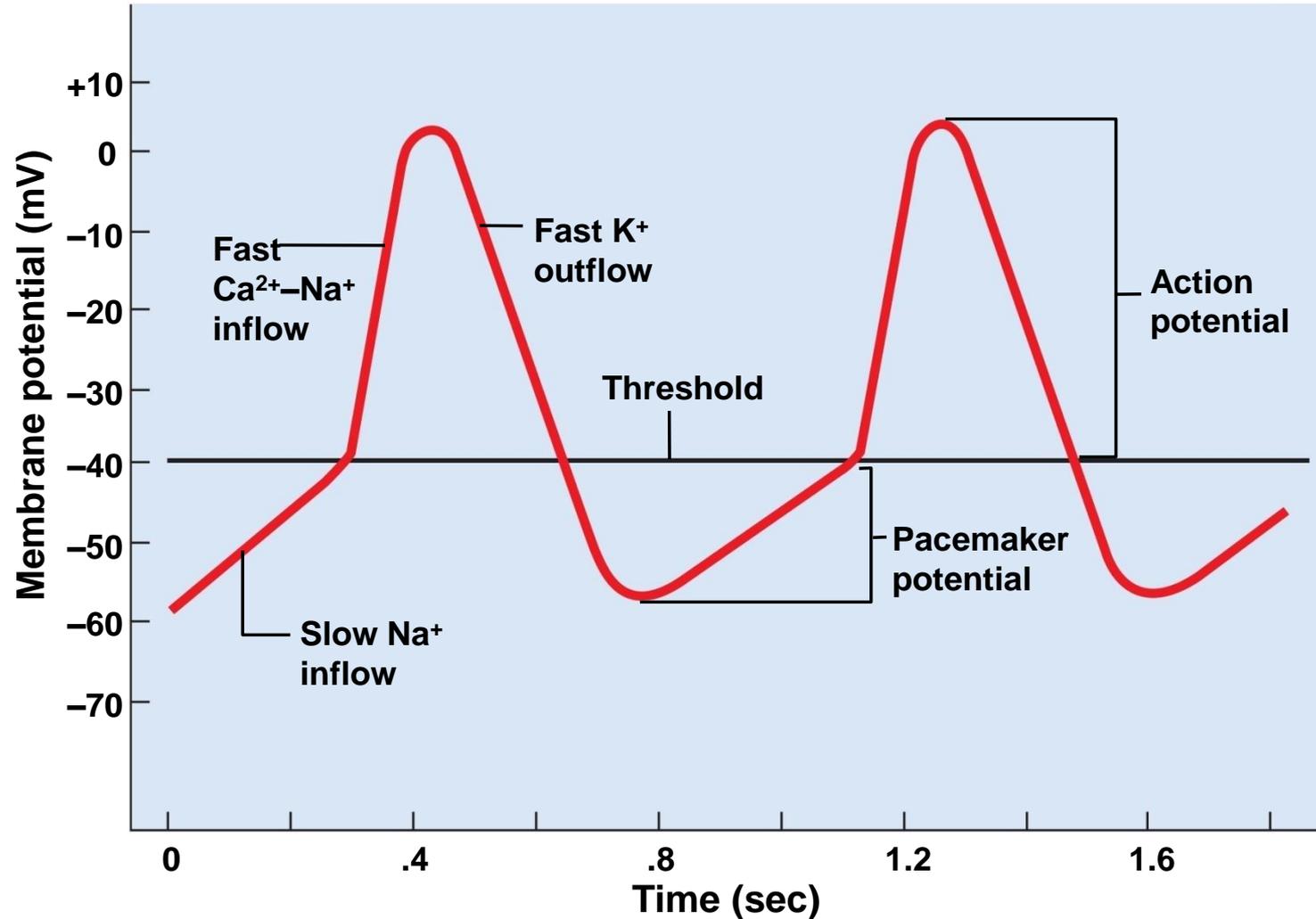


Figure 19.13

Impulse Conduction to the Myocardium

- Signal from **SA node** stimulates two atria to contract almost simultaneously
 - Reaches AV node in 50 ms
- Signal slows down through **AV node**
 - Thin cardiocytes with fewer gap junctions
 - Delays signal 100 ms which allows the ventricles time to fill

Impulse Conduction to the Myocardium

- Signals travel very quickly through **AV bundle** and **Purkinje fibers**
 - Entire ventricular myocardium depolarizes and contracts in near unison
 - Signals reach papillary muscles slightly later than rest of myocardium
- **Ventricular systole progresses up from the apex of the heart**
 - Spiral arrangement of myocardium twists ventricles slightly; like someone wringing out a towel

Electrical Behavior of the Myocardium

- **Cardiocytes have a stable resting potential of -90 mV, and depolarize only when stimulated**
- **Three phases to cardiocyte action potential:**
depolarization, plateau, repolarization
 - **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 ms, sustains contraction for expulsion of blood from heart
 - Voltage-gated slow Ca^{2+} channels open admitting Ca^{2+} which triggers opening of Ca^{2+} channels on sarcoplasmic reticulum (SR)
 - Ca^{2+} (mostly from the SR) binds to troponin triggering contraction

Electrical Behavior of the Myocardium

- **Cardiocyte action potential (Continued)**
 - **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 ms (compared to 1 to 2 ms in skeletal muscle)
 - Prevents wave summation and tetanus which would stop the pumping action of the heart

Electrical Behavior of the Myocardium

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)

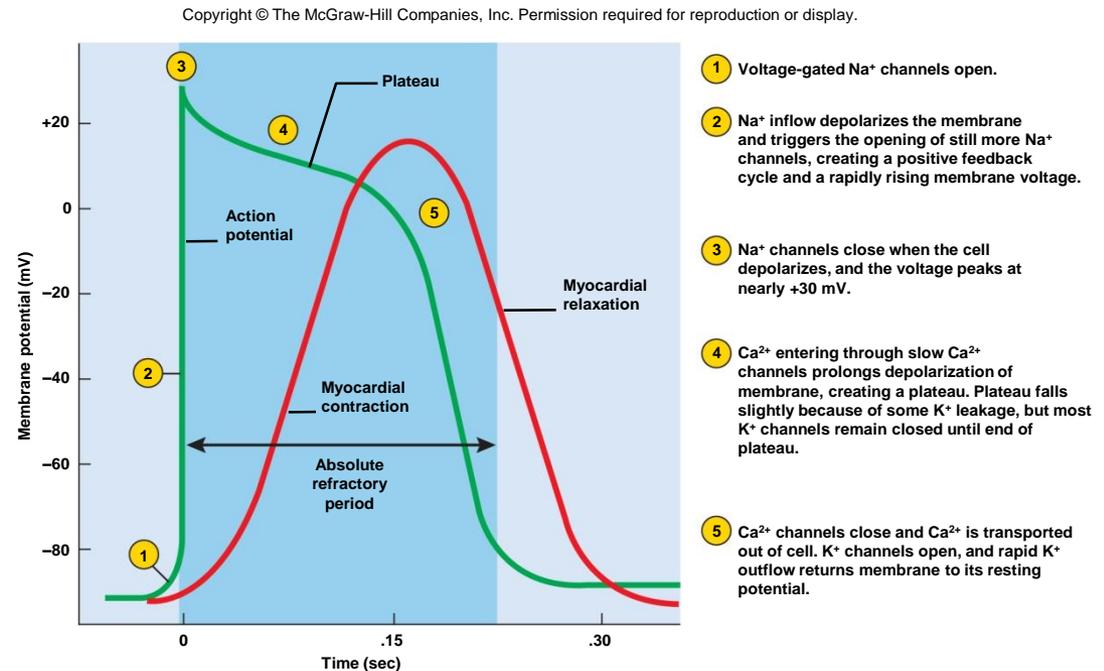


Figure 19.14

The Electrocardiogram

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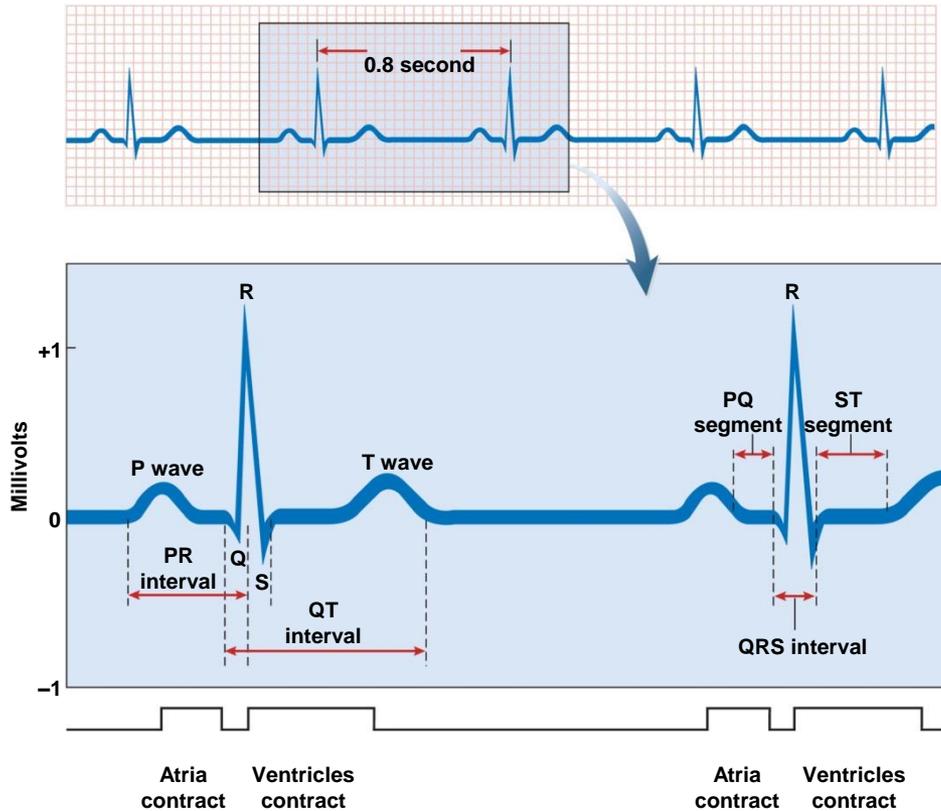


Figure 19.15

- **Electrocardiogram (ECG or EKG)**

- Composite of all action potentials of nodal and myocardial cells detected, amplified and recorded by electrodes on arms, legs, and chest

The Electrocardiogram

- **P wave**
 - SA node fires, **atria depolarize** and contract
 - Atrial systole begins 100 ms after SA signal
- **QRS complex**
 - **Ventricular depolarization**
 - Complex shape of spike due to different thickness and shape of the two ventricles
- **ST segment**—ventricular systole
 - Corresponds to plateau in myocardial action potential
- **T wave**
 - **Ventricular repolarization** and relaxation

The Electrocardiogram

1. Atrial depolarization begins
2. Atrial depolarization complete (atria contracted)
3. Ventricles begin to depolarize at apex; atria repolarize (atria relaxed)
4. Ventricular depolarization complete (ventricles contracted)
5. Ventricles begin to repolarize at apex
6. Ventricular repolarization complete (ventricles relaxed)

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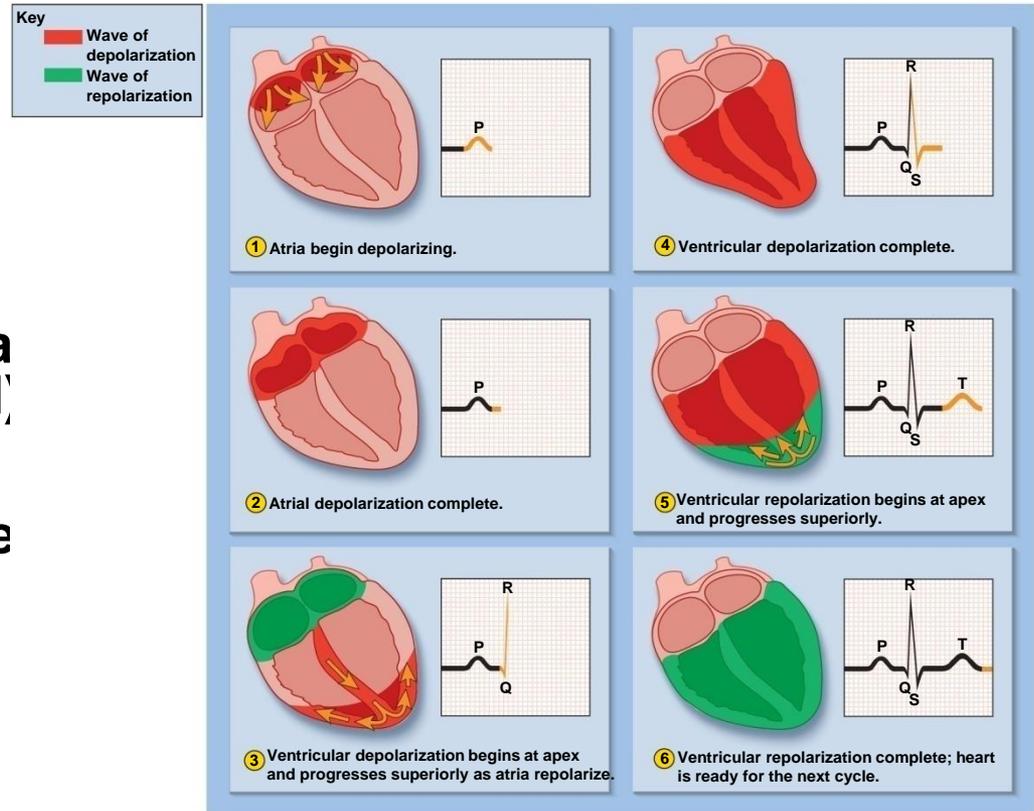
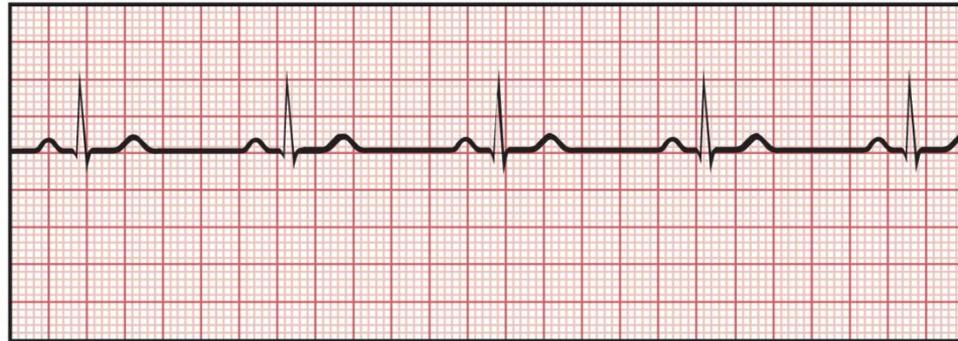


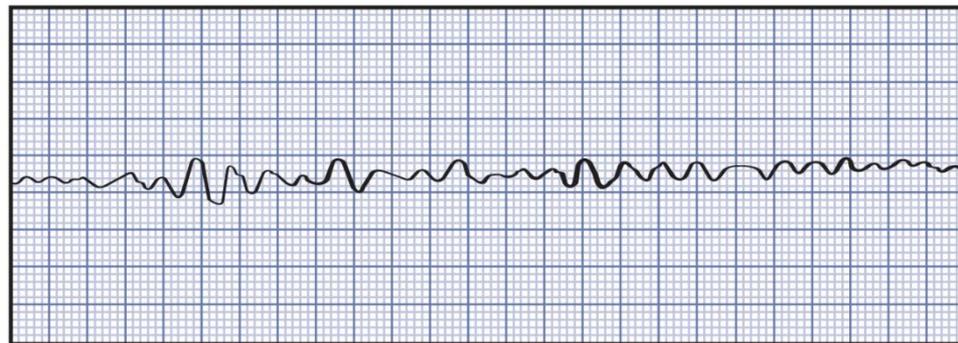
Figure 19.16

ECGs: Normal and Abnormal

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(a) Sinus rhythm (normal)



(b) Ventricular fibrillation

- **Deviations of ECG from normal can indicate:**
 - Myocardial infarction (MI)
 - Abnormalities in conduction pathways
 - Heart enlargement
 - Electrolyte and hormone imbalances

Figure 19.17a,b

Cardiac Arrhythmias

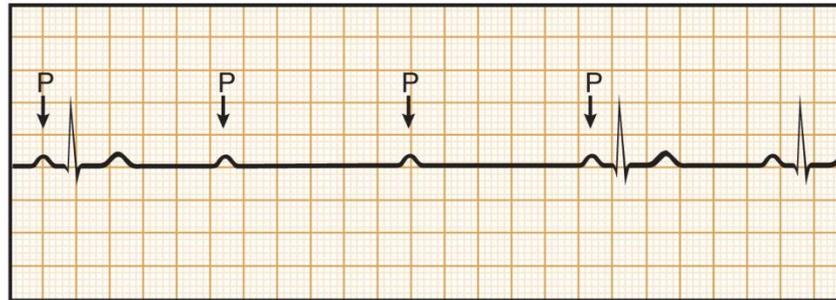
- **Ventricular fibrillation**
 - Serious **arrhythmia** caused by electrical signals traveling randomly
 - Heart cannot pump blood; no coronary perfusion
 - Hallmark of heart attack (MI)
 - Kills quickly if not stopped
 - **Defibrillation**—strong electrical shock with intent to depolarize entire myocardium and reset heart to sinus rhythm
 - Not a cure for artery disease, but may allow time for other corrective action

The Cardiac Rhythm

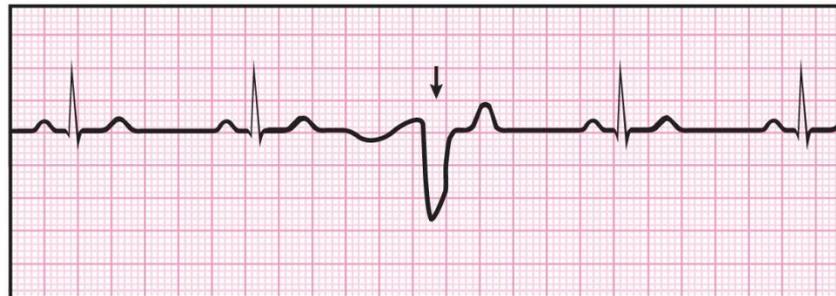
- **Other arrhythmias**

- Heart block—failure of conduction system to conduct
- Premature ventricular contraction—extra beats due to a ventricular ectopic focus

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(d) Heart block



(e) Premature ventricular contraction

Figure 19.17d,e

Blood Flow, Heart Sounds, and the Cardiac Cycle

- **Expected Learning Outcomes**

- 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.

Blood Flow, Heart Sounds, and the Cardiac Cycle

- **Cardiac cycle**—one complete contraction and relaxation of all four chambers of the heart
- **Questions to consider: How does pressure affect blood flow? How are heart sounds produced?**

Principles of Pressure and Flow

- Two main variables govern fluid movement: **pressure** causes **flow** and **resistance** opposes it
 - Fluid will only flow if there is a **pressure gradient (pressure difference)**
 - Fluid flows from high-pressure point to low-pressure point
 - Pressure measured in mm Hg with a manometer (sphygmomanometer for BP)

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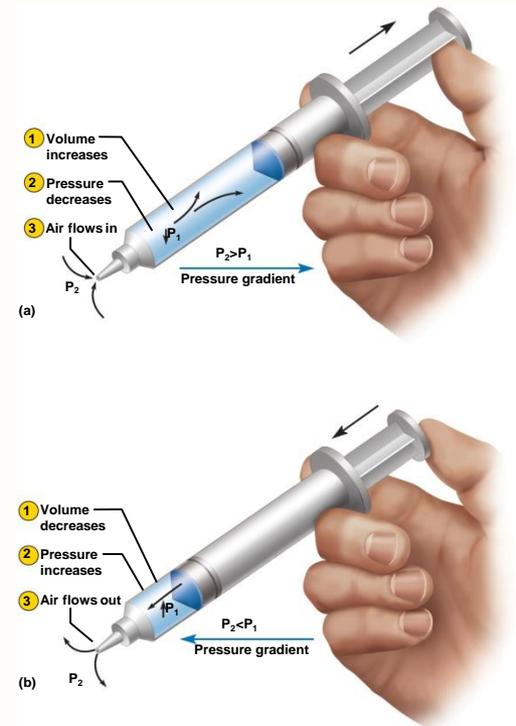


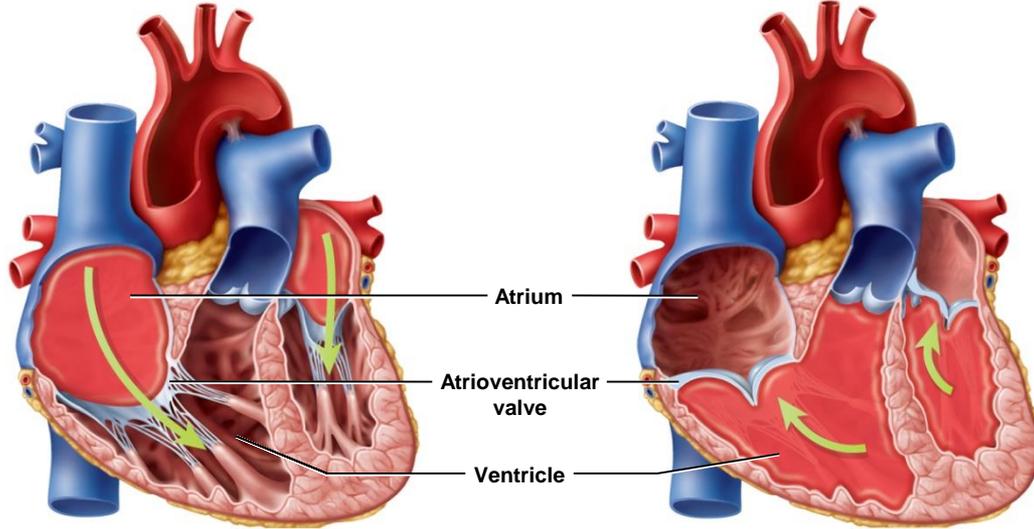
Figure 19.18

Pressure Gradients and Flow

- **Events occurring on left side of heart**
 - When ventricle relaxes and expands, its internal pressure falls
 - If mitral valve is open, blood flows into left ventricle
 - When ventricle contracts, internal pressure rises
 - AV valves close, 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 the Heart Valves

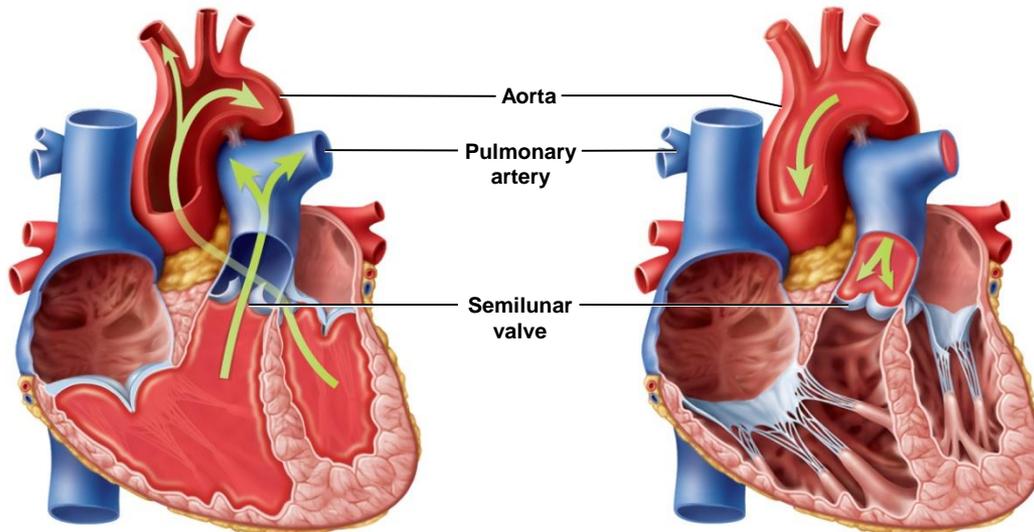
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Atrioventricular valves open

Atrioventricular valves closed

(a)



Semilunar valves open

Semilunar valves closed

(b)

Figure 19.19

Valvular Insufficiency Disorders

- **Valvular insufficiency (incompetence)**—any failure of a valve to prevent reflux (regurgitation), the backward flow of blood
 - **Valvular stenosis:** cusps are stiffened and opening is constricted by scar tissue
 - Result of **rheumatic fever**, autoimmune attack on the mitral and aortic valves
 - Heart overworks and may become enlarged
 - **Heart murmur**—abnormal heart sound produced by regurgitation of blood through incompetent valves

Valvular Insufficiency Disorders

(Continued)

- **Mitral valve prolapse:** insufficiency in which one or both mitral valve cusps bulge into atria during ventricular contraction
 - Hereditary in 1 out of 40 people
 - May cause chest pain and shortness of breath

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

Phases of the Cardiac Cycle

- **Ventricular filling (during diastole)**
- **Isovolumetric contraction (during systole)**
- **Ventricular ejection (during systole)**
- **Isovolumetric relaxation (during diastole)**
- **The entire cardiac cycle (all four of these phases) are completed in less than 1 second**

Phases of the Cardiac Cycle

- **Ventricular filling**
 - Ventricles expand and their pressure drops below that of the atria
 - AV valves open and blood flows into the ventricles
 - Filling occurs in three phases:
 - **Rapid ventricular filling:** first one-third
 - **Diastasis:** second one-third; slower filling
 - P wave occurs at the end of diastasis
 - **Atrial systole:** final one-third; atria contract
 - End-diastolic volume (EDV) achieved in each ventricle (about 130 mL of blood)

Phases of the Cardiac Cycle

- **Isovolumetric contraction**
 - Atria repolarize, relax and remain in diastole for rest of cardiac cycle
 - **Ventricles depolarize**, causing QRS complex, and begin to contract
 - **AV valves close** as ventricular blood surges back against the cusps
 - **Heart sound S₁** occurs at the beginning of this phase
 - “Isovolumetric” because although ventricles contract, they do not eject blood
 - Pressures in aorta and pulmonary trunk are still greater than those in the ventricles
 - Cardiocytes exert force, but with all four valves closed, the blood cannot go anywhere

Phases of the Cardiac Cycle

- **Ventricular ejection**

- Begins when ventricular pressure exceeds arterial pressure and semilunar valves open
- Pressure peaks in left ventricle at about 120 mm Hg and 25 mm Hg in the right
- First: **rapid ejection**—blood spurts out of ventricles quickly
- Then: **reduced ejection**—slower flow with lower pressure
- Ejection lasts about 200 to 250 ms - corresponds to plateau phase of cardiac action potential
- T wave of ECG occurs late in this phase
- **Stroke volume (SV)** is about 70 mL
 - **Ejection fraction** is about 54% of EDV (130 mL)
 - 60 mL remaining blood is **end-systolic volume (ESV) = EDV - SV**

Phases of the Cardiac Cycle

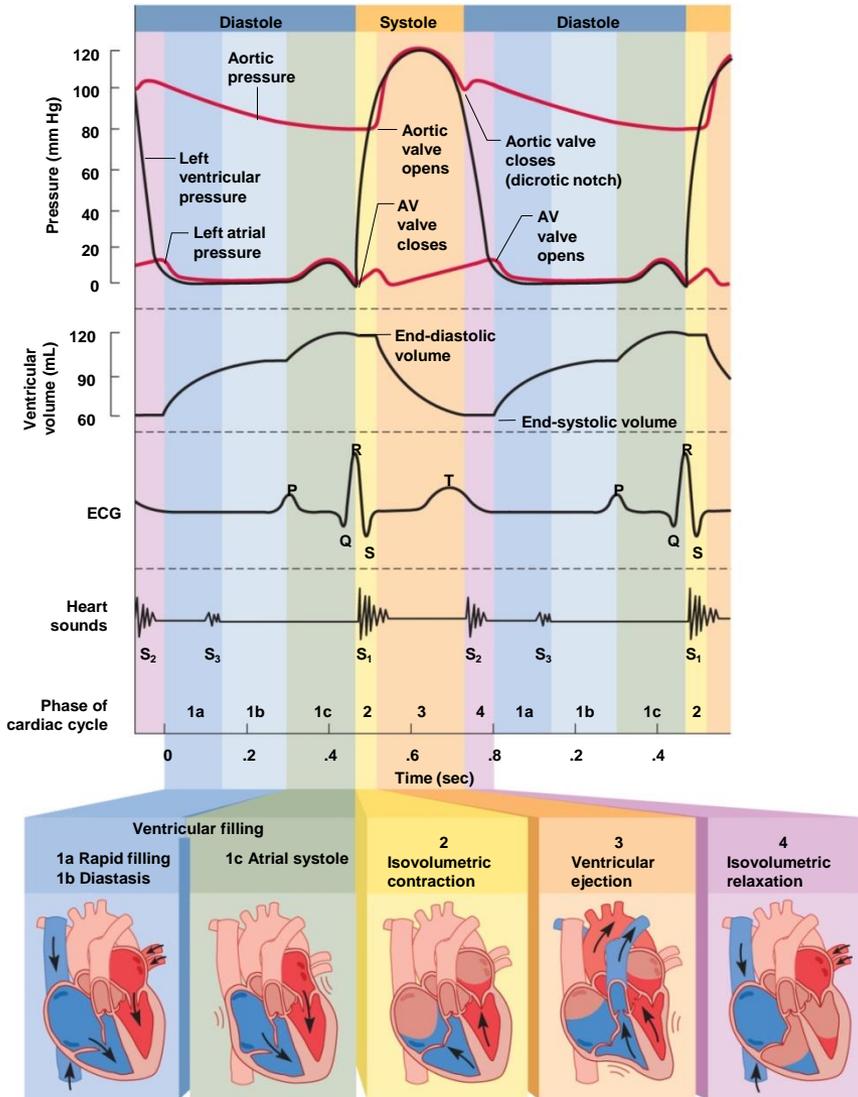
- **Isovolumetric relaxation**
 - T wave ends and ventricles begin to expand
 - Blood from aorta and pulmonary trunk briefly flows backward filling cusps and **closing semilunar valves**
 - Creates pressure rebound that appears as **dicrotic notch** in graph of artery pressure
 - **Heart sound S₂** occurs
 - “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**

Phases of the Cardiac Cycle

- **In a resting person**
 - **Atrial systole** lasts about **0.1 second**
 - **Ventricular systole** lasts about **0.3 second**
 - **Quiescent period**, when all four chambers are in diastole, lasts about **0.4 second**
- **Total duration of the cardiac cycle** is therefore **0.8 second** in a heart beating **75 bpm**

Events of the Cardiac Cycle

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- 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
<hr/>	
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

Overview of Volume Changes

- **Normally, right and left sides of heart eject the same volume of blood even though they are under different pressure**
- **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

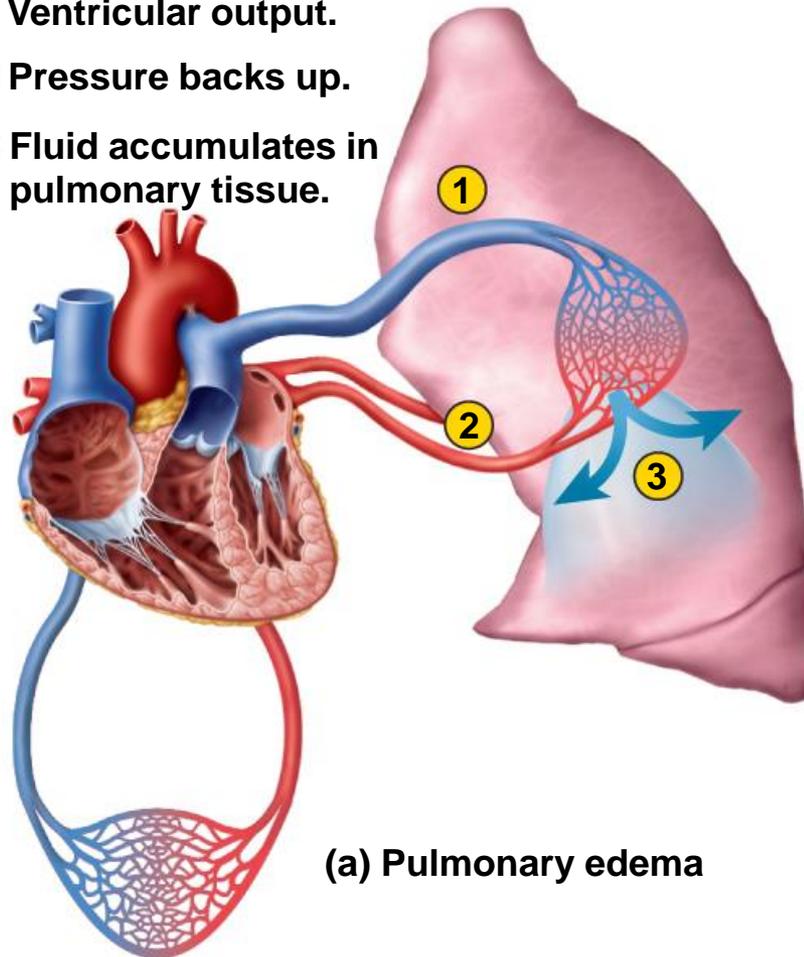
Overview of Volume Changes

- **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**

Overview of Volume Changes

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



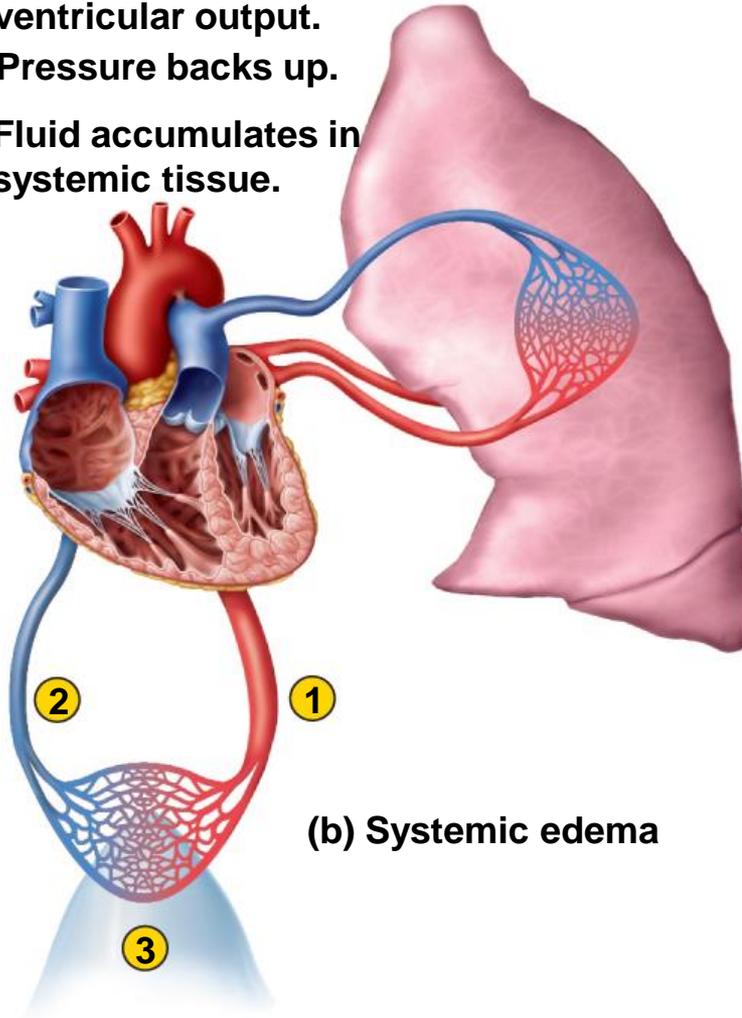
(a) Pulmonary edema

- If the left ventricle pumps less blood than the right, the blood pressure backs up into the lungs and causes **pulmonary edema**

Overview of Volume Changes

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



(b) Systemic edema

- If the right ventricle pumps less blood than the left, pressure backs up in the systemic circulation and causes **systemic edema**

Figure 19.21b

Cardiac Output

- **Expected Learning Outcomes**
 - 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

- **Cardiac output (CO)**—amount ejected by each ventricle in 1 minute
- **Cardiac output = heart rate x stroke volume**
 - About **4 to 6 L/min at rest**
 - A RBC leaving the left ventricle will arrive back at the left ventricle in about 1 minute
 - Vigorous exercise increases CO to 21 L/min for a fit person and up to 35 L/min for a world-class athlete
- **Cardiac reserve**—the difference between a person's maximum and resting CO
 - Increases with fitness, decreases with disease

Heart Rate

- **Pulse**—surge of pressure produced by heart beat that can be felt by palpating a superficial artery
 - Infants have HR of 120 bpm or more
 - Young adult females average 72 to 80 bpm
 - Young adult males average 64 to 72 bpm
 - Heart rate rises again in the elderly

Heart Rate

- **Tachycardia**—resting adult heart rate above 100 bpm
 - Stress, anxiety, drugs, heart disease, or fever
 - Loss of blood or damage to myocardium
- **Bradycardia**—resting adult heart rate of less than 60 bpm
 - In sleep, low body temperature, and endurance-trained athletes
- **Positive chronotropic agents**—factors that raise the heart rate
- **Negative chronotropic agents**—factors that lower the heart rate

Chronotropic Effects of the Autonomic Nervous System

- **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**

Chronotropic Effects of the Autonomic Nervous System

- **Sympathetic** postganglionic fibers are adrenergic
 - They release **norepinephrine**
 - Bind to **β -adrenergic fibers** in the heart
 - Activate **cAMP second-messenger system** in cardiocytes and nodal cells
 - Lead to **opening of Ca^{2+} channels** in plasma membrane

Chronotropic Effects of the Autonomic Nervous System

- **Sympathetic** postganglionic fibers are adrenergic (continued)
 - Increased Ca^{2+} inflow **accelerates depolarization of SA node**
 - **cAMP accelerates uptake of Ca^{2+} by sarcoplasmic reticulum** allowing cardiocytes to **relax more quickly**
 - By accelerating both contraction and relaxation, norepinephrine (through cAMP) increases heart rate as high as 230 bpm
 - But at excessively high heart rates (>180 bpm) diastole becomes too brief for adequate filling
 - Both stroke volume and cardiac output are reduced

Chronotropic Effects of the Autonomic Nervous System

- **Parasympathetic vagus nerves** have cholinergic, inhibitory effects on 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**

Chronotropic Effects of the Autonomic Nervous System

- Without influence from cardiac centers, the heart has an **intrinsic firing rate of 100 bpm**
- **Vagal tone**—holds down the heart rate to 70 to 80 bpm at rest
 - Steady background firing rate of the **vagus nerves**

Chronotropic Effects of the Autonomic Nervous System

- **Inputs to cardiac centers in medulla** are diverse
 - Sources include **higher brain centers** such as cerebral cortex, limbic system, hypothalamus
 - Sensory or emotional stimuli
 - Medulla also receives input from muscles, joints, arteries, and brainstem
 - **Proprioceptors** in muscles and joints inform cardiac centers about changes in activity, so HR increases before metabolic demands on muscle arise

Chronotropic Effects of the Autonomic Nervous System

Inputs to cardiac centers (Continued)

- **Baroreceptors** 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

Chronotropic Effects of the Autonomic Nervous System

- **Inputs to cardiac centers (Continued)**
 - **Chemoreceptors**
 - In aortic arch, carotid arteries, and medulla oblongata
 - Sensitive to blood pH, CO₂ and O₂ levels
 - More important in respiratory control than cardiac control, but will trigger an increase in heart rate when high CO₂ levels (hypercapnia) lead to acidosis
 - Also respond to hypoxemia (oxygen deficiency in blood) usually by slowing down the heart
 - **Baroreflexes and chemoreflexes**—responses to fluctuation in blood pressure and chemistry—are both negative feedback loops

Chronotropic Effects of Chemicals

- **Several chemicals affect heart rate**
 - Autonomic neurotransmitters (NE and Ach)
 - Blood-borne adrenal catecholamines (NE and epinephrine) are potent cardiac stimulants
 - **Nicotine** stimulates catecholamine secretion
 - **Thyroid hormone** increases number of adrenergic receptors on heart so it is more responsive to adrenergic stimulation
 - **Caffeine** inhibits cAMP breakdown, prolonging adrenergic effect

Chronotropic Effects of Chemicals

- **Electrolytes**

- K^+ has greatest chronotropic effect

- **Hyperkalemia**—excess K^+ diffuses into cardiocytes

- Myocardium less excitable, heart rate slows and becomes irregular

- **Hypokalemia**—deficiency in K^+

- 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

- The other factor in cardiac output, besides heart rate, is **stroke volume (SV)**
- **Three variables govern stroke volume**
 - Preload
 - Contractility
 - Afterload
- **Examples**
 - Increased preload or contractility increases stroke volume
 - Increased afterload decreases stroke volume

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 the heart: $SV \propto EDV$**
 - Stroke volume is proportional to the end diastolic volume
 - Ventricles eject almost as much blood as they receive
 - The more they are stretched, the harder they contract
 - Relates to the length-tension relationship of striated muscle

Contractility

- **Contractility** refers to how hard the myocardium contracts for a given preload
- **Positive inotropic agents** increase contractility
 - **Hypercalcemia** can cause strong, prolonged contractions and even cardiac arrest in systole
 - **Catecholamines** increase calcium levels
 - **Glucagon** stimulates cAMP production
 - **Digitalis** 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**—sum of all forces opposing ejection of blood from ventricle
- **Largest part of afterload is blood pressure in aorta and pulmonary trunk**
 - Opposes the opening of semilunar valves
 - Limits stroke volume
- **Hypertension** increases afterload and opposes ventricular ejection

Afterload

- **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

Exercise and Cardiac Output

- **Increases 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**: an accumulation of lipid deposits that degrade the arterial wall and obstruct the lumen
 - Begins when endothelium damaged by hypertension, diabetes, or other causes

Coronary Artery Disease

(Continued)

- Monocytes penetrate walls of damaged vessels and transform into macrophages
 - Absorb cholesterol and fats to be called **foam cells**
 - 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

Coronary Artery Disease

- Causes **angina pectoris**, intermittent chest pain, by obstructing 75% or more of the blood flow
- **Immune cells of atheroma stimulate inflammation**
 - May rupture, resulting in traveling clots or fatty emboli
- **Cause coronary artery spasms due to lack of secretion of nitric oxide (vasodilator)**
- **Inflammation transforms atheroma into a hardened complicated plaque**
 - One cause of **arteriosclerosis**

Coronary Artery Disease

- **Major risk factor for CAD is excess of low-density lipoprotein (LDL) in the blood combined with defective LDL receptors in arterial walls**
 - LDLs—protein-coated droplets of cholesterol, neutral fats, free fatty acids, and phospholipids
 - Dysfunctional receptors in arterial cells cause them to accumulate excess cholesterol

Coronary Artery Disease

- **Unavoidable risk factors: heredity, aging, being male**
- **Preventable risk factors: obesity, smoking, lack of exercise, anxious personality, stress, aggression, and diet**
- **Treatment**
 - Coronary bypass surgery
 - Uses great saphenous vein from leg or small thoracic arteries
 - Balloon or laser angioplasty
 - Insertion of a stent to prevent restenosis