

A&P Refresher- Heart

Note: Not all slides will be covered

- Goal is to provide quick background/review to hopefully begin to understand some of the pathologies on first 2 slides

Diseases and Disorders

Heart

Condition	Description
Inflammation of Heart Tissue	
Endocarditis	Inflammation of the endocardium; affects the valves more severely than other areas of the endocardium; may lead to scarring, causing stenosed or incompetent valves
Pericarditis	Inflammation of the pericardium; see Clinical Impact, "Pericarditis and Cardiac Tamponade"
Cardiomyopathy	Disease of the myocardium of unknown cause or occurring secondarily to other disease; results in weakened cardiac muscle, causing all chambers of the heart to enlarge; may eventually lead to congestive heart failure
Rheumatic heart disease	Results from a streptococcal infection in young people; toxin produced by the bacteria can cause rheumatic fever several weeks after the infection that can result in rheumatic endocarditis
Reduced Blood Flow to Cardiac Muscle	
Coronary heart disease	Reduces the amount of blood the coronary arteries can deliver to the myocardium
Coronary thrombosis	Formation of blood clot in a coronary artery
Myocardial infarction	Damaged cardiac muscle tissue resulting from lack of blood flow to the myocardium; often referred to as a heart attack; see Clinical Impact, "Angina, Infarctions, and the Treatment of Blocked coronary Arteries"
Congenital Heart Diseases (occur at birth)	
Septal defect	Hole in the septum between the left and right sides of the heart, allowing blood to flow from one side of the heart to the other and greatly reducing the heart's pumping effectiveness
Patent ductus arteriosus	Ductus arteriosus fails to close after birth, allowing blood to flow from the aorta to the pulmonary trunk under a higher pressure, which damages the lungs; also, the left ventricle must work harder to maintain adequate systemic pressure
Stenosis of the heart valve	Narrowed opening through one or more of the heart valves; aortic or pulmonary semilunar stenosis increases the heart's workload; bicuspid valve stenosis causes blood to back up in the left atria and lungs, resulting in edema of the lungs; tricuspid valve stenosis results in similar blood flow problems and edema in the peripheral tissues
Incompetent heart valve	Heart valves do not close correctly, and blood flows through in the reverse direction; see Clinical Impact, "Abnormal Heart Sounds"
Cyanosis (<i>sī-ā-no'sis</i> ; <i>cyan</i> , blue + <i>osis</i> , condition of)	Symptom of inadequate heart function in babies with congenital heart disease; the infant's skin appears blue because of low oxygen levels in the blood in peripheral blood vessels
Heart Failure	Progressive weakening of the heart muscle, reducing the heart's pumping action; hypertension leading to heart failure due to increased afterload; advanced age, malnutrition, chronic infections, toxins, severe anemias, hyperthyroidism, and hereditary factors can lead to heart failure

TABLE 20.1**Major Cardiac Arrhythmias**

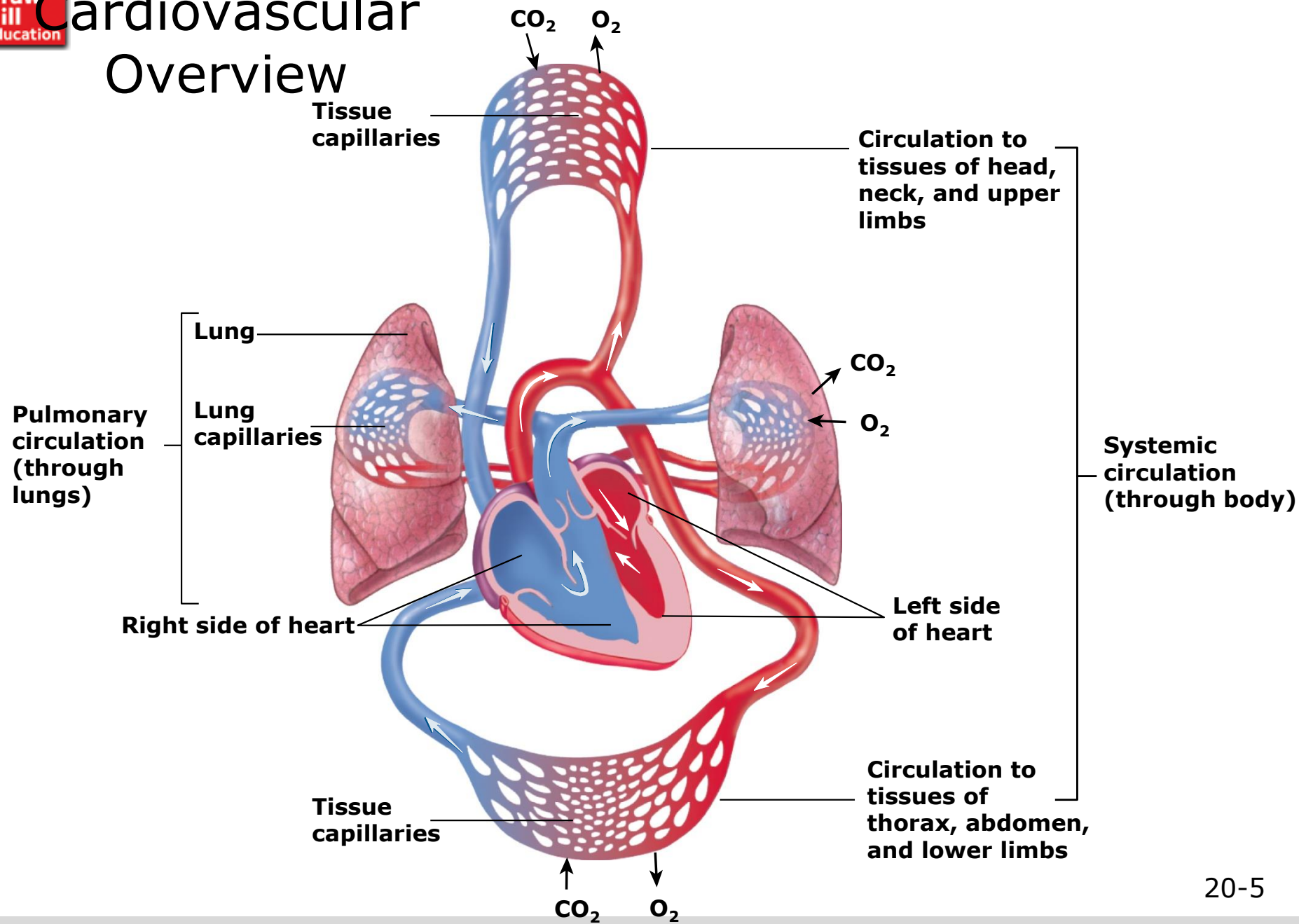
Conditions	Symptoms	Possible Causes
Abnormal Heart Rhythms		
Tachycardia	Heart rate in excess of 100 beats per minute (bpm)	Elevated body temperature; excessive sympathetic stimulation; toxic conditions
Paroxysmal atrial tachycardia	Sudden increase in heart rate to 95–150 bpm for a few seconds or even for several hours; P wave precedes every QRS complex; P wave inverted and superimposed on T wave	Excessive sympathetic stimulation; abnormally elevated permeability of slow channels
Ventricular tachycardia	Frequently causes fibrillation	Often associated with damage to AV node or ventricular muscle
Abnormal Rhythms Resulting from Ectopic Action Potentials		
Atrial flutter	300 P waves/min; 125 QRS complexes/min, resulting in two or three P waves (atrial contraction) for every QRS complex (ventricular contraction)	Ectopic action potentials in the atria
Atrial fibrillation	No P waves; normal QRS complexes; irregular timing; ventricles constantly stimulated by atria; reduced pumping effectiveness and filling time	Ectopic action potentials in the atria
Ventricular fibrillation	No QRS complexes; no rhythmic contraction of the myocardium; many patches of asynchronously contracting ventricular muscle	Ectopic action potentials in the ventricles
Bradycardia	Heart rate less than 60 bpm	Elevated stroke volume in athletes; excessive vagal stimulation; carotid sinus syndrome
Sinus arrhythmia	Heart rate varies 5% during respiratory cycle and up to 30% during deep respiration.	Cause not always known; occasionally caused by ischemia or inflammation or associated with cardiac failure
SA node block	Cessation of P wave; new low heart rate due to AV node acting as pacemaker; normal QRS complex and T wave	Ischemia; tissue damage due to infarction; causes unknown
AV Node Block		
First-degree	PR interval greater than 0.2 second	Inflammation of AV bundle
Second-degree	PR interval 0.25–0.45 second; some P waves trigger QRS complexes and others do not; 2:1, 3:1, and 3:2 P wave/QRS complex ratios may occur	Excessive vagal stimulation
Third-degree (complete heart block)	P wave dissociated from QRS complex; atrial rhythm approximately 100 bpm; ventricular rhythm less than 40 bpm	Ischemia of AV nodal fibers or compression of AV bundle
Premature atrial contractions	Occasional shortened intervals between contractions; frequently occurs in healthy people P wave superimposed on QRS complex	Excessive smoking; lack of sleep; too much caffeine; alcoholism
Premature ventricular contractions (PVCs)	Prolonged QRS complex; exaggerated voltage because only one ventricle may depolarize; inverted T wave; increased probability of fibrillation	Ectopic foci in ventricles; lack of sleep; too much caffeine, irritability; occasionally occurs with coronary thrombosis

Abbreviations: SA = sinoatrial; AV = atrioventricular.

Functions of the Heart

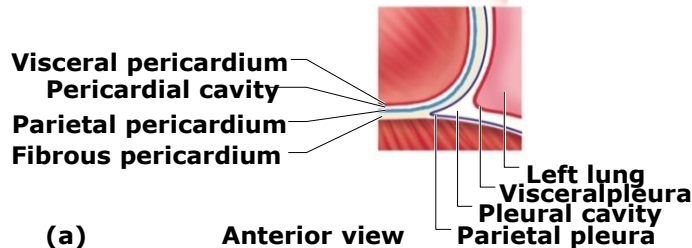
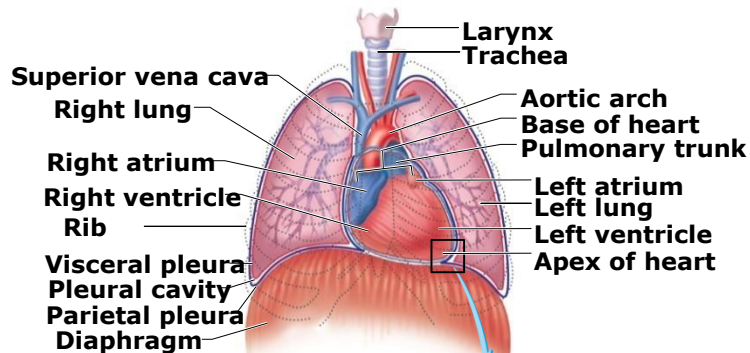
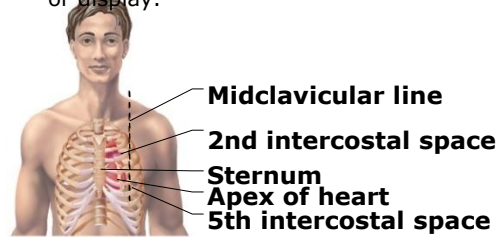
1. Generating blood pressure
2. Routing blood: separates pulmonary and systemic circulations
3. Ensuring one-way blood flow: valves
4. Regulating blood supply
 - Changes in contraction rate and force match blood delivery to changing metabolic needs

Cardiovascular Overview



Size, Shape, Location of the Heart

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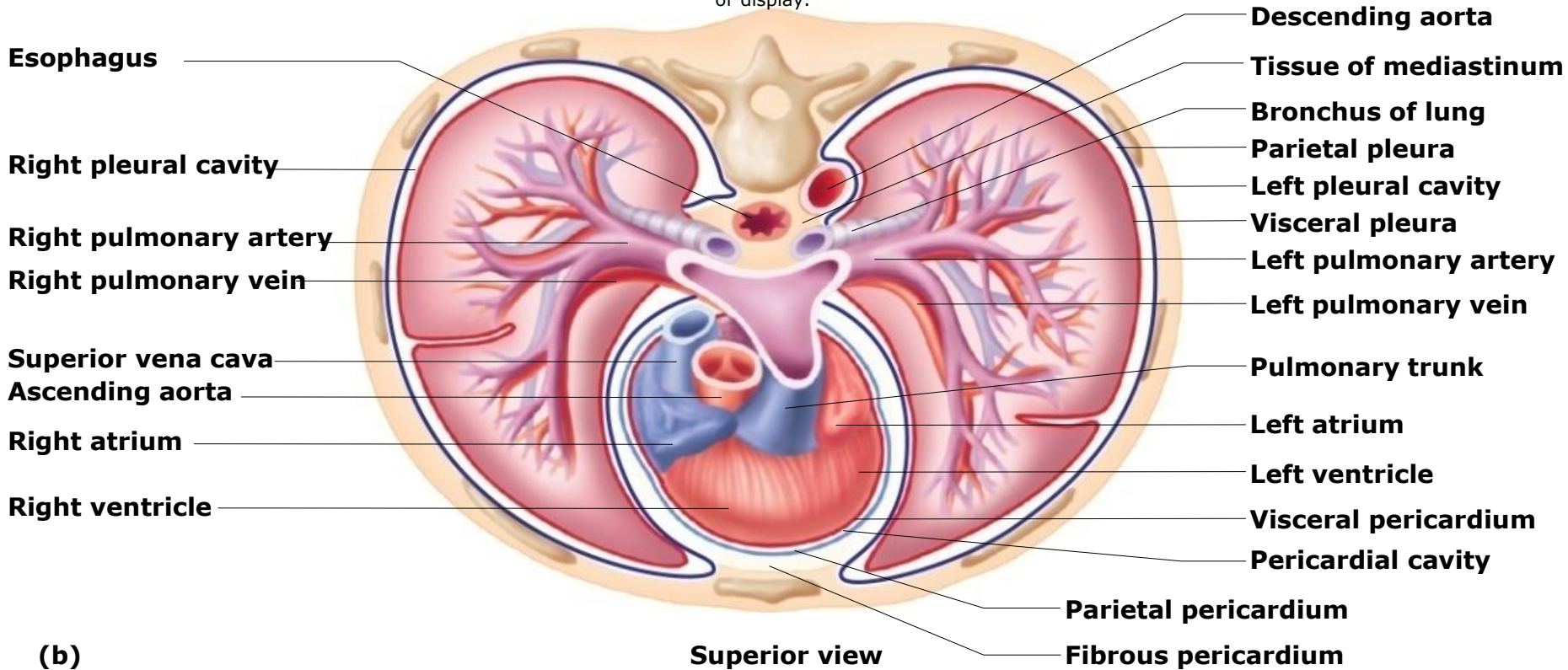


(a) Anterior view

- Size of a closed fist
- Shape
 - **Apex:** Blunt rounded point of cone
 - **Base:** Flat part at opposite of end of cone
- Located in thoracic cavity in **mediastinum** (central core of the thoracic cavity; everything in the thoracic cavity except the lungs.)
 - Important clinically when using a stethoscope, performing an ECG, or performing CPR

Heart Cross Section

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Anatomy of the Heart

- **Pericardium** or pericardial sac
 - **Fibrous pericardium**: tough fibrous outer layer. Prevents over distention; acts as anchor
 - **Serous pericardium**: thin, transparent, inner layer. Simple squamous epithelium
 - **Parietal pericardium**: lines the fibrous outer layer
 - **Visceral pericardium** (epicardium): covers heart surface
 - The two are continuous and have a pericardial cavity between them filled with pericardial fluid

Anatomy of the Heart

Pericardium

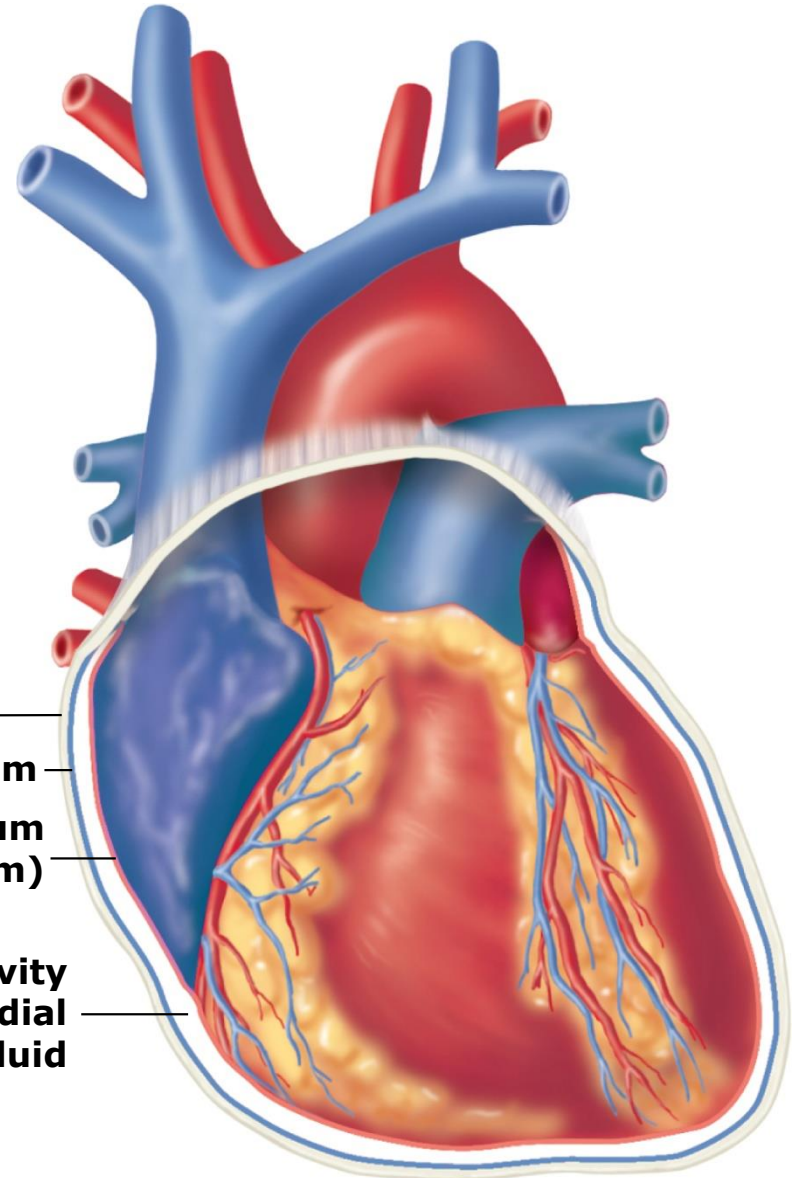
Fibrous pericardium

Serous pericardium

Parietal pericardium

Visceral pericardium
(or epicardium)

Pericardial cavity
filled with pericardial
fluid

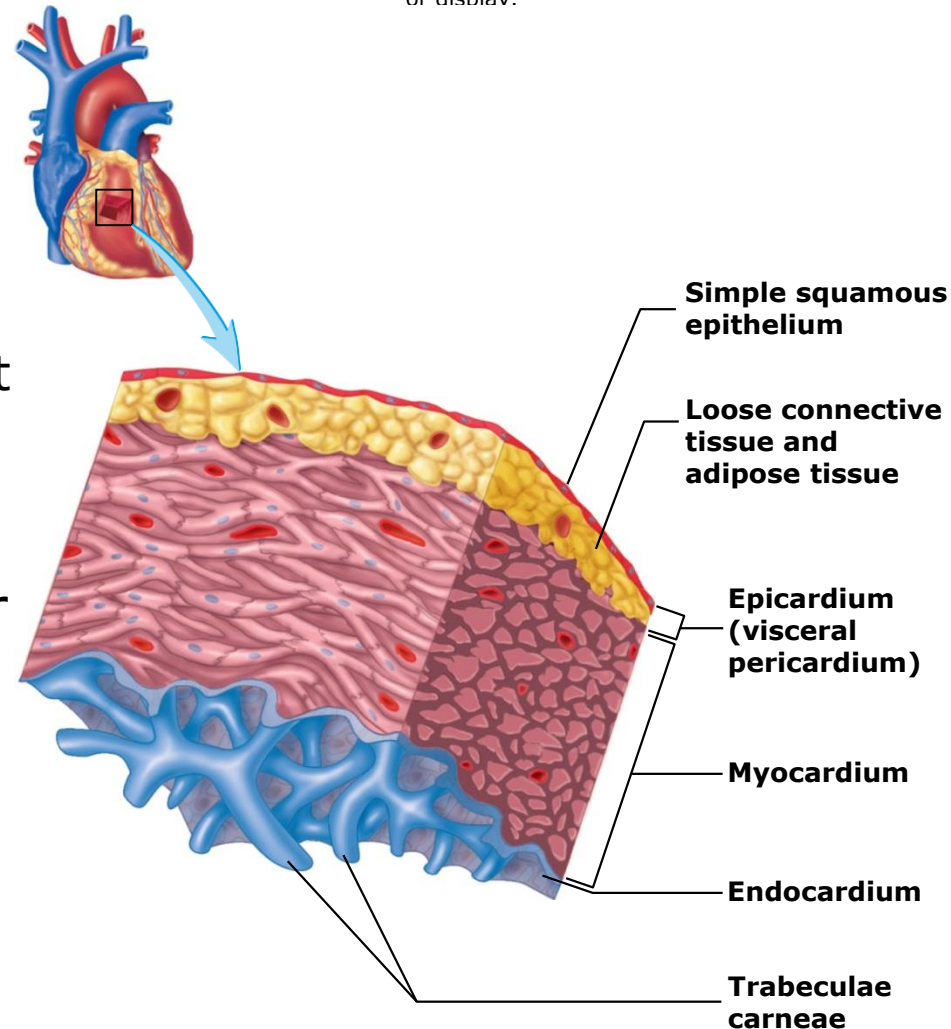


Anterior view

Heart Wall

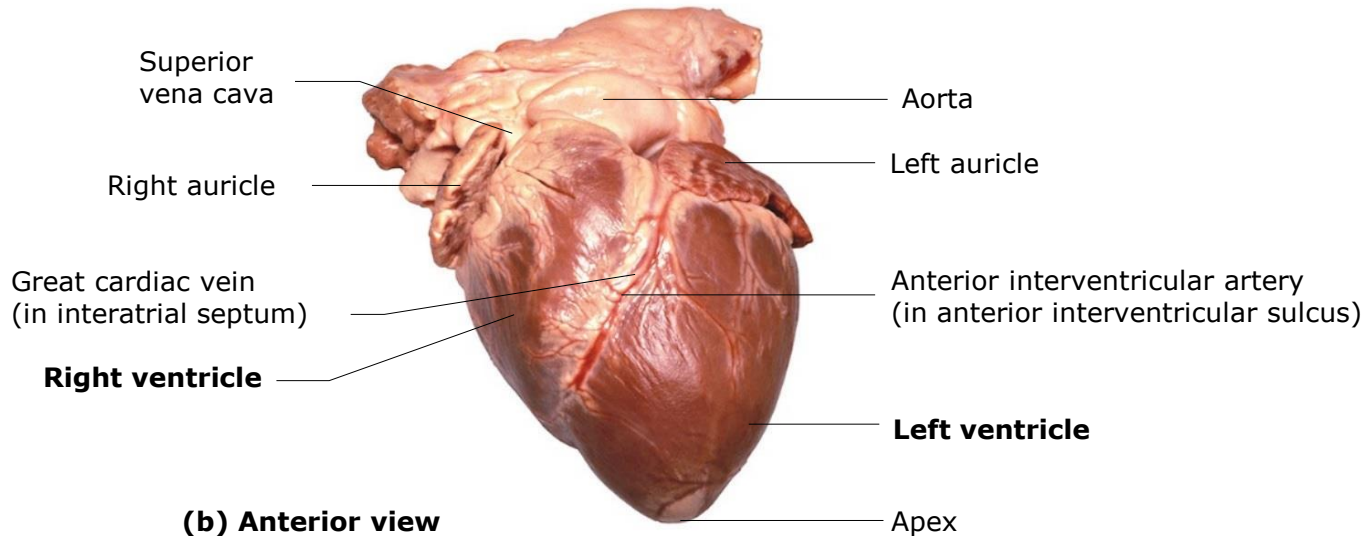
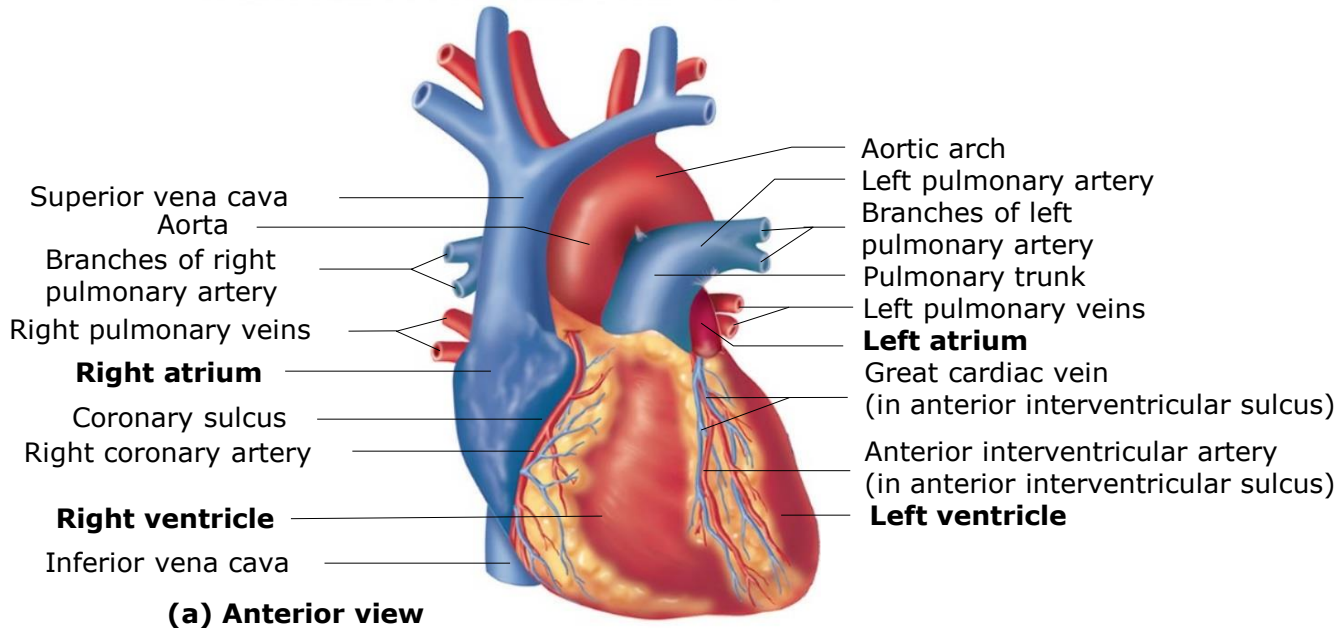
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- Three layers of tissue
 - **Epicardium:** Serous membrane; smooth outer surface of heart
 - **Myocardium:** Middle layer composed of cardiac muscle cell and responsibility for heart contracting
 - **Endocardium:** Smooth inner surface of heart chambers
- **Pectinate muscles:** muscular ridges in auricles and right atrial wall
- **Trabeculae carneae:** muscular ridges and columns on inside walls of ventricles



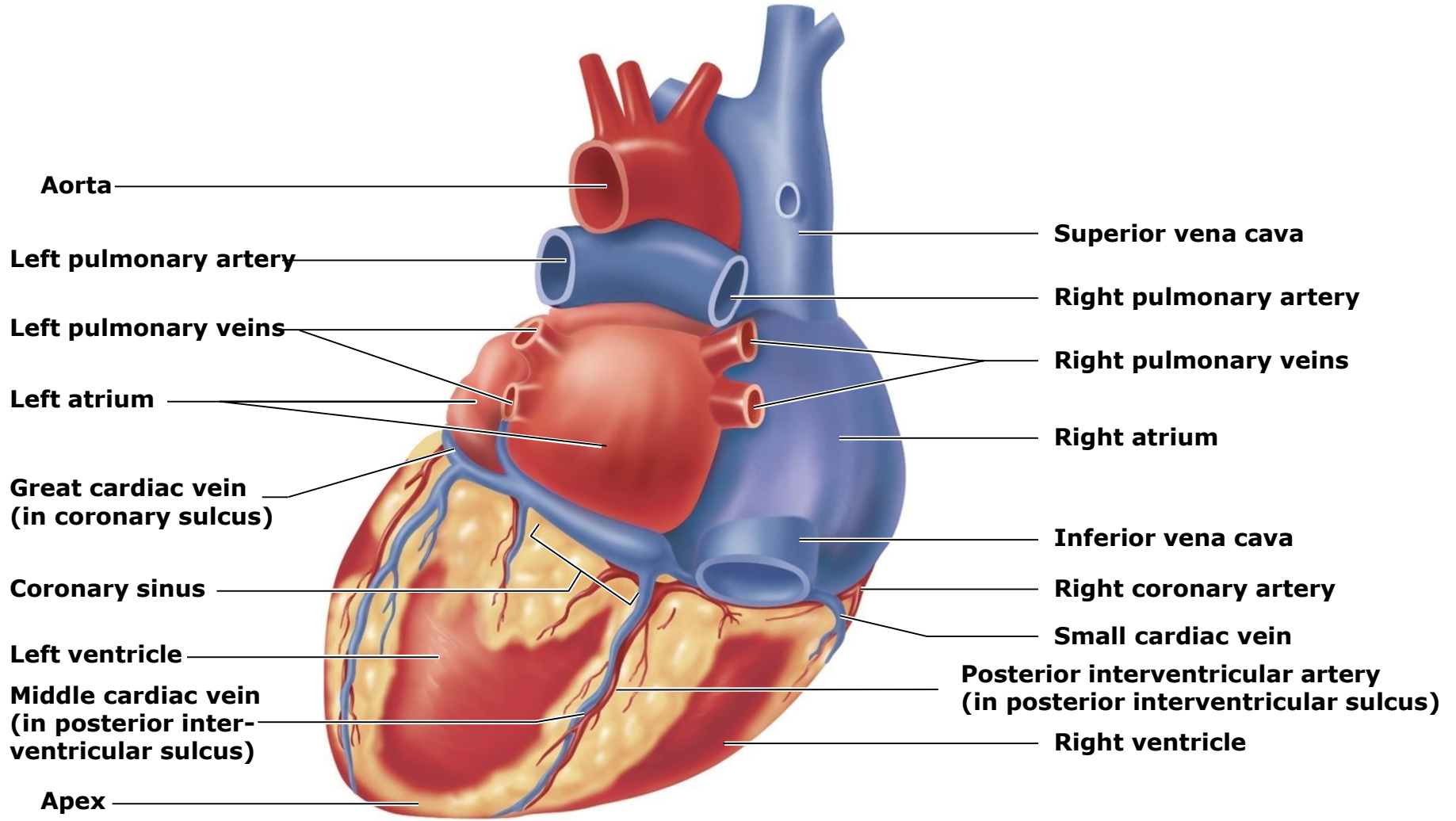
External Anatomy

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External Anatomy

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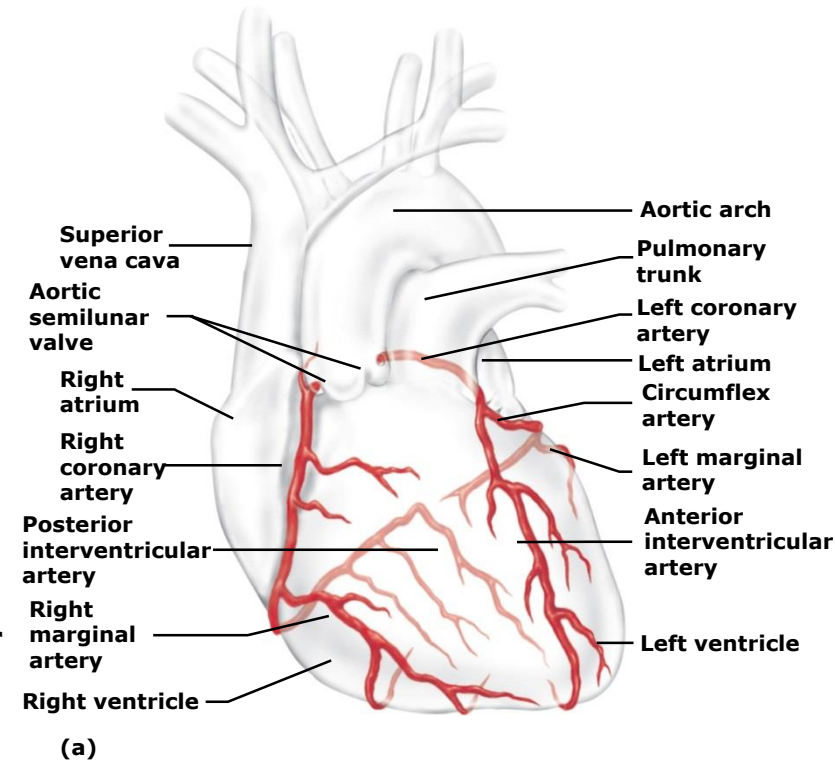


(c) Posterior view

Coronary Circulation: Arteries

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- **Right coronary artery** exits aorta just superior to point where aorta exits heart; lies in coronary sulcus. Smaller than left. Extends to posterior aspect of heart
 - **Right marginal artery** to lateral wall of right ventricle
 - **Posterior interventricular artery** lies in posterior interventricular sulcus, supplies posterior and inferior aspects of heart

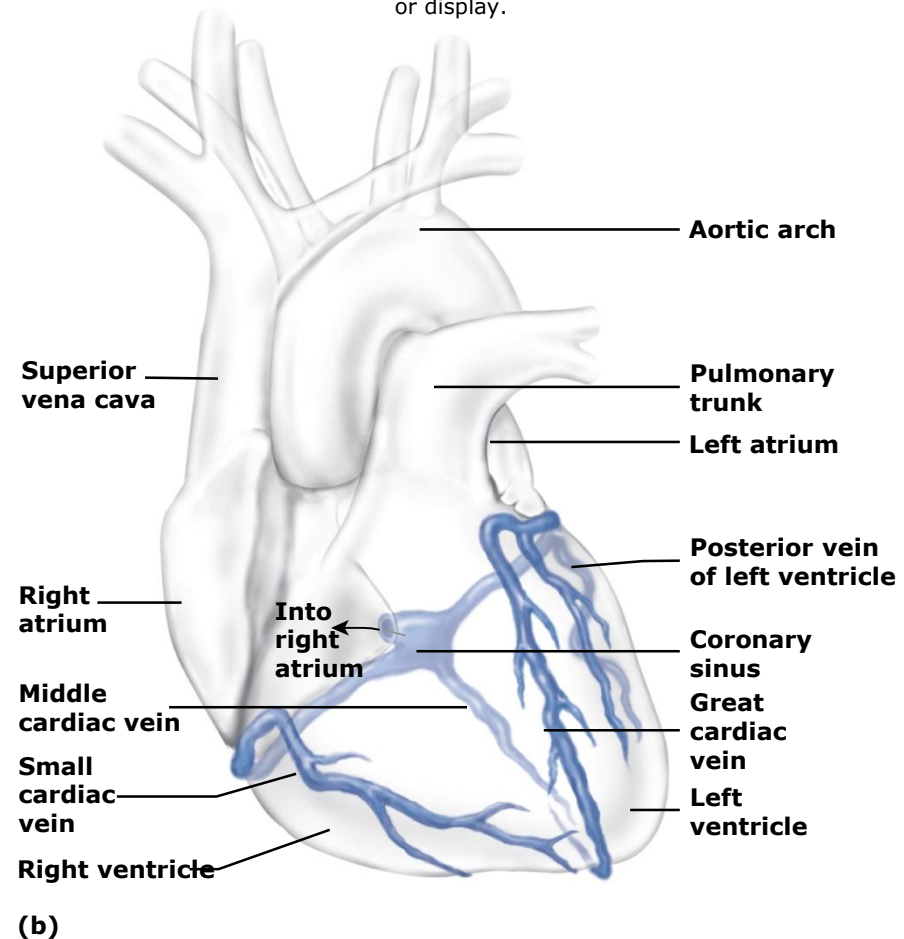


- **Left coronary artery** exits aorta near right coronary. Branches
 - **Anterior interventricular artery** (left anterior descending artery) in anterior interventricular sulcus
 - **Left marginal artery** supplies lateral wall of left ventricle
 - **Circumflex artery** extends to posterior aspect

Coronary Circulation: Veins

- **Great cardiac vein** and small cardiac vein drain right margin of heart
- **Coronary sinus:** veins empty here then into the right atrium
- Number of small veins drain the rest of the heart

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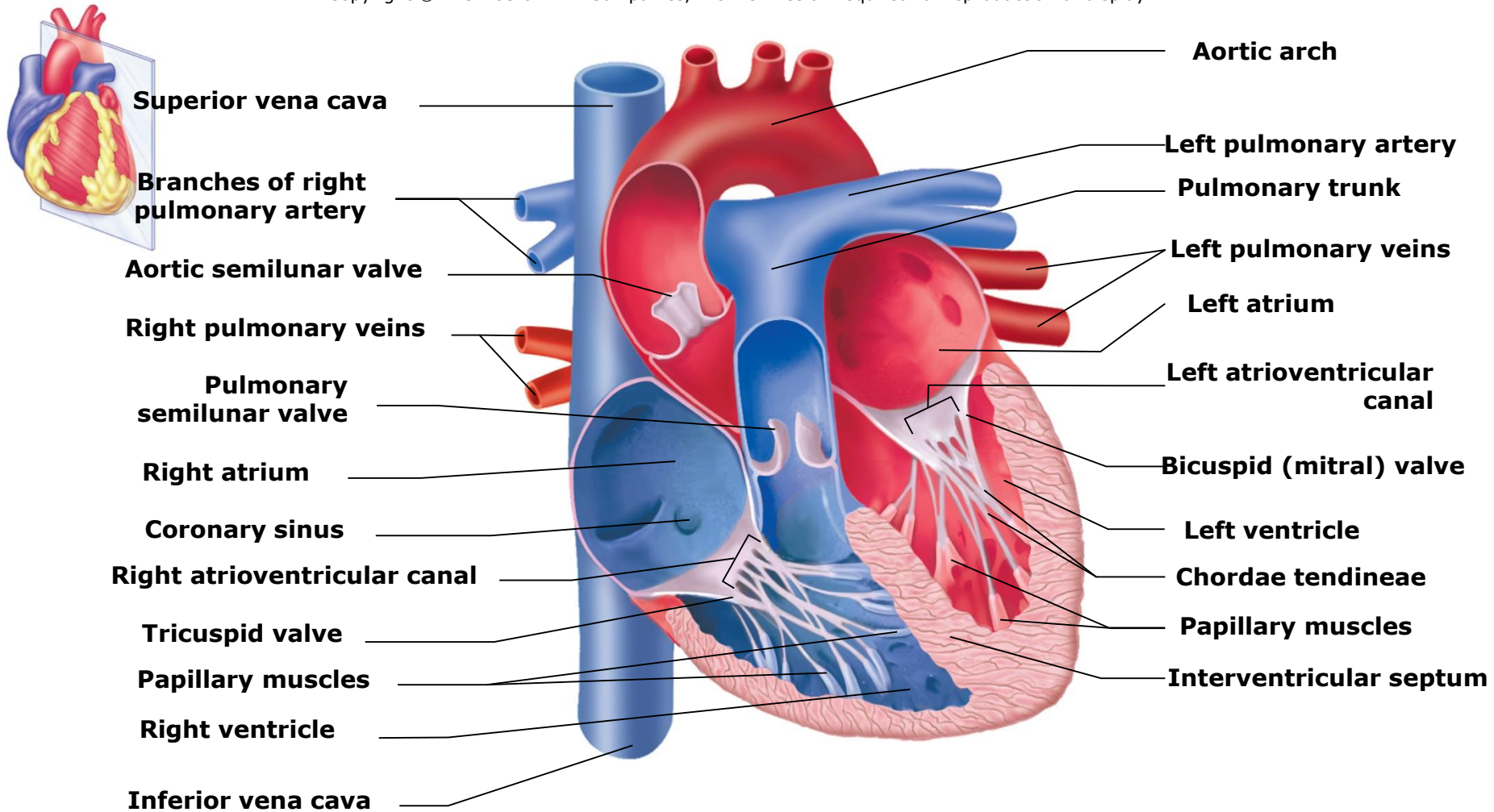


Heart Chambers

- Atria
 - **Right atrium:** three major openings to receive blood returning from the body (superior vena cava, inferior vena cava, coronary sinus)
 - **Left atrium:** four openings that receive blood from pulmonary veins
 - **Interatrial septum:** wall between the atria. Contains a depression, the foramen ovale, a remnant of the fetal opening between the atria
- Ventricles
 - **Atrioventricular canals:** openings between atria and respective ventricles
 - **Right ventricle** opens to pulmonary trunk
 - **Left ventricle** opens to aorta
 - **Interventricular septum** between the two.

Heart Chambers

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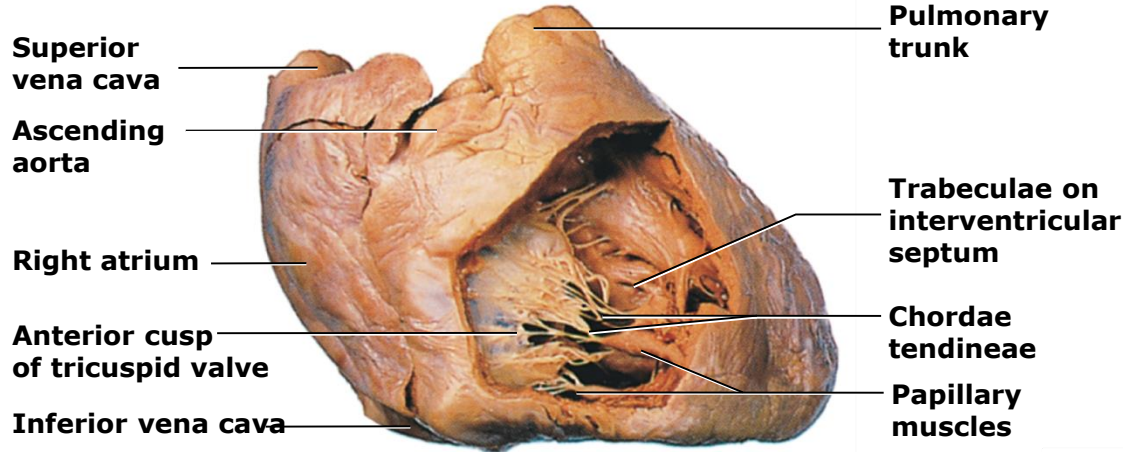


Structure of the Heart Valves

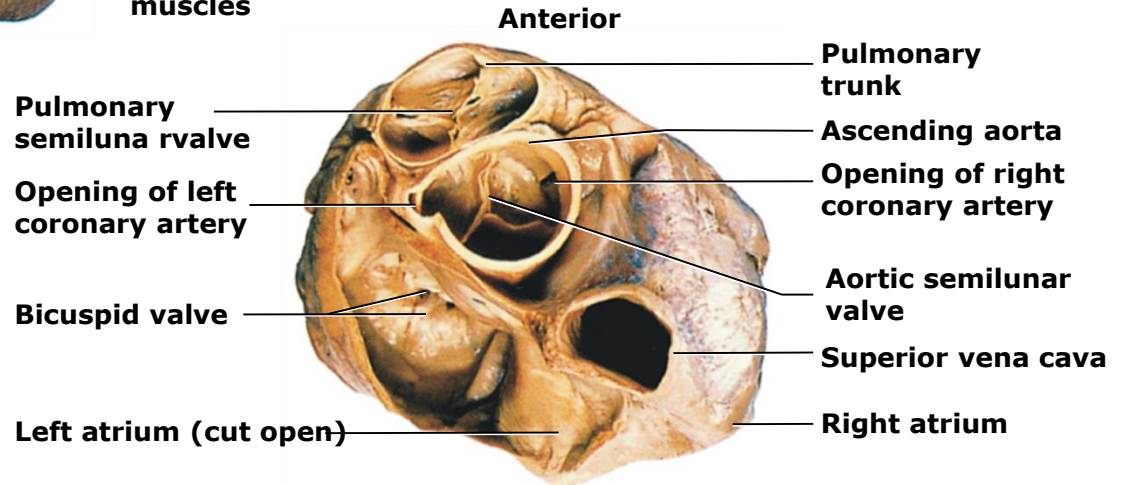
- **Atrioventricular valves** (AV valves). Each valve has leaf-like cusps that are attached to cone-shaped papillary muscles by tendons (chordae tendineae). Right has three cusps (tricuspid). Left has two cusps (bicuspid, mitral). When valve is open, canal is atrioventricular canal.
- **Semilunar valves**. Right (pulmonary); left (aortic). Each cusp is shaped like a cup. When cusps are filled, valve is closed; when cusps are empty, valve is open.

Heart Chambers

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(a) Anterior view

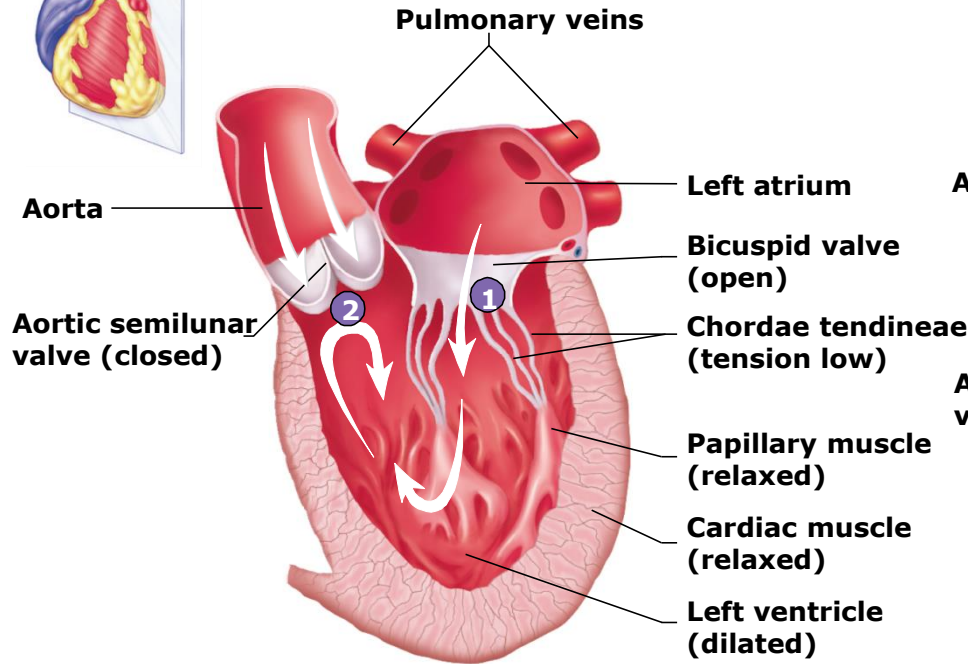
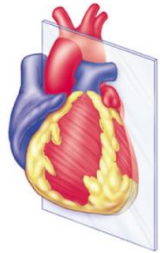


(b) Superior view

b. © McMinn & Hutchings, Color Atlas of Human Anatomy/Mosby

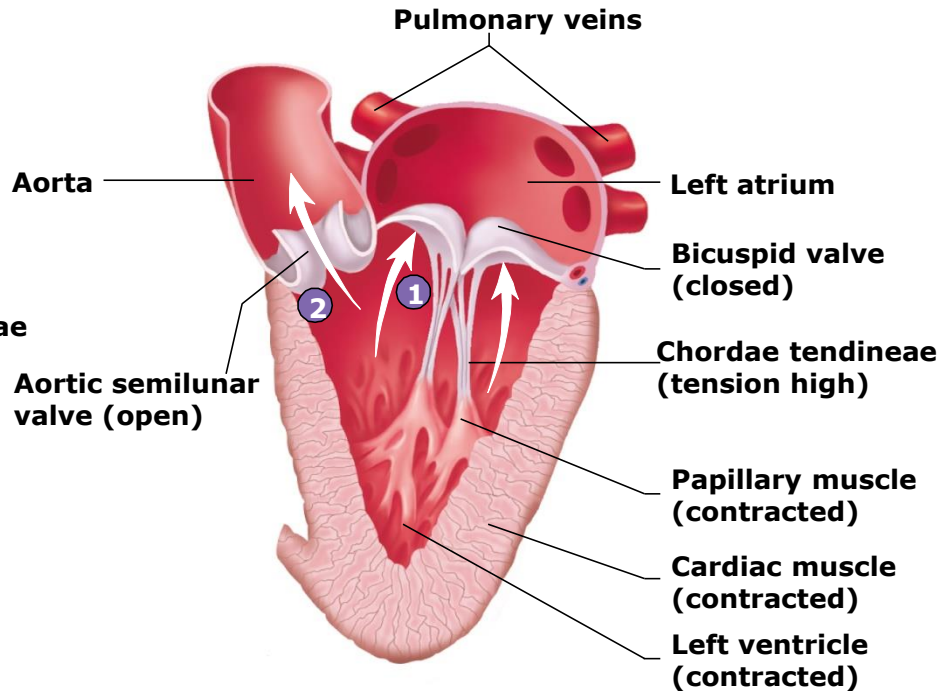
Function of Heart Valves

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(a) Valve positions when blood is flowing into the left ventricle.

- 1 The bicuspid valve is open. The cusps of the valve are pushed by the blood into the ventricle.
- 2 The aortic semilunar valve is closed. The cusps of the valve overlap as they are pushed by the blood in the aorta toward the ventricle.

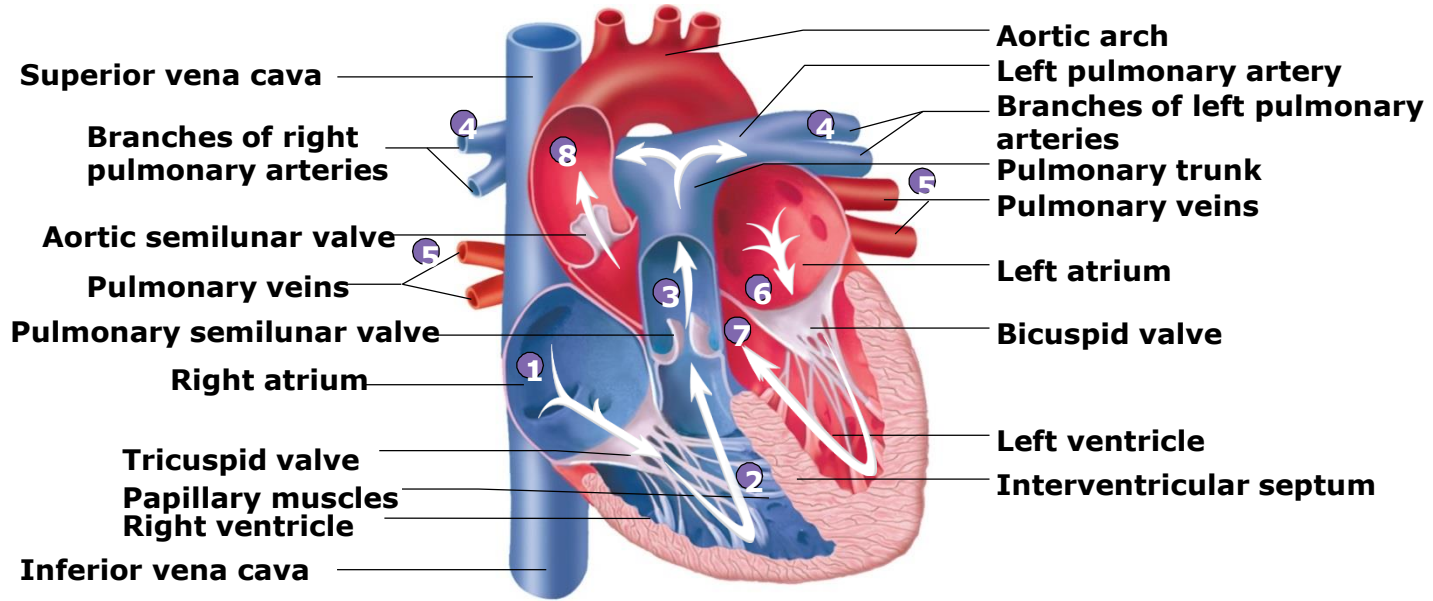


(b) Valve positions when blood is flowing out of the left ventricle.

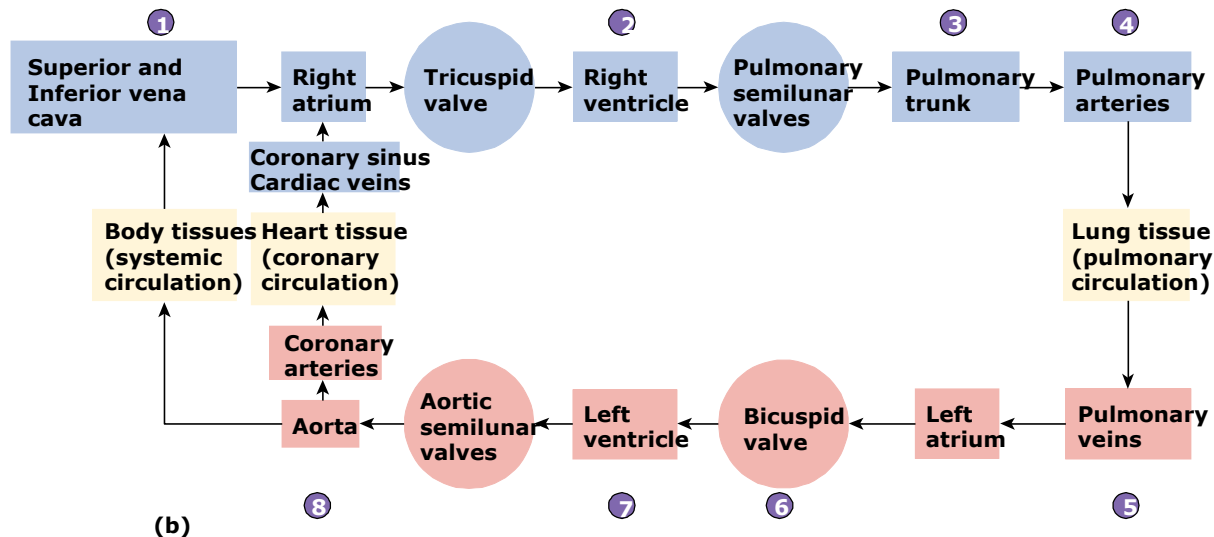
- 1 The bicuspid valve is closed. The cusps of the valves overlap as they are pushed by the blood toward the left atrium.
- 2 The aortic semilunar valve is open. The cusps of the valve are pushed by the blood toward the aorta.

Route of Blood Flow Through Heart

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(a)



(b)

Histology

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• Heart Skeleton

Skeleton of the heart, including fibrous rings around valves

Bicuspid valve

Pulmonary semilunar valve

Aortic semilunar valve

Tricuspid valve

Cardiac muscle of the right ventricle

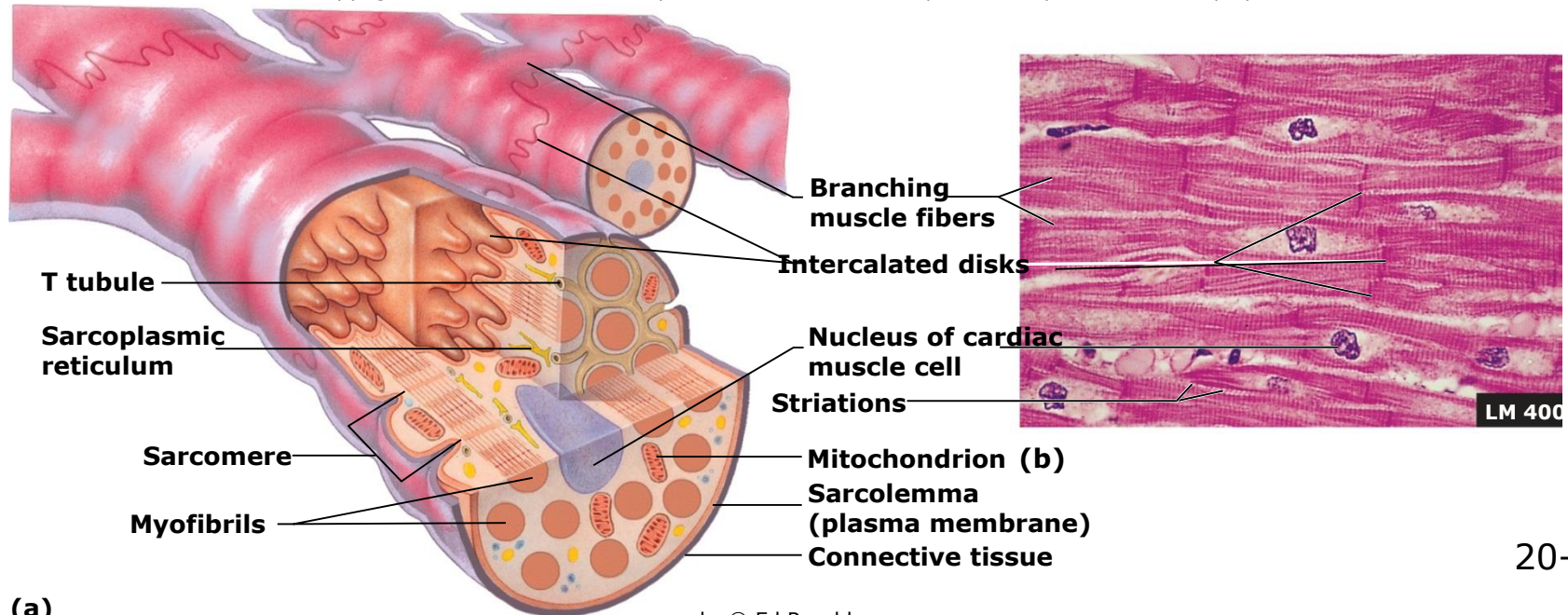
Cardiac muscle of the left ventricle

- Consists of plate of fibrous connective tissue between atria and ventricles
- Fibrous rings around valves to support
- Serves as electrical insulation between atria and ventricles
- Provides site for muscle attachment

Cardiac Muscle

- Elongated, branching cells containing 1-2 centrally located nuclei
- Contains actin and myosin myofilaments
- **Intercalated disks:** specialized cell-cell contacts.
 - Cell membranes interdigitate
 - Desmosomes hold cells together
 - Gap junctions allow action potentials to move from one cell to the next.
- Electrically, cardiac muscle of the atria and of the ventricles behaves as single unit

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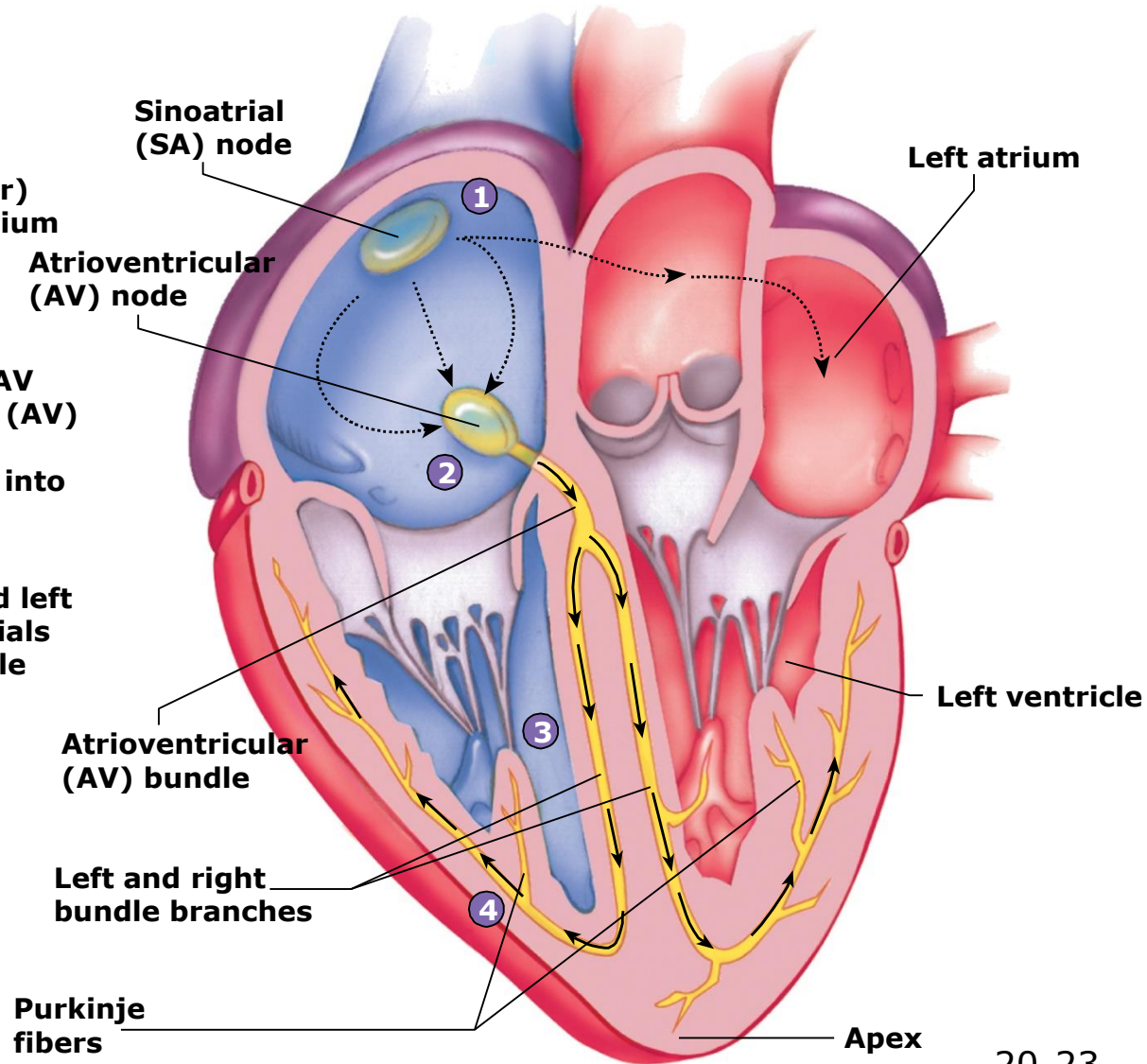
(a)

b: © Ed Reschke

Conducting System

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- 1** Action potentials originate in the sinoatrial (SA) node (the pacemaker) and travel across the wall of the atrium (arrows) from the SA node to the atrioventricular (AV) node.
- 2** Action potentials pass through the AV node and along the atrioventricular (AV) bundle, which extends from the AV node, through the fibrous skeleton, into the interventricular septum.
- 3** The AV bundle divides into right and left bundle branches, and action potentials descend to the apex of each ventricle along the bundle branches.
- 4** Action potentials are carried by the Purkinje fibers from the bundle branches to the ventricular walls and papillary muscles.

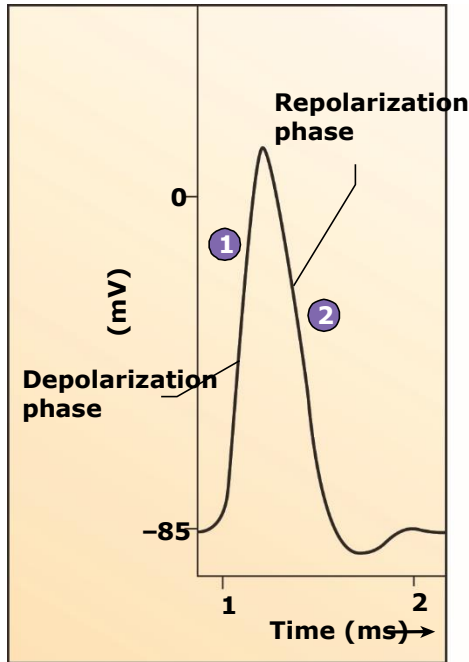


Conducting System

- **SA node:** sinoatrial node. Medial to opening of superior vena cava. The pacemaker. Specialized cardiac muscle cells. Generate spontaneous action potentials. Action potentials pass to atrial muscle cells and to the AV node
- **AV node:** atrioventricular node. Medial to the right atrioventricular valve. Action potentials conducted more slowly here than in any other part of system. Ensures ventricles receive signal to contract after atria have contracted
- **AV bundle:** passes through hole in cardiac skeleton to reach interventricular septum
- **Right and left bundle branches:** extend beneath endocardium to apices of right and left ventricles
- **Purkinje fibers:** Large diameter cardiac muscle cells with few myofibrils. Many gap junctions. Conduct action potential to ventricular muscle cells

Electrical Properties

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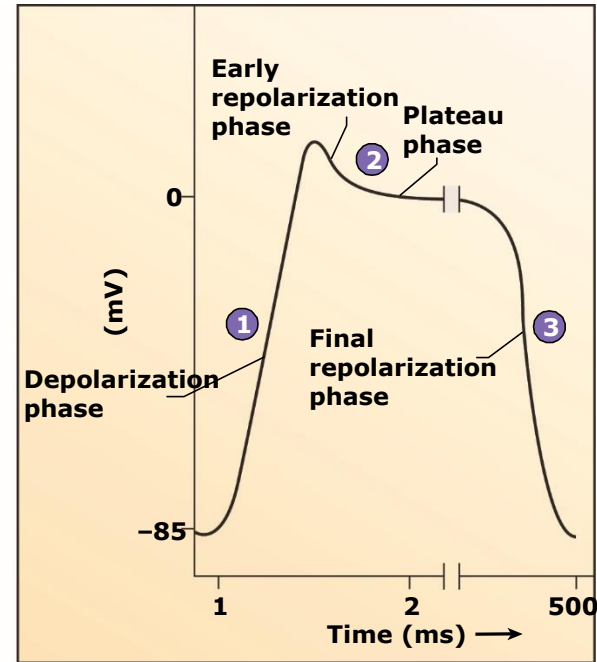
Permeability changes during an action potential in skeletal muscle:

1 Depolarization phase

- Voltage-gated Na^+ channels open.
- Voltage-gated K^+ channels begin to open.

2 Repolarization phase

- Voltage-gated Na^+ channels close.
- Voltage-gated K^+ channels continue to open.
- Voltage-gated K^+ channels close at the end of repolarization and return the membrane potential to its resting value.



Permeability changes during an action potential in cardiac muscle:

1 Depolarization phase

- Voltage-gated Na^+ channels open.
- Voltage-gated K^+ channels close.
- Voltage-gated Ca^{2+} channels begin to open.

2 Early repolarization and plateau phases

- Voltage-gated Na^+ channels close.
- Some voltage-gated K^+ channels open, causing early repolarization.
- Voltage-gated Ca^{2+} channels are open, producing the plateau by slowing further repolarization.

3 Final repolarization phase

- Voltage-gated Ca^{2+} channels close.
- Many voltage-gated K^+ channels open.

Differences Between Skeletal and Cardiac Muscle Physiology

- Cardiac: action potentials conducted from cell to cell. In skeletal, action potential conducted along length of single fiber
- Cardiac: rate of action potential propagation is slow because of gap junctions and small diameter of fibers. In skeletal it is faster due to larger diameter fibers.
- Cardiac: calcium-induced calcium release (CICR). Movement of Ca^{2+} through plasma membrane and T tubules into sarcoplasm stimulates release of Ca^{2+} from sarcoplasmic reticulum

Autorhythmicity of Cardiac Muscle

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Permeability changes in pacemaker cells

1 Pacemaker potential

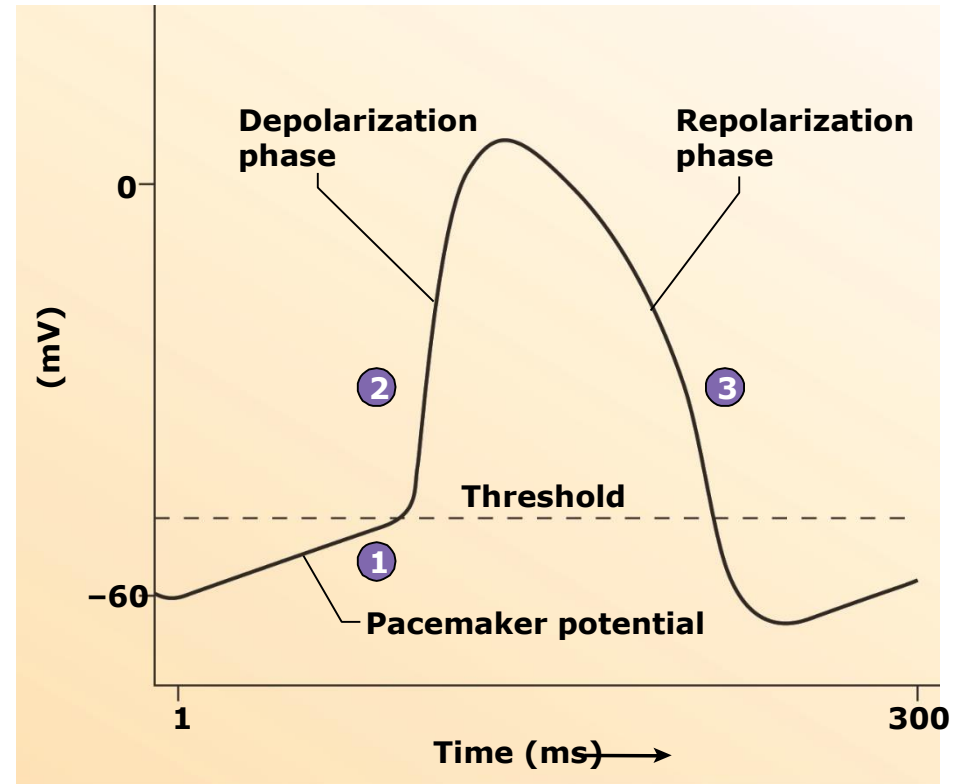
- A small number of Na^+ channels are open.
- Voltage-gated K^+ channels that opened in the repolarization phase of the previous action potential are closing.
- Voltage-gated Ca^{2+} channels begin to open.

2 Depolarization phase

- Voltage-gated Ca^+ channels are open.
- Voltage-gated K^+ channels are closed.

3 Repolarization phase

- Voltage-gated Ca^{2+} channels close.
- Voltage-gated K^+ channels open.



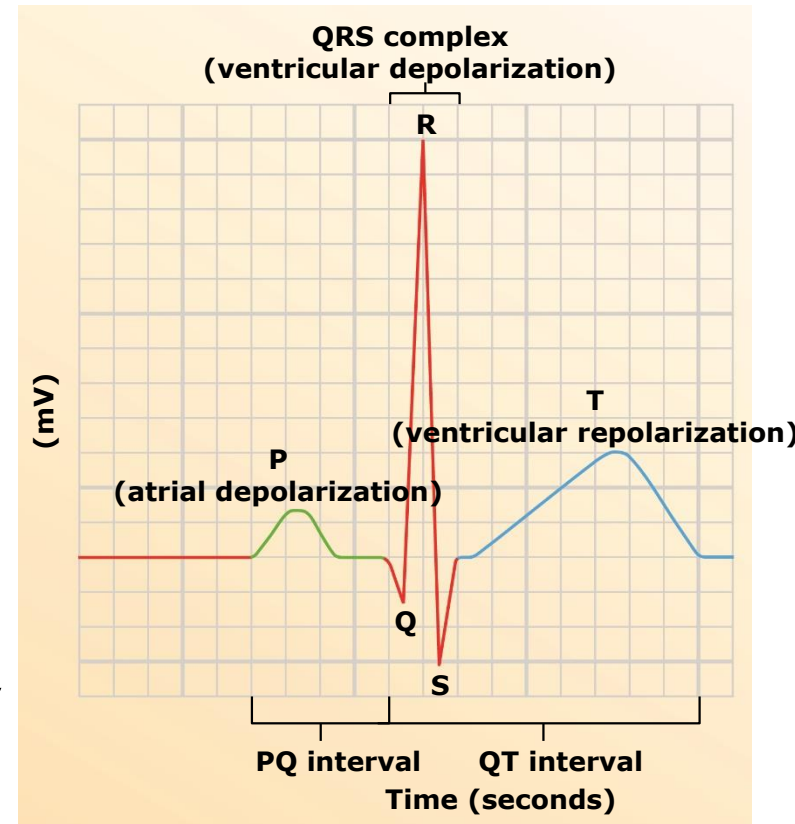
Refractory Period

- **Absolute:** Cardiac muscle cell completely insensitive to further stimulation
- **Relative:** Cell exhibits reduced sensitivity to additional stimulation
- Long refractory period prevents tetanic contractions

Electrocardiogram

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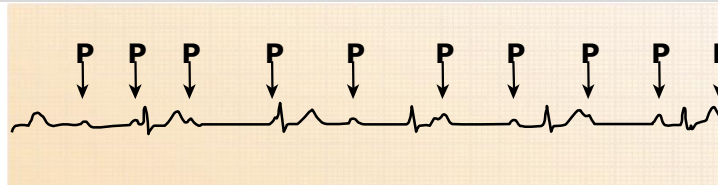
- Record of electrical event in the myocardium that can be correlated with mechanical events
- **P wave**: depolarization of atrial myocardium and signals onset of atrial contraction
- **QRS complex**: ventricular depolarization and signals onset of ventricular contraction. Repolarization of atria simultaneously.
- **T wave**: repolarization of ventricles; precedes ventricular relaxation



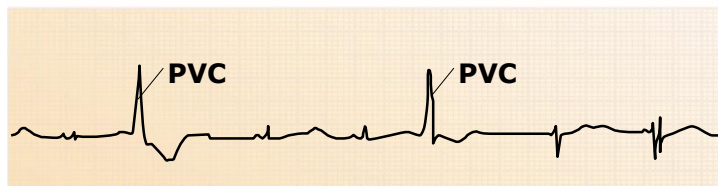
- **PQ interval** or PR interval: 0.16 sec; atria contract and begin to relax, ventricles begin to contract
- **QT interval**: 0.36 sec; ventricles contract and begin to relax



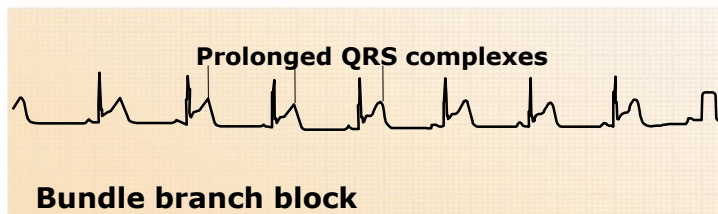
Electrocardiogram



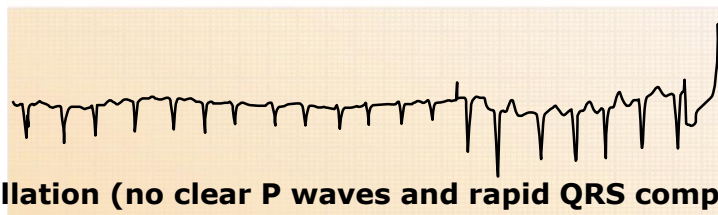
Complete heart block (P waves and QRS complexes are not coordinated)



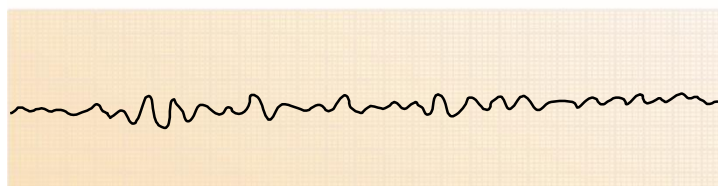
Premature ventricular contraction (PVC) (no P waves precede PVCs)



Bundle branch block



Atrial fibrillation (no clear P waves and rapid QRS complexes)



Ventricular fibrillation (no P, QRS, or T waves)

TABLE 20.1**Major Cardiac Arrhythmias**

Conditions	Symptoms	Possible Causes
Abnormal Heart Rhythms		
Tachycardia	Heart rate in excess of 100 beats per minute (bpm)	Elevated body temperature; excessive sympathetic stimulation; toxic conditions
Paroxysmal atrial tachycardia	Sudden increase in heart rate to 95–150 bpm for a few seconds or even for several hours; P wave precedes every QRS complex; P wave inverted and superimposed on T wave	Excessive sympathetic stimulation; abnormally elevated permeability of slow channels
Ventricular tachycardia	Frequently causes fibrillation	Often associated with damage to AV node or ventricular muscle
Abnormal Rhythms Resulting from Ectopic Action Potentials		
Atrial flutter	300 P waves/min; 125 QRS complexes/min, resulting in two or three P waves (atrial contraction) for every QRS complex (ventricular contraction)	Ectopic action potentials in the atria
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Ventricular fibrillation	No QRS complexes; no rhythmic contraction of the myocardium; many patches of asynchronously contracting ventricular muscle	Ectopic action potentials in the ventricles
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Second-degree	PR interval 0.25–0.45 second; some P waves trigger QRS complexes and others do not; 2:1, 3:1, and 3:2 P wave/QRS complex ratios may occur	Excessive vagal stimulation
Third-degree (complete heart block)	P wave dissociated from QRS complex; atrial rhythm approximately 100 bpm; ventricular rhythm less than 40 bpm	Ischemia of AV nodal fibers or compression of AV bundle
Premature atrial contractions	Occasional shortened intervals between contractions; frequently occurs in healthy people P wave superimposed on QRS complex	Excessive smoking; lack of sleep; too much caffeine; alcoholism
Premature ventricular contractions (PVCs)	Prolonged QRS complex; exaggerated voltage because only one ventricle may depolarize; inverted T wave; increased probability of fibrillation	Ectopic foci in ventricles; lack of sleep; too much caffeine, irritability; occasionally occurs with coronary thrombosis

Abbreviations: SA = sinoatrial; AV = atrioventricular.

Cardiac Cycle

- Heart is two pumps that work together, right and left half
- Repetitive contraction (**systole**) and relaxation (**diastole**) of heart chambers
- Blood moves through circulatory system from areas of higher to lower pressure.
 - Contraction of heart produces the pressure

Period of Isovolumetric Contraction

- Begins at the completion of the QRS complex.
- Ventricular muscles start to contract, increasing the pressure inside the ventricles. This causes the AV valves to close, which is the beginning of ventricular systole. The semilunar valves were closed in the previous diastole and remain closed during this event.
- 120-130 mL of blood are in the ventricles, left from the last diastole when the atria emptied into the ventricles. This is referred to as the end diastolic volume.

Period of Ejection

- Pressure in the ventricle has increased to the point where it is greater than the pressure in the pulmonary trunk/aorta. This pushes the cusps of the semilunar valves against the walls of the vessels, opening the valve.
- Blood is ejected from the ventricles.
- The pressures in the two ventricles are different: 120 mm Hg in the left ventricle; 25 mm Hg in the right ventricle. Remember: blood in the left ventricle must be pumped to the whole body; blood in the right ventricle is pumped to the lungs.
- After the first initial spurt, pressure starts to drop.
- At the end of the period of ejection, 50-60 mL remain: end-systolic volume.

Period of Isovolumetric Relaxation

- Completion of T wave results in ventricular repolarization and relaxation.
- Ventricular pressure falls very rapidly.
- Pulmonary trunk/aorta pressure is higher than ventricular pressure.
- Elastic recoil of the arteries causes blood to flow back toward the relaxed ventricles: the semilunar valves close, which is the beginning of ventricular diastole.
- Note that the AV valves are also closed.

Passive Ventricular Filling

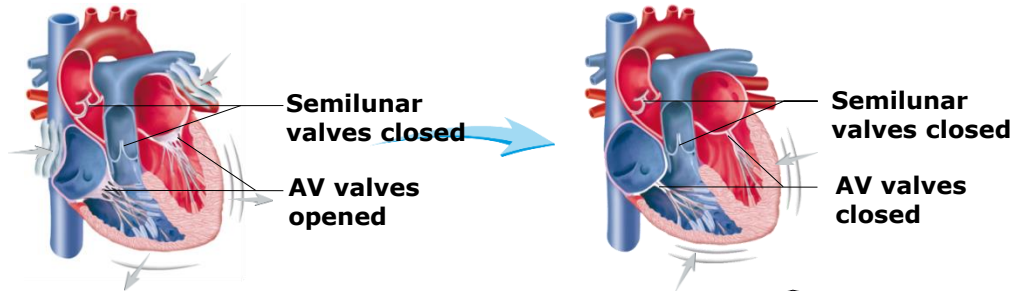
- While the ventricles were in systole, the atria were filling with blood.
- Atrial pressure rises above ventricular pressure and the AV valves open.
- Blood flows into the relaxed ventricles, accounting for most of the ventricular filling (70%).

Active Ventricular Filling

- Depolarization of the SA node generates action potentials that spread over the atria (P wave) and the atria contract. This completes ventricular filling.
- At rest, contraction of atria not necessary for heart function.
- During exercise, atrial contraction necessary for function as heart pumps 300-400%.

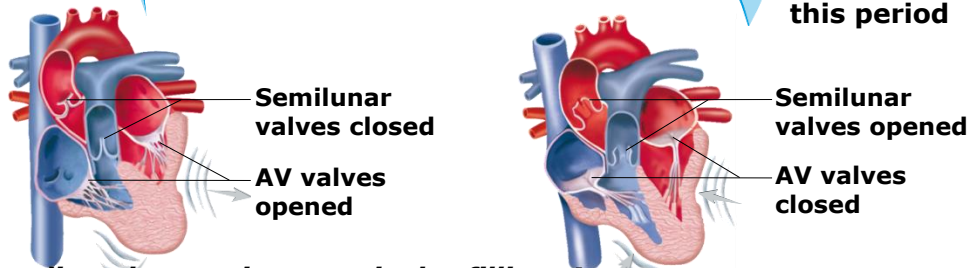
Cardiac Cycle

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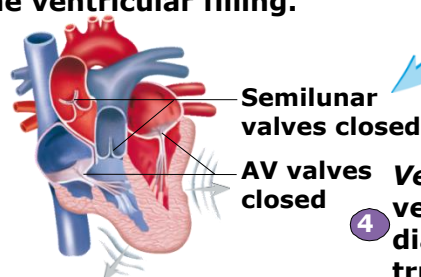
1 Atrial systole: active ventricular filling. The atria contract, increasing atrial pressure and completing ventricular filling while the ventricles are relaxed.

2 Ventricular systole: period of isovolumetric contraction. The atria are relaxed, and blood flows into them from the veins. ventricular contraction causes ventricular pressure to increase and causes the AV valves to close, which is the beginning of ventricular systole. The semilunar valves were closed in the previous diastole and remain closed during this period



5 Ventricular diastole: passive ventricular filling. As ventricular relaxation continues, the AV valves open, and blood flows from the atria into the relaxing ventricles, accounting for most of the ventricular filling.

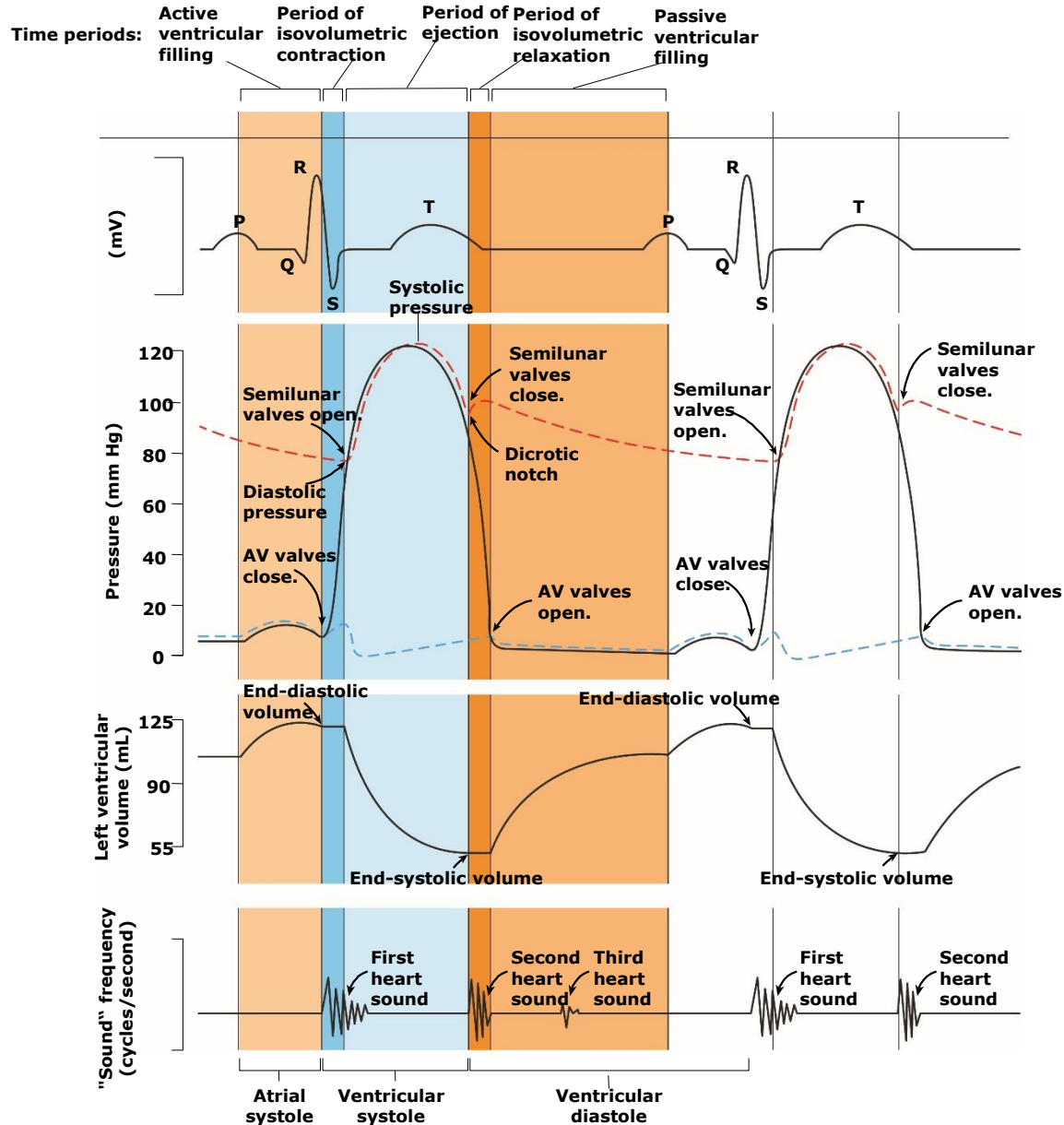
3 Ventricular systole: period of ejection. Continued ventricular contraction causes a greater increase in ventricular pressure, which pushes blood out of the ventricles, causing the semilunar valves to open.



4 Ventricular diastole: period of isovolumetric relaxation. As the ventricles begin to relax at the beginning of ventricular diastole, blood flowing back from the aorta and pulmonary trunk toward the relaxing ventricles causes the semilunar valves to close. Note that the AV valves are closed also.

Events Occurring During the Cardiac Cycle

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TABLE 20.2 Summary of the Events of the Cardiac Cycle

	Atrial Systole	Atrial Diastole
	Ventricular Diastole	Ventricular Systole
	Active Ventricular Filling	Period of Isovolumetric Contraction
Time Period	Contraction of the atria pumps blood into the ventricles.	The ventricles begin to contract, but ventricular volume does not change.
Condition of Valves	The semilunar valves are closed; the AV valves are opened (figure 20.18, <i>step 1</i>).	The semilunar valves are closed; the AV valves are closed (figure 20.18, <i>step 2</i>).
ECG	The P wave is completed and the atria are stimulated to contract. Action potentials are delayed in the AV node for 0.11 second, allowing time for the atria to contract. The QRS complex begins as action potentials are propagated from the AV node to the ventricles.	The QRS complex is completed, and the ventricles are depolarized. As a result, the ventricles begin to contract. Atrial repolarization is masked by the QRS complex. The atria are relaxed (atrial diastole).
Atrial Pressure Graph	Atrial contraction (systole) causes an increase in atrial pressure, and blood is forced to flow from the atria into the ventricles.	Atrial pressure decreases in the relaxed atria. When atrial pressure is less than venous pressure, blood flows into the atria. Atrial pressure increases briefly as the contracting ventricles push blood back toward the atria.
Ventricular Pressure Graph	Atrial contraction (systole) and the movement of blood into the ventricles cause a slight increase in ventricular pressure.	Ventricular contraction causes an increase in ventricular pressure, which causes blood to flow toward the atria, closing the AV valves. Ventricular pressure increases rapidly.
Aortic Pressure Graph	Aortic pressure gradually decreases as blood runs out of the aorta into other systemic blood vessels.	Just before the semilunar valves open, pressure in the aorta decreases to its lowest value, called the diastolic pressure (approximately 80 mm Hg).
Volume Graph	Atrial contraction (systole) completes ventricular filling during the last one-third of diastole. The amount of blood in a ventricle at the end of ventricular diastole is called the end-diastolic volume .	During the period of isovolumetric contraction , ventricular volume does not change because the semilunar and AV valves are closed.
Sound Graph		Blood flowing from the ventricles toward the atria closes the AV valves. Vibrations of the valves and the turbulent flow of blood produce the first heart sound , which marks the beginning of ventricular systole

Abbreviation: AV = atrioventricular.

TABLE 20.2

Atrial Diastole		
Ventricular Systole	Ventricular Diastole	
Period of Ejection	Period of Isovolumetric Relaxation	Passive Ventricular Filling
The ventricles continue to contract, and blood is pumped out of the ventricles.	The ventricles relax, but ventricular volume does not change.	Blood flows into the ventricles because blood pressure is higher in the veins and atria than in the relaxed ventricles.
The semilunar valves are opened; the AV valves are closed (figure 20.18, <i>step 3</i>).	The semilunar valves are closed; the AV valves are closed (figure 20.18, <i>step 4</i>).	The semilunar valves are closed; the AV valves are opened (see figure 20.18, <i>step 5</i>).
The T wave results from ventricular repolarization.	The T wave is completed, and the ventricles are repolarized. The ventricles relax.	The P wave is produced when the SA node generates action potentials and a wave of depolarization begins to propagate across the atria.
Atrial pressure increases gradually as blood flows from the veins into the relaxed atria.	Atrial pressure continues to increase gradually as blood flows from the veins into the relaxed atria	After the AV valves open, atrial pressure decreases as blood flows out of the atria into the relaxed ventricles.
Ventricular pressure becomes greater than pressure in the aorta as the ventricles continue to contract. The semilunar valves are pushed open as blood flows out of the ventricles. Ventricular pressure peaks as the ventricles contract maximally; then pressure decreases as blood flow out of the ventricles decreases	Elastic recoil of the aorta pushes blood back toward the heart, causing the semilunar valves to close. After closure of the semilunar valves, the pressure in the relaxing ventricles rapidly decreases.	No significant change occurs in ventricular pressure during this time period.
As ventricular contraction forces blood into the aorta, pressure in the aorta increases to its highest value, called the systolic pressure (approximately 120 mm Hg).	After the semilunar valves close, elastic recoil of the aorta causes a slight increase in aortic pressure, producing the dicrotic notch and dicrotic wave .	Aortic pressure gradually decreases as blood runs out of the aorta into other systemic blood vessels.
After the semilunar valves open, blood volume decreases as blood flows out of the ventricles during the period of ejection . The amount of blood left in a ventricle at the end of the period of ejection is called the end-systolic volume .	During the period of isovolumetric relaxation , ventricular volume does not change because the semilunar and AV valves are closed.	After the AV valves open, blood flows from the atria and veins into the ventricles because of pressure differences. Most ventricular filling occurs during the first one-third of diastole. Little ventricular filling occurs during the middle one-third of diastole.
	Blood flowing from the ventricles toward the aorta and pulmonary trunk closes the semilunar valves. Vibrations of the valves and the turbulent flow of blood produce the second heart sound , which marks the beginning of ventricular diastole.	Sometimes the turbulent flow of blood into the ventricles produces a third heart sound .

Heart Sounds

- **First heart sound** or **“lubb”**
 - Atrioventricular valves and surrounding fluid vibrations as valves close at beginning of ventricular systole
- **Second heart sound** or **“dupp”**
 - Results from closure of aortic and pulmonary semilunar valves at beginning of ventricular diastole, lasts longer
- **Third heart sound** (occasional)
 - Caused by turbulent blood flow into ventricles and detected near end of first one-third of diastole

Aortic Pressure Curve

- **Dicrotic notch** (incisura): when the aortic semilunar valve closes, pressure within the aorta increases slightly
- Blood pressure measurement taken in the arm is a reflection of aortic pressures, not ventricular

20.8 Mean Arterial Pressure

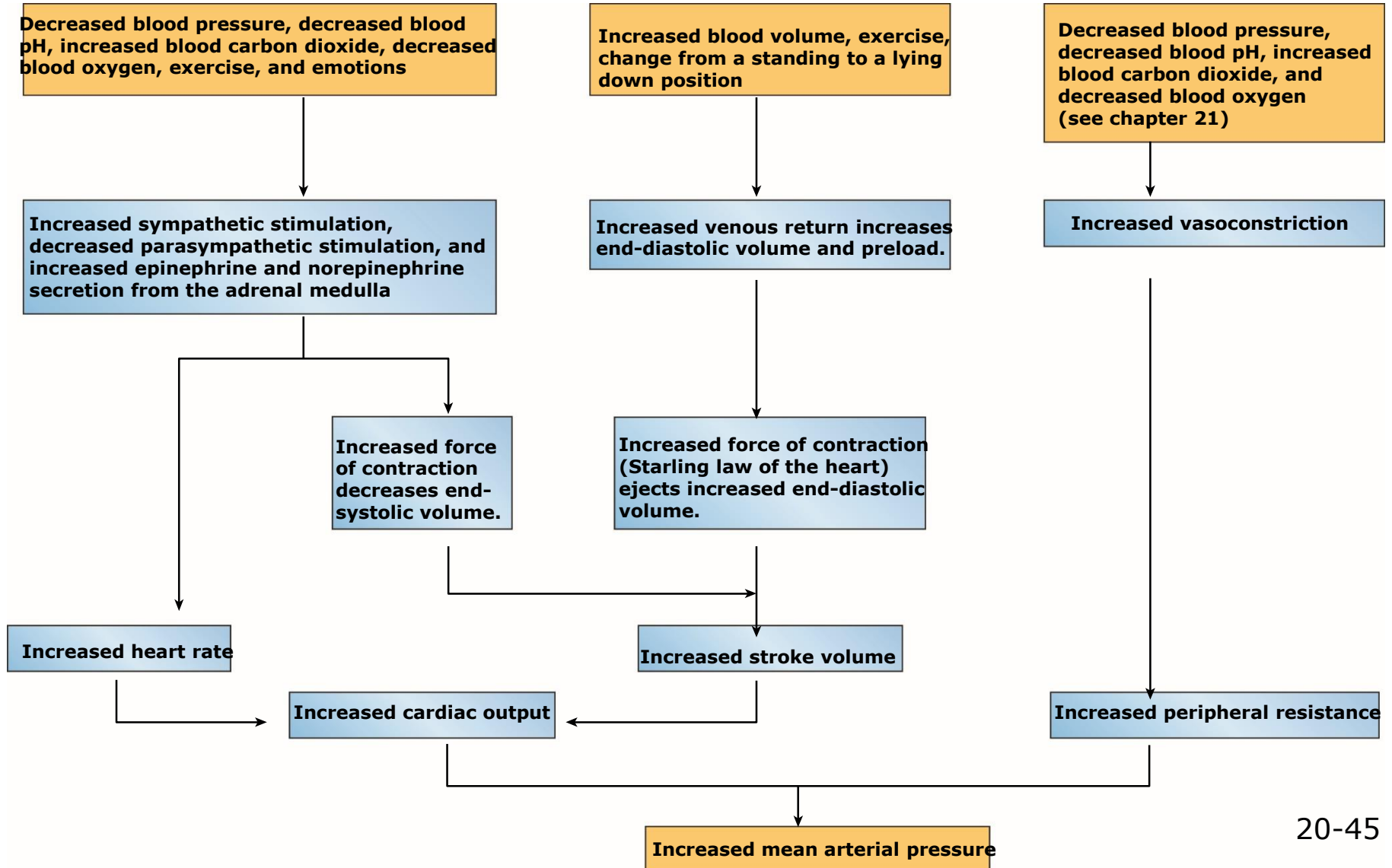
- Average blood pressure in aorta
- $MAP = CO \times PR$
 - CO is amount of blood pumped by heart per minute
 - $CO = SV \times HR$
 - SV: Stroke volume (blood pumped during each heart beat)
 - HR: Heart rate (number of times heart beats per minute)
 - Cardiac reserve: Difference between CO at rest and maximum CO
 - PR is total resistance against which blood must be pumped

Factors Affecting MAP

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Factors Affecting Cardiac Output

Factors Affecting Peripheral Resistance



20.9 Regulation of the Heart

- **Intrinsic regulation:** Results from normal functional characteristics, not on neural or hormonal regulation
 - **Preload:** Starling's law of the heart
 - Preload is the amount of stretch of the ventricular walls. The greater the stretch (preload), the greater the force of contraction.
 - **Afterload:** pressure the contracting ventricles must produce to overcome the pressure in the aorta and move blood into the aorta. Heart not as sensitive to this as it is to changes in preload.

Regulation of the Heart

- **Extrinsic regulation:** Involves neural and hormonal control
 - Parasympathetic stimulation
 - Supplied by vagus nerve, decreases heart rate, acetylcholine is secreted and hyperpolarizes the heart
 - Sympathetic stimulation
 - Supplied by cardiac nerves. Innervate the SA and AV nodes, coronary vessels and the atrial and ventricular myocardium. Increases heart rate and force of contraction. Epinephrine and norepinephrine released.
 - Increased heart beat causes increased cardiac output. Increased force of contraction causes a lower end-systolic volume; heart empties to a greater extent. Limitations: heart has to have time to fill.
 - Hormonal Control. Epinephrine and norepinephrine from the adrenal medulla. Occurs in response to increased physical activity, emotional excitement, stress

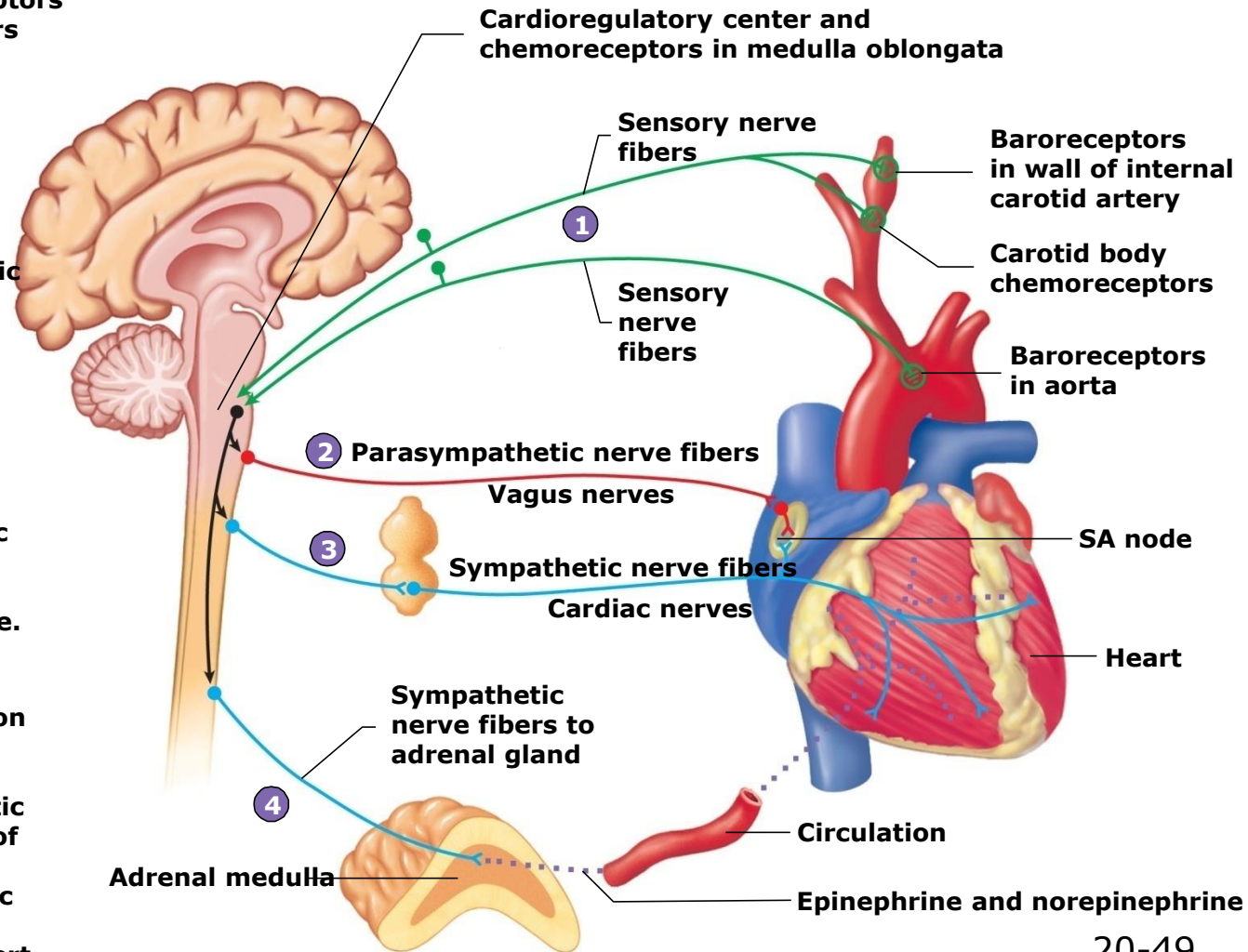
Heart and Homeostasis

- Effect of blood pressure
 - Baroreceptors monitor blood pressure; in walls of internal carotids and aorta. This sensory information goes to centers in the medulla oblongata
- Effect of pH, carbon dioxide, oxygen
 - Receptors that measure pH and carbon dioxide levels found in hypothalamus
 - Chemoreceptors monitoring oxygen levels found in aorta and internal carotids. Prolonged lowered oxygen levels causes increased heart rate, which increases blood pressure and can thus deliver more oxygen to the tissues.
- Effect of extracellular ion concentration
 - Increase or decrease in extracellular K^+ decreases heart rate
- Effect of body temperature
 - Heart rate increases when body temperature increases, heart rate decreases when body temperature decreases

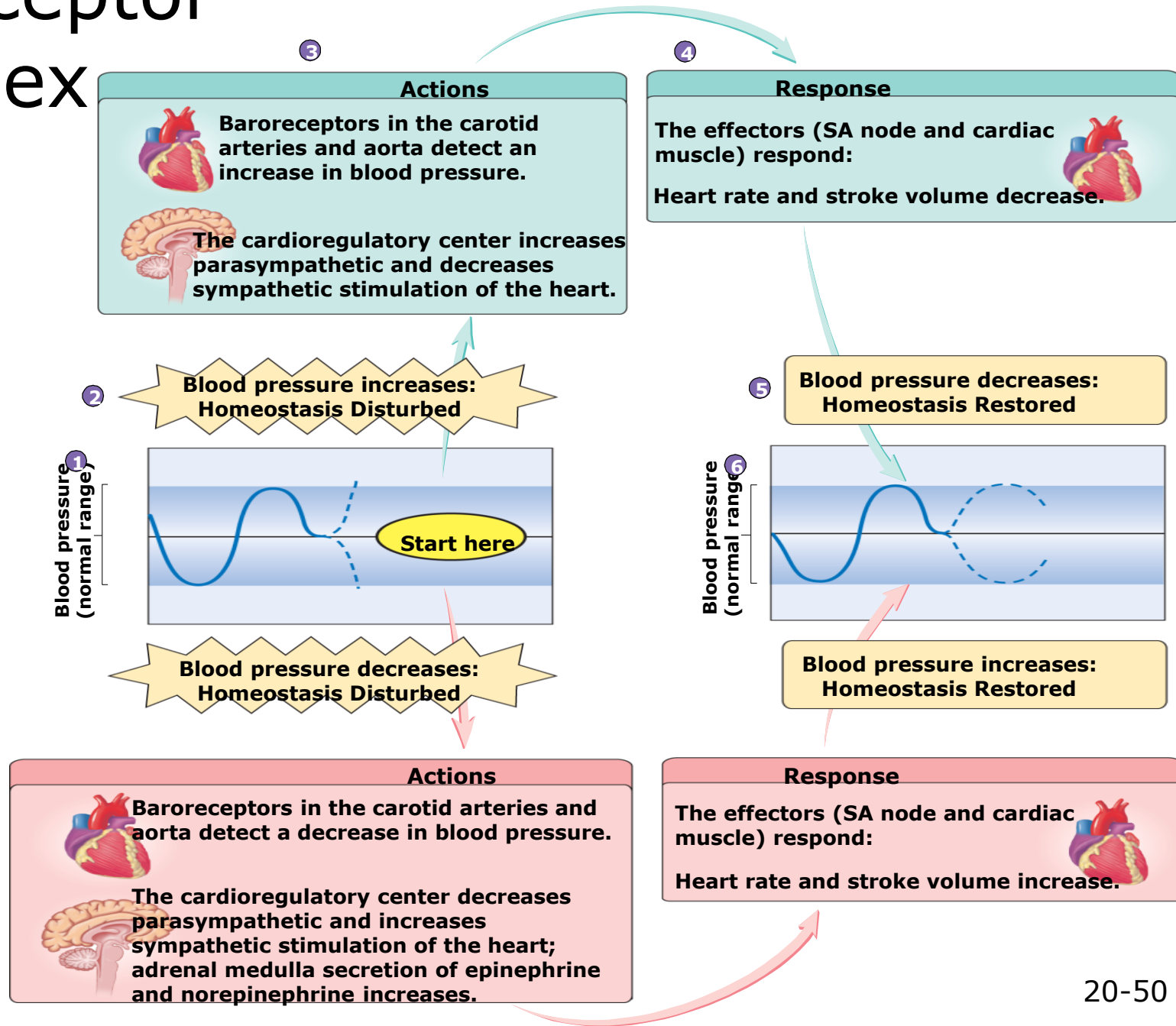
Baroreceptor and Chemoreceptor Reflexes

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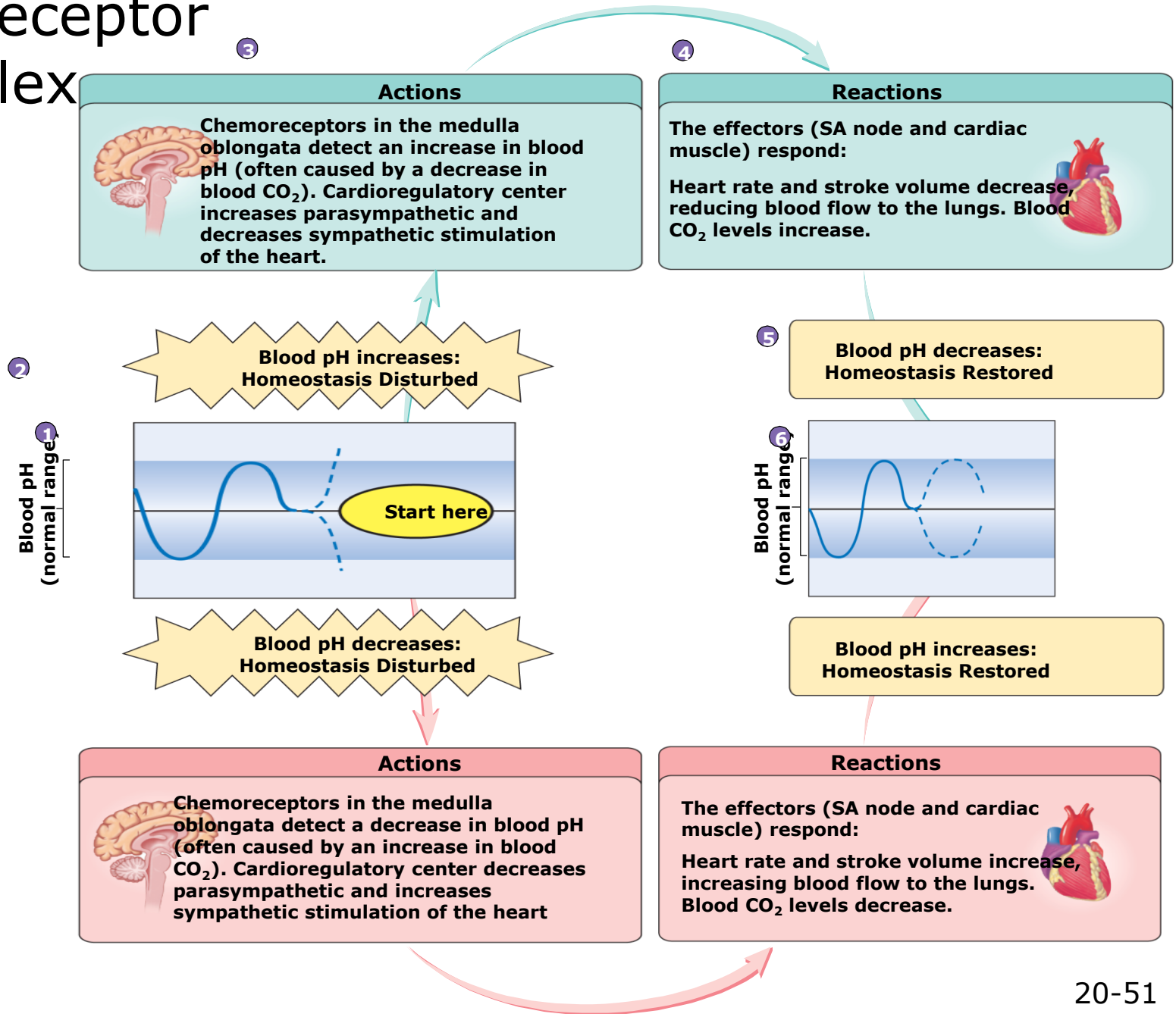
- 1** Sensory neurons (green) carry action potentials from baroreceptors and carotid body chemoreceptors to the cardioresgulatory center. Chemoreceptors in the medulla oblongata also influence the cardioresgulatory center.
- 2** The cardioresgulatory center controls the frequency of action potentials in the parasympathetic neurons (red) extending to the heart through the vagus nerves. The parasympathetic neurons decrease the heart rate.
- 3** The cardioresgulatory center controls the frequency of action potentials in the sympathetic neurons (blue). The sympathetic neurons extend through the cardiac nerves and increase the heart rate and the stroke volume.
- 4** The cardioresgulatory center influences the frequency of action potentials in the sympathetic neurons (blue) extending to the adrenal medulla. The sympathetic neurons increase the secretion of epinephrine and some norepinephrine into the systemic circulation. Epinephrine and Norepinephrine increase the heart Rate and stroke volume.



Baroreceptor Reflex



Chemoreceptor Reflex



Diseases and Disorders

Heart

Condition	Description
Inflammation of Heart Tissue	
Endocarditis	Inflammation of the endocardium; affects the valves more severely than other areas of the endocardium; may lead to scarring, causing stenosed or incompetent valves
Pericarditis	Inflammation of the pericardium; see Clinical Impact, "Pericarditis and Cardiac Tamponade"
Cardiomyopathy	Disease of the myocardium of unknown cause or occurring secondarily to other disease; results in weakened cardiac muscle, causing all chambers of the heart to enlarge; may eventually lead to congestive heart failure
Rheumatic heart disease	Results from a streptococcal infection in young people; toxin produced by the bacteria can cause rheumatic fever several weeks after the infection that can result in rheumatic endocarditis
Reduced Blood Flow to Cardiac Muscle	
Coronary heart disease	Reduces the amount of blood the coronary arteries can deliver to the myocardium
Coronary thrombosis	Formation of blood clot in a coronary artery
Myocardial infarction	Damaged cardiac muscle tissue resulting from lack of blood flow to the myocardium; often referred to as a heart attack; see Clinical Impact, "Angina, Infarctions, and the Treatment of Blocked coronary Arteries"
Congenital Heart Diseases (occur at birth)	
Septal defect	Hole in the septum between the left and right sides of the heart, allowing blood to flow from one side of the heart to the other and greatly reducing the heart's pumping effectiveness
Patent ductus arteriosus	Ductus arteriosus fails to close after birth, allowing blood to flow from the aorta to the pulmonary trunk under a higher pressure, which damages the lungs; also, the left ventricle must work harder to maintain adequate systemic pressure
Stenosis of the heart valve	Narrowed opening through one or more of the heart valves; aortic or pulmonary semilunar stenosis increases the heart's workload; bicuspid valve stenosis causes blood to back up in the left atria and lungs, resulting in edema of the lungs; tricuspid valve stenosis results in similar blood flow problems and edema in the peripheral tissues
Incompetent heart valve	Heart valves do not close correctly, and blood flows through in the reverse direction; see Clinical Impact, "Abnormal Heart Sounds"
Cyanosis (<i>sī-ā-no'sis</i> ; <i>cyan</i> , blue + <i>osis</i> , condition of)	Symptom of inadequate heart function in babies with congenital heart disease; the infant's skin appears blue because of low oxygen levels in the blood in peripheral blood vessels
Heart Failure	Progressive weakening of the heart muscle, reducing the heart's pumping action; hypertension leading to heart failure due to increased afterload; advanced age, malnutrition, chronic infections, toxins, severe anemias, hyperthyroidism, and hereditary factors can lead to heart failure