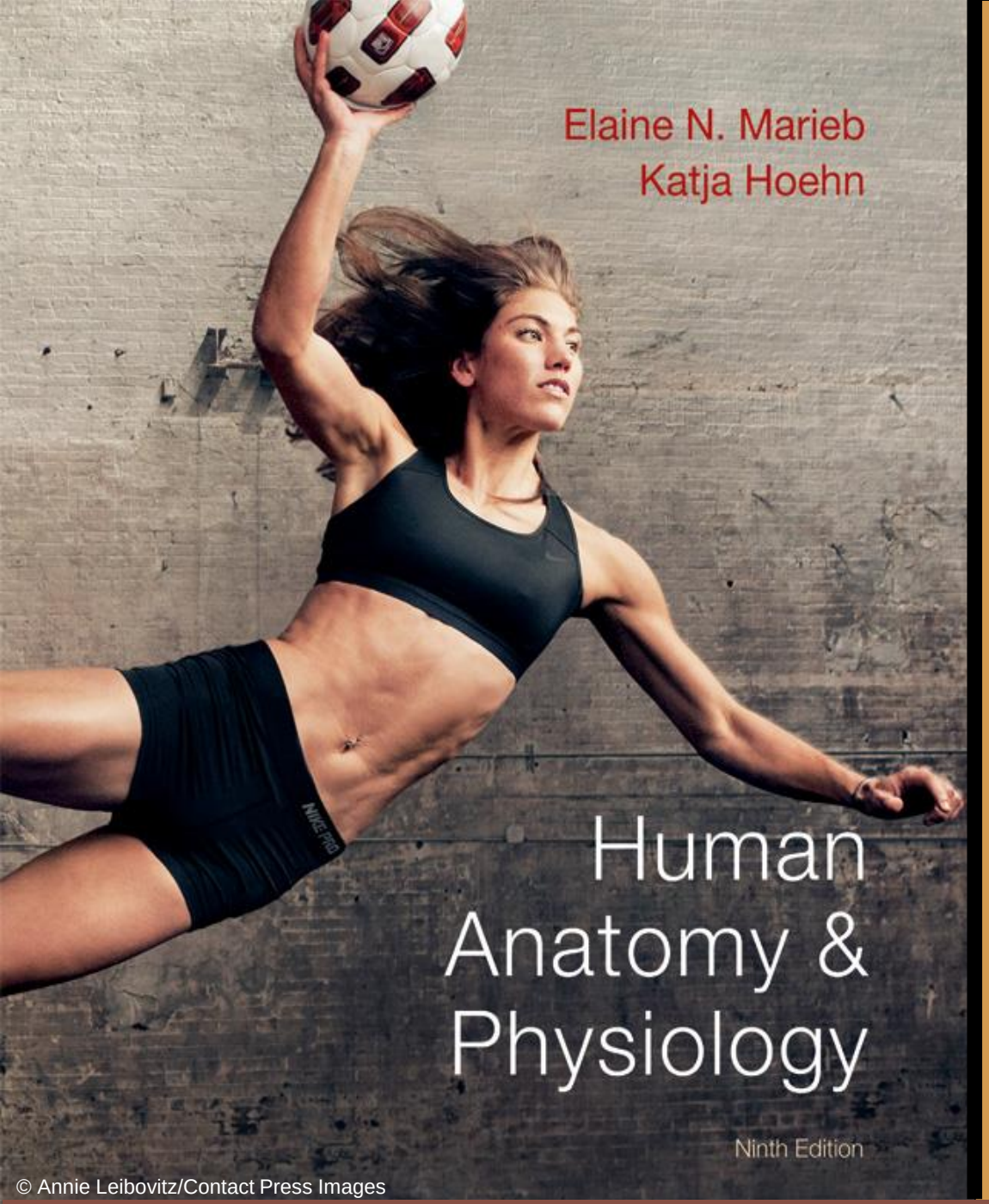

PowerPoint® Lecture Slides

prepared by
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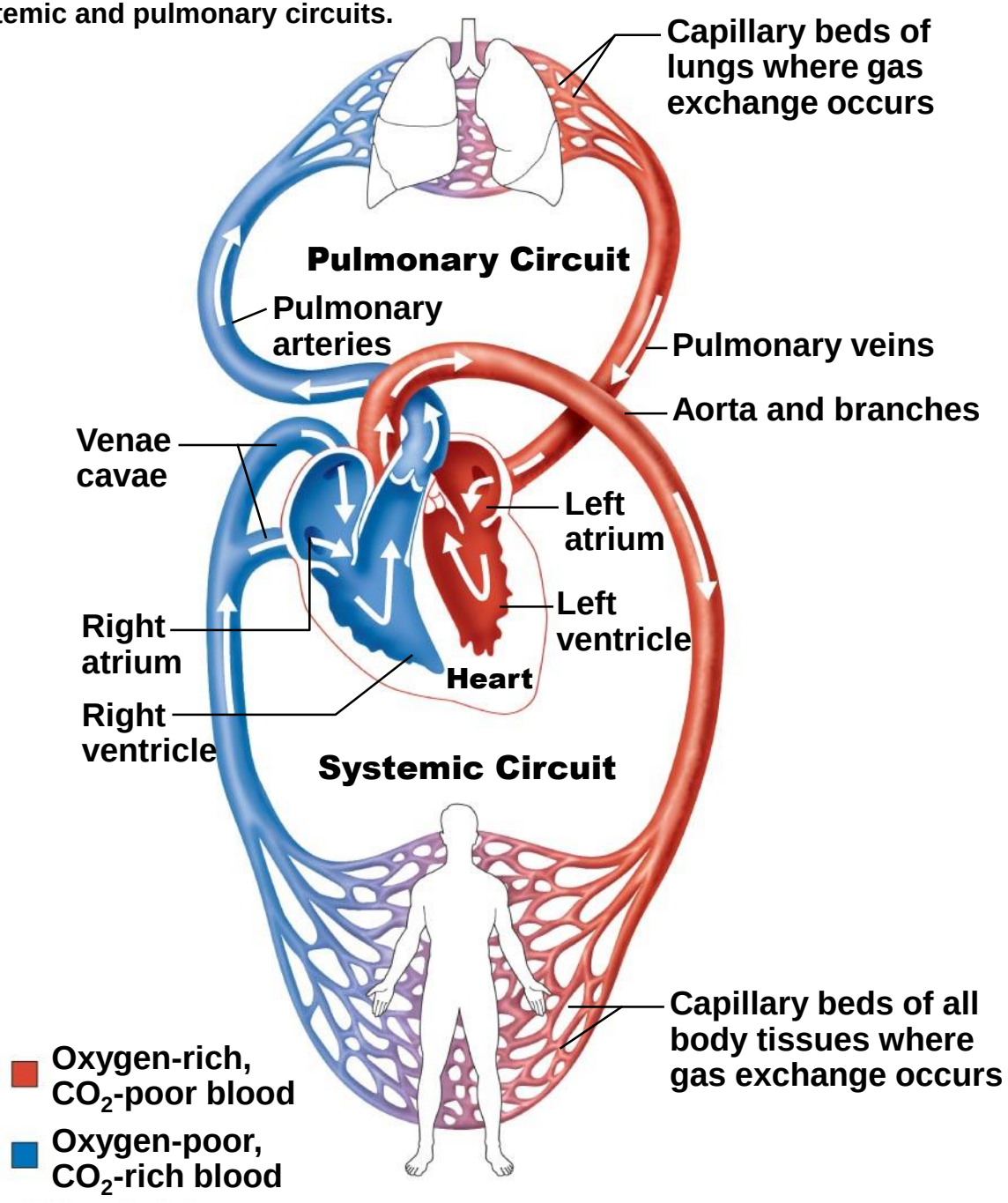
CHAPTER 18

The Cardiovascular System: The Heart: Part A

The Pulmonary and Systemic Circuits

- Heart is transport system; two side-by-side pumps
  - *Right side* receives oxygen-poor blood from tissues
    - Pumps to lungs to get rid of CO_2, pick up O_2, via **pulmonary circuit**
  - *Left side* receives oxygenated blood from lungs
    - Pumps to body tissues via **systemic circuit**

Figure 18.1 The systemic and pulmonary circuits.



Heart Anatomy

- Approximately size of fist
- Location:
  - In **mediastinum** between second rib and fifth intercostal space
  - On superior surface of diaphragm
  - Two-thirds of heart to left of midsternal line
  - Anterior to vertebral column, posterior to sternum

Animation: Rotatable heart

Heart Anatomy

- **Base** (posterior surface) leans toward right shoulder
- **Apex** points toward left hip
- **Apical impulse** palpated between fifth and sixth ribs, just below left nipple

Figure 18.2a Location of the heart in the mediastinum.

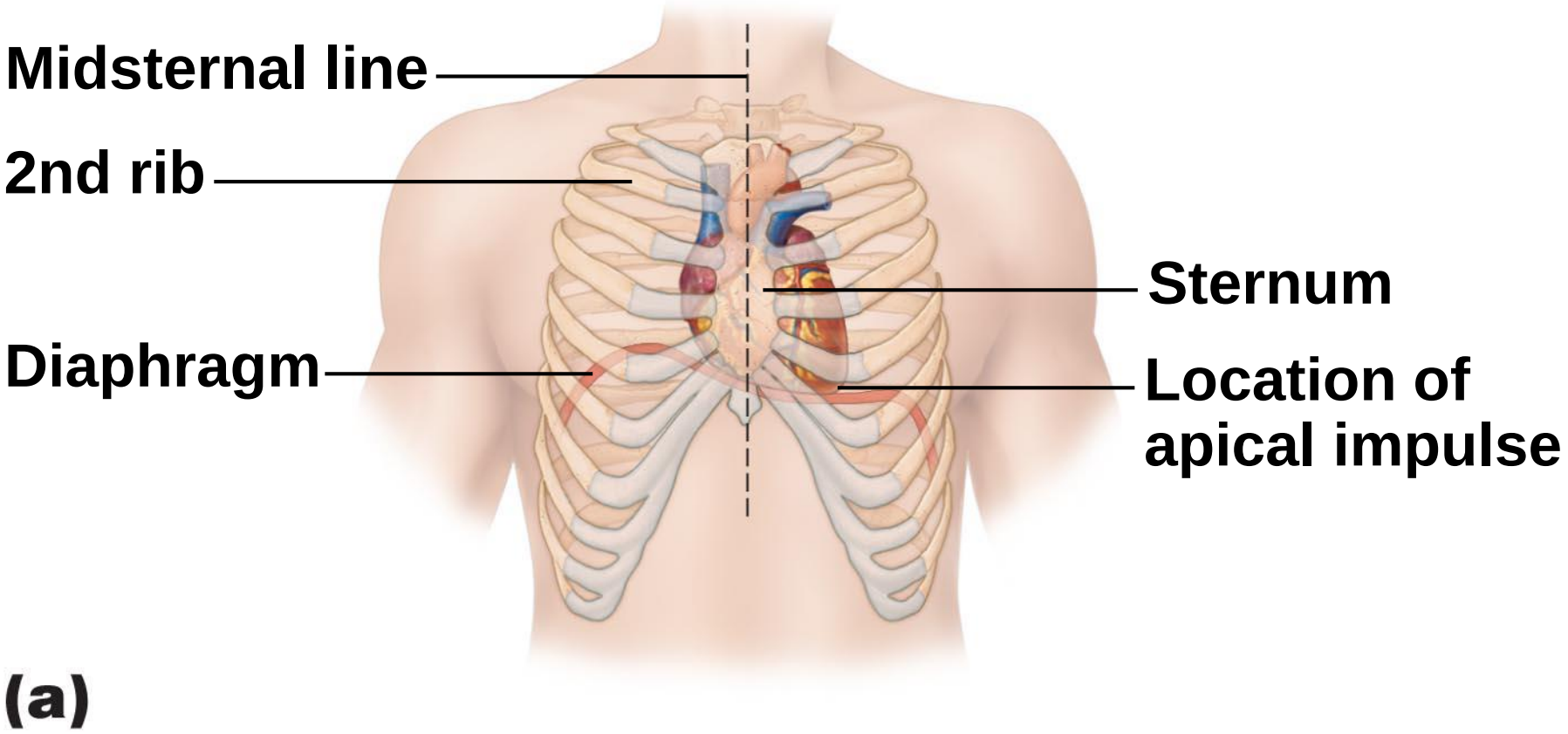
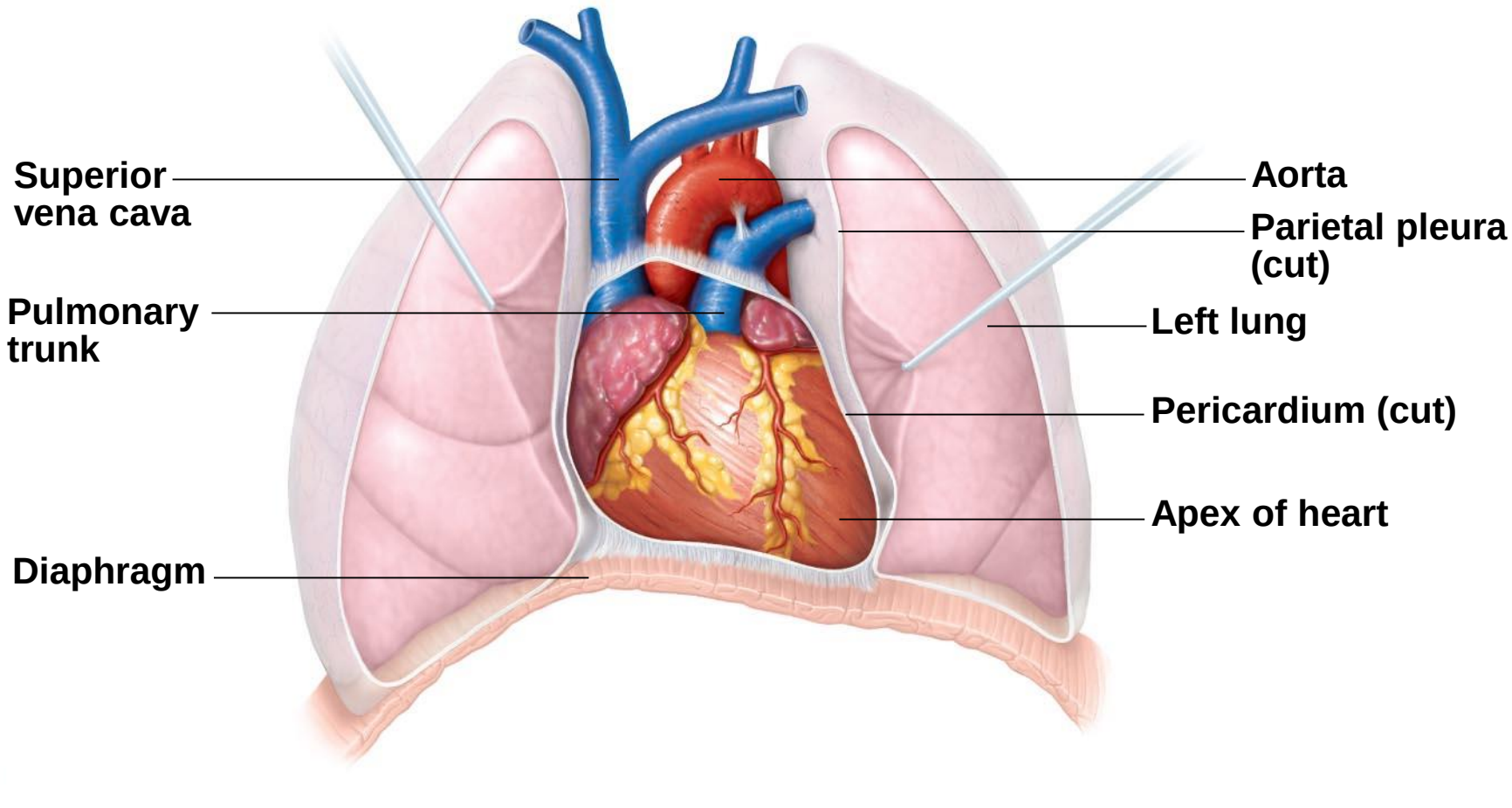


Figure 18.2c Location of the heart in the mediastinum.



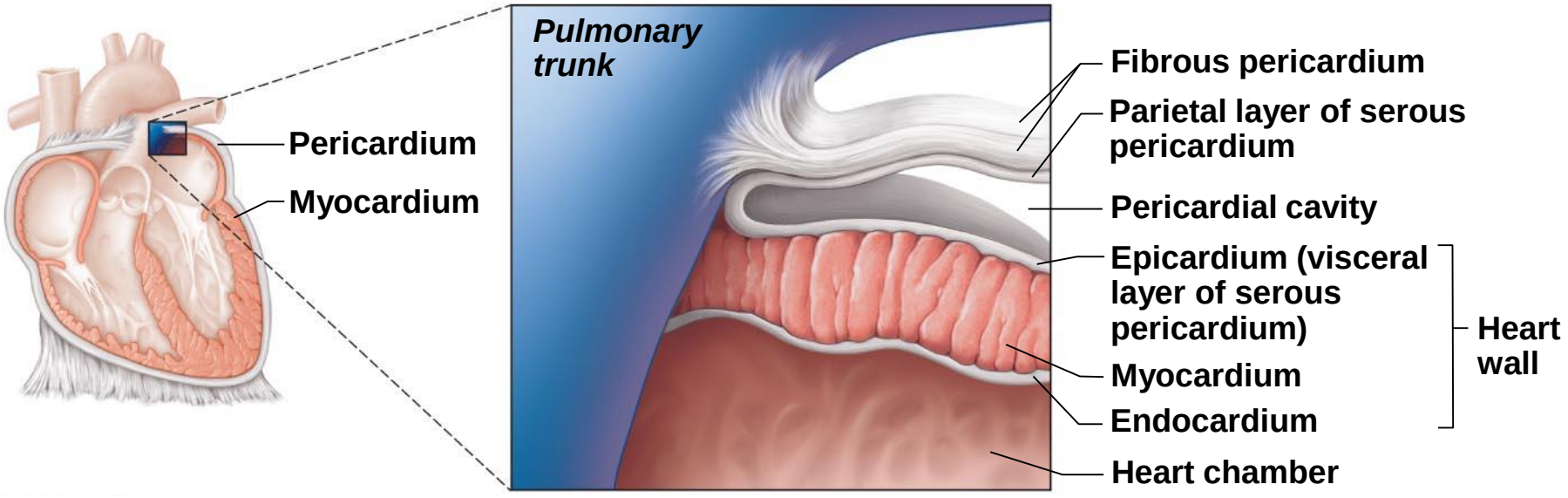
Coverings of the Heart: Pericardium

- Double-walled sac
- Superficial **fibrous pericardium**
  - Protects, anchors to surrounding structures, and prevents overfilling

Pericardium

- Deep two-layered **serous pericardium**
  - **Parietal layer** lines internal surface of fibrous pericardium
  - **Visceral layer** (**epicardium**) on external surface of heart
  - Two layers separated by fluid-filled **pericardial cavity** (decreases friction)

Figure 18.3 The pericardial layers and layers of the heart wall.



Homeostatic Imbalance

- *Pericarditis*
  - Inflammation of pericardium
  - Roughens membrane surfaces → **pericardial friction rub** (creaking sound) heard with stethoscope
  - *Cardiac tamponade*
    - Excess fluid sometimes compresses heart → limited pumping ability

Layers of the Heart Wall

- Three layers of heart wall:
  - **Epicardium**
  - **Myocardium**
  - **Endocardium**

- Epicardium
  - Visceral layer of serous pericardium

Layers of the Heart Wall

- Myocardium
  - Spiral bundles of contractile cardiac muscle cells
  - Cardiac skeleton: crisscrossing, interlacing layer of connective tissue
    - Anchors cardiac muscle fibers
    - Supports great vessels and valves
    - Limits spread of action potentials to specific paths

Layers of the Heart Wall

- Endocardium continuous with endothelial lining of blood vessels
  - Lines heart chambers; covers cardiac skeleton of valves

Figure 18.3 The pericardial layers and layers of the heart wall.

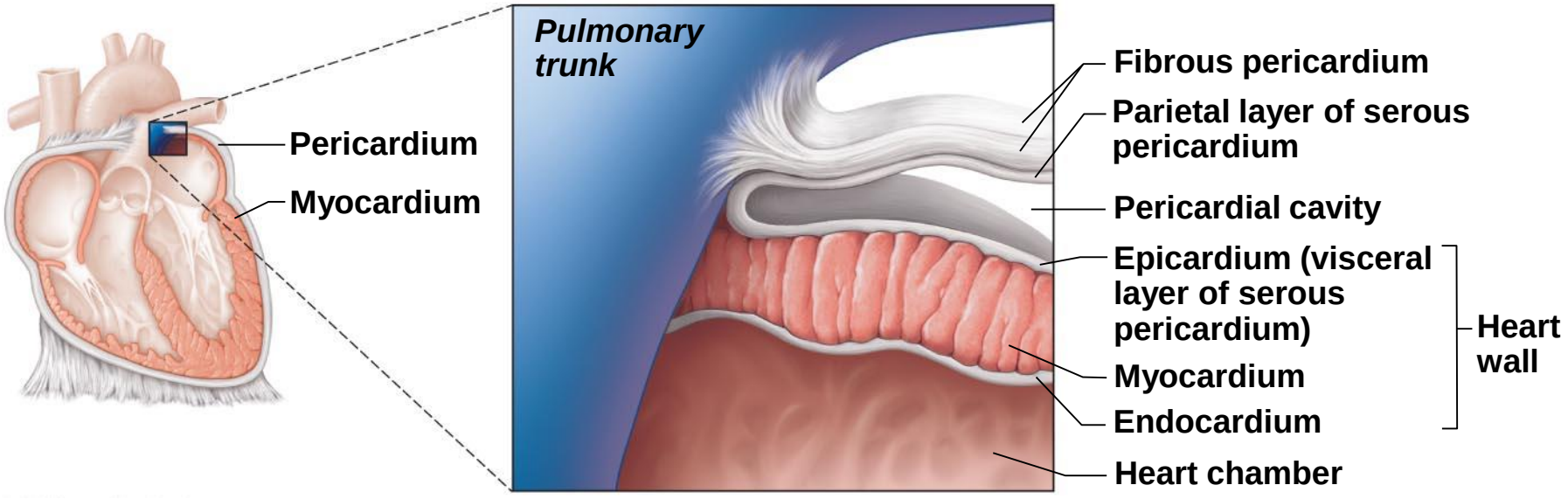
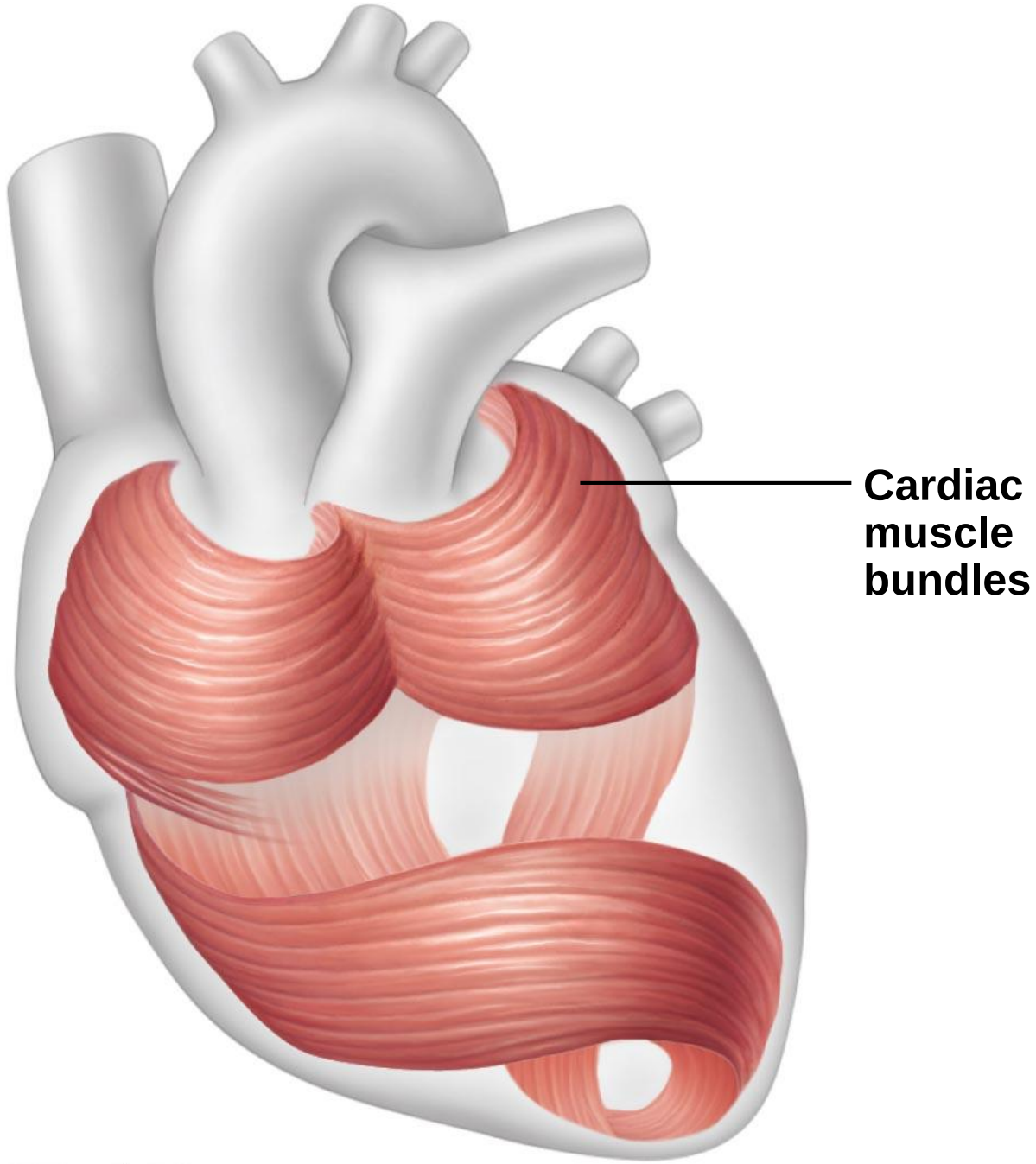


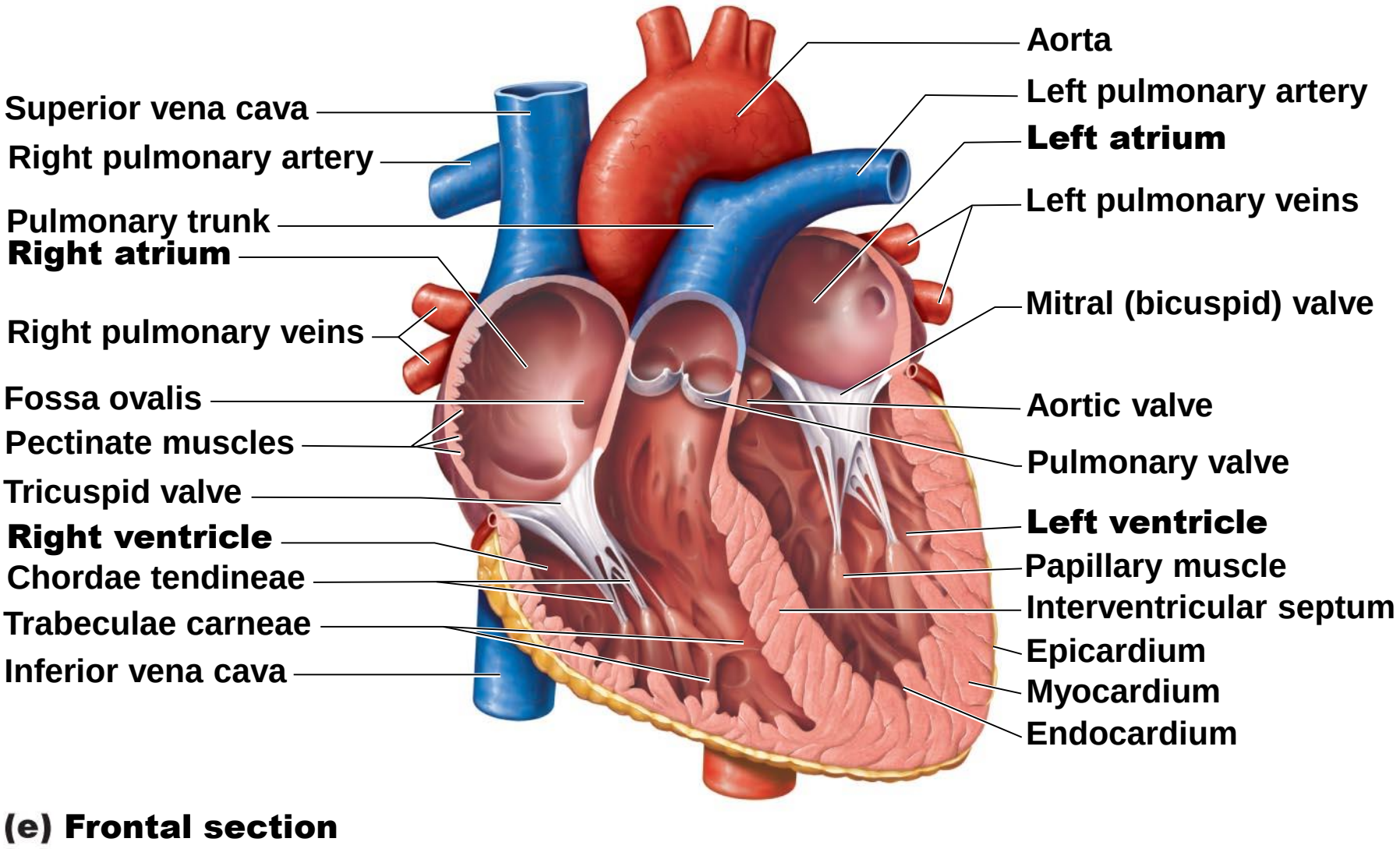
Figure 18.4 The circular and spiral arrangement of cardiac muscle bundles in the myocardium of the heart.



Chambers

- Four chambers:
  - Two superior **atria**
  - Two inferior **ventricles**
- **Interatrial septum** – separates atria
  - **Fossa ovalis** – remnant of foramen ovale of fetal heart
- **Interventricular septum** – separates ventricles

Figure 18.5e Gross anatomy of the heart.



Atria: The Receiving Chambers

- Small, thin-walled
- Contribute little to propulsion of blood
- Three veins empty into right atrium:
  - **Superior vena cava, inferior vena cava, coronary sinus**
- Four pulmonary veins empty into left atrium

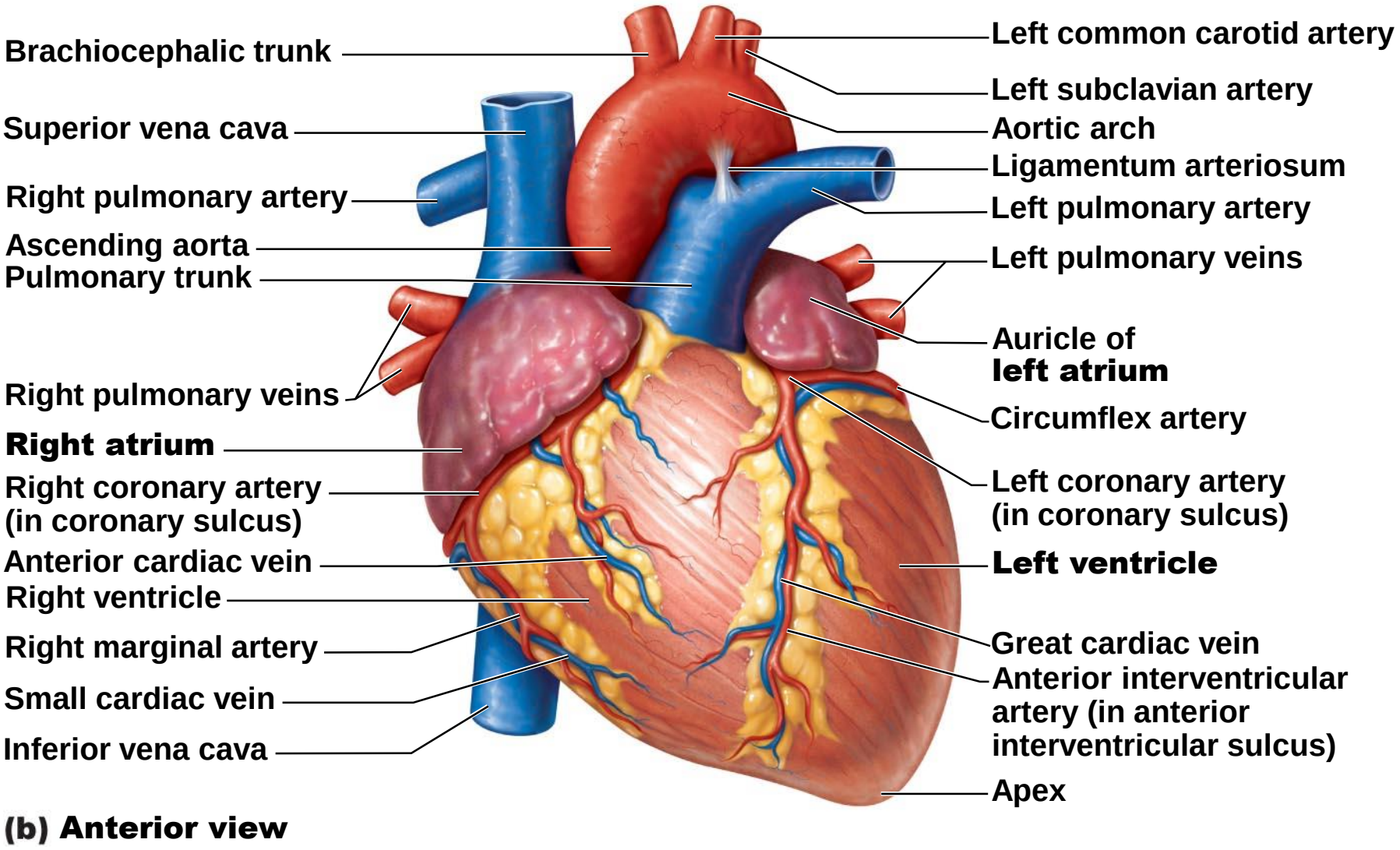
Ventricles: The Discharging Chambers

- Most of the volume of heart
- **Right ventricle** - most of anterior surface
- **Left ventricle** – posteroinferior surface
- **Trabeculae carneae** – irregular ridges of muscle on walls
- **Papillary muscles** – anchor **chordae tendineae**

Ventricles: The Discharging Chambers

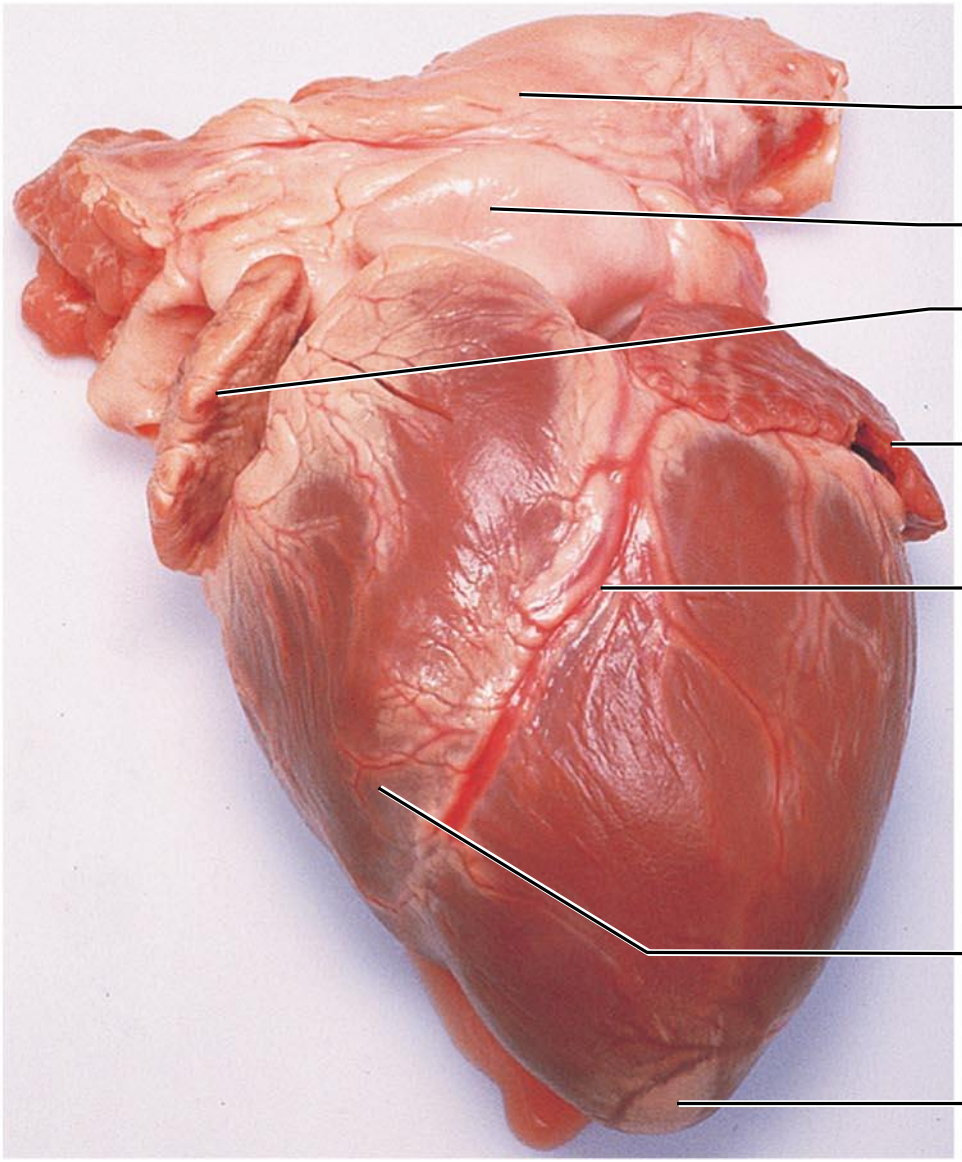
- Thicker walls than atria
- Actual pumps of heart
- Right ventricle
  - Pumps blood into **pulmonary trunk**
- Left ventricle
  - Pumps blood into **aorta** (largest artery in body)

Figure 18.5b Gross anatomy of the heart.



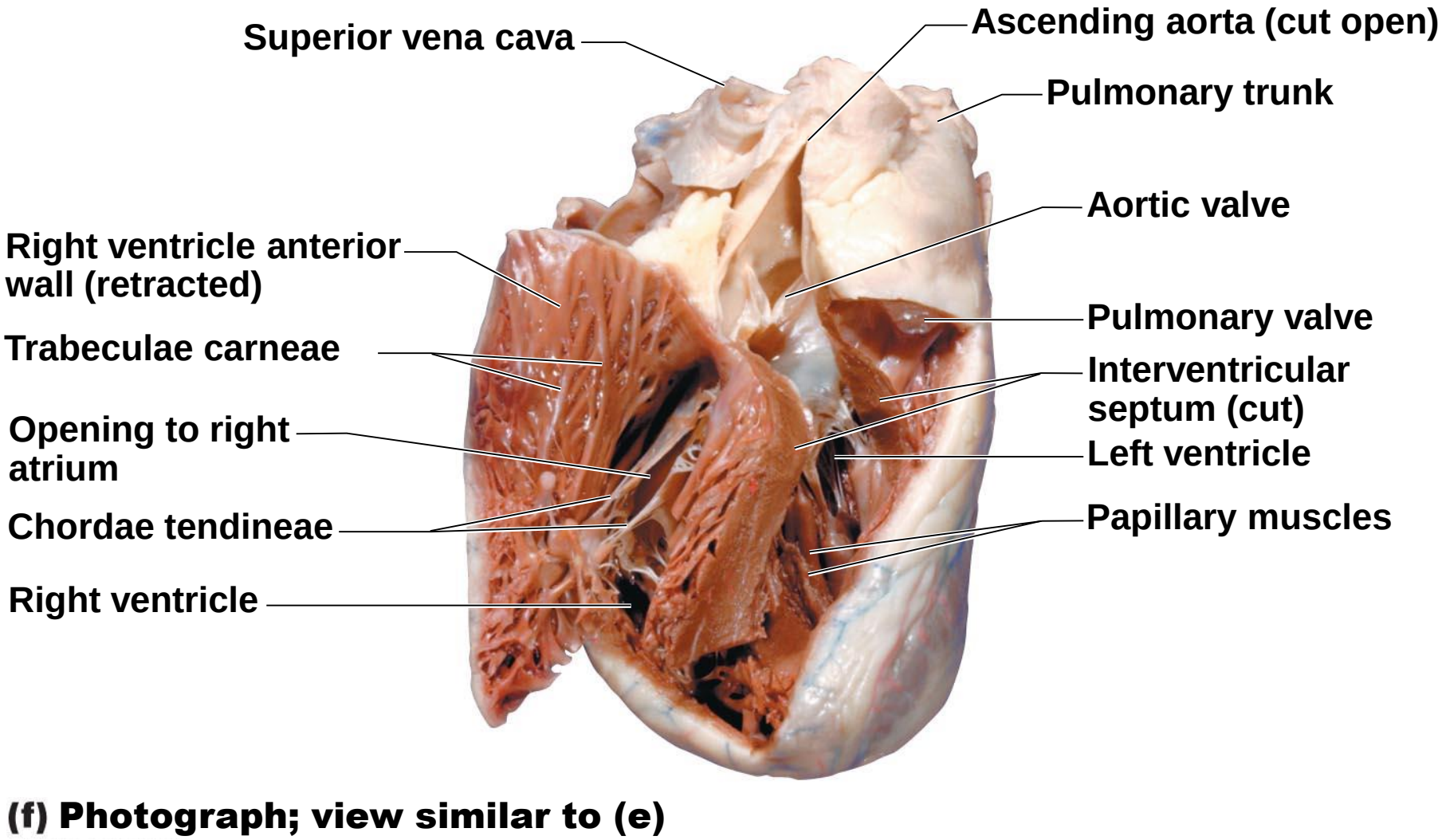
(b) Anterior view

Figure 18.5a Gross anatomy of the heart.



(a) Anterior aspect (pericardium removed)

Figure 18.5f Gross anatomy of the heart.

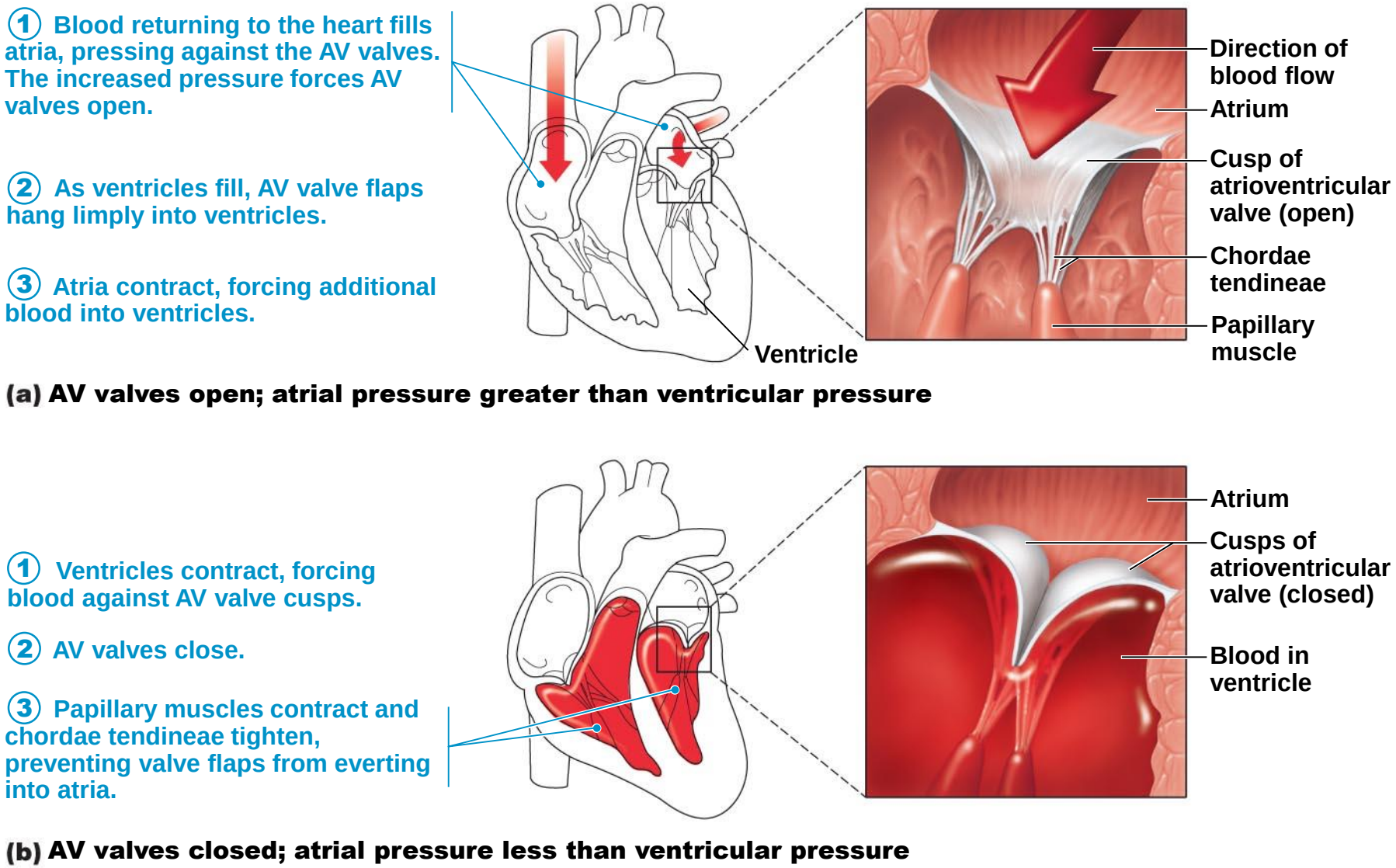


(f) Photograph; view similar to (e)

Heart Valves

- Ensure unidirectional blood flow through heart
- Open and close in response to pressure changes
- Two **atrioventricular (AV) valves**
  - Prevent backflow into atria when ventricles contract
  - **Tricuspid valve** (right AV valve)
  - **Mitral valve** (left AV valve, bicuspid valve)
  - **Chordae tendineae** anchor cusps to **papillary muscles**
    - Hold valve flaps in closed position

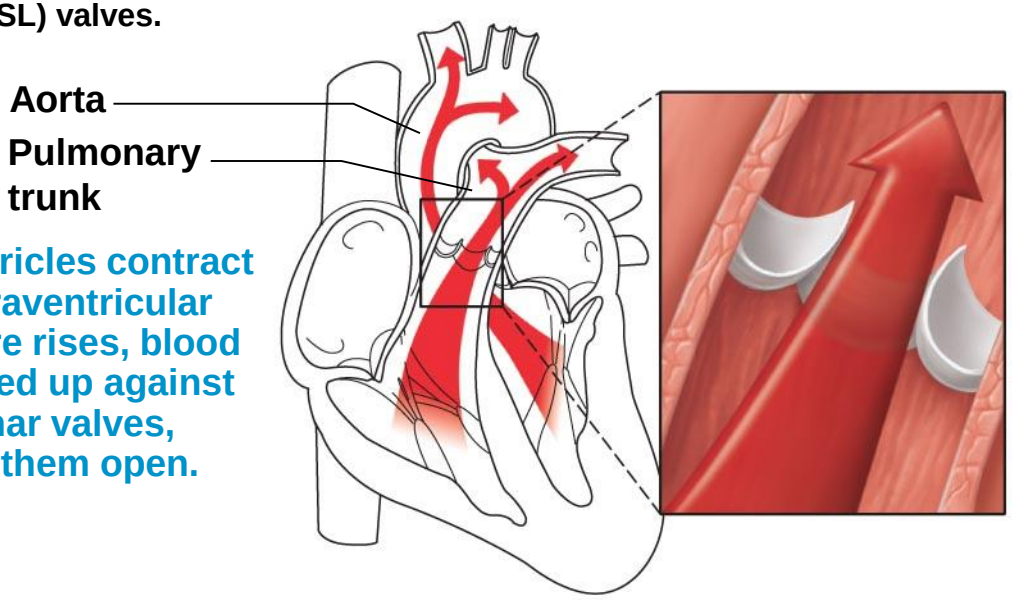
Figure 18.7 The atrioventricular (AV) valves.



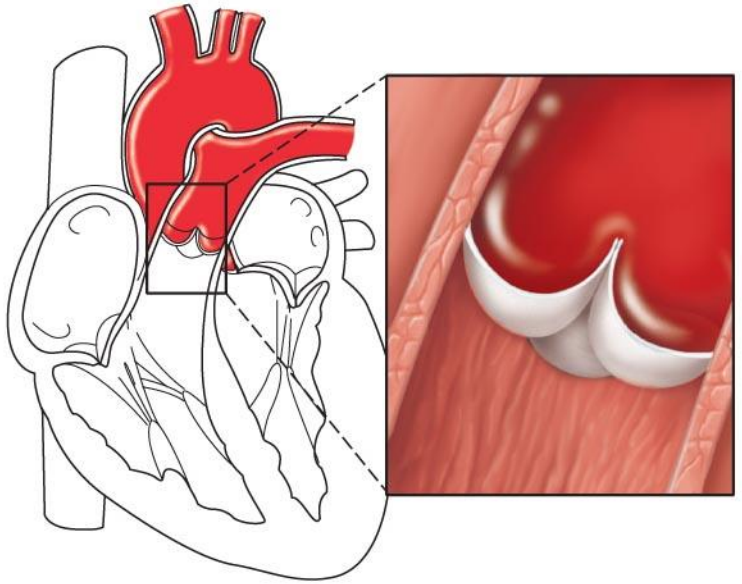
Heart Valves

- Two **semilunar (SL) valves**
  - Prevent backflow into ventricles when ventricles relax
  - Open and close in response to pressure changes
  - **Aortic semilunar valve**
  - **Pulmonary semilunar valve**

Figure 18.8 The semilunar (SL) valves.



(a) Semilunar valves open



(b) Semilunar valves closed

Figure 18.6a Heart valves.

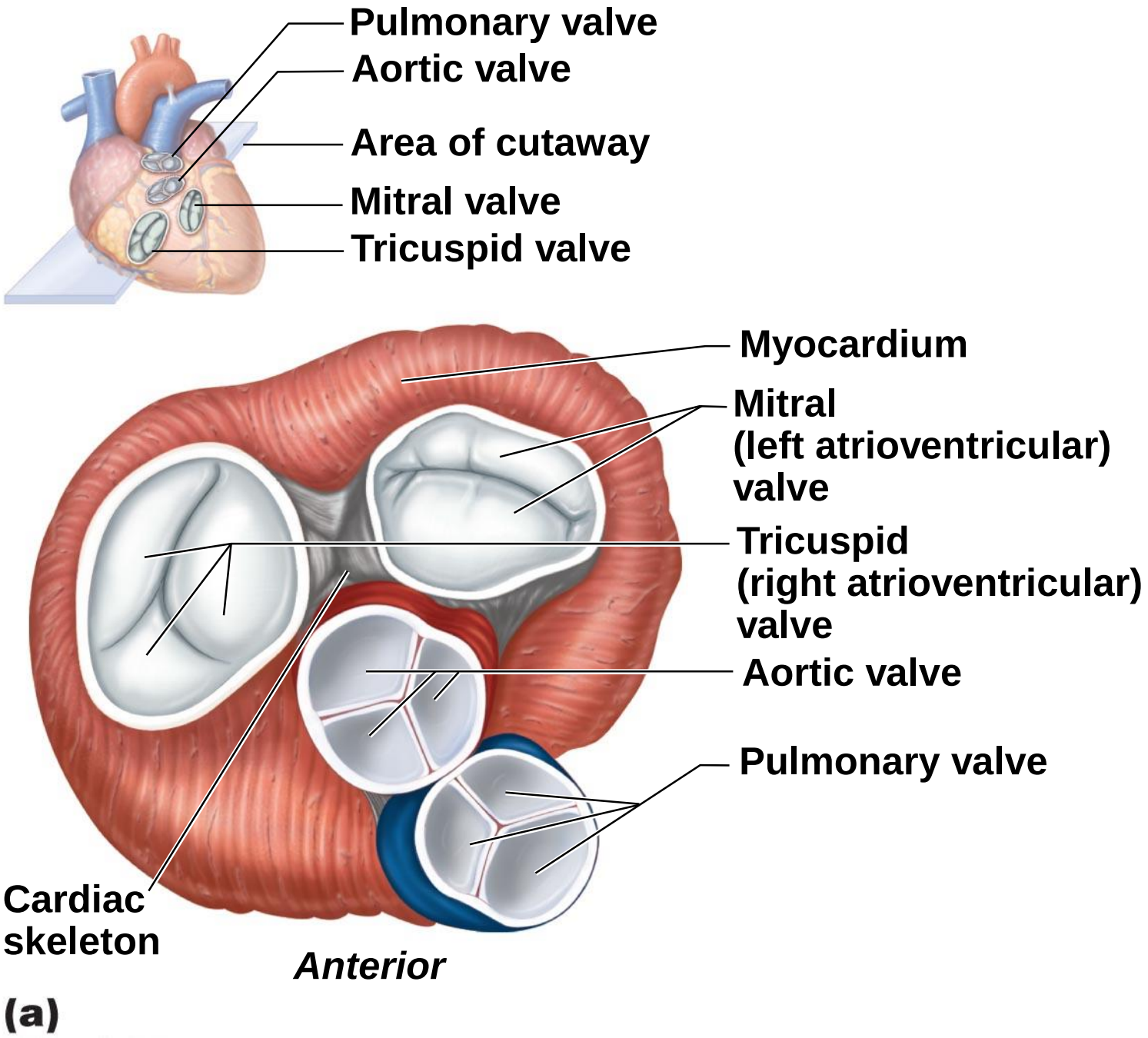
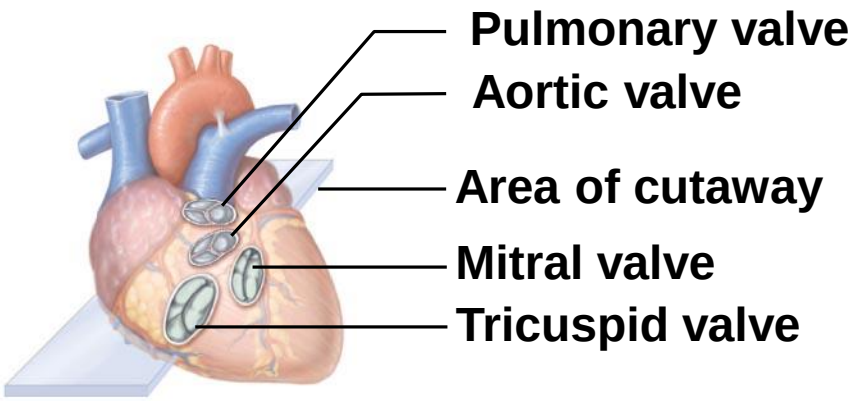
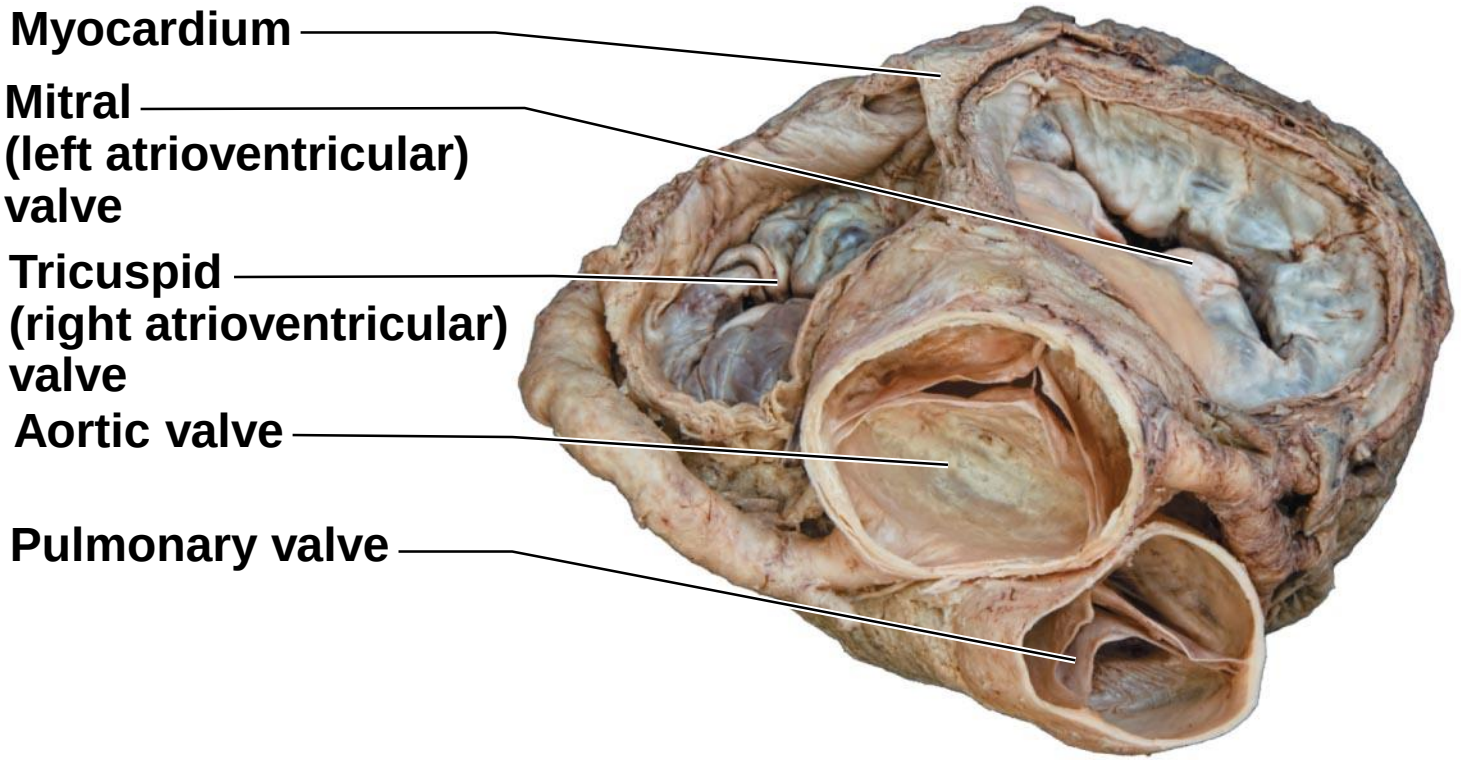


Figure 18.6b Heart valves.





(b)

Figure 18.6c Heart valves.

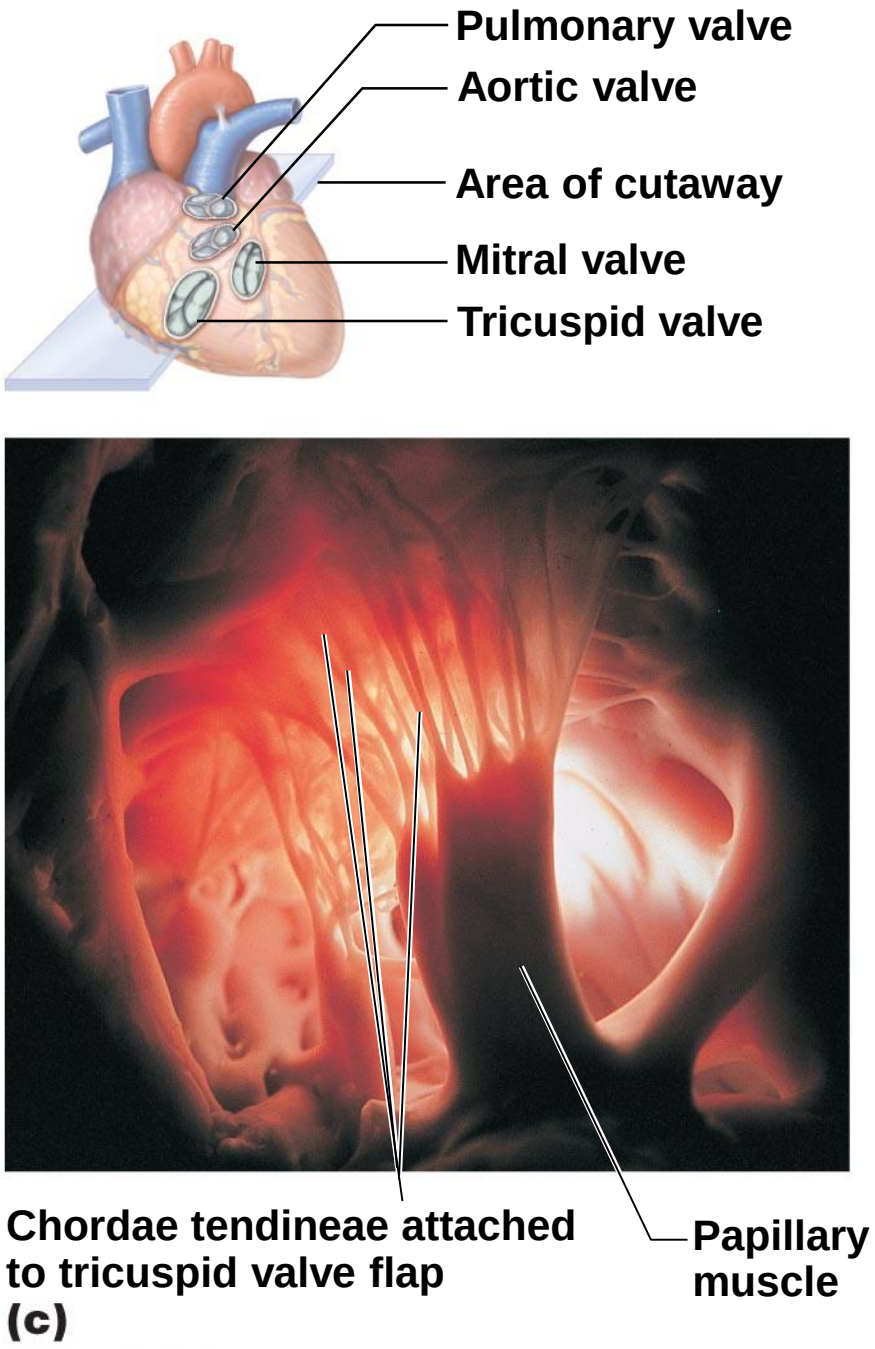
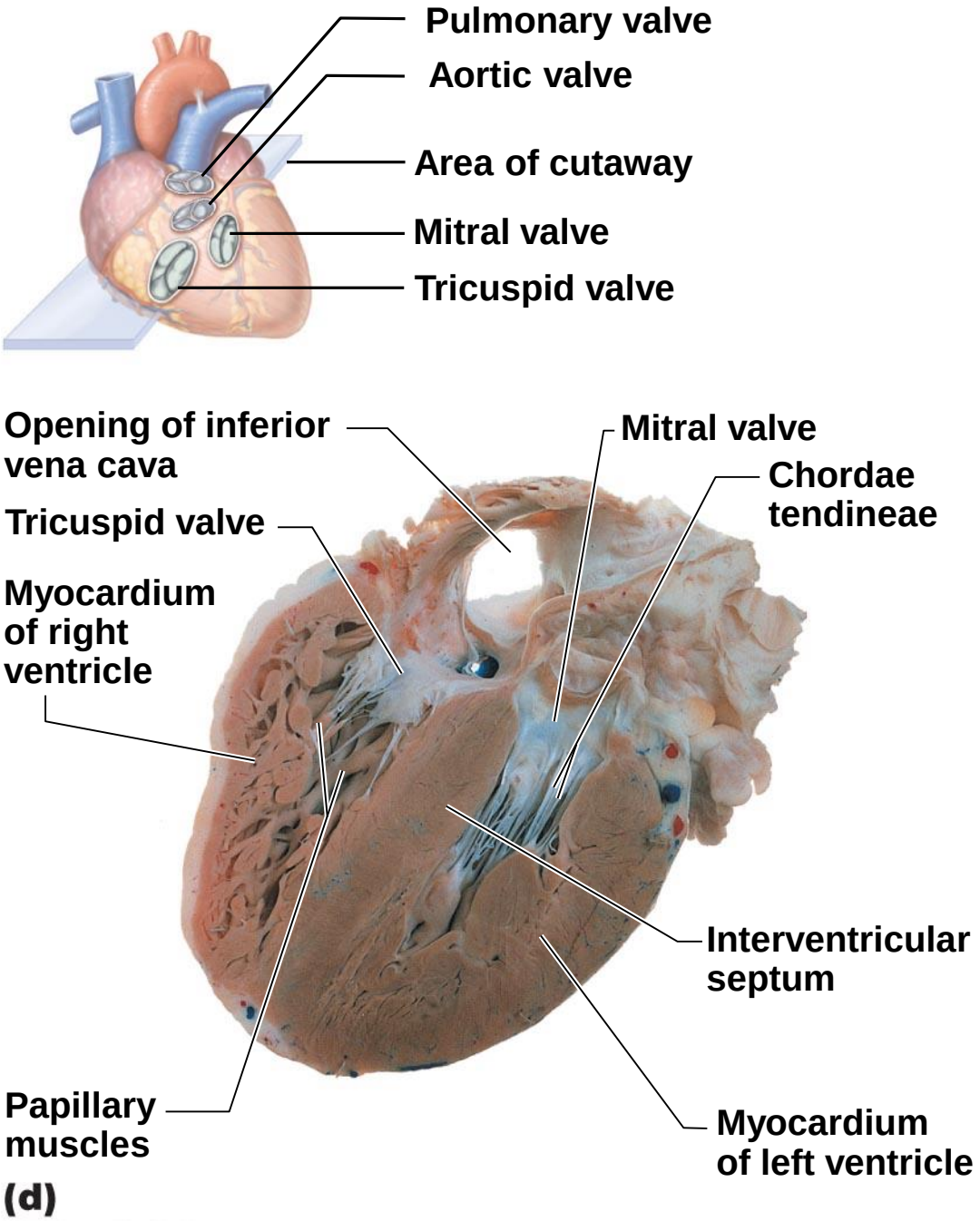


Figure 18.6d Heart valves.



Homeostatic Imbalance

- Two conditions severely weaken heart:
  - *Incompetent valve*
    - Blood backflows so heart repumps same blood over and over
  - *Valvular stenosis*
    - Stiff flaps – constrict opening → heart must exert more force to pump blood
- Valve replaced with mechanical, animal, or cadaver valve

Pathway of Blood Through the Heart

- **Pulmonary circuit**
  - Right atrium → tricuspid valve → right ventricle
  - Right ventricle → pulmonary semilunar valve → pulmonary trunk → pulmonary arteries → lungs
  - Lungs → pulmonary veins → left atrium

Pathway of Blood Through the Heart

- **Systemic circuit**
  - Left atrium → mitral valve → left ventricle
  - Left ventricle → aortic semilunar valve → aorta
  - Aorta → systemic circulation

PLAY **Animation: Rotatable heart (sectioned)**

Figure 18.9 The heart is a double pump, each side supplying its own circuit.

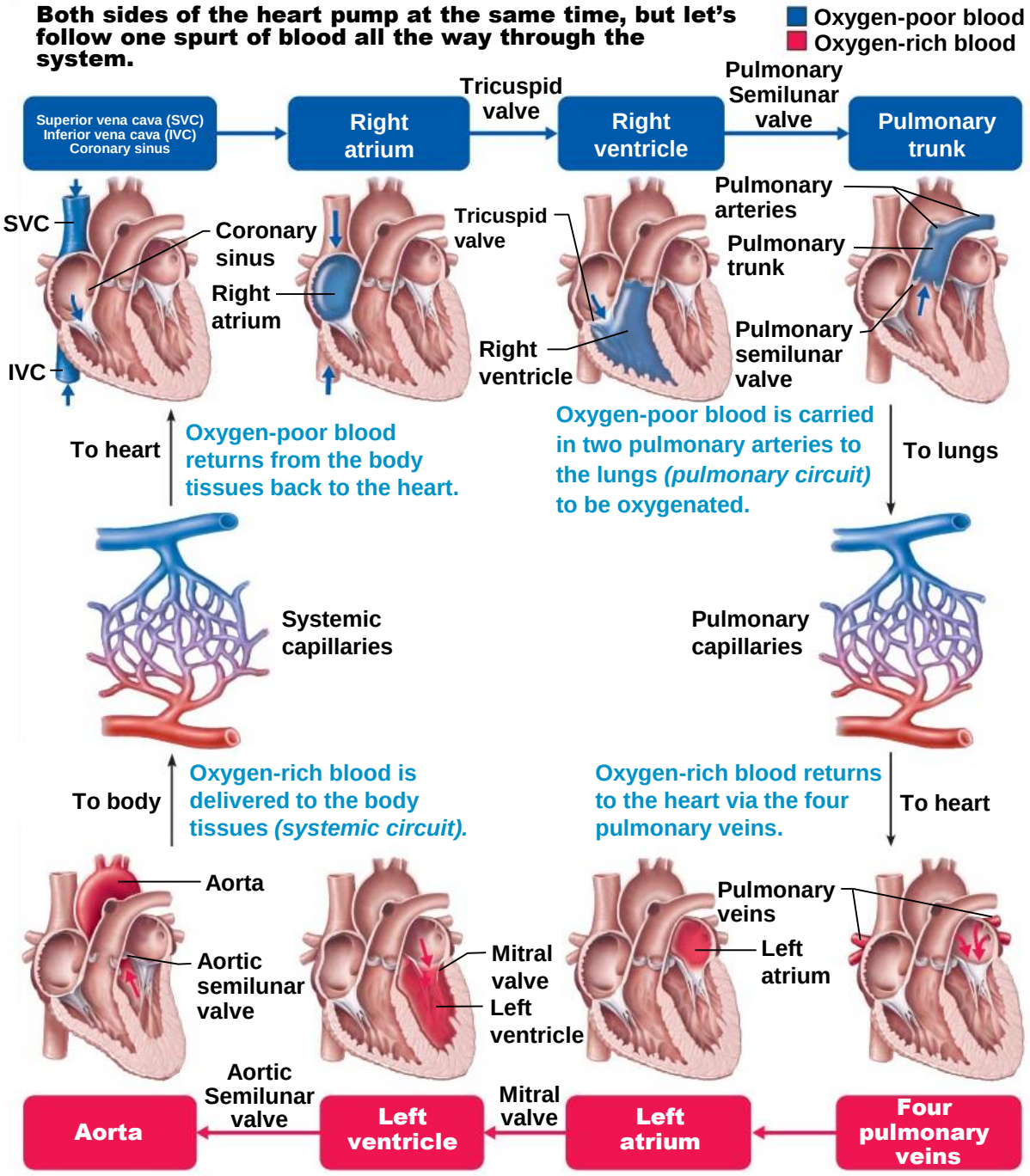


Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Superior vena cava (SVC)
Inferior vena cava (IVC)
Coronary sinus

Oxygen-poor blood

Oxygen-rich blood

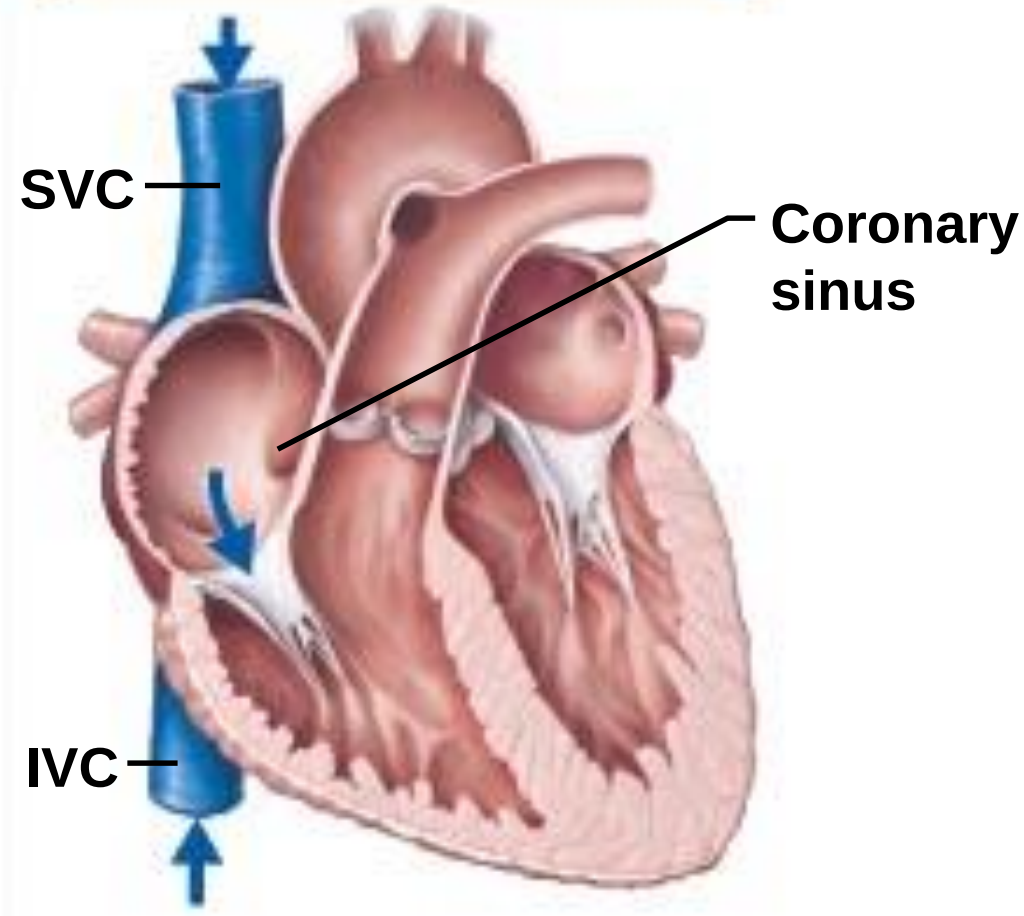
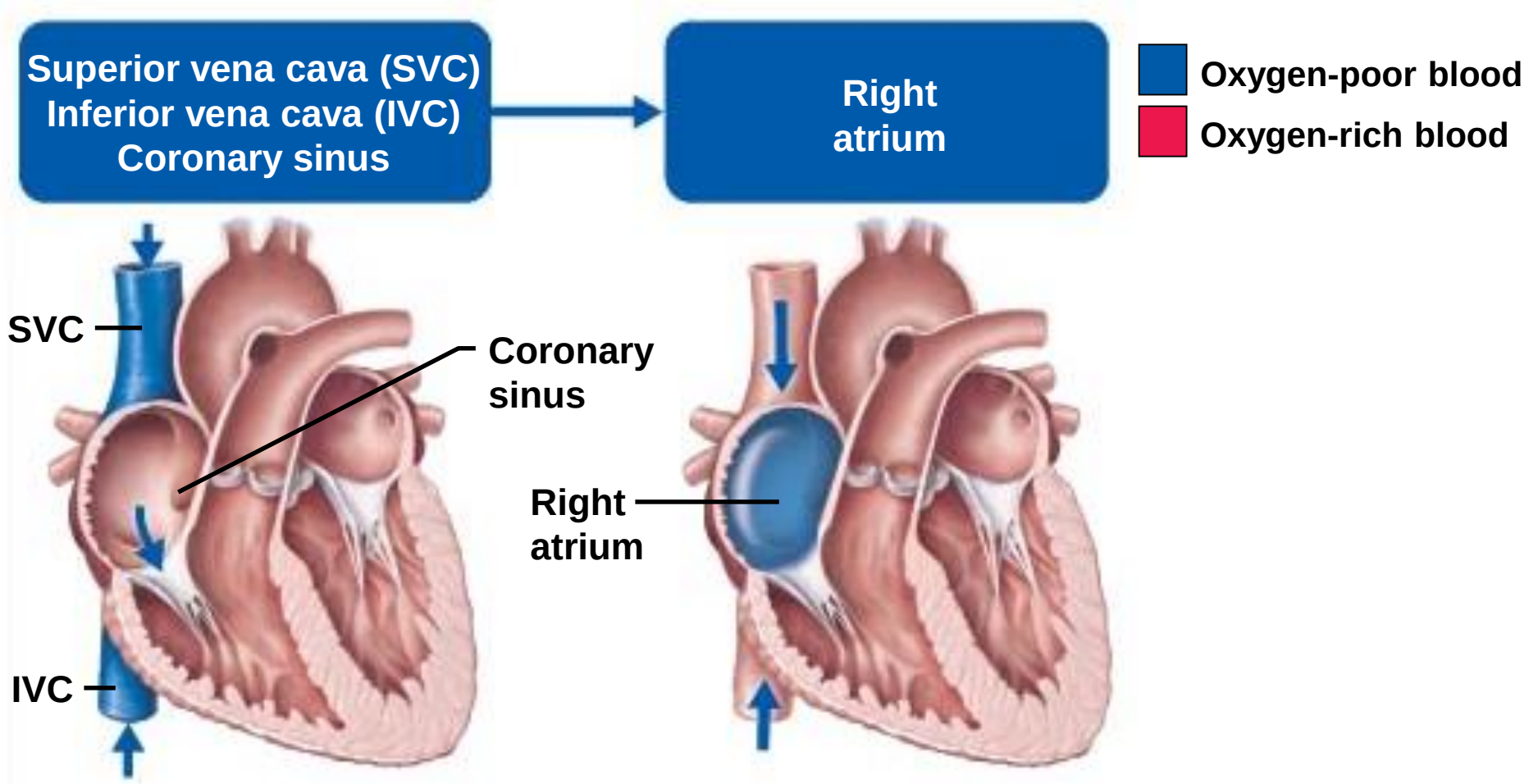


Figure 18.9 The heart is a double pump, each side supplying its own circuit.



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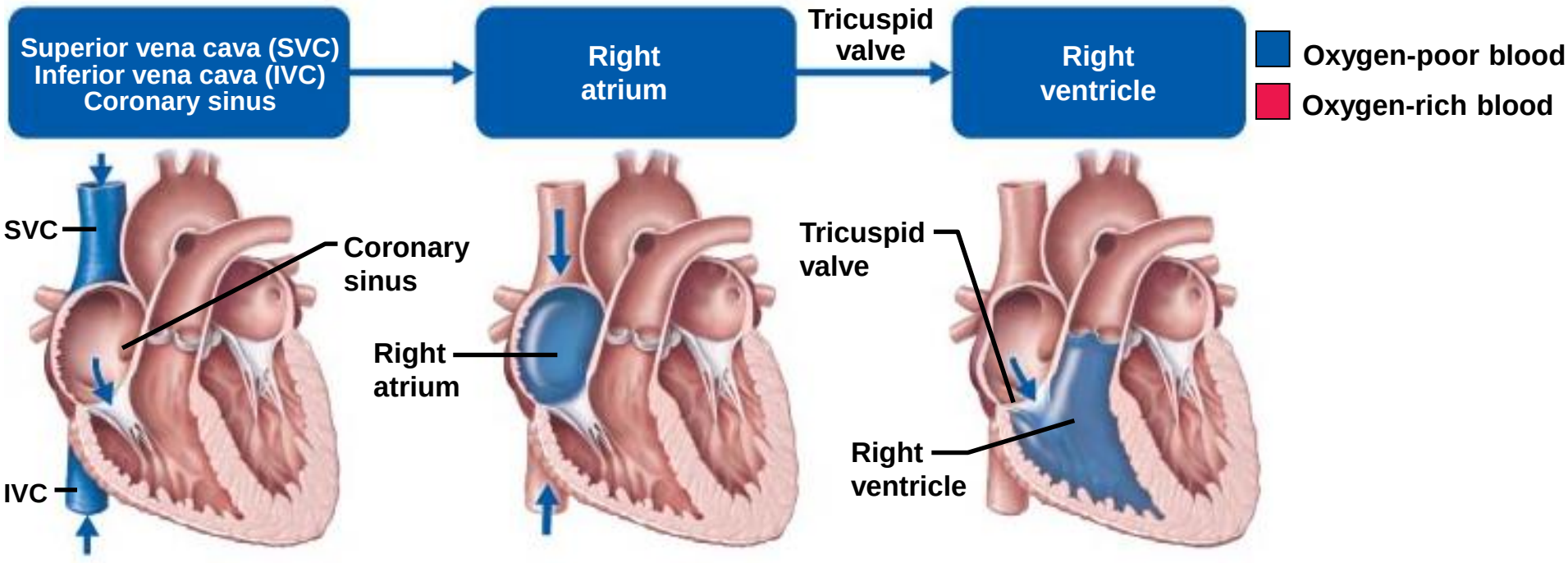

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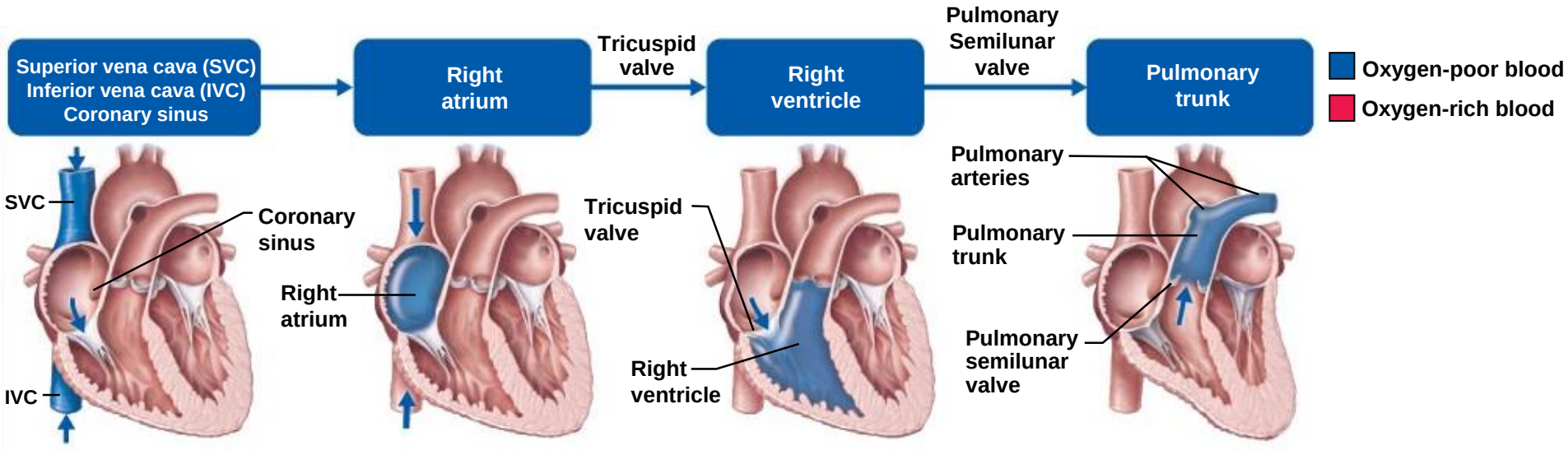

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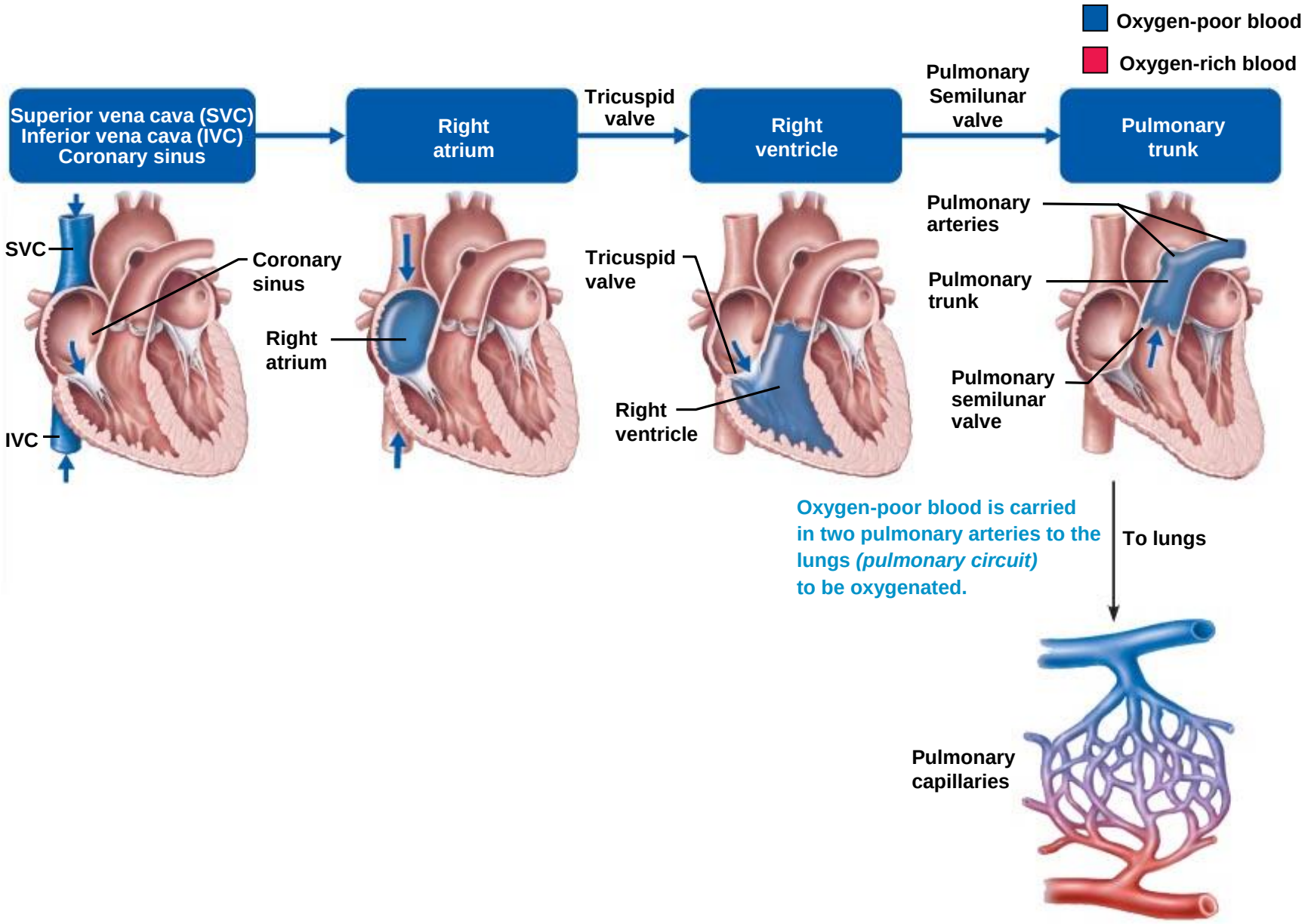


Figure 18.9 The heart is a double pump, each side supplying its own circuit.

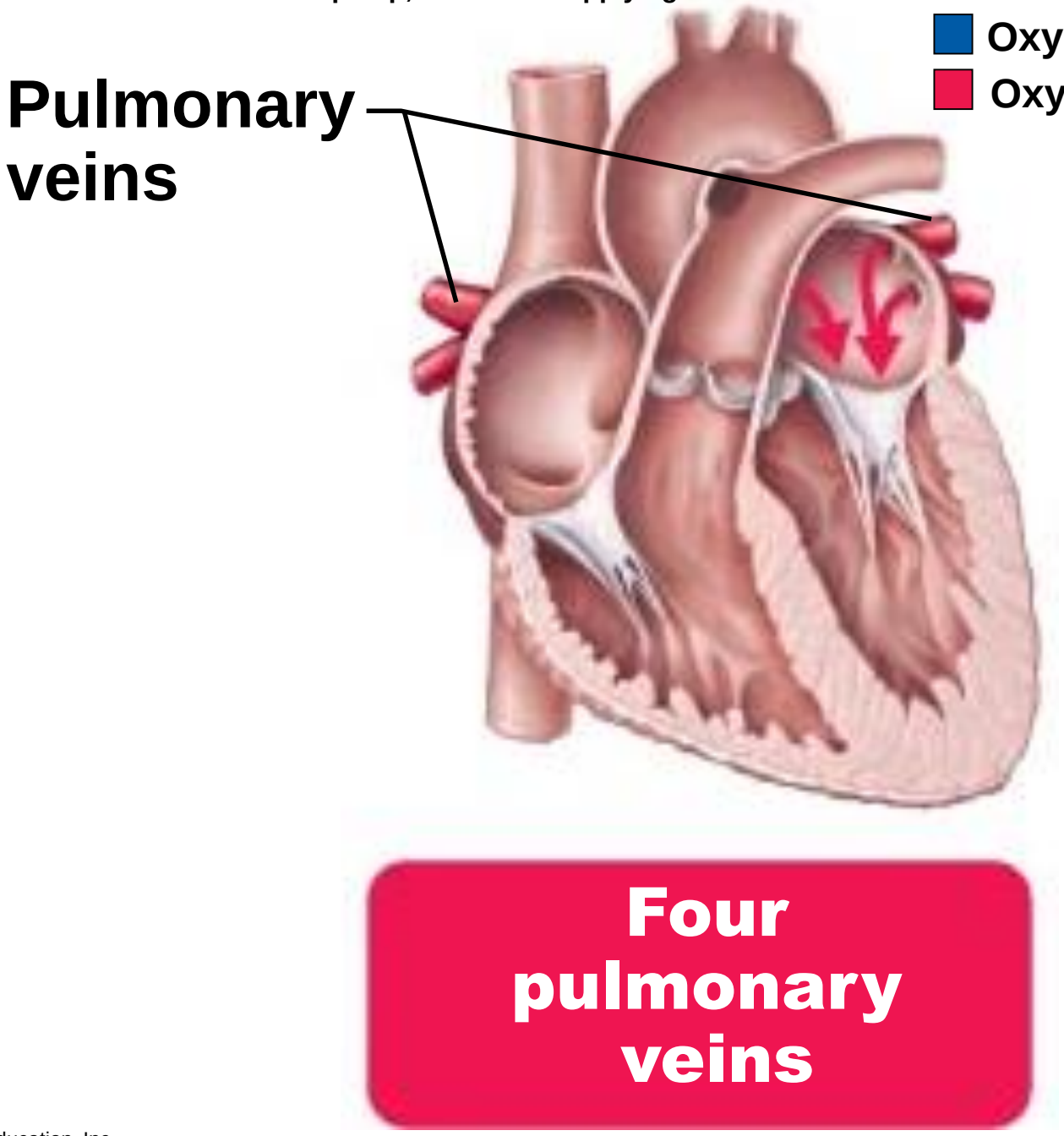
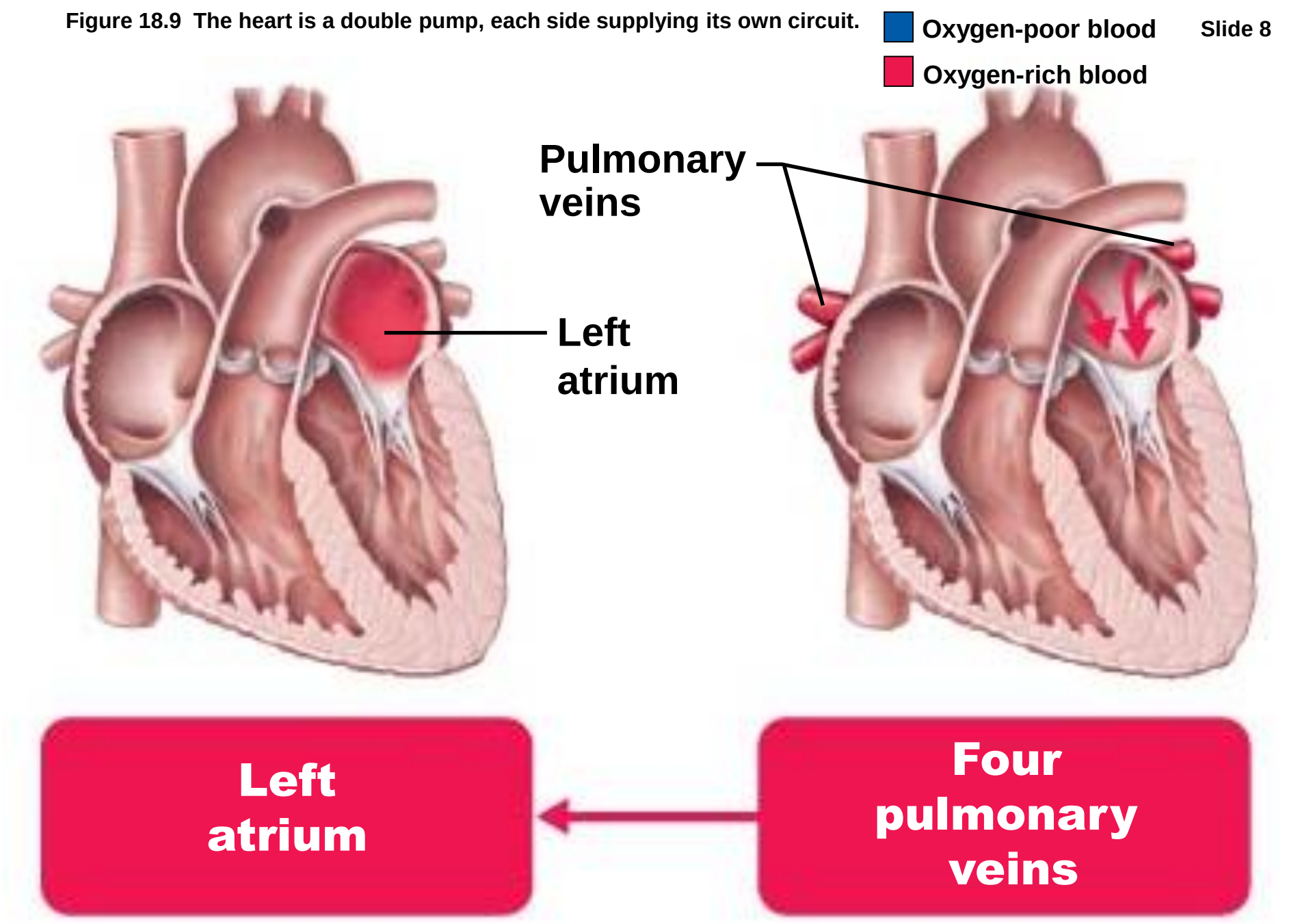


Figure 18.9 The heart is a double pump, each side supplying its own circuit.

Slide 8



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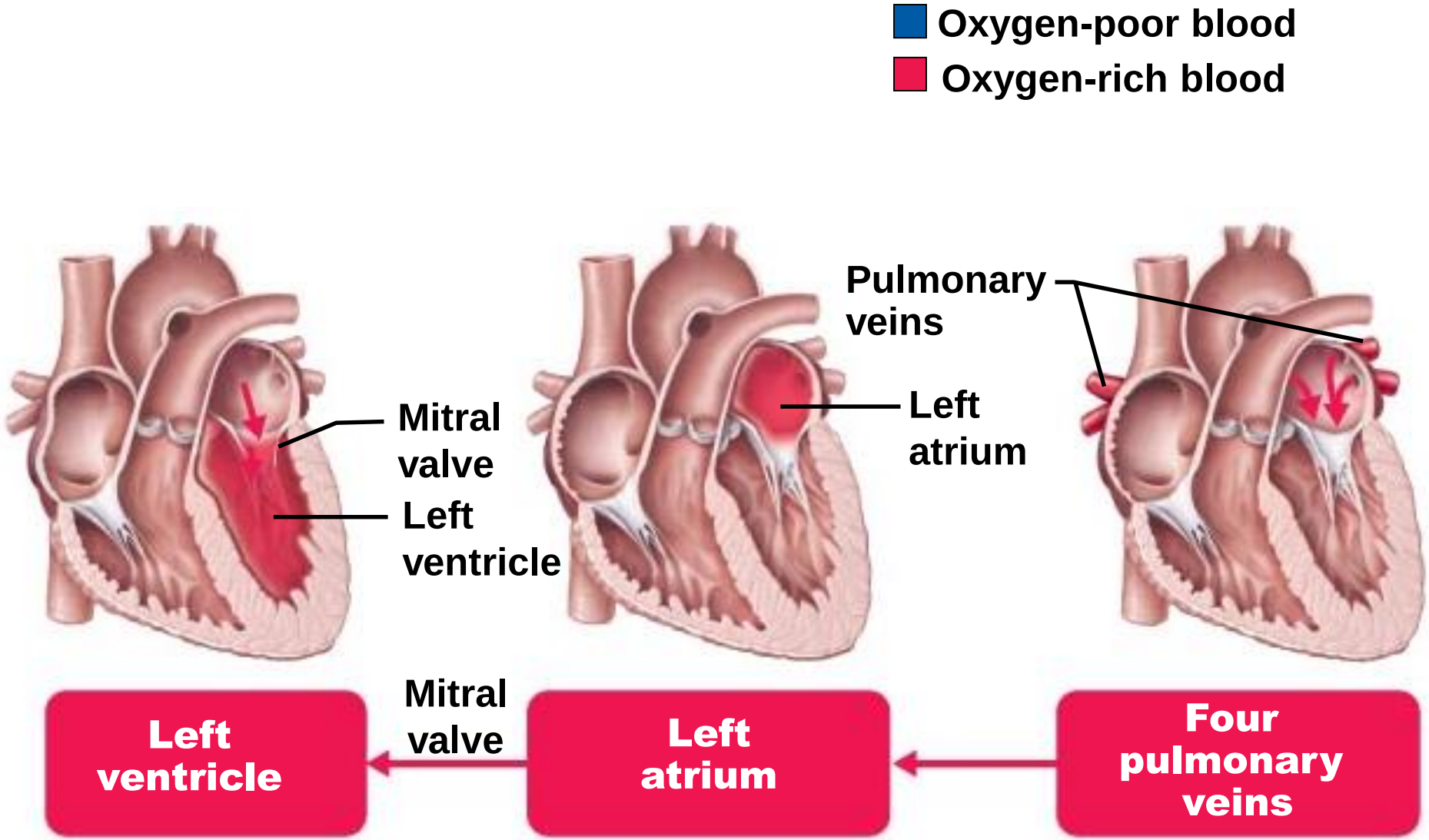

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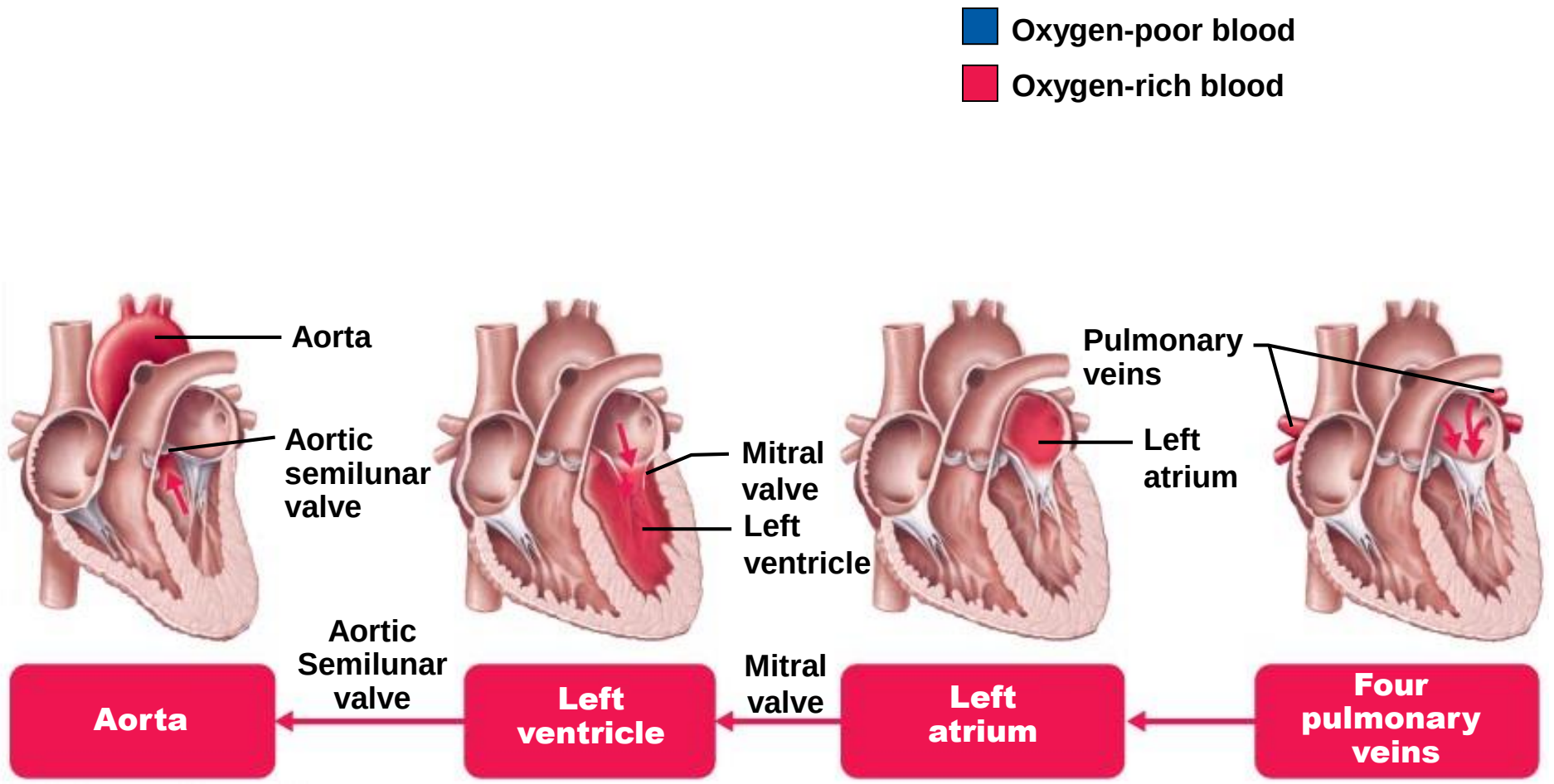

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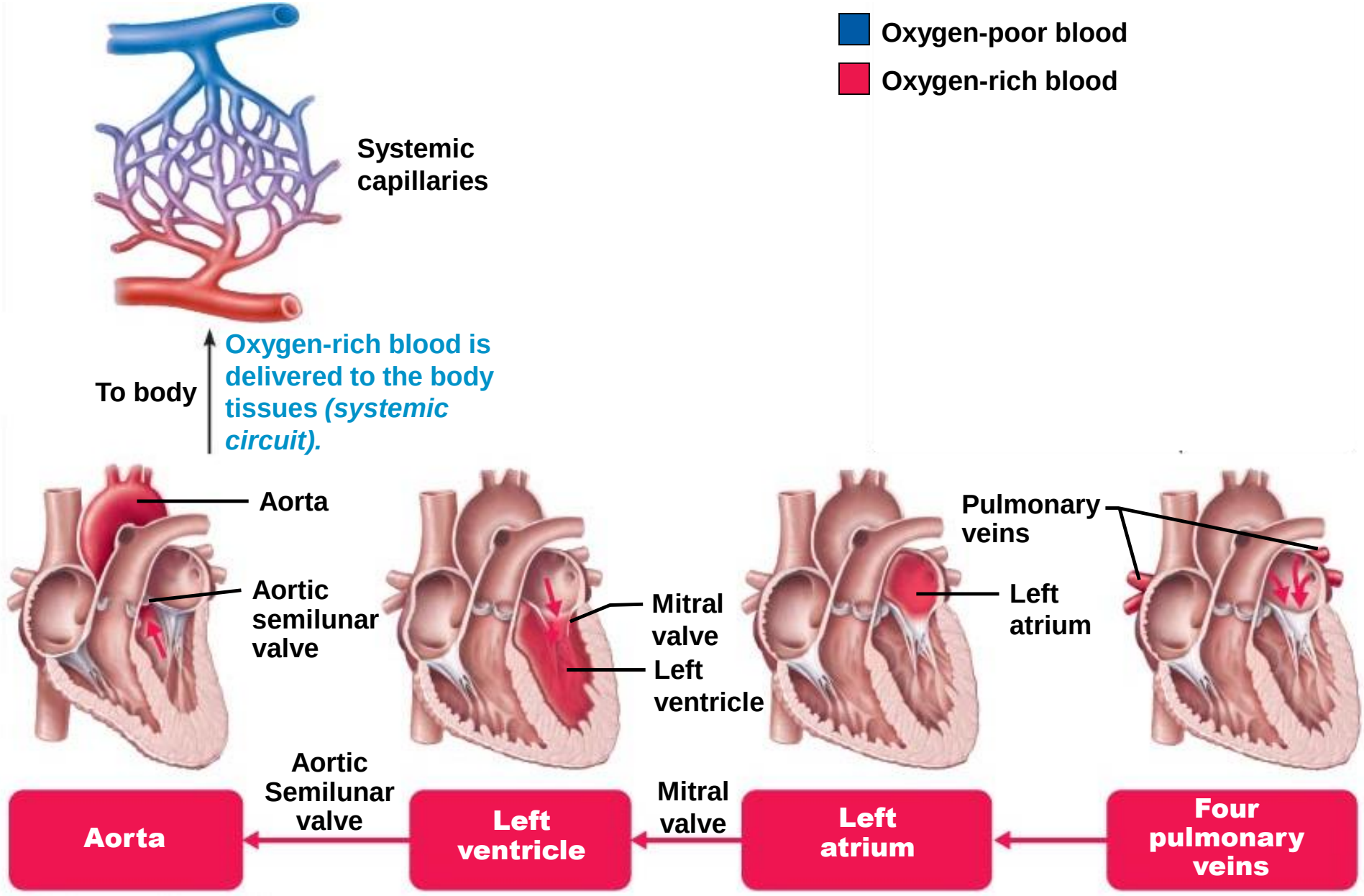

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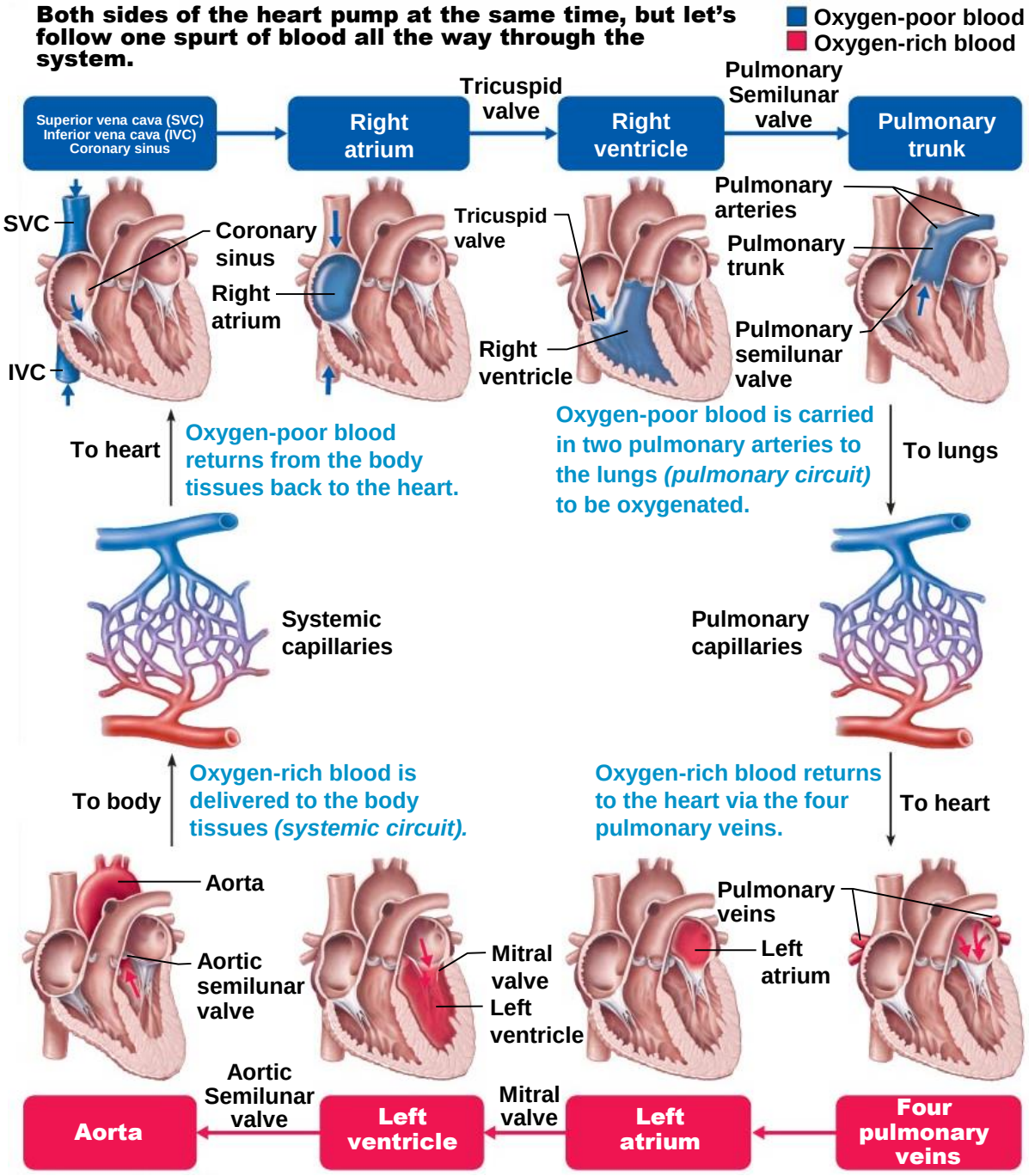
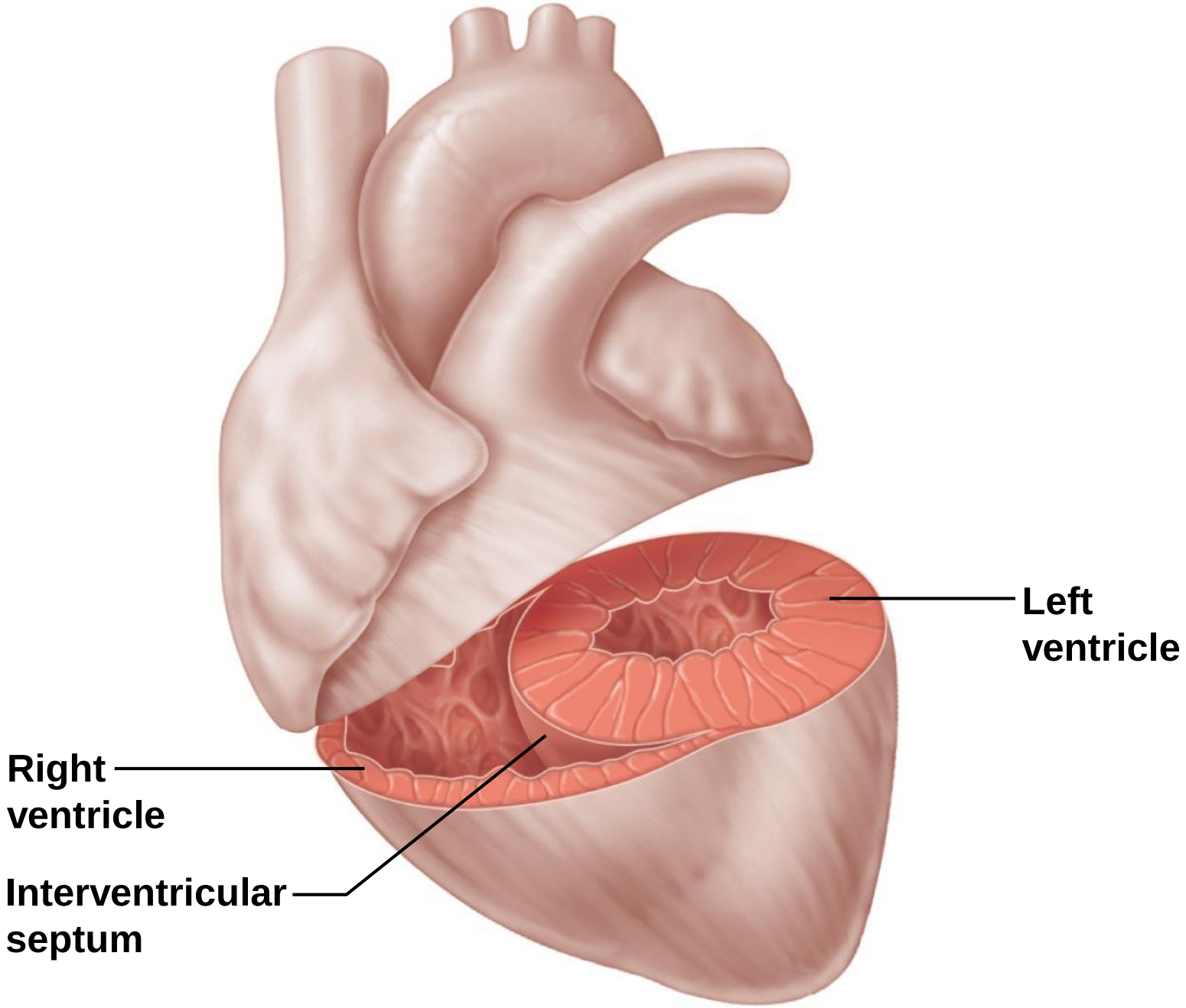


Pathway of Blood Through the Heart

- Equal volumes of blood pumped to pulmonary and systemic circuits
- Pulmonary circuit short, low-pressure circulation
- Systemic circuit long, high-friction circulation
- Anatomy of ventricles reflects differences
  - Left ventricle walls 3X thicker than right
    - Pumps with greater pressure

Figure 18.10 Anatomical differences between the right and left ventricles.



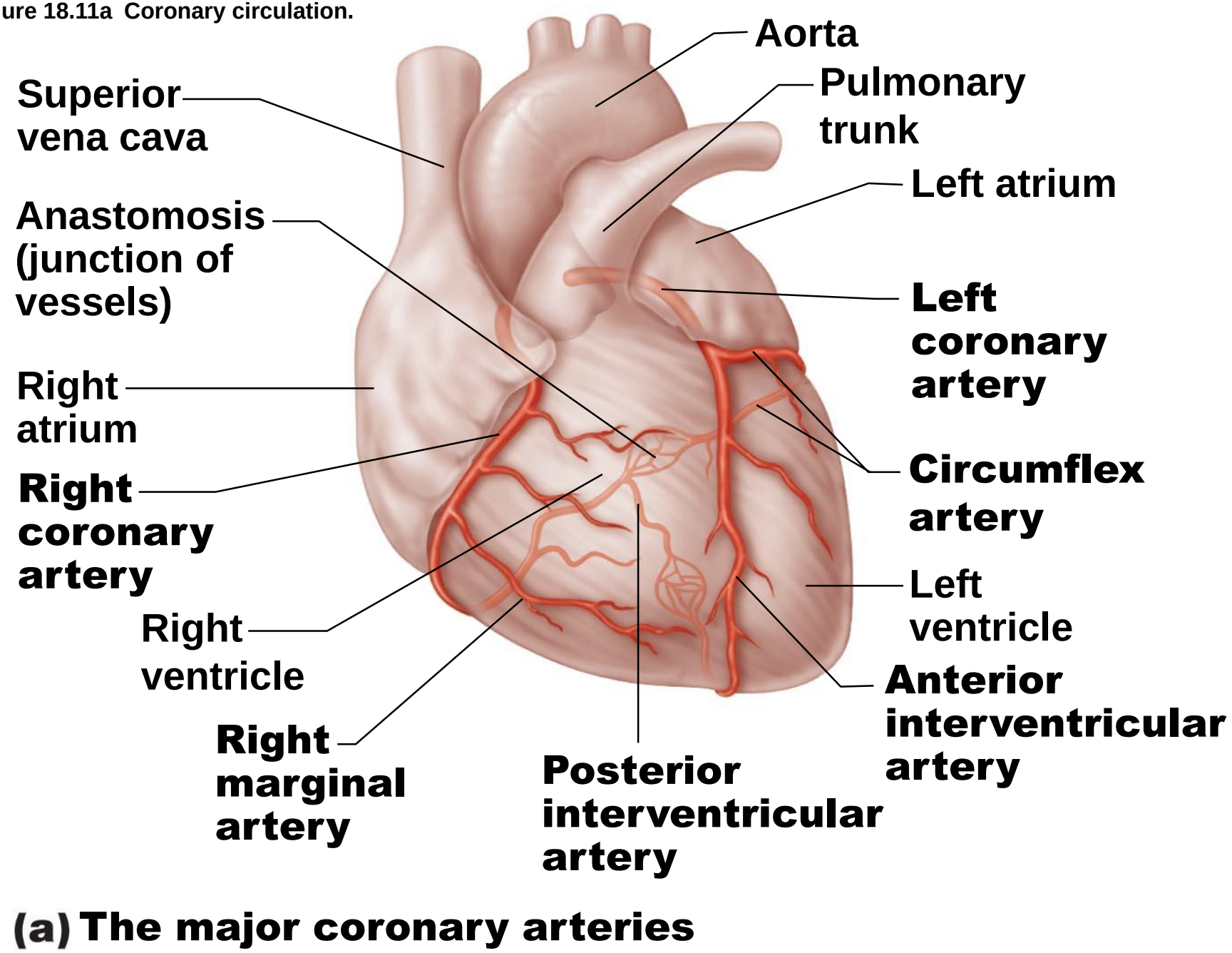
Coronary Circulation

- Functional blood supply to heart muscle itself
  - Delivered when heart relaxed
  - Left ventricle receives most blood supply
- Arterial supply varies among individuals
- Contains many anastomoses (junctions)
  - Provide additional routes for blood delivery
  - Cannot compensate for coronary artery occlusion

Coronary Circulation: Arteries

- Arteries arise from base of aorta
- **Left coronary artery** branches → **anterior interventricular artery** and **circumflex artery**
  - Supplies interventricular septum, anterior ventricular walls, left atrium, and posterior wall of left ventricle
- **Right coronary artery** branches → **right marginal artery** and **posterior interventricular artery**
  - Supplies right atrium and most of right ventricle

Figure 18.11a Coronary circulation.



Coronary Circulation: Veins

- **Cardiac veins** collect blood from capillary beds
- **Coronary sinus** empties into right atrium; formed by merging cardiac veins
  - **Great cardiac vein** of anterior interventricular sulcus
  - **Middle cardiac vein** in posterior interventricular sulcus
  - **Small cardiac vein** from inferior margin
- Several **anterior cardiac veins** empty directly into right atrium anteriorly

Figure 18.11b Coronary circulation.

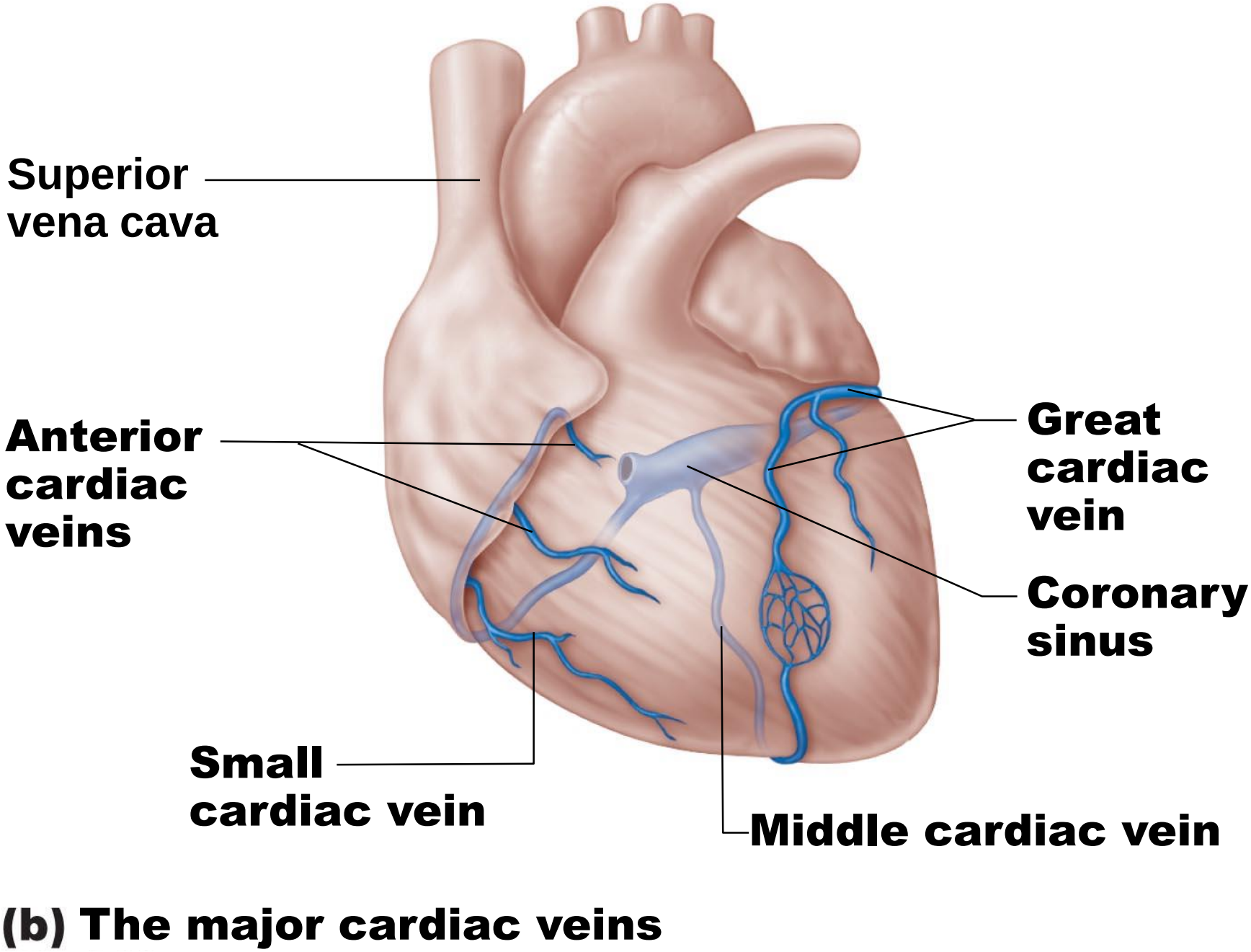
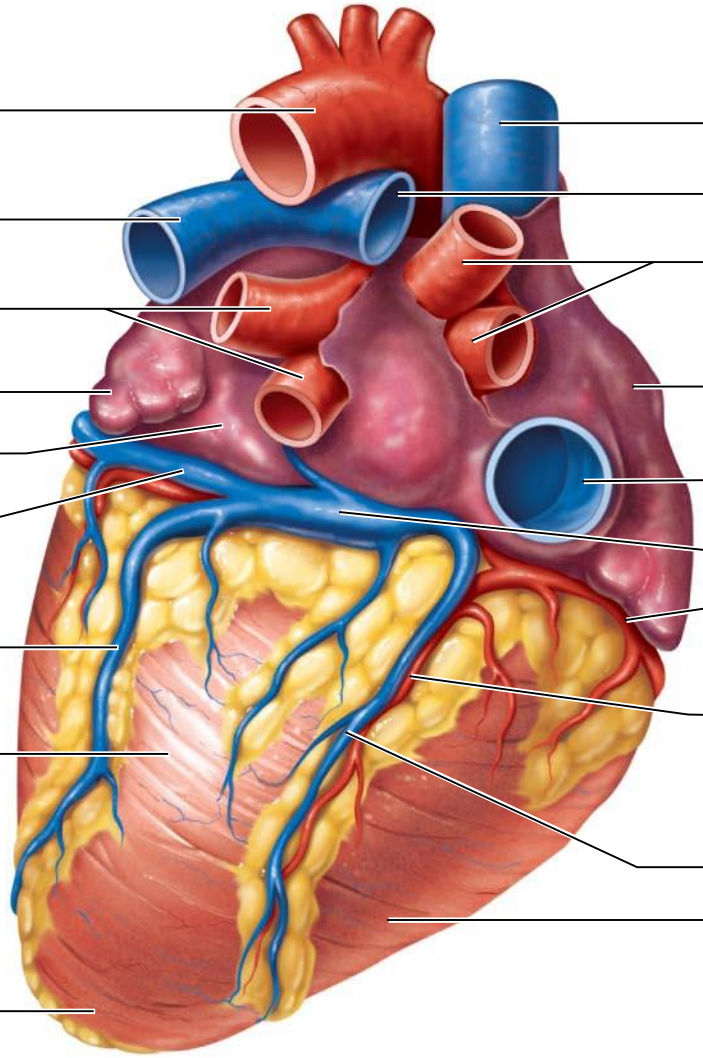


Figure 18.5d Gross anatomy of the heart.



Aorta

Left pulmonary artery

Left pulmonary veins

Auricle of left atrium

Left atrium

Great cardiac vein

Posterior vein of left ventricle

Left ventricle

Apex

Superior vena cava

Right pulmonary artery

Right pulmonary veins

Right atrium

Inferior vena cava

Coronary sinus

Right coronary artery (in coronary sulcus)

Posterior interventricular artery (in posterior interventricular sulcus)

Middle cardiac vein

Right ventricle

(d) Posterior surface view

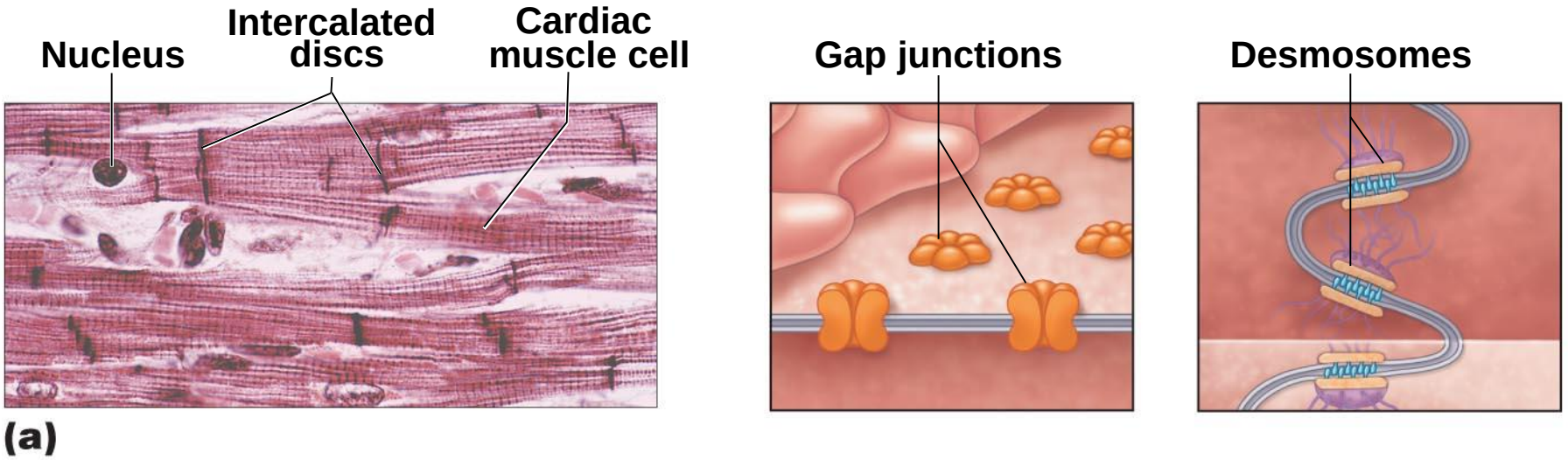
Homeostatic Imbalances

- **Angina pectoris**
  - Thoracic pain caused by fleeting deficiency in blood delivery to myocardium
  - Cells weakened
- **Myocardial infarction** (heart attack)
  - Prolonged coronary blockage
  - Areas of cell death repaired with noncontractile scar tissue

Microscopic Anatomy of Cardiac Muscle

- Cardiac muscle cells striated, short, branched, fat, interconnected, 1 (perhaps 2) central nuclei
- Connective tissue matrix (endomysium) connects to cardiac skeleton
  - Contains numerous capillaries
- T tubules wide, less numerous; SR simpler than in skeletal muscle
- Numerous large mitochondria (25–35% of cell volume)

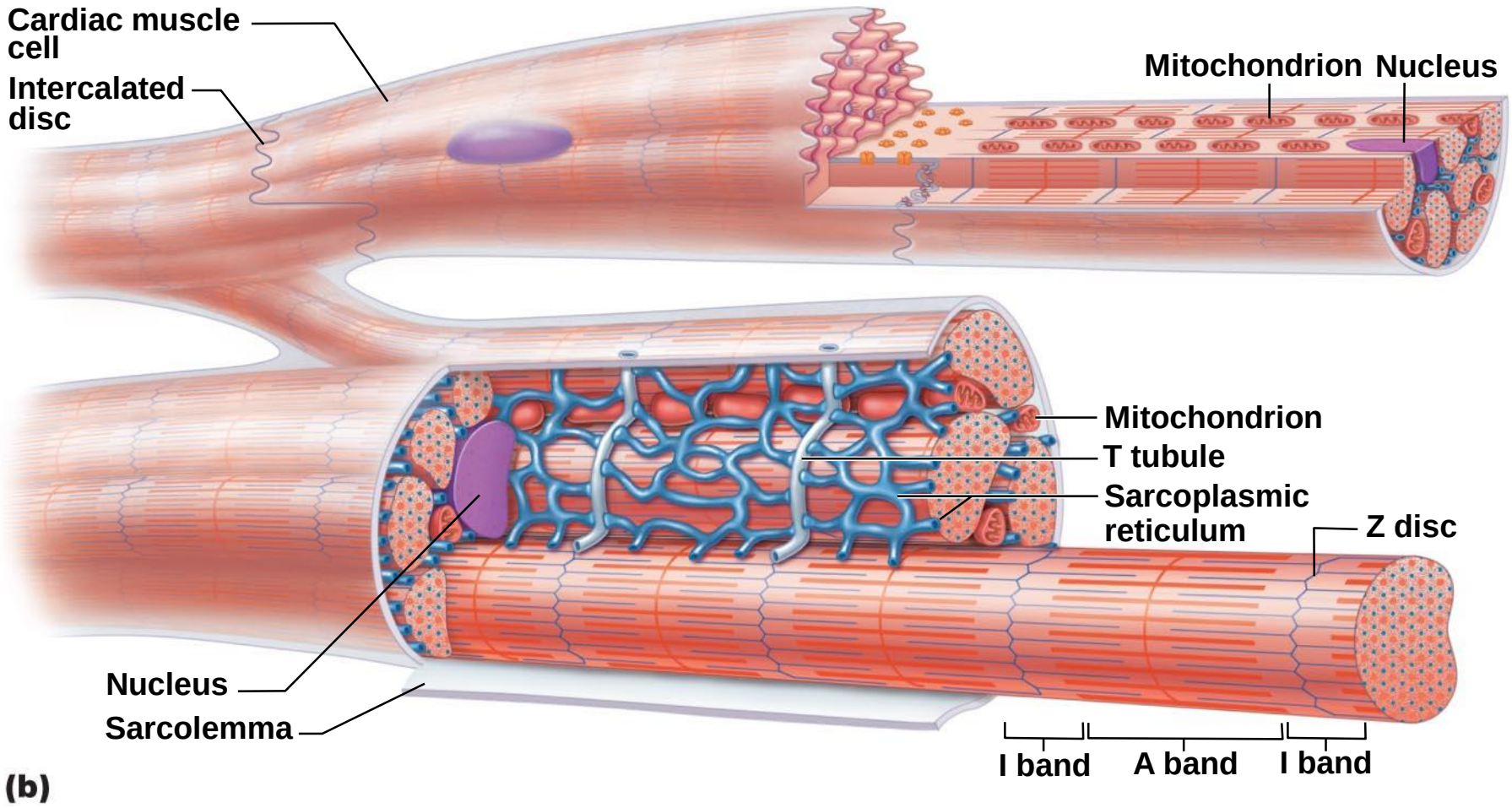
Figure 18.12a Microscopic anatomy of cardiac muscle.



Microscopic Anatomy of Cardiac Muscle

- **Intercalated discs** - junctions between cells - anchor cardiac cells
  - *Desmosomes* prevent cells from separating during contraction
  - *Gap junctions* allow ions to pass from cell to cell; electrically couple adjacent cells
    - Allows heart to be **functional syncytium**
      - Behaves as single coordinated unit

Figure 18.12b Microscopic anatomy of cardiac muscle.



Cardiac Muscle Contraction

- Three differences from skeletal muscle:
  - ~1% of cells have automaticity (autorhythmicity)
    - Do not need nervous system stimulation
    - Can depolarize entire heart
  - All cardiomyocytes contract as unit, or none do
  - Long absolute refractory period (250 ms)
    - Prevents tetanic contractions

Cardiac Muscle Contraction

- Three similarities with skeletal muscle:
  - Depolarization opens few voltage-gated fast Na^+ channels in sarcolemma →
    - Reversal of membrane potential from –90 mV to +30 mV
    - Brief; Na channels close rapidly
  - Depolarization wave down T tubules → SR to release Ca^{2+} →
  - Excitation-contraction coupling occurs
    - Ca^{2+} binds troponin → filaments slide

Cardiac Muscle Contraction

- More differences
  - Depolarization wave also opens **slow Ca^{2+} channels** in sarcolemma → SR to release its Ca^{2+}
  - Ca^{2+} surge prolongs the depolarization phase (plateau)

Cardiac Muscle Contraction

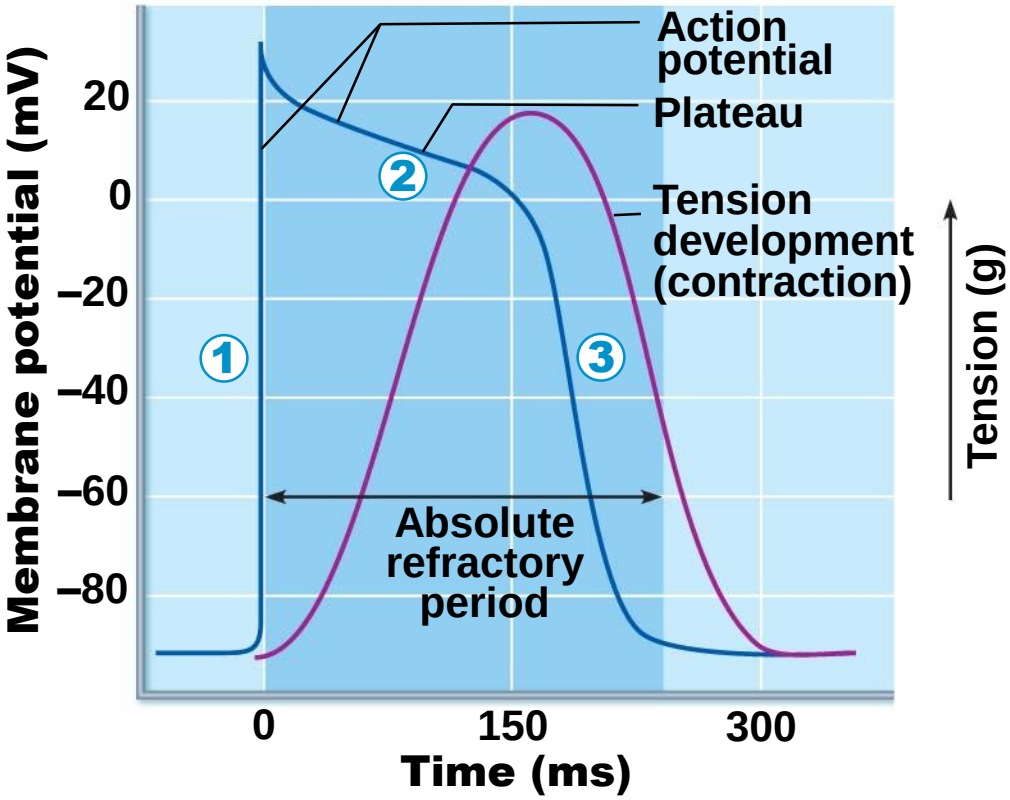
- More differences
  - Action potential and contractile phase last much longer than a neuron.
    - Allow blood ejection from heart
  - Repolarization result of inactivation of Ca^{2+} channels and opening of voltage-gated K^{+} channels
    - Ca^{2+} pumped back to SR and extracellularly

Table 10-2 Characteristics of the major types of muscle fibers in vertebrates

	Striated muscle		Smooth (nonstriated) muscle	
Property/component	Skeletal	Cardiac	Multi-unit	Single-unit
Visible banding pattern	Yes	Yes	No	No
Myosin thick filaments and actin thin filaments	Yes	Yes	Yes	Yes
Tropomyosin and troponin	Yes	Yes	No	No
Transverse tubules	Yes	Yes	No	No
Sarcoplasmic reticulum	Well developed	Well developed	Very little	Very little
Mechanism of contraction	Sliding of thick and thin filaments past each other	Sliding of thick and thin filaments past each other	Sliding of thick and thin filaments past each other	Sliding of thick and thin filaments past each other
Innervation	Somatic nerves	Autonomic nerves	Autonomic nerves	Autonomic nerves
Initiation of contraction*	Neurogenic	Myogenic	Neurogenic	Myogenic
Source of Ca^{2+} for activation†	SR	ECF and SR	ECF and SR	ECF and SR
Gap junctions between fibers?	No	Yes	No	Yes
Speed of contraction	Fast or slow depending on fiber type	Slow	Very slow	Very slow
Clear-cut relationship between length and tension	Yes	Yes	No	No

*Neurogenic muscles contract only when stimulated by synaptic input from a neuron. Myogenic muscles endogenously produce depolarizing membrane potentials, allowing them to contract independently of any neuronal input.
†SR, sarcoplasmic reticulum; ECF, extracellular fluid.
Source: Adapted from Sherwood, 2001.

Figure 18.13 The action potential of contractile cardiac muscle cells.

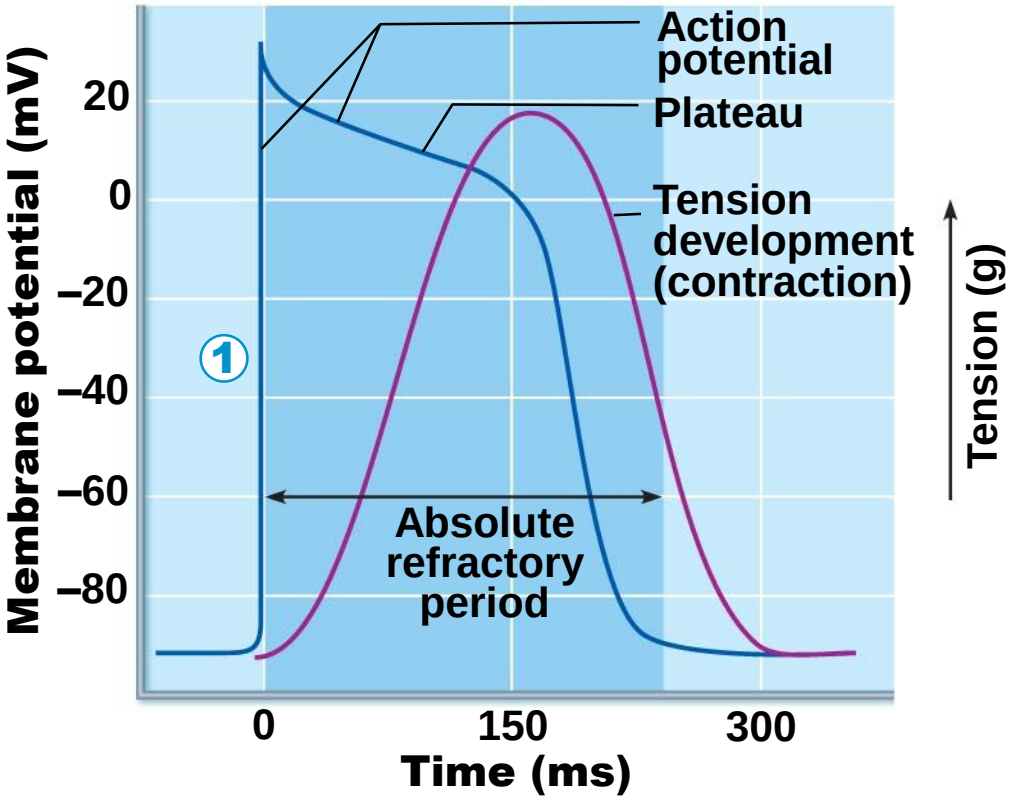


① **Depolarization** is due to Na^+ influx through fast voltage-gated Na^+ channels. A positive feedback cycle rapidly opens many Na^+ channels, reversing the membrane potential. Channel inactivation ends this phase.

② **Plateau phase** is due to Ca^{2+} influx through slow Ca^{2+} channels. This keeps the cell depolarized because few K^+ channels are open.

③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its resting voltage.

Figure 18.13 The action potential of contractile cardiac muscle cells.



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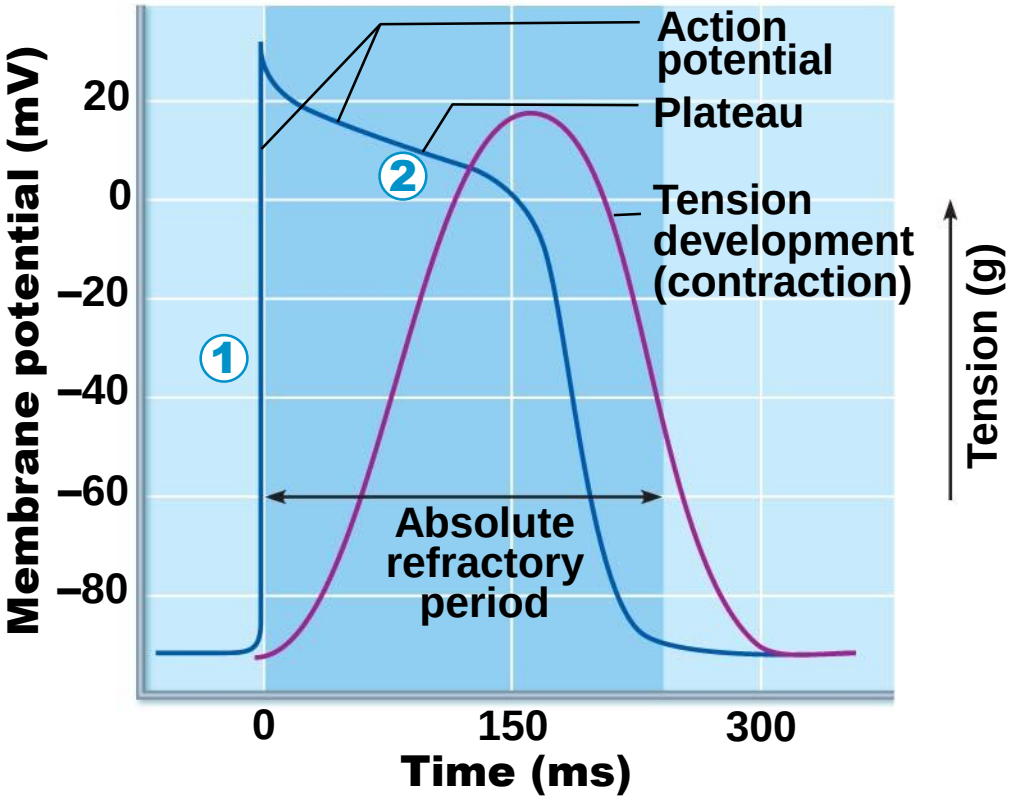

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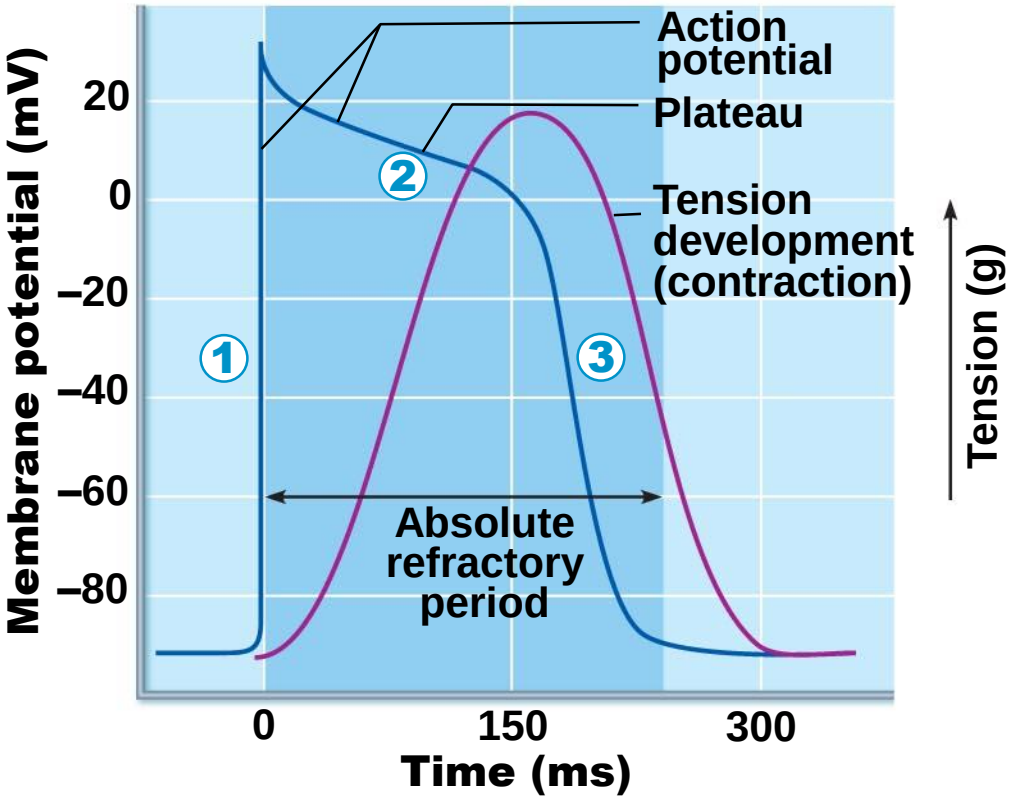

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③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its resting voltage.

Energy Requirements

- Cardiac muscle
  - Has many mitochondria
    - Great dependence on aerobic respiration
    - Little anaerobic respiration ability
  - Readily switches fuel source for respiration
    - Even uses lactic acid from skeletal muscles

Homeostatic Imbalance

- Ischemic cells → anaerobic respiration → lactic acid →
  - High H^+ concentration → high Ca^{2+} concentration
    - → Mitochondrial damage → decreased ATP production
    - → Gap junctions close → fatal arrhythmias

Heart Physiology: Electrical Events

- Heart depolarizes and contracts without nervous system stimulation
  - Rhythm can be altered by autonomic nervous system

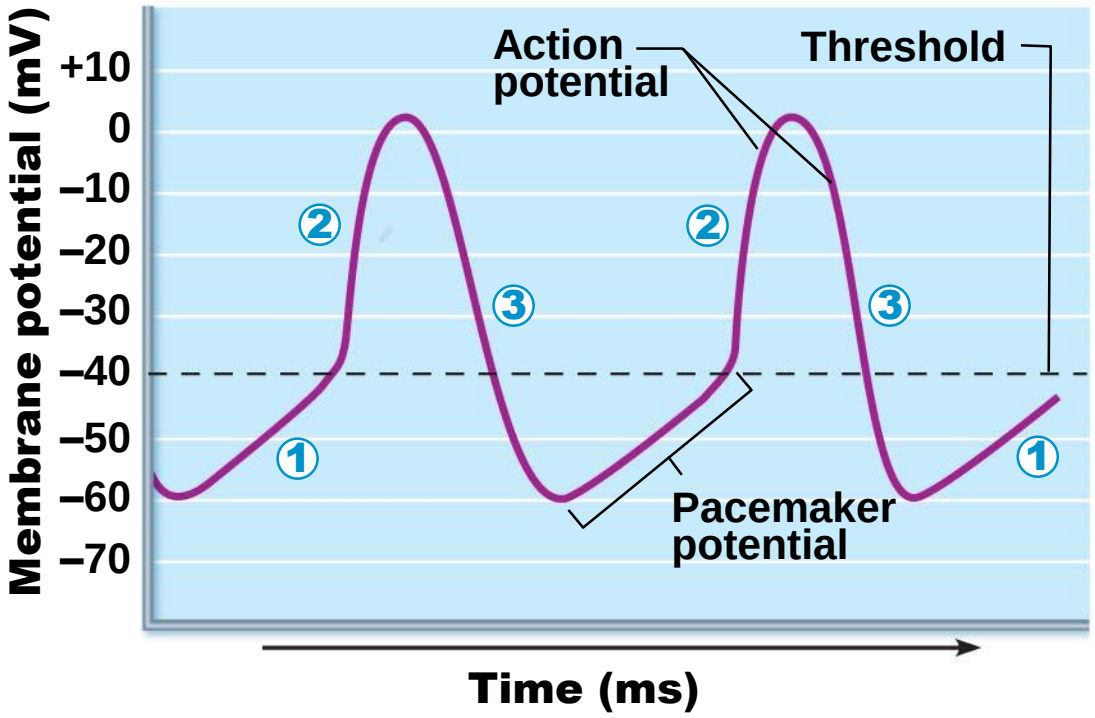
Pacemaker (Autorhythmic) Cells

- Have unstable resting membrane potentials (pacemaker potentials or prepotentials) due to opening of slow Na^+ channels
  - Continuously depolarize
- At threshold, Ca^{2+} channels open
- Explosive Ca^{2+} influx produces the rising phase of the action potential
- Repolarization results from inactivation of Ca^{2+} channels and opening of voltage-gated K^+ channels

Action Potential Initiation by Pacemaker Cells

- Three parts of action potential:
  - **Pacemaker potential**
    - Repolarization closes K^+ channels and opens slow Na^+ channels → ion imbalance →
  - **Depolarization**
    - Ca^{2+} channels open → huge influx → rising phase of action potential
  - **Repolarization**
    - K^+ channels open → efflux of K^+

Figure 18.14 Pacemaker and action potentials of pacemaker cells in the heart.



① **Pacemaker potential** This slow depolarization is due to both opening of Na^+ channels and closing of K^+ channels. Notice that the membrane potential is never a flat line.

② **Depolarization** The action potential begins when the pacemaker potential reaches threshold. Depolarization is due to Ca^{2+} influx through Ca^{2+} channels.

③ **Repolarization** is due to Ca^{2+} channels inactivating and K^+ channels opening. This allows K^+ efflux, which brings the membrane potential back to its most negative voltage.

Sequence of Excitation

- Cardiac pacemaker cells pass impulses, in order, across heart in ~220 ms
  - **Sinoatrial node →**
  - **Atrioventricular node →**
  - **Atrioventricular bundle →**
  - **Right and left bundle branches →**
  - **Subendocardial conducting network (Purkinje fibers)**

Heart Physiology: Sequence of Excitation

- Sinoatrial (SA) node
  - **Pacemaker** of heart in right atrial wall
    - Depolarizes faster than rest of myocardium
  - Generates impulses about 75X/minute (**sinus rhythm**)
    - Inherent rate of 100X/minute tempered by extrinsic factors
- Impulse spreads across atria, and to AV node

Heart Physiology: Sequence of Excitation

- Atrioventricular (AV) node
  - In inferior interatrial septum
  - Delays impulses approximately 0.1 second
    - Because fibers are smaller diameter, have fewer gap junctions
    - Allows atrial contraction prior to ventricular contraction
  - Inherent rate of 50X/minute in absence of SA node input

Heart Physiology: Sequence of Excitation

- Atrioventricular (AV) bundle (bundle of His)
  - In superior interventricular septum
  - Only electrical connection between atria and ventricles
    - Atria and ventricles not connected via gap junctions

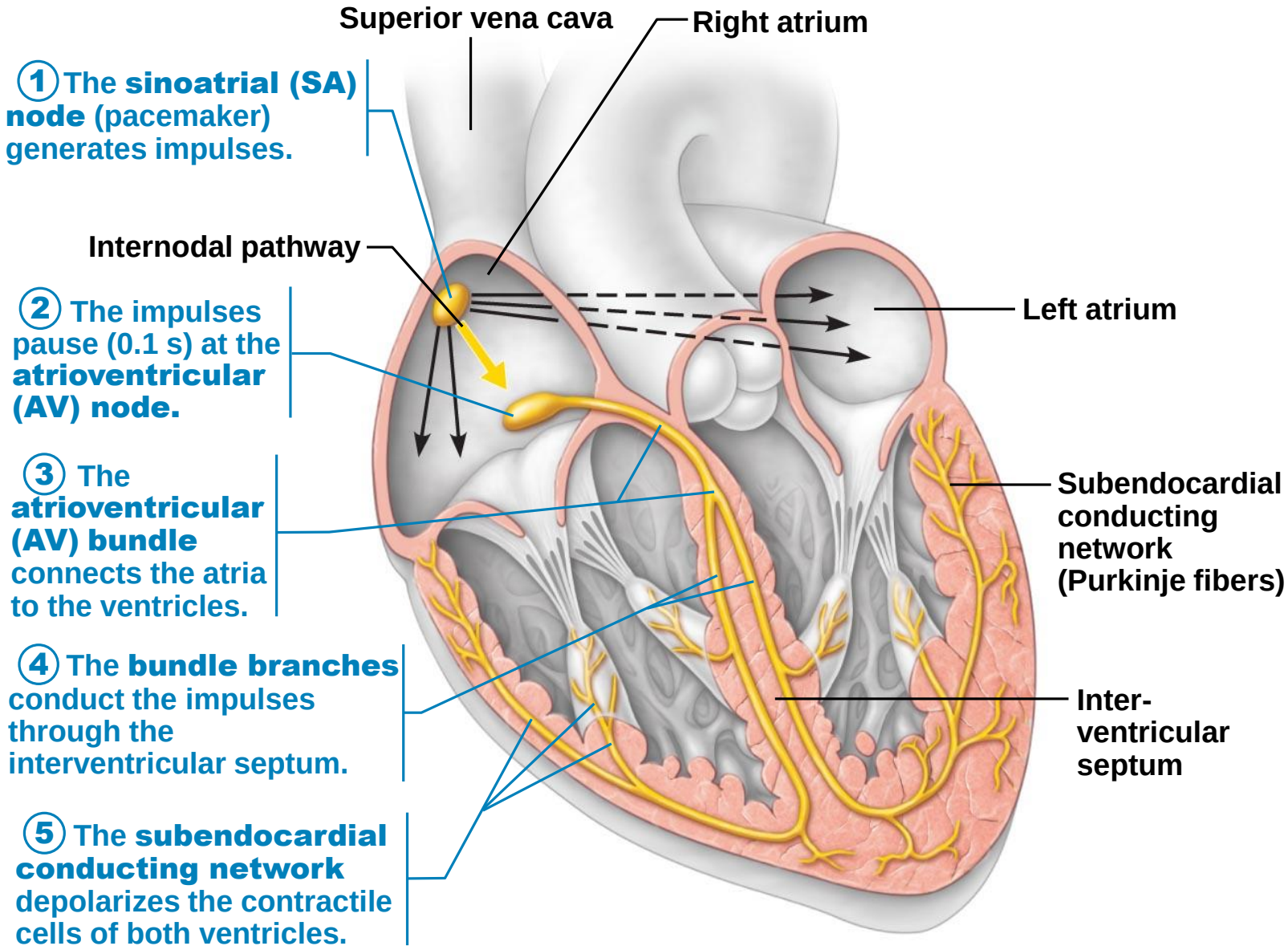
Heart Physiology: Sequence of Excitation

- Right and left bundle branches
  - Two pathways in interventricular septum
  - Carry impulses toward apex of heart

Heart Physiology: Sequence of Excitation

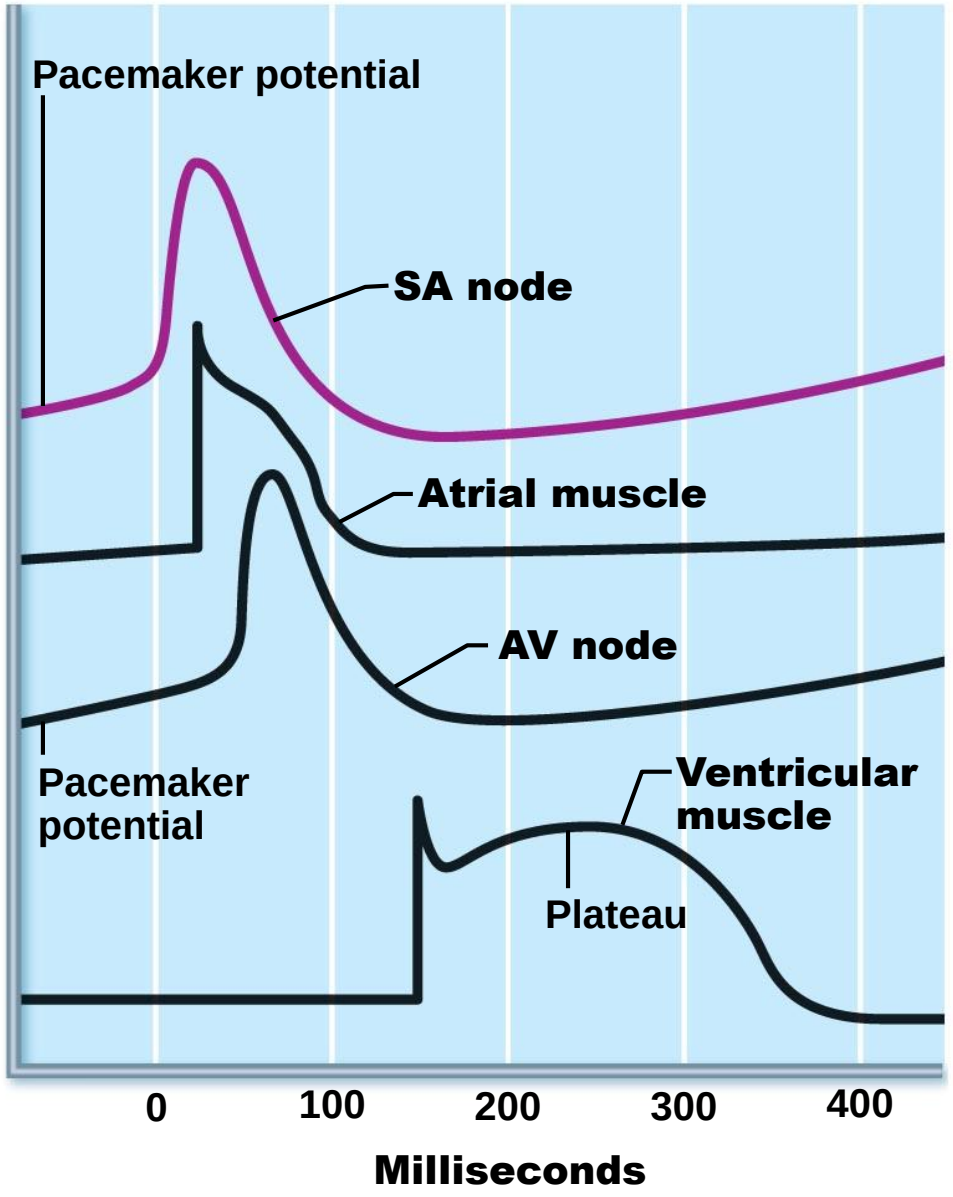
- Subendocardial conducting network
  - Complete pathway through interventricular septum into apex and ventricular walls
  - More elaborate on left side of heart
  - AV bundle and subendocardial conducting network depolarize 30X/minute in absence of AV node input
- Ventricular contraction immediately follows from apex toward atria

Figure 18.15a Intrinsic cardiac conduction system and action potential succession during one heartbeat.



(a) Anatomy of the intrinsic conduction system showing the sequence of electrical excitation

Figure 18.15b Intrinsic cardiac conduction system and action potential succession during one heartbeat.



(b) Comparison of action potential shape at various locations

Homeostatic Imbalances

- Defects in intrinsic conduction system may cause
  - **Arrhythmias** - irregular heart rhythms
  - Uncoordinated atrial and ventricular contractions
  - **Fibrillation** - rapid, irregular contractions; useless for pumping blood → circulation ceases → brain death
    - Defibrillation to treat

Homeostatic Imbalances

- Defective SA node may cause
  - **Ectopic focus** - abnormal pacemaker
  - AV node may take over; sets **junctional rhythm** (40–60 beats/min)
- **Extrasystole** (premature contraction)
  - Ectopic focus sets high rate
  - Can be from excessive caffeine or nicotine

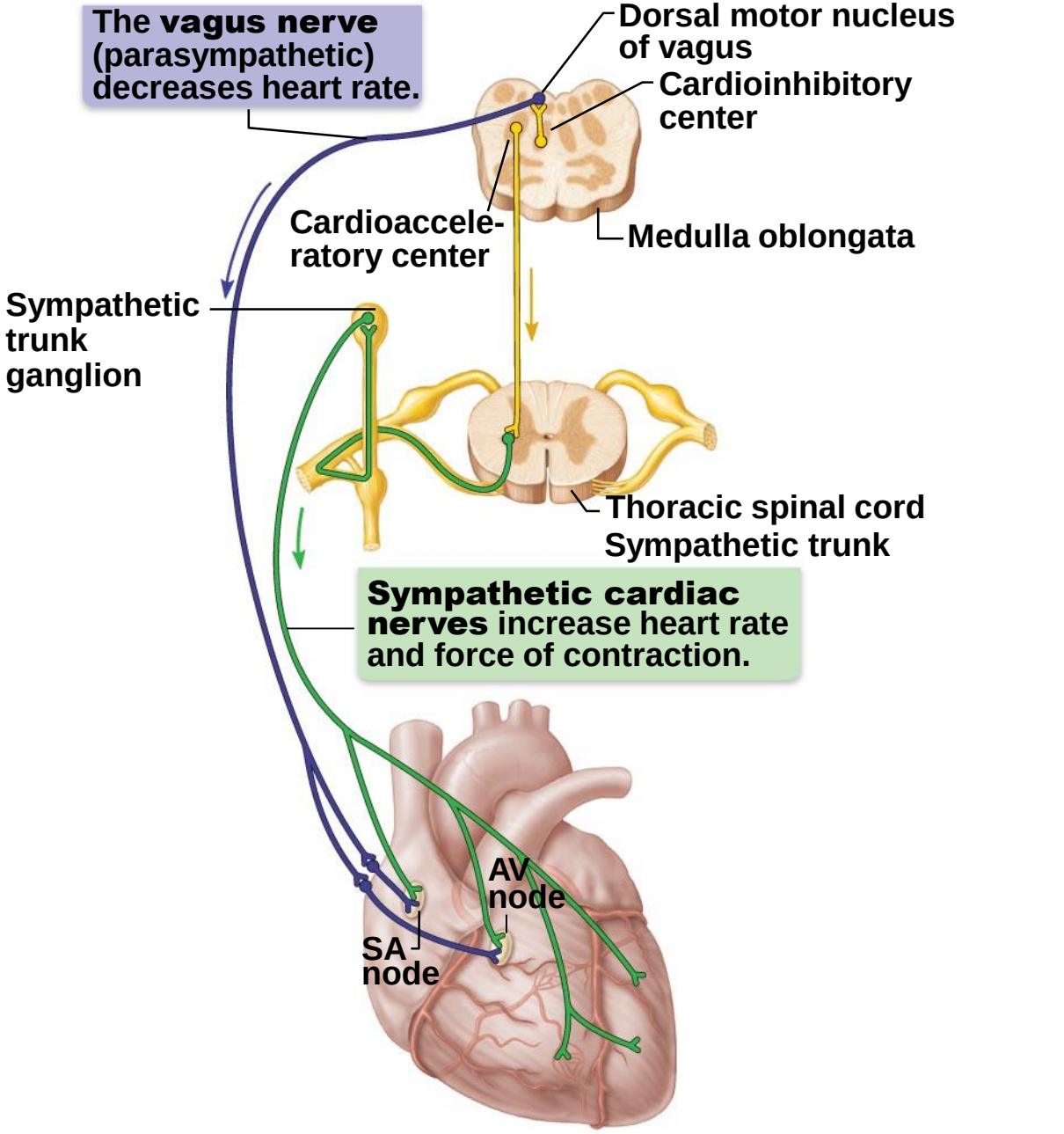
Homeostatic Imbalance

- To reach ventricles, impulse must pass through AV node
- Defective AV node may cause
  - **Heart block**
    - Few (partial) or no (total) impulses reach ventricles
      - Ventricles beat at intrinsic rate – too slow for life
  - Artificial pacemaker to treat

Extrinsic Innervation of the Heart

- Heartbeat modified by ANS via cardiac centers in medulla oblongata
  - Sympathetic → ↑ rate and force
  - Parasympathetic → ↓ rate
  - **Cardioacceleratory center** – sympathetic – affects SA, AV nodes, heart muscle, coronary arteries
  - **Cardioinhibitory center** – parasympathetic – inhibits SA and AV nodes via vagus nerves

Figure 18.16 Autonomic innervation of the heart.



Electrocardiography

- Electrocardiogram (ECG or EKG)
  - Composite of all action potentials generated by nodal and contractile cells at given time
- Three waves:
  - **P wave** – depolarization SA node → atria
  - **QRS complex** - ventricular depolarization and atrial repolarization
  - **T wave** - ventricular repolarization

Figure 18.17 An electrocardiogram (ECG) tracing.

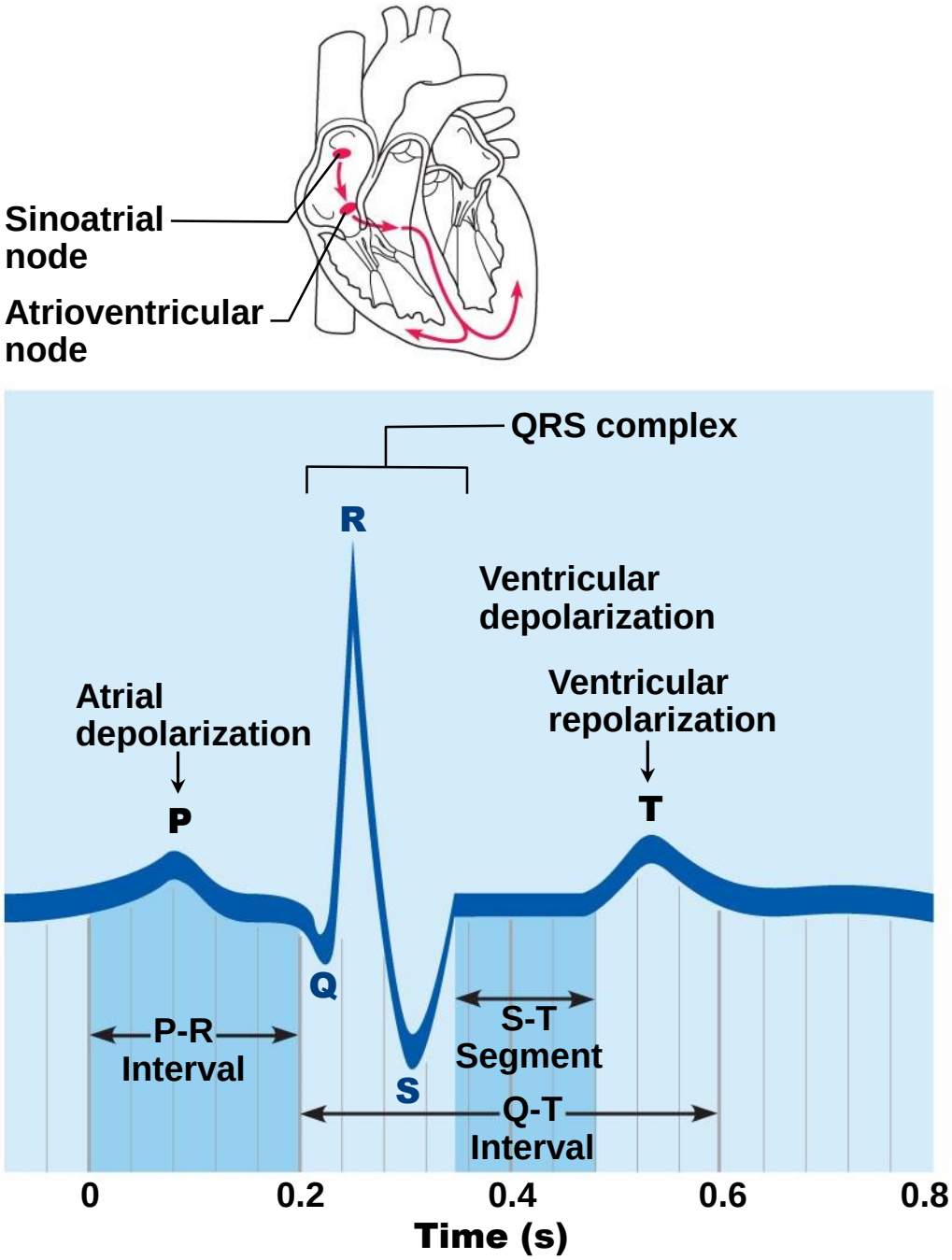


Figure 18.18 The sequence of depolarization and repolarization of the heart related to the deflection waves of an ECG tracing.

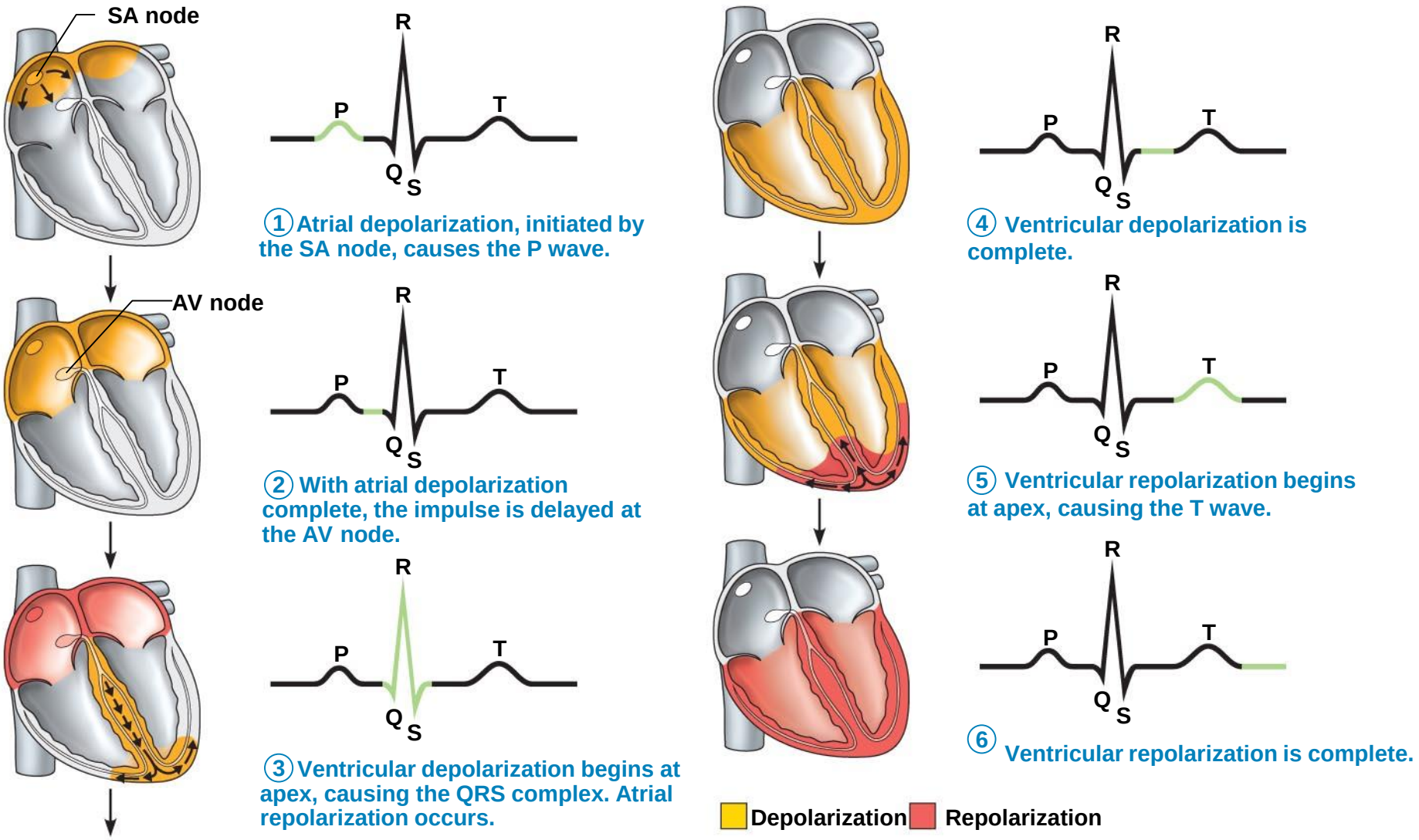
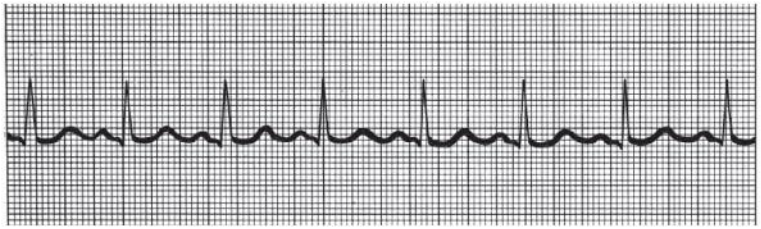
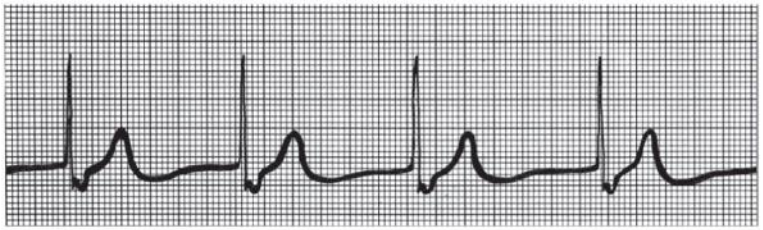


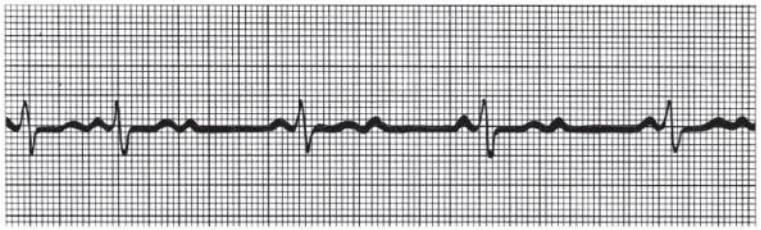
Figure 18.19 Normal and abnormal ECG tracings.



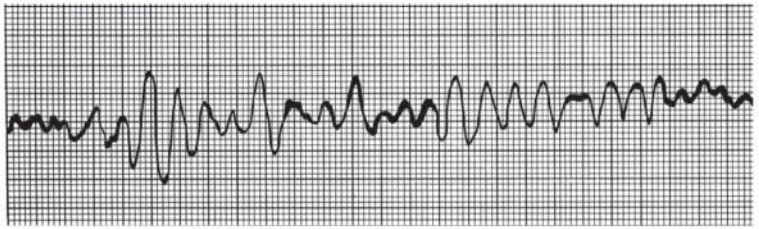
(a) Normal sinus rhythm.



(b) Junctional rhythm. The SA node is nonfunctional, P waves are absent, and the AV node paces the heart at 40–60 beats/min.



(c) Second-degree heart block. Some P waves are not conducted through the AV node; hence more P than QRS waves are seen. In this tracing, the ratio of P waves to QRS waves is mostly 2:1.



(d) Ventricular fibrillation. These chaotic, grossly irregular ECG deflections are seen in acute heart attack and electrical shock.

Electrocardiography

- **P-R interval**
  - Beginning of atrial excitation to beginning of ventricular excitation
- **S-T segment**
  - Entire ventricular myocardium depolarized
- **Q-T interval**
  - Beginning of ventricular depolarization through ventricular repolarization

Heart Sounds

- Two sounds (lub-dup) associated with closing of heart valves
  - First as AV valves close; beginning of systole
  - Second as SL valves close; beginning of ventricular diastole
  - Pause indicates heart relaxation
- **Heart murmurs** - abnormal heart sounds; usually indicate incompetent or stenotic valves

Cardiac cycle related to sounds & values:

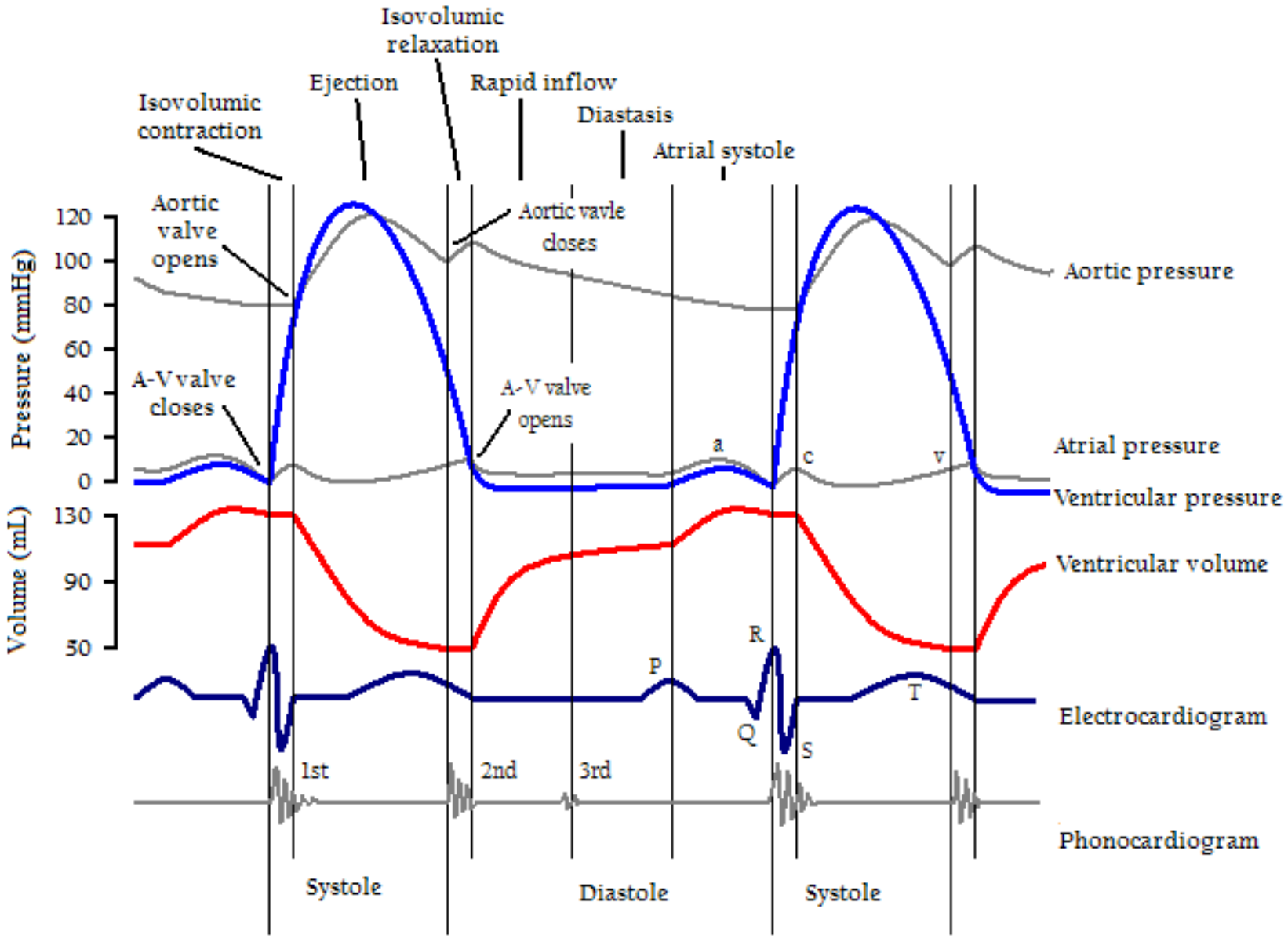
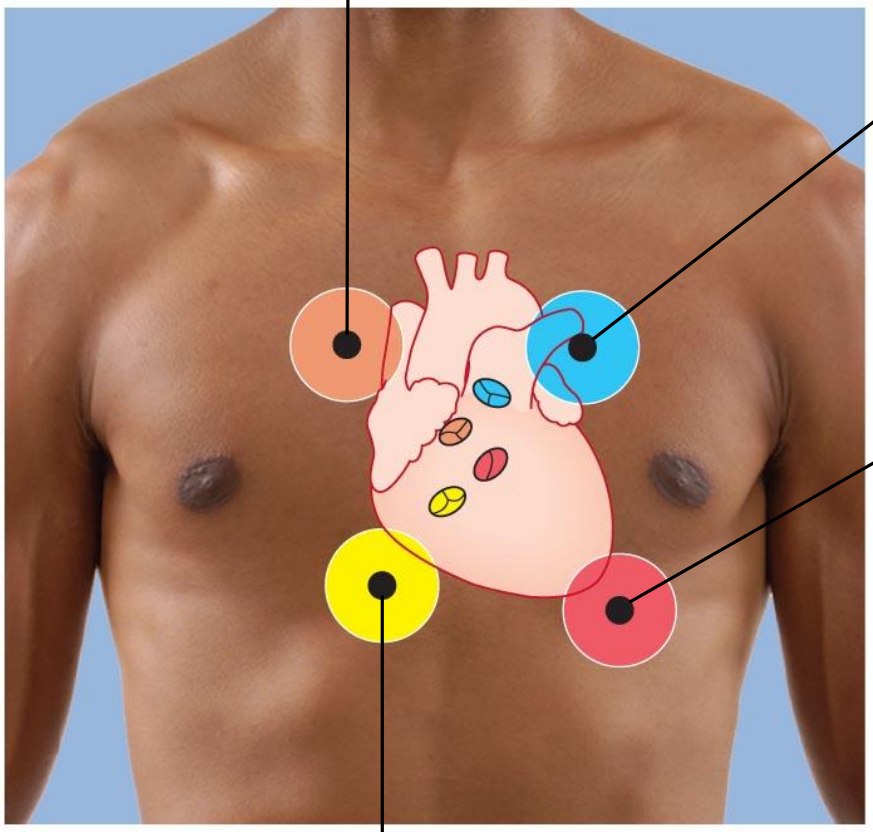


Figure 18.20 Areas of the thoracic surface where the sounds of individual valves can best be detected.



Aortic valve sounds heard in 2nd intercostal space at right sternal margin

Pulmonary valve sounds heard in 2nd intercostal space at left sternal margin

Mitral valve sounds heard over heart apex (in 5th intercostal space) in line with middle of clavicle

Tricuspid valve sounds typically heard in right sternal margin of 5th intercostal space

Mechanical Events: The Cardiac Cycle

- **Cardiac cycle**
  - Blood flow through heart during one complete heartbeat: atrial systole and diastole followed by ventricular systole and diastole
  - **Systole**—contraction
  - **Diastole**—relaxation
  - Series of pressure and blood volume changes

Phases of the Cardiac Cycle

- 1. Ventricular filling—takes place in mid-to-late diastole
  - AV valves are open; pressure low
  - 80% of blood passively flows into ventricles
  - Atrial systole occurs, delivering remaining 20%
  - **End diastolic volume** (EDV): volume of blood in each ventricle at end of ventricular diastole

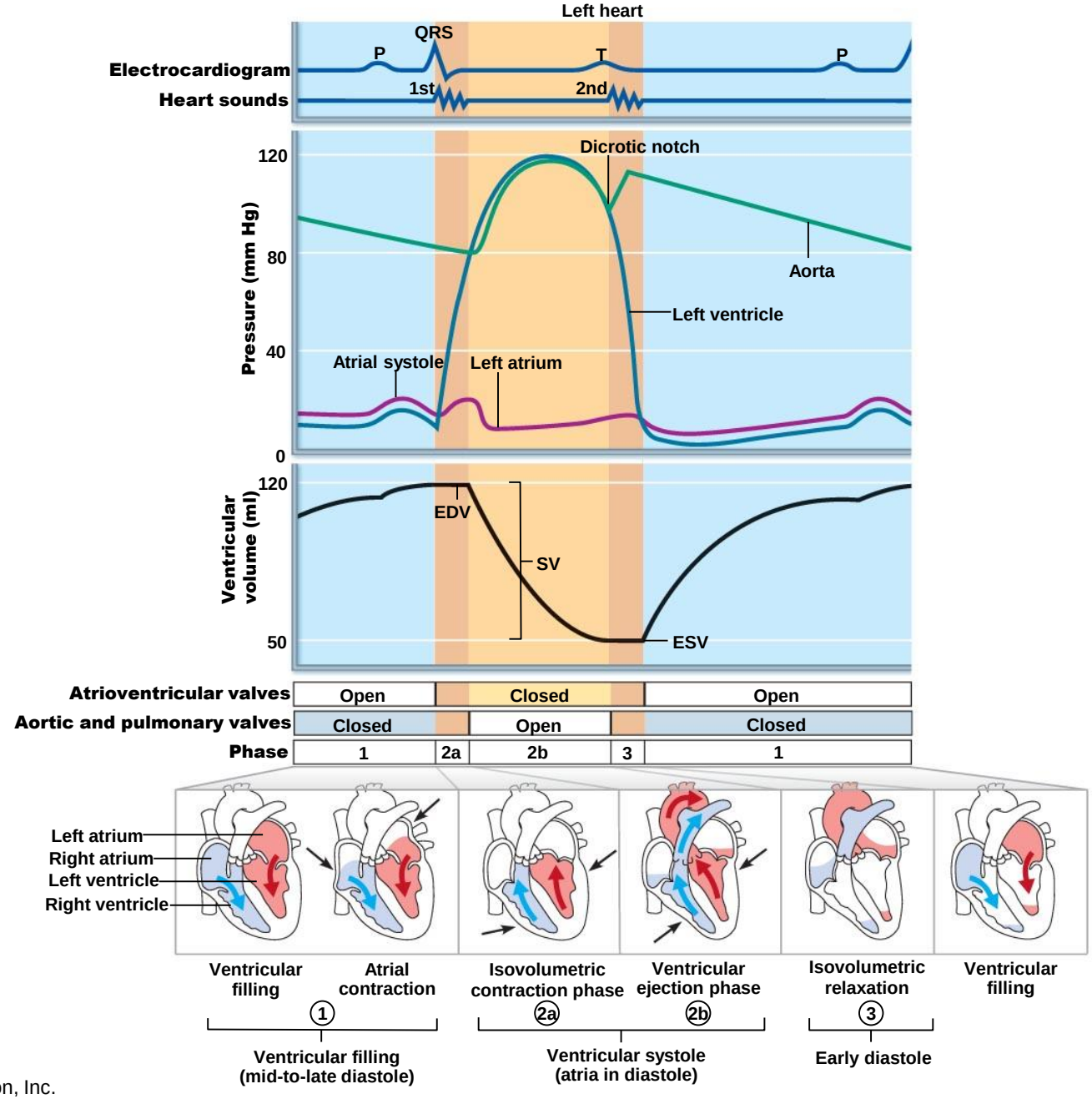
Phases of the Cardiac Cycle

- 2. Ventricular systole
  - Atria relax; ventricles begin to contract
  - Rising ventricular pressure → closing of AV valves
  - **Isovolumetric contraction phase** (all valves are closed)
  - In ejection phase, ventricular pressure exceeds pressure in large arteries, forcing SL valves open
  - **End systolic volume** (ESV): volume of blood remaining in each ventricle after systole

Phases of the Cardiac Cycle

- 3. **Isovolumetric relaxation** - early diastole
  - Ventricles relax; atria relaxed and filling
  - Backflow of blood in aorta and pulmonary trunk closes SL valves
    - Causes **dicrotic notch** (brief rise in aortic pressure as blood rebounds off closed valve)
    - Ventricles totally closed chambers
  - When atrial pressure exceeds that in ventricles → AV valves open; cycle begins again at step 1

Figure 18.21 Summary of events during the cardiac cycle.



Cardiac Output (CO)

- Volume of blood pumped by each ventricle in one minute
- **CO** = **heart rate** (**HR**) × **stroke volume** (**SV**)
  - HR = number of beats per minute
  - SV = volume of blood pumped out by one ventricle with each beat
- Normal – 5.25 L/min

Cardiac Output (CO)

- At rest
  - CO (ml/min) = HR (75 beats/min) × SV (70 ml/beat)
    = 5.25 L/min
  - CO increases if either/both SV or HR increased
  - Maximal CO is 4–5 times resting CO in nonathletic people
  - Maximal CO may reach 35 L/min in trained athletes
  - **Cardiac reserve** - difference between resting and maximal CO

Regulation of Stroke Volume

- SV = EDV – ESV
  - EDV affected by length of ventricular diastole and venous pressure
  - ESV affected by arterial BP and force of ventricular contraction
- Three main factors affect SV:
  - **Preload**
  - **Contractility**
  - **Afterload**

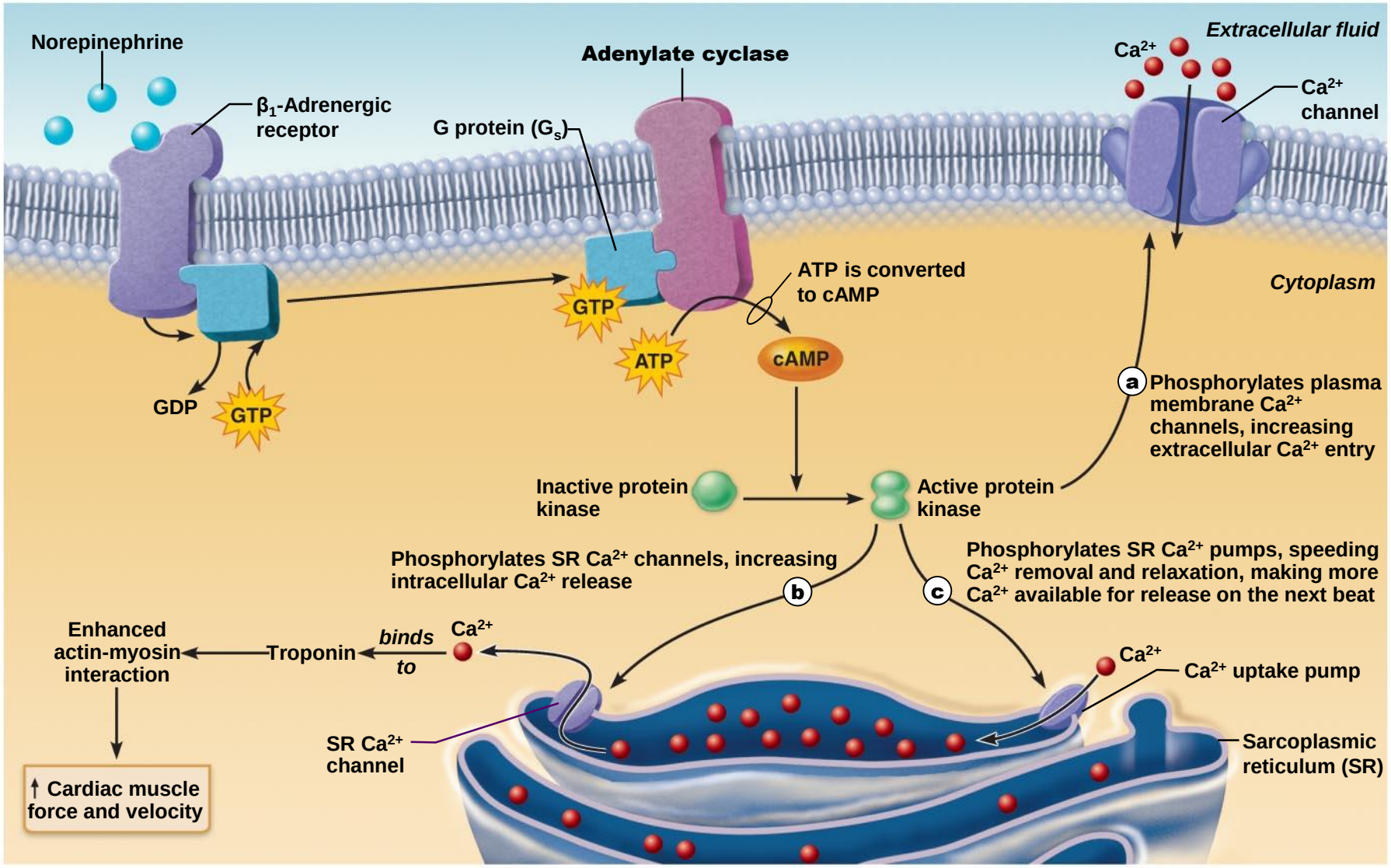
Regulation of Stroke Volume

- Preload: degree of stretch of cardiac muscle cells before they contract (**Frank-Starling law of heart**)
  - Cardiac muscle exhibits a length-tension relationship
  - At rest, cardiac muscle cells shorter than optimal length
  - Most important factor stretching cardiac muscle is **venous return** – amount of blood returning to heart
    - Slow heartbeat and exercise increase venous return
    - Increased venous return distends (stretches) ventricles and increases contraction force

Regulation of Stroke Volume

- Contractility—contractile strength at given muscle length, independent of muscle stretch and EDV
- Increased by
  - Sympathetic stimulation → increased Ca^{2+} influx → more cross bridges
  - Positive inotropic agents
    - Thyroxine, glucagon, epinephrine, digitalis, high extracellular Ca^{2+}
- Decreased by negative inotropic agents
  - Acidosis, increased extracellular K^{+}, calcium channel blockers

Figure 18.23 Norepinephrine increases heart contractility via a cyclic AMP secondmessenger system.



Regulation of Stroke Volume

- **Afterload** - pressure ventricles must overcome to eject blood
- Hypertension increases afterload, resulting in increased ESV and reduced SV

Regulation of Heart Rate

- Positive chronotropic factors increase heart rate
- Negative chronotropic factors decrease heart rate

Autonomic Nervous System Regulation

- Sympathetic nervous system activated by emotional or physical stressors
  - Norepinephrine causes pacemaker to fire more rapidly (and increases contractility)
    - Binds to β_1-adrenergic receptors → ↑ HR
    - ↑ contractility; faster relaxation
      - Offsets lower EDV due to decreased fill time

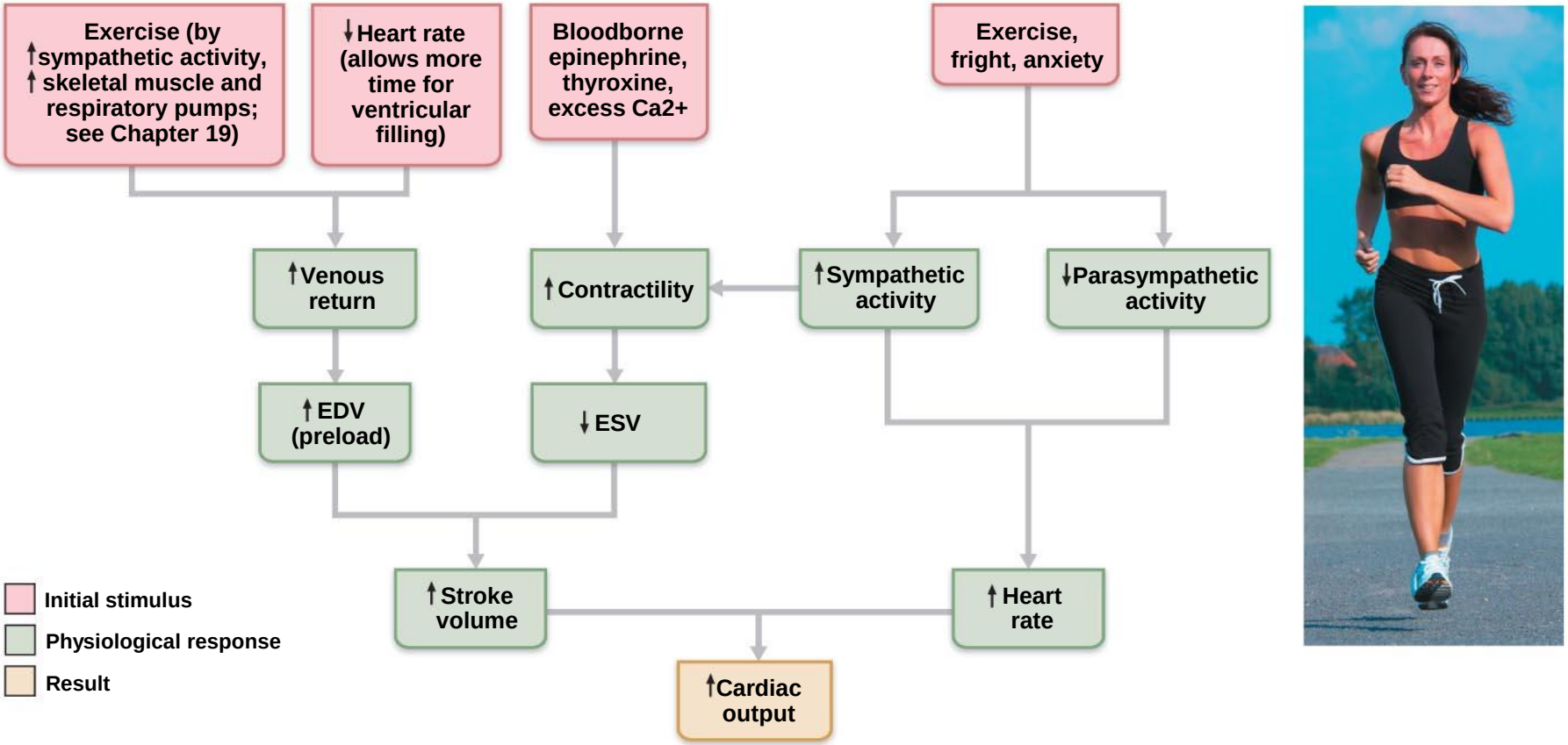
Autonomic Nervous System Regulation

- Parasympathetic nervous system opposes sympathetic effects
  - Acetylcholine hyperpolarizes pacemaker cells by opening K^+ channels → slower HR
  - Little to no effect on contractility
- Heart at rest exhibits **vagal tone**
  - Parasympathetic dominant influence

Autonomic Nervous System Regulation

- Atrial (Bainbridge) reflex - sympathetic reflex initiated by increased venous return, hence increased atrial filling
  - Stretch of atrial walls stimulates SA node → ↑ HR
  - Also stimulates atrial stretch receptors, activating sympathetic reflexes

Figure 18.22 Factors involved in determining cardiac output.



Chemical Regulation of Heart Rate

- Hormones
  - Epinephrine from adrenal medulla increases heart rate and contractility
  - Thyroxine increases heart rate; enhances effects of norepinephrine and epinephrine
- Intra- and extracellular ion concentrations (e.g., Ca^{2+} and K^{+}) must be maintained for normal heart function

Homeostatic Imbalance

- **Hypocalcemia** → depresses heart
- **Hypercalcemia** → increased HR and contractility
- **Hyperkalemia** → alters electrical activity → heart block and cardiac arrest
- **Hypokalemia** → feeble heartbeat; arrhythmias

Other Factors that Influence Heart Rate

- Age
  - Fetus has fastest HR
- Gender
  - Females faster than males
- Exercise
  - Increases HR
- Body temperature
  - Increases with increased temperature

Homeostatic Imbalances

- **Tachycardia** - abnormally fast heart rate (>100 beats/min)
  - If persistent, may lead to fibrillation
- **Bradycardia** - heart rate slower than 60 beats/min
  - May result in grossly inadequate blood circulation in nonathletes
  - May be desirable result of endurance training

Homeostatic Imbalance

- **Congestive heart failure (CHF)**
  - Progressive condition; CO is so low that blood circulation inadequate to meet tissue needs
  - Reflects weakened myocardium caused by
    - Coronary atherosclerosis—clogged arteries
    - Persistent high blood pressure
    - Multiple myocardial infarcts
    - Dilated cardiomyopathy (DCM)

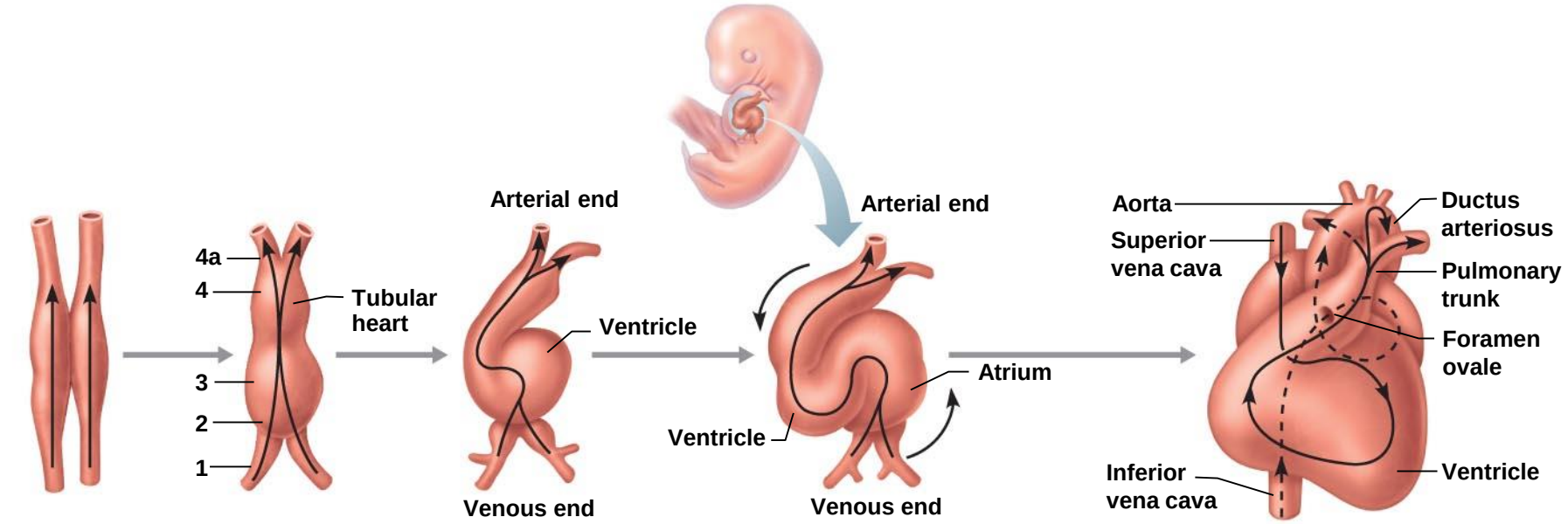
Homeostatic Imbalance

- Pulmonary congestion
  - Left side fails → blood backs up in lungs
- Peripheral congestion
  - Right side fails → blood pools in body organs → edema
- Failure of either side ultimately weakens other
- Treat by removing fluid, reducing afterload, increasing contractility

Developmental Aspects of the Heart

- Embryonic heart chambers
  - Sinus venosus
  - Atrium
  - Ventricle
  - Bulbus cordis

Figure 18.24 Development of the human heart.



(a) Day 20: Endothelial tubes begin to fuse.

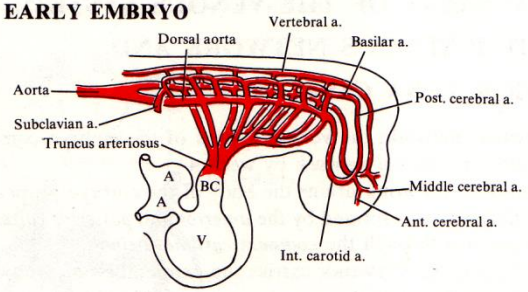
(b) Day 22: Heart starts pumping.

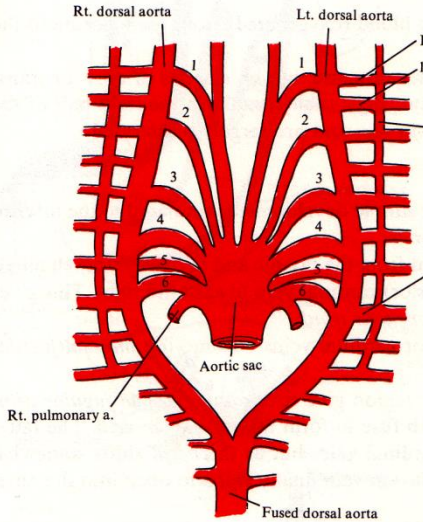
(c) Day 24: Heart continues to elongate and starts to bend.

(d) Day 28: Bending continues as ventricle moves caudally and atrium moves cranially.

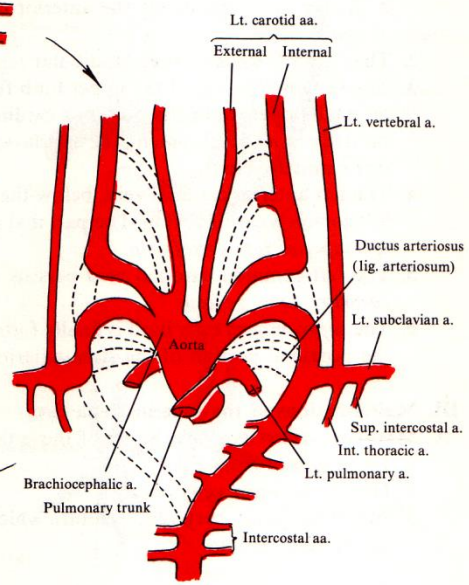
(e) Day 35: Bending is complete.

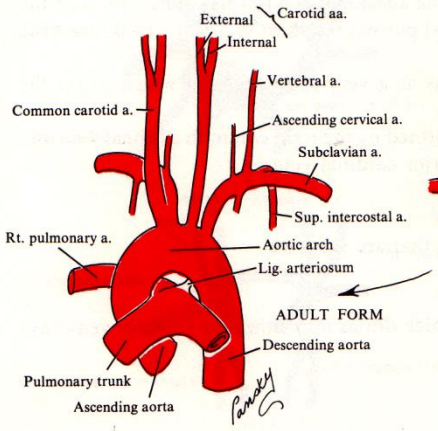
DEVELOPING ARTERIES IN HEAD REGION OF EARLY EMBRYO





BRANCHIAL (PHARYNGEAL) ARCH ARTERIES





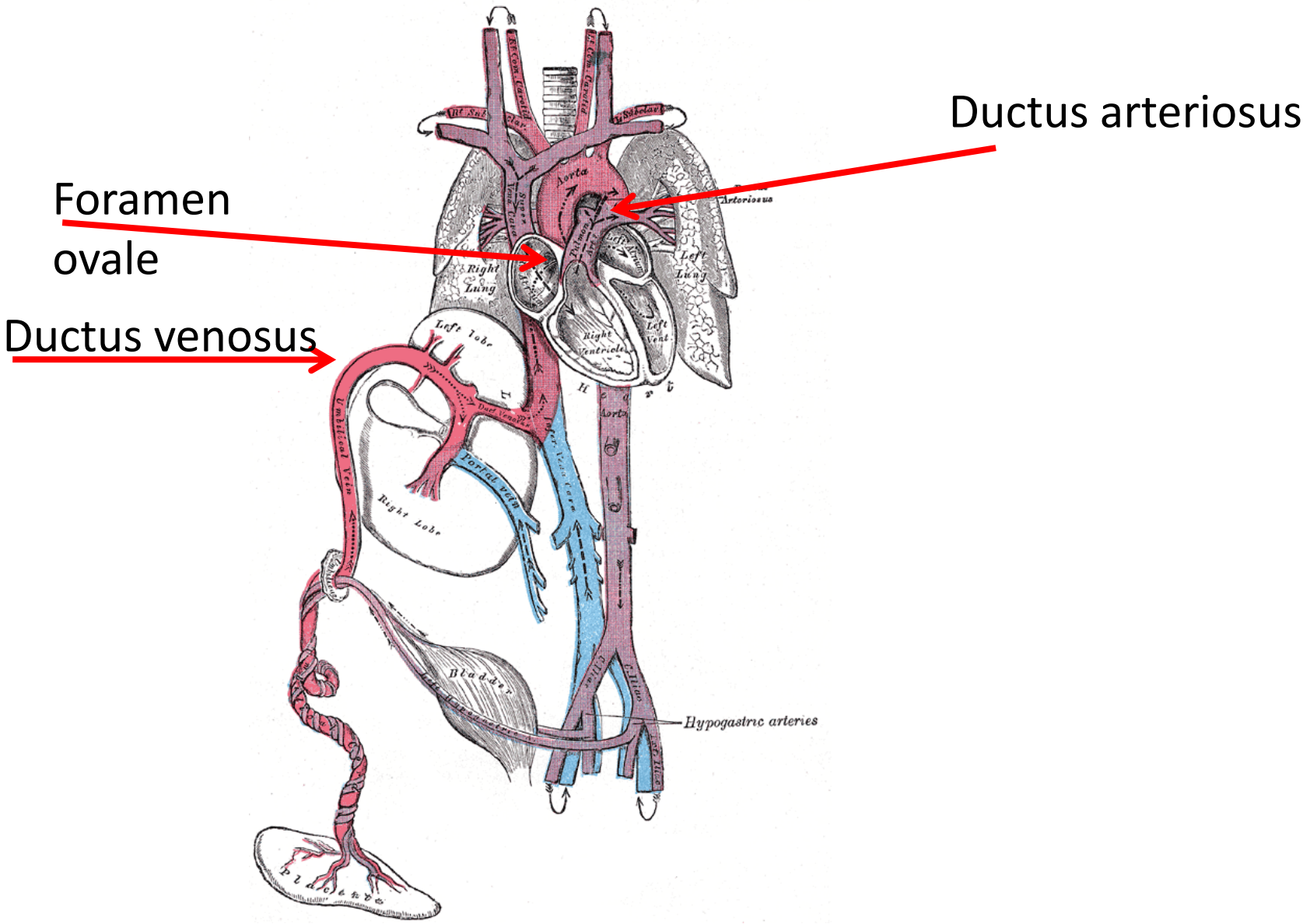
Developmental Aspects of the Heart

- Fetal heart structures that bypass pulmonary circulation
  - **Foramen ovale** connects two atria
    - Remnant is **fossa ovalis** in adult
  - **Ductus arteriosus** connects pulmonary trunk to aorta
    - Remnant - **ligamentum arteriosum** in adult
  - Close at or shortly after birth

Developmental Aspects of the Heart

- Congenital heart defects
  - Most common birth defects; treated with surgery
  - Most are one of two types:
    - Mixing of oxygen-poor and oxygen-rich blood, e.g., septal defects, patent ductus arteriosus
    - Narrowed valves or vessels → increased workload on heart, e.g., coarctation of aorta
  - **Tetralogy of Fallot**
    - Both types of disorders present

Fetus Heart: Before & After Birth



Sounds

Normal

http://www.easyauscultation.com/cases-listing-details.aspx?caseID=7

Ventricular septal defect

Indomethacin

In humans, postnatal indomethacin can cause closure of the ductus arteriosus, and is used therapeutically when this structure remains patent in preterm neonates (Heymann et al., ’76).

Ductal constriction can also occur in utero after maternal indomethacin administration (Moise et al., ’88).

In addition, infants exposed to prenatal indomethacin were more likely to require surgical ligation of their PDA due to either a lack of response to postnatal indomethacin or a reopening of the duct after initial closure.

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The human heart beats more than 3.5 billion times in an average lifetime.

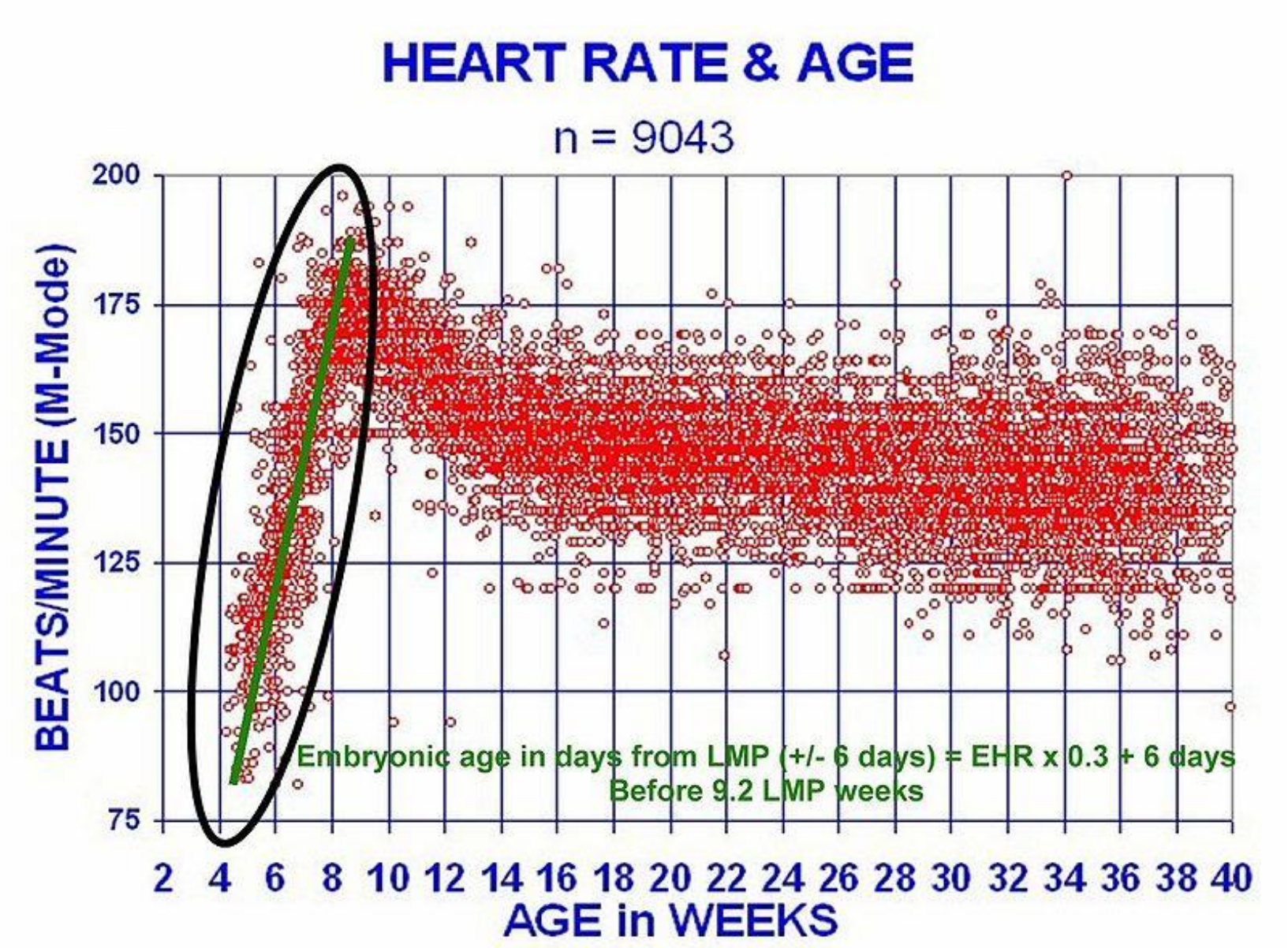
The human embryonic heart begins beating approximately 21 days after conception.

The human heart begins beating at a rate near the mother's, about 75-80 BPM.

The embryonic heart rate (EHR) then accelerates linearly for the first month of beating, peaking at 165-185 BPM during the early 7th week. This acceleration is approximately 3.3 BPM per day, or about 10 BPM every three days
(**increase of 100 BPM in the first month**).

$$\text{Age in days} = \text{EHR}(0.3) + 6$$

After peaking at about 9.2 weeks after the normal menstrual period (LMP), it decelerates to about 150 BPM (+/-25 BPM) during the 15th week after the LMP.



Terry J. DuBose, M.S., RDMS; Director Diagnostic Medical Sonography Program

Congenital Heart Diseases: Neonate & Young Infant

- **Significant congenital heart disease (CHD) may be diagnosed at virtually any age.**

- **Some conditions always are discovered in neonates; others rarely are identified during infancy.**

Table 1. **Mendelian Gene Syndromes Associated with Congenital Heart Anomalies***

Etiologic Syndrome	Frequency of Cardiac Anomalies† All (%)	Distinctive or Most Common	Distinguishing Features
Autosomal Dominant			
Adams-Oliver syndrome	20	Left-sided obstruction (eg, COA, parachute MVP), TOF	Scalp cutis aplasia, terminal transverse limb defects
Alagille syndrome	95	(P)PS, TOF/TOF with PA, ASD, VSD	Bile duct paucity, chronic cholestasis, butterfly vertebrae, posterior embryotoxon
Char syndrome	60	PDA	Anomalies on fifth finger, supernumerary nipple
Cornelia de Lange syndrome	25	VSD, ASD, PS, TOF	Upper limb deficiency, GI anomalies
Holt-Oram syndrome	80	ASD ± other CVM, VSD, TA, TOF, PAPVC, conduction defect	Upper limb malformations
Neurofibromatosis	2	PSV, ASV, COA, HCM	Café au lait macules, optic glioma, scoliosis, pseudarthrosis, neurofibromas
Noonan syndrome	85	PSV, ASD, AVSD partial, COA, HCM	Short, webbed neck; pectus deformity; cryptorchidism
Rubinstein-Taybi syndrome	35	PDA, ASD, VSD, left-sided obstruction (eg, COA, HLHS)	Broad thumbs and great toes
Williams syndrome	60	SVAS, PS, other left-sided obstructions (eg, ASV, MS, COA)	Hypercalcemia, hypodontia, hypoplastic nails
Autosomal Recessive			
Ellis-van Creveld syndrome	60	AVSD, common atrium, ASD primum	Short limbs, polydactyly, hypoplastic nails, dental anomalies
Fryns syndrome	50	ASD, VSD, conotruncal	Diaphragmatic hernia, distal digital hypoplasia
Keutel syndrome	70	(P)PS	Short digits, mixed hearing loss, cartilage calcification
Smith-Lemli-Opitz syndrome	45	ASD, VSD, complete AVSD, TAPVC	Two- to three-toe syndactyly, cleft palate, lung anomalies, genital anomalies
X-linked Recessive			
Simpson-Golabi-Behmel syndrome	25	ASD; VSD; rare, variable cardiomyopathy	Macrosomia, cleft palate, supernumerary nipples, hernias, hypospadias, poly/syndactyly
Suspected Gene Etiology			
Cardio-facio-cutaneous syndrome	75	ASD, HCM	Sparse, curly hair; low, rotated ears; hyperkeratosis
Hall-Hittner syndrome (CHARGE association)	80	Conotruncal/arch, assorted CVMs	Coloboma, choanal atresia, genital anomalies, ear anomalies
Costello syndrome	60	MVP, AV, thickening HCM, arrhythmia (atrial tachycardia)	Skin/joint laxity, fine/curly hair, deep palm creases, ulnar deviation, papillomata
PHACES syndrome	100	COA; IAA, A right; double, cervical aortic arch	Posterior fossa malformations, hemangiomas, eye anomalies
Ritscher-Schinzel syndrome (3C)	100	TOF, DORV, AVSD	Posterior fossa malformations, cleft palate, coloboma

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Table 2. **Incidence of Most Common Cardiac Malformations***

Malformation	Prevalence (per 10,000 Births)	
	Metropolitan Atlanta Congenital Defect Program, 1995–1997	Baltimore–Washington Infant Study, 1981–1989
Heterotaxy, L-TGA	1.6	1.4
Outflow tract defects, total		
Tetralogy of Fallot	4.7	3.3
D-TGA	2.4	2.3
Double-outlet right ventricle	2.2	0.7
Truncus arteriosus	0.6	0.5
Atrioventricular septal defect		
With Down syndrome	2.4	2.3
Without Down syndrome	1	1
Ebstein anomaly	0.6	0.6
Total APVC	0.6	0.7
Right-sided obstruction		
Peripheral pulmonic stenosis	7	Not available
Pulmonic stenosis, atresia	5.9	5.4
Pulmonic atresia/intact septum	0.6	0.6
Tricuspid atresia	0.3	0.4
Left-sided obstruction		
Coarctation of the aorta	3.5	1.4
Hypoplastic left heart	2.1	1.8
Aortic valve stenosis	0.8	0.8
Aortic arch atresia or hypoplasia	0.6	Not available
Septal defects		
Ventricular septal defect	24.9	11.2
Atrial septal defect	10	3.2
Patent ductus arteriosus	8.1	0.9
Other major heart defects	9.7	–
Total	90.2	48.4

APVC=anomalous pulmonary venous connection, TGA=transposition of the great arteries.
*Reprinted from Lin AE, Holly HA. Genetic epidemiology of cardiovascular malformations. *Progr Pediatr Cardiol.* 2005;20:113–126 with permission from Elsevier.

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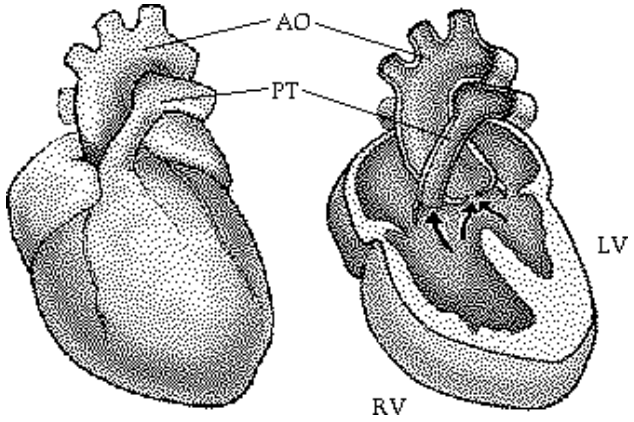
Tetralogy of Fallot



This condition results from a single error: the conus septum develops too far anteriorly giving rise to two unequally proportioned vessels- - a large aorta and a smaller stenotic pulmonary trunk.

The four main characteristics of Tetralogy of Fallot are:
(1) pulmonary stenosis
(2) ventricular septal defect (VSD) of the membranous portion (the septum is displaced too far anteriorly to contribute to the septum)
(3) overriding aorta (the aorta straddles the VSD)

Tricuspid Atresia:
Total Correction: mortality less than 3%

Transposition of the great arteries
Total Correction: mortality less than 2%

Pulmonic stenosis
Total Correction: mortality less than 1%

Truncus Arteriosus (various types)
Mortality is > 10%

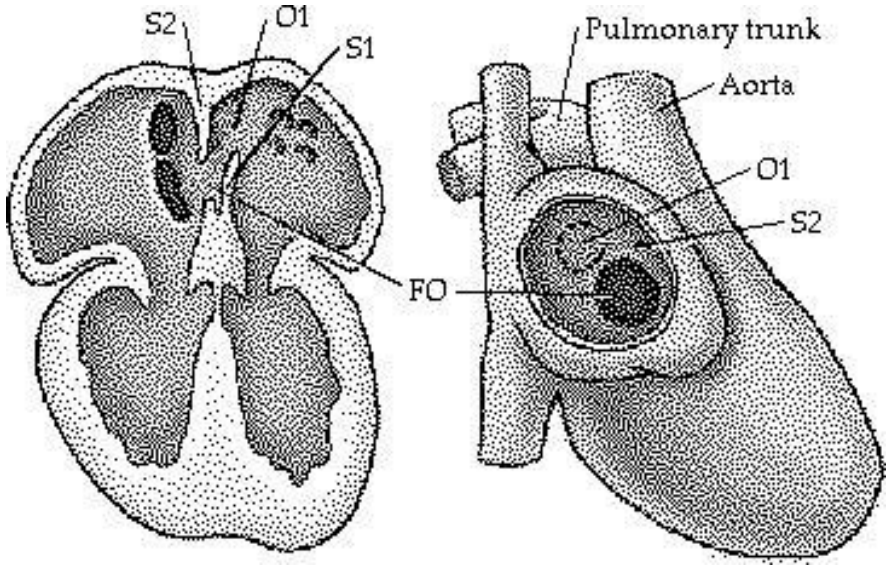
Hypoplastic left heart syndrome
Mortality ~10%

Perry et al. 2010. Maternal Child Nursing Care, 4th Edition. Chapter 48

Septal defects

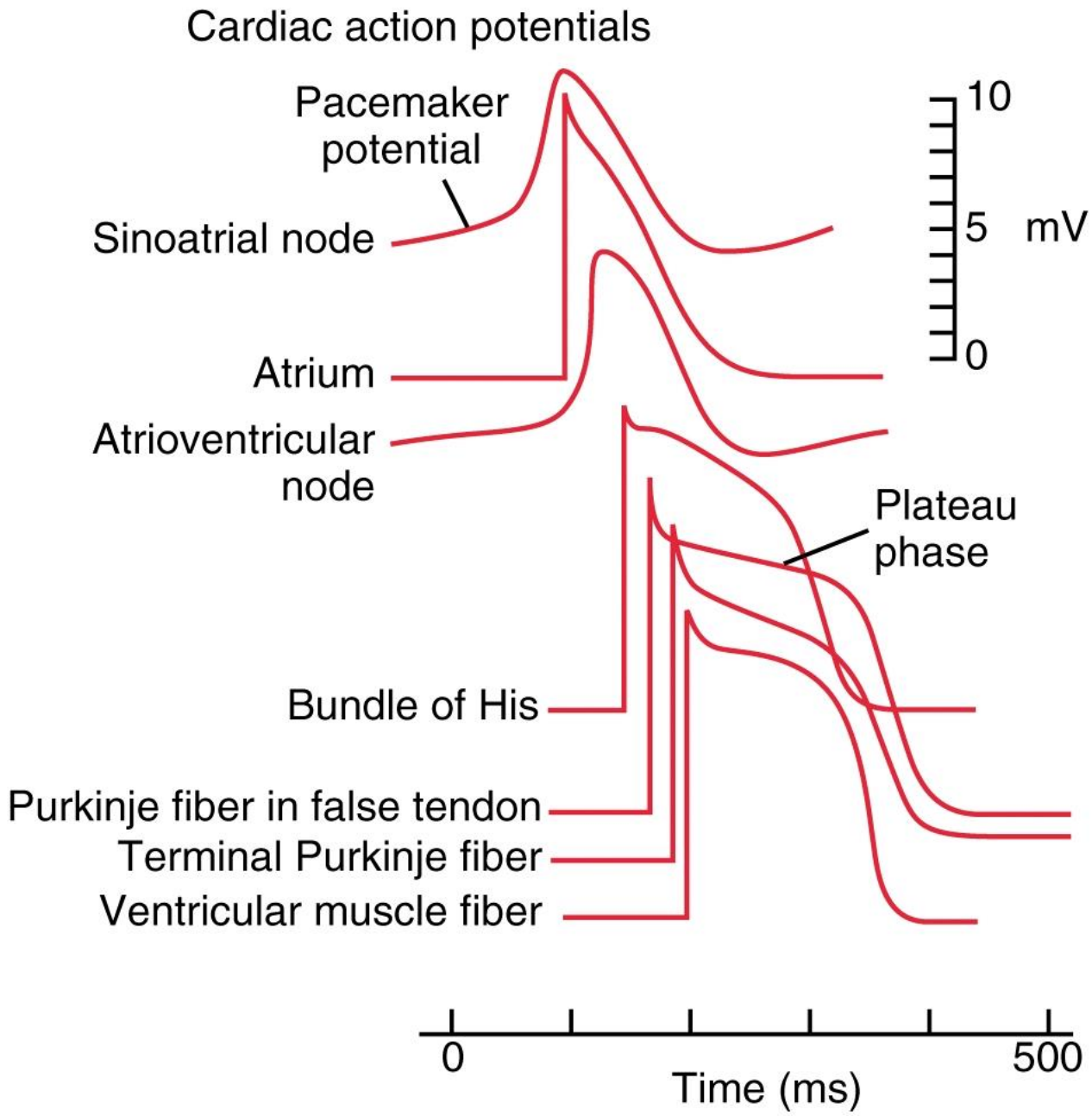
At the end of the seventh week -final stage of development.
Fetus does not use its lungs, most of the blood is diverted to the systemic circulation. Foramen ovale and the septum primum
At birth the child will use its lungs for the first time: more blood pulmonary circulation.
The pressure increase in the left atrium will force septum primum to wall- two septa fuse

(common atrial septum)
O1 = Ostium primum
S1 = Septum primum
FO = Foramen ovale
S2 = Septum secundum

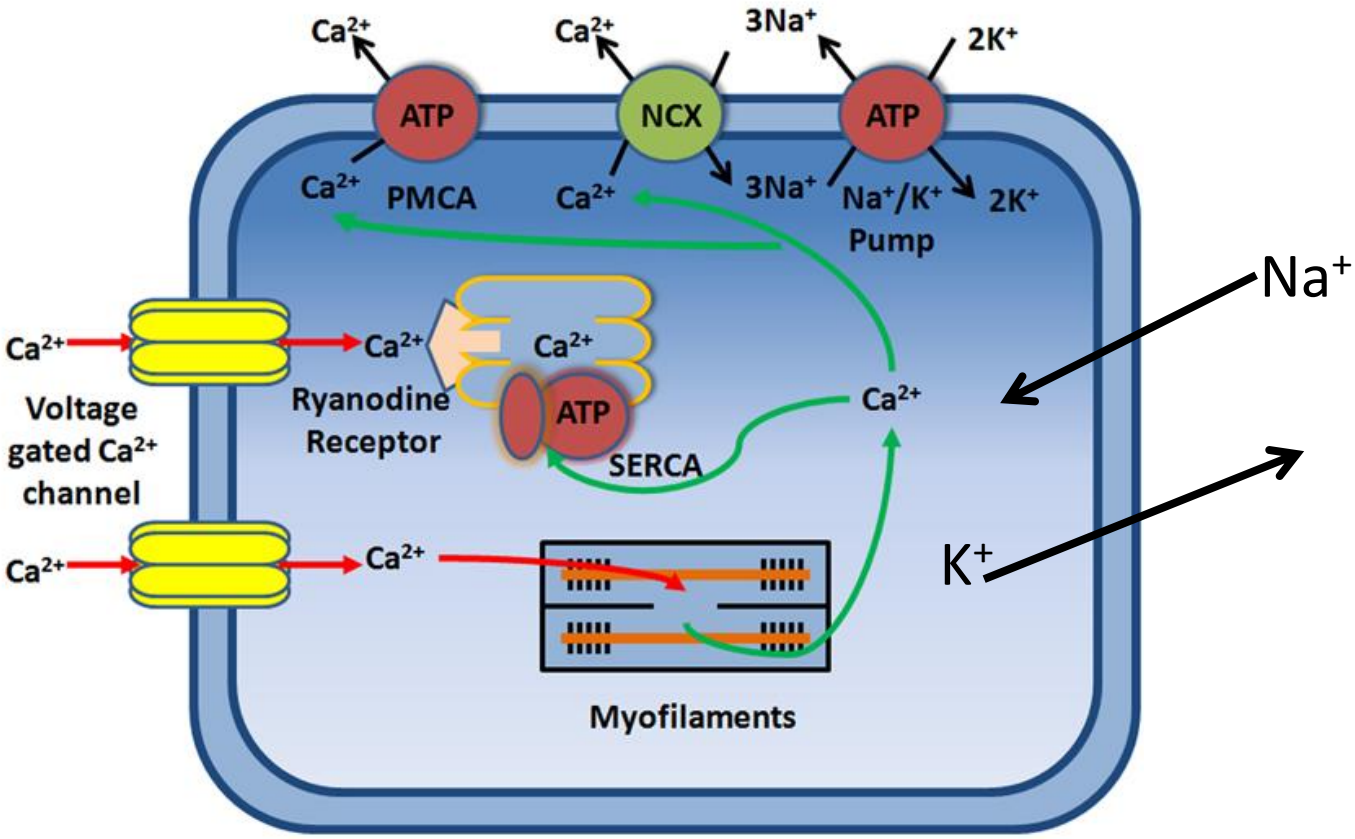


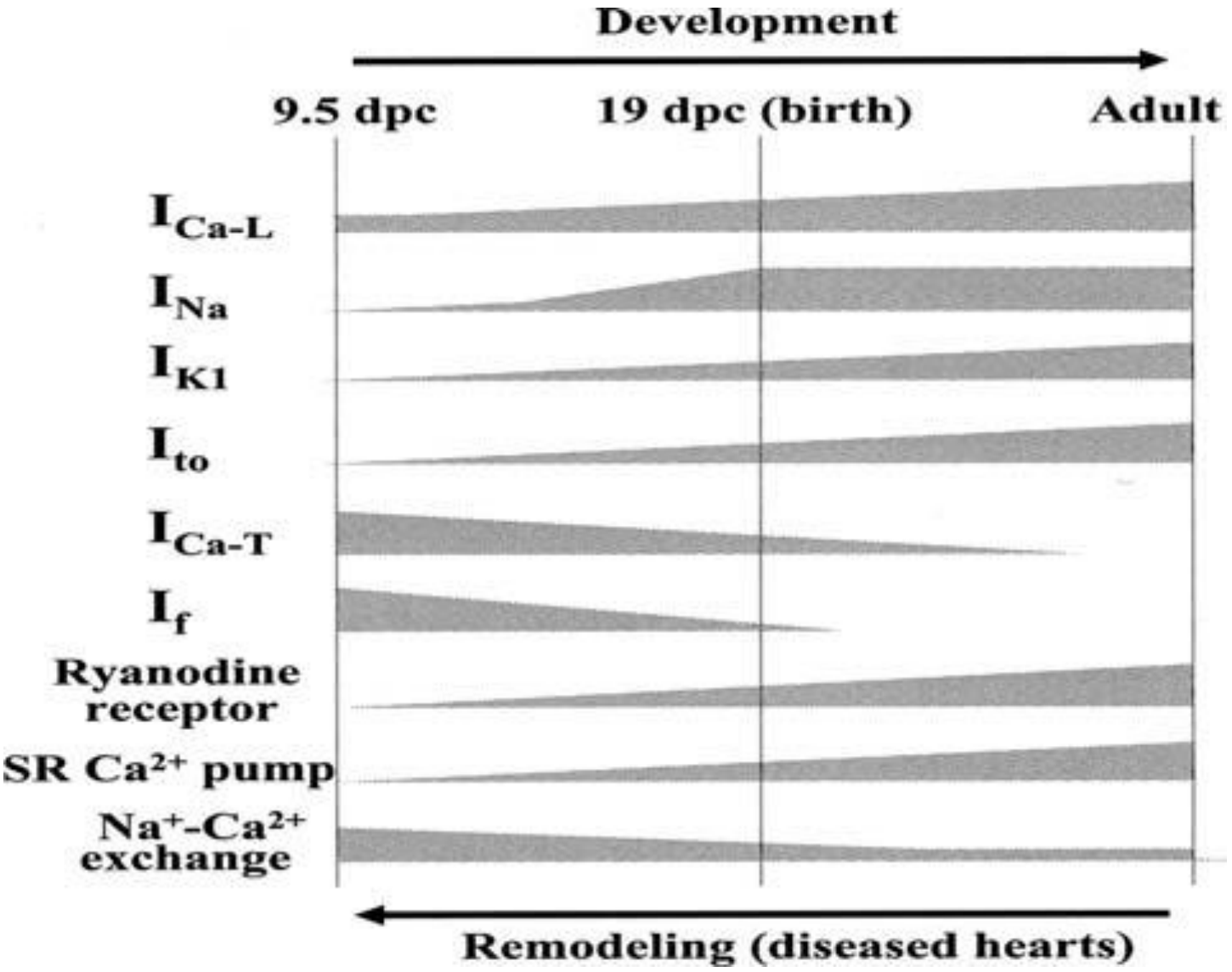
ECG: Neonate

- Arrhythmias in fetuses and newborns are relatively common, occurring in up to 90% of newborns and in 1% to 3% of pregnancies *(NeoReviews Vol.9 No.6 2008 e242,2008 American Academy of Pediatrics, Fetal and Neonatal Arrhythmias)*
- Weak arterial pulses and right heart overload in the **electrocardigram** suggested the diagnosis of hypoplasia of the left heart. Impaired coronary perfusion to portions of the right and left ventricular myocardium. Pulmonary vasoconstriction from hypoxia. Myocardial ischemia on the **electrocardiogram** ***(The Journal of Pediatrics Volume 81 (2): 243-250)***
- SIDS: A prolonged QT interval may be an important cause for SIDS. *(Schwartz et al., The New England Journal of Medicine, 1998 338(24):1709-1714.*


Cardiac action potentials
Pacemaker potential
Sinoatrial node
Atrium
Atrioventricular node
Bundle of His
Purkinje fiber in false tendon
Terminal Purkinje fiber
Ventricular muscle fiber
Plateau phase
10
5
mV
0
0
500
Time (ms)

MUSCLE CONTRACTION





Brugada syndrome : Genetic disease, abnormal ECG sudden cardiac death (Sudden Unexpected Death Syndrome -SUDS). First described in 1992, ventricular fibrillation mutation in Na+ ion channel

Ion channels

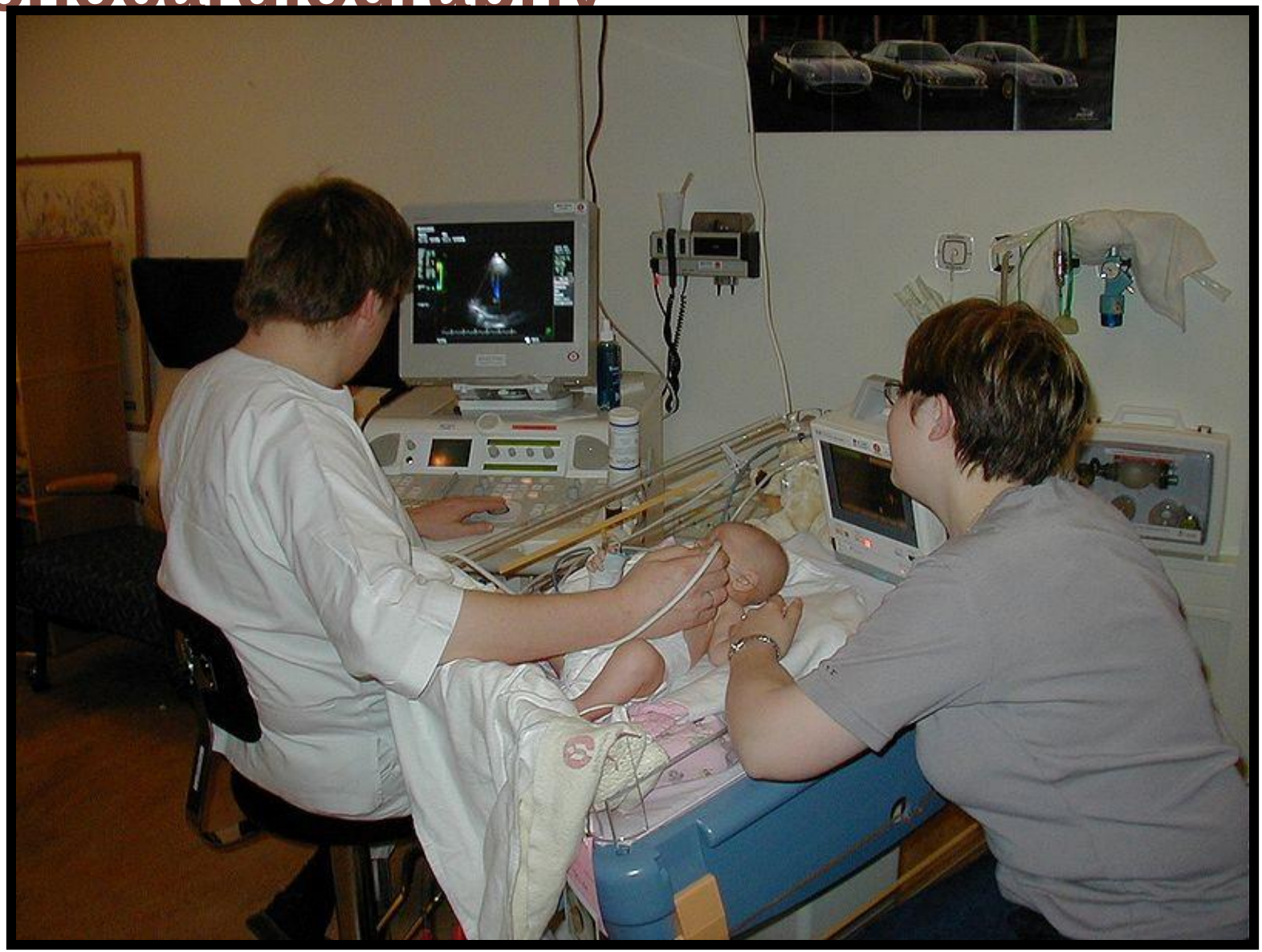
- Recent evidence indicates that between 5 and 15% of SIDS cases carry potentially lethal **loss-of-function mutations in cardiac channelopathy genes**.

(Future Cardiol. 2009 Mar;5(2):201-7. Sudden infant death syndrome and cardiac arrhythmias. Morris JA, Harrison L, Brodison A, Lauder R. Department of Pathology, Royal Lancaster Infirmary, Lancaster LA1 4RP, UK.)

- Morphological changes in the mitochondrial network likely accompany the uncoupling with mitochondrial fission dampening the signals leading to **cardiomyocyte death**.

(J Bioenerg Biomembr. 2009 Apr;41(2):133-6. Uncouple my heart: the benefits of inefficiency.

Sonographer: Pediatric echocardiography



Age-Related Changes Affecting the Heart

- Sclerosis and thickening of valve flaps
- Decline in cardiac reserve
- Fibrosis of cardiac muscle
- Atherosclerosis