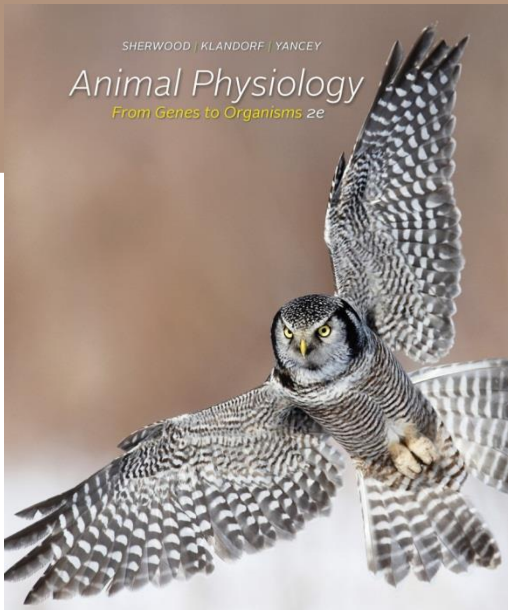

Lauralee Sherwood
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Chapter 9
Circulatory Systems

Kip McGilliard • Eastern Illinois University

9.1 Evolution of Circulation

- Circulatory systems in multicellular organisms supplement **diffusion** with **bulk transport**

- Components of a circulatory system

  - **Fluid** that carries transported molecules and cells

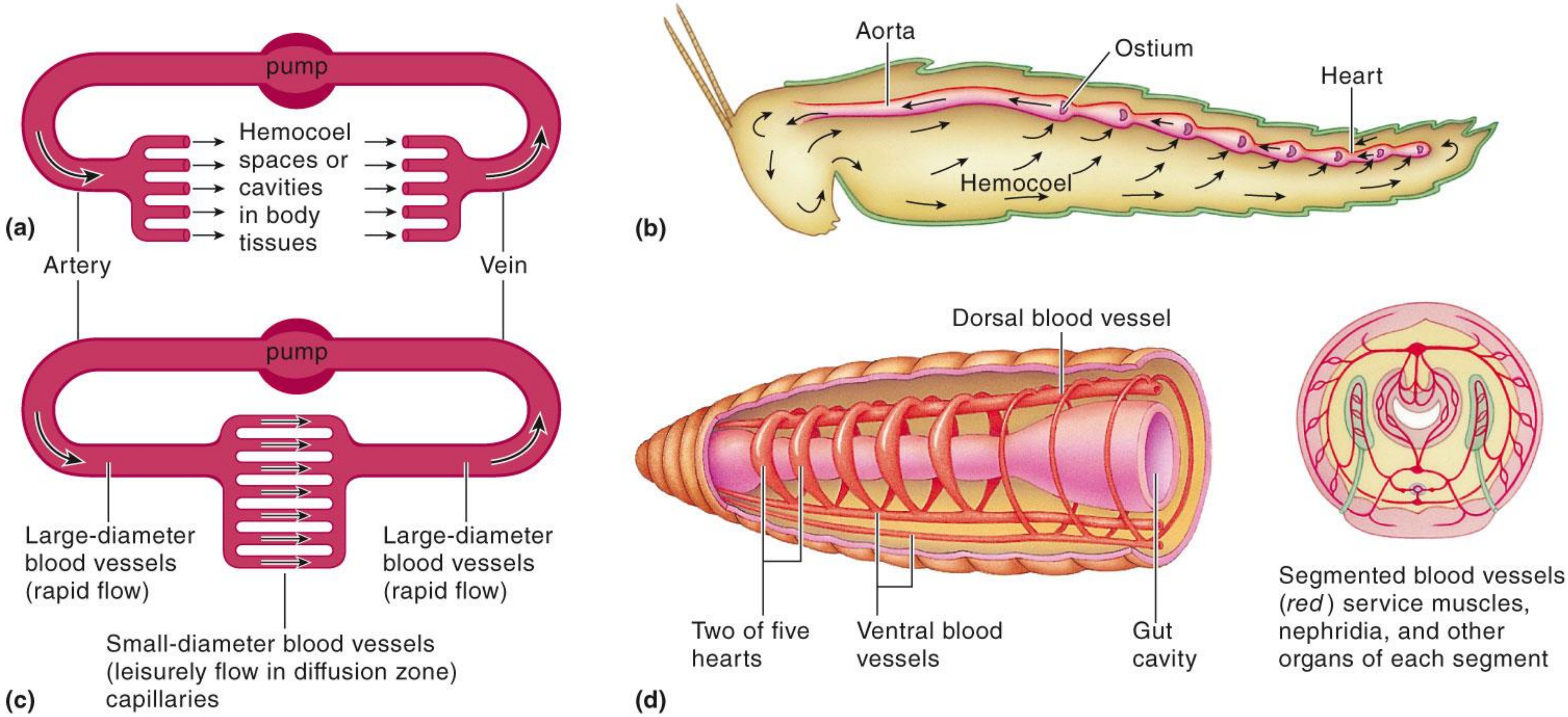
  - A **pump** to move the fluids

  - **Vessels** to carry fluid between the pump and body tissues

9.1 Evolution of Circulation

- Open vs. closed circulatory systems
  - **Open systems**
    - **Hemolymph** moves through vessels that open into extracellular spaces
  - **Closed systems**
    - **Blood** is pumped from a heart through vessels that return blood to the heart
    - **Capillaries** are the primary structure distinguishing a closed from an open system

9.1 Evolution of Circulation



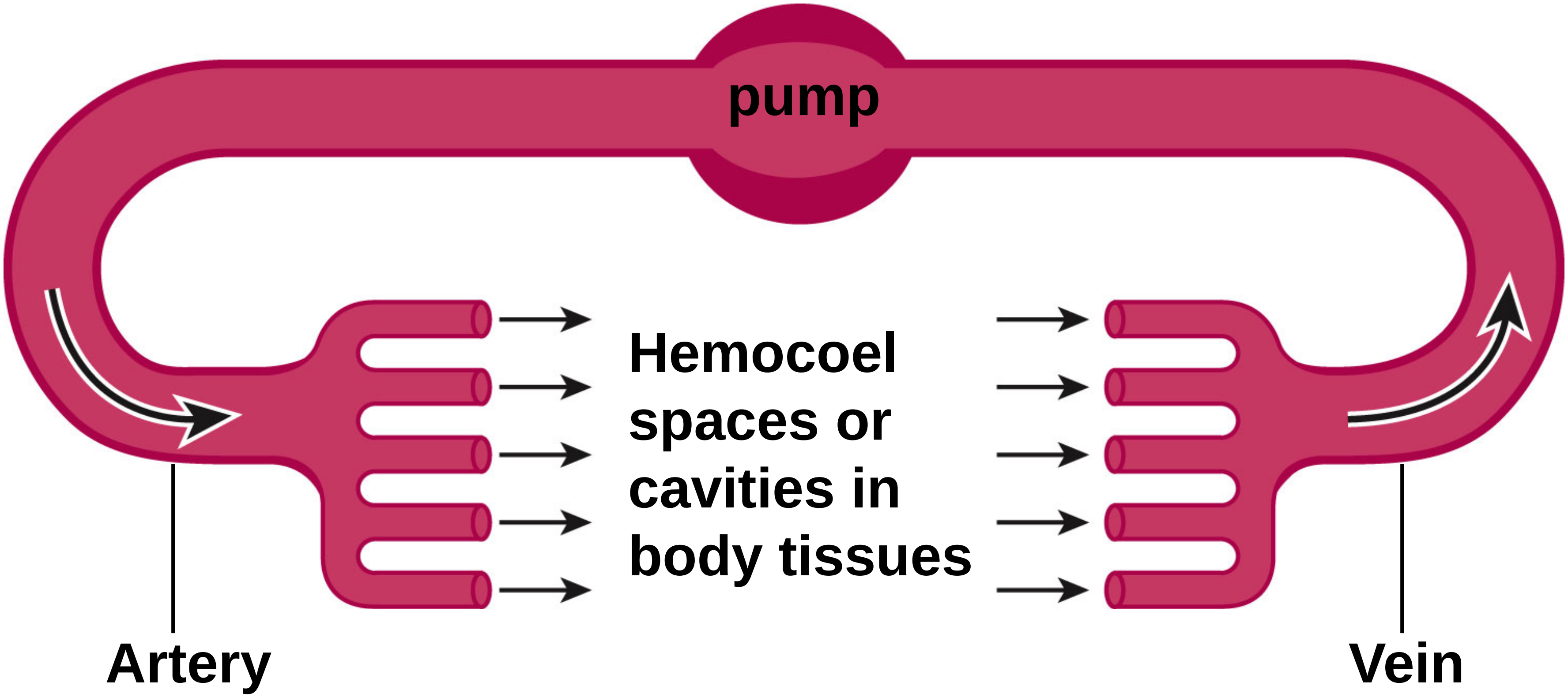


Figure 9-2a p387

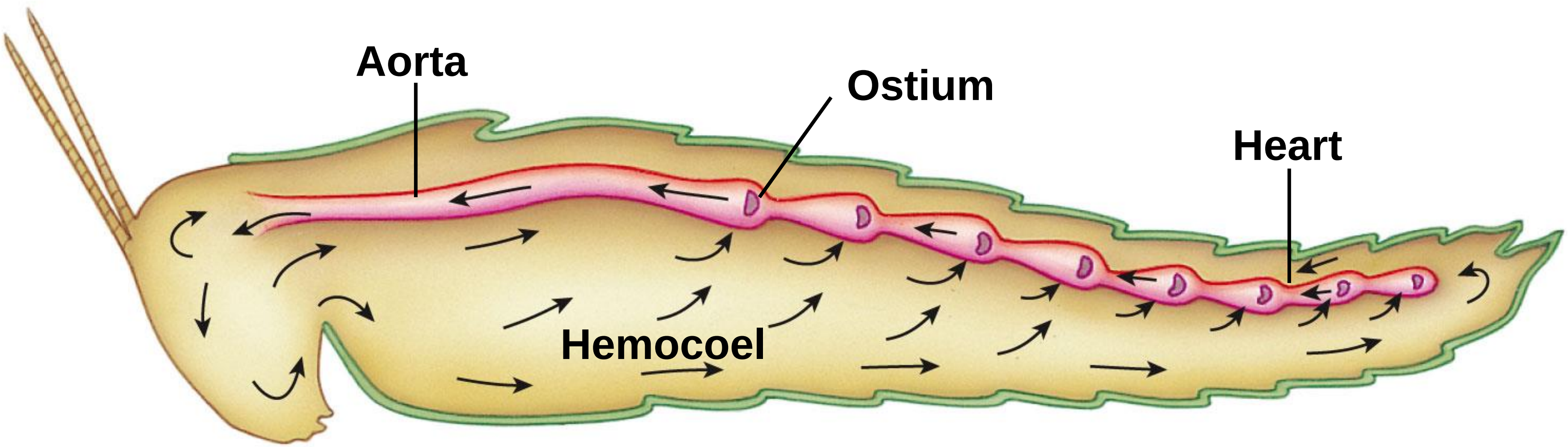


Figure 9-2b p387

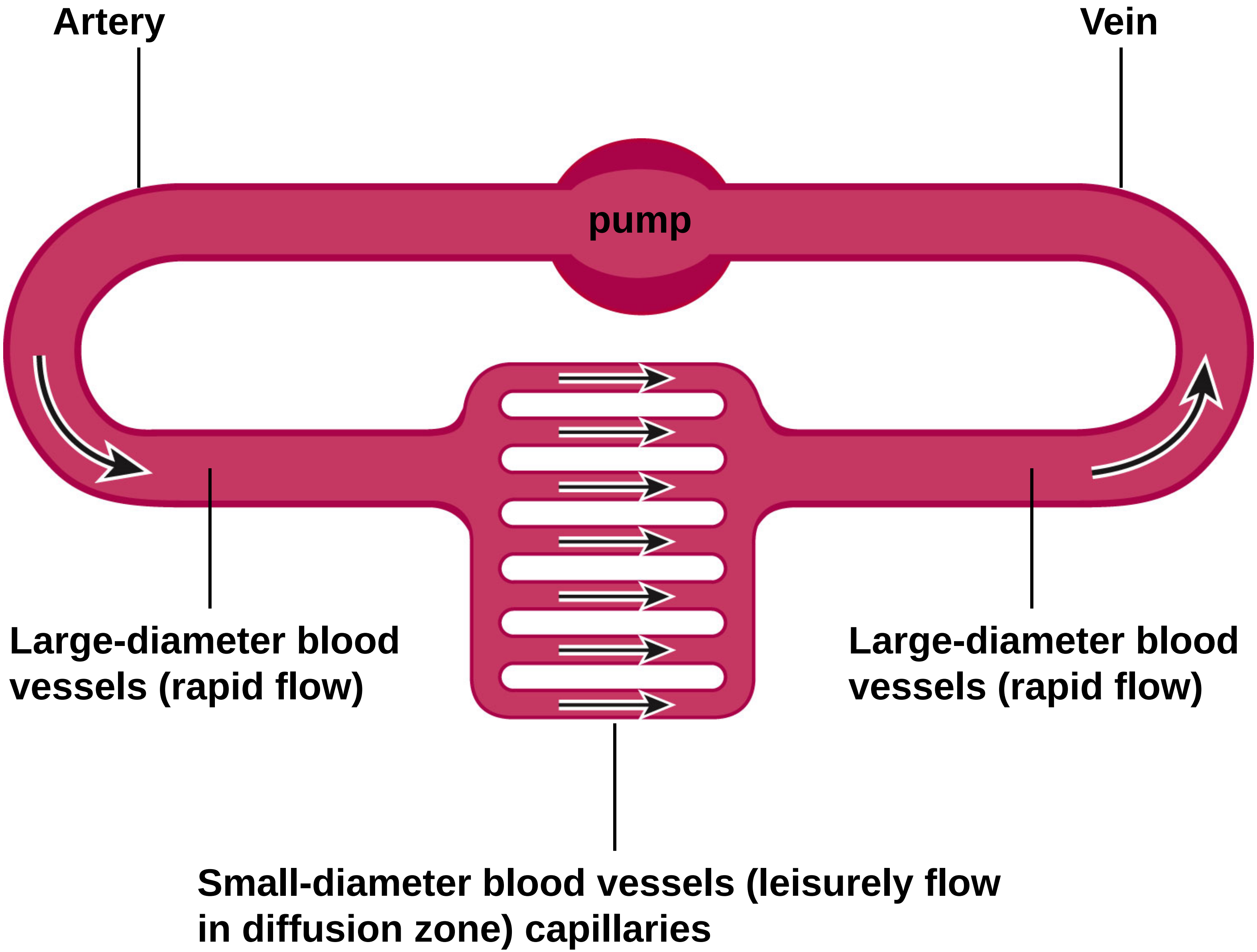
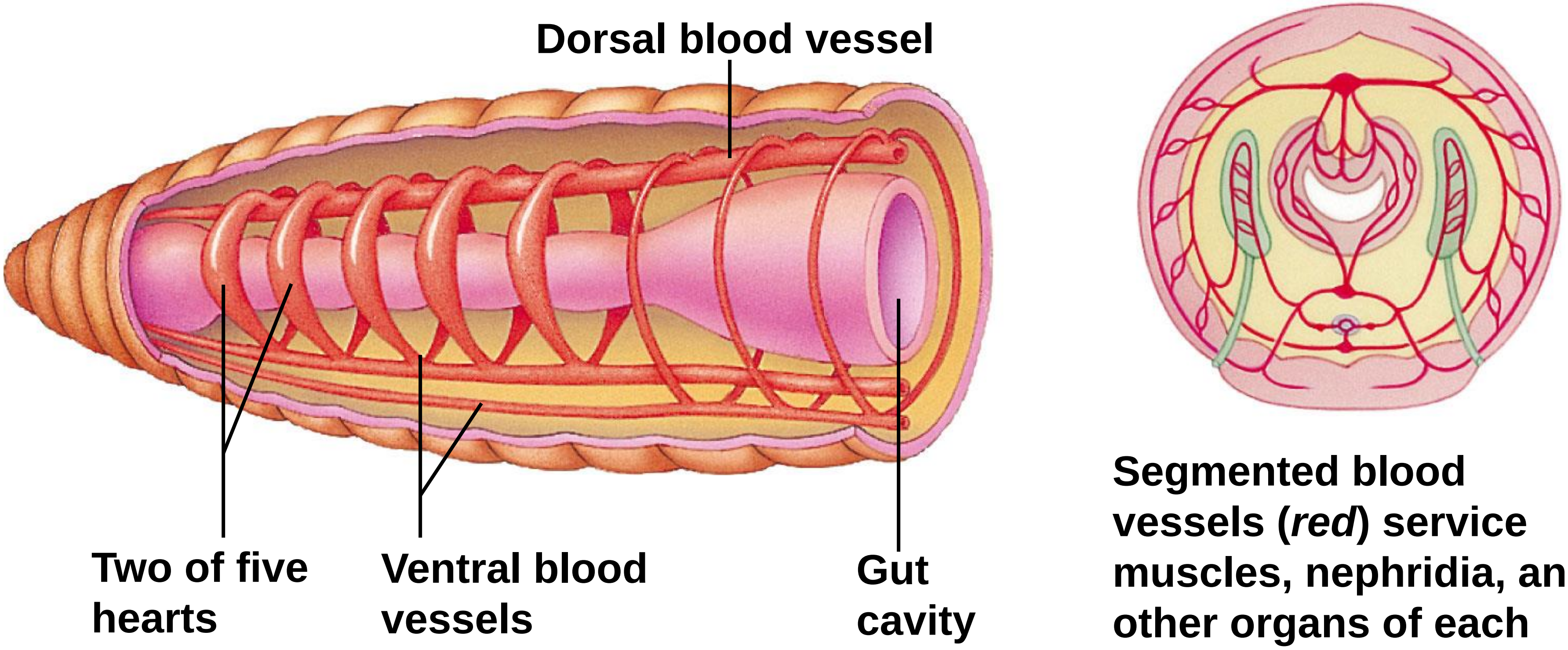


Figure 9-2c p387



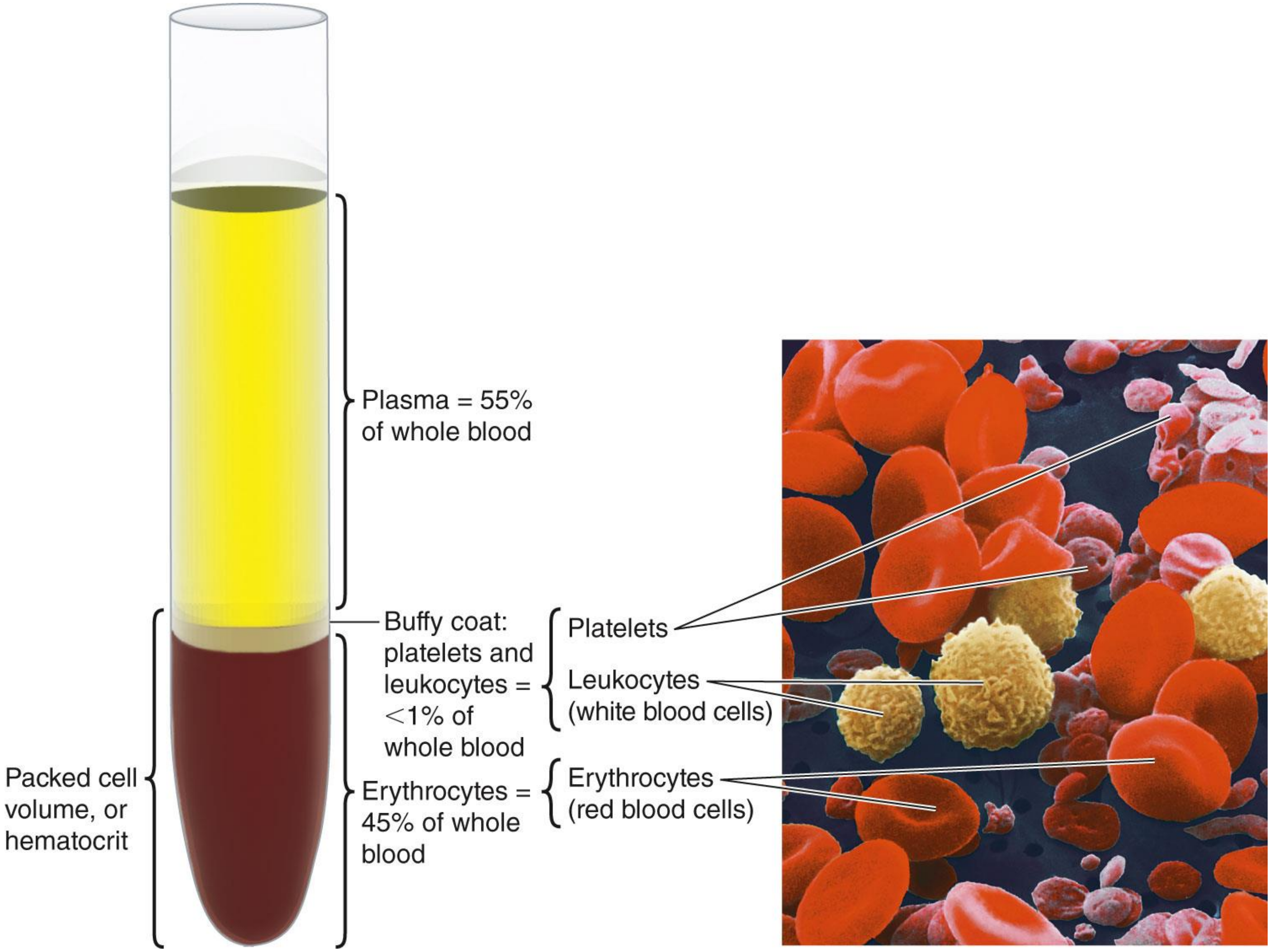
Segmented blood vessels (*red*) service muscles, nephridia, and other organs of each segment

Figure 9-2d p387

9.2 Circulatory Fluids and Cells

- Circulatory fluids are divided into plasma and cellular elements
  - **Hematocrit** is the percentage of blood volume occupied by cells
    - 45% in human males, 42% in human females
  - **Plasma** is an aqueous medium for transport of inorganic ions, gases and organic solutes
    - 90% **water**
    - **Plasma proteins** are the most plentiful organic solutes (6 - 8% of plasma)
    - **Lipoproteins** carry **energy lipids** (triglycerides) and **structural lipids** (phospholipids and cholesterol)

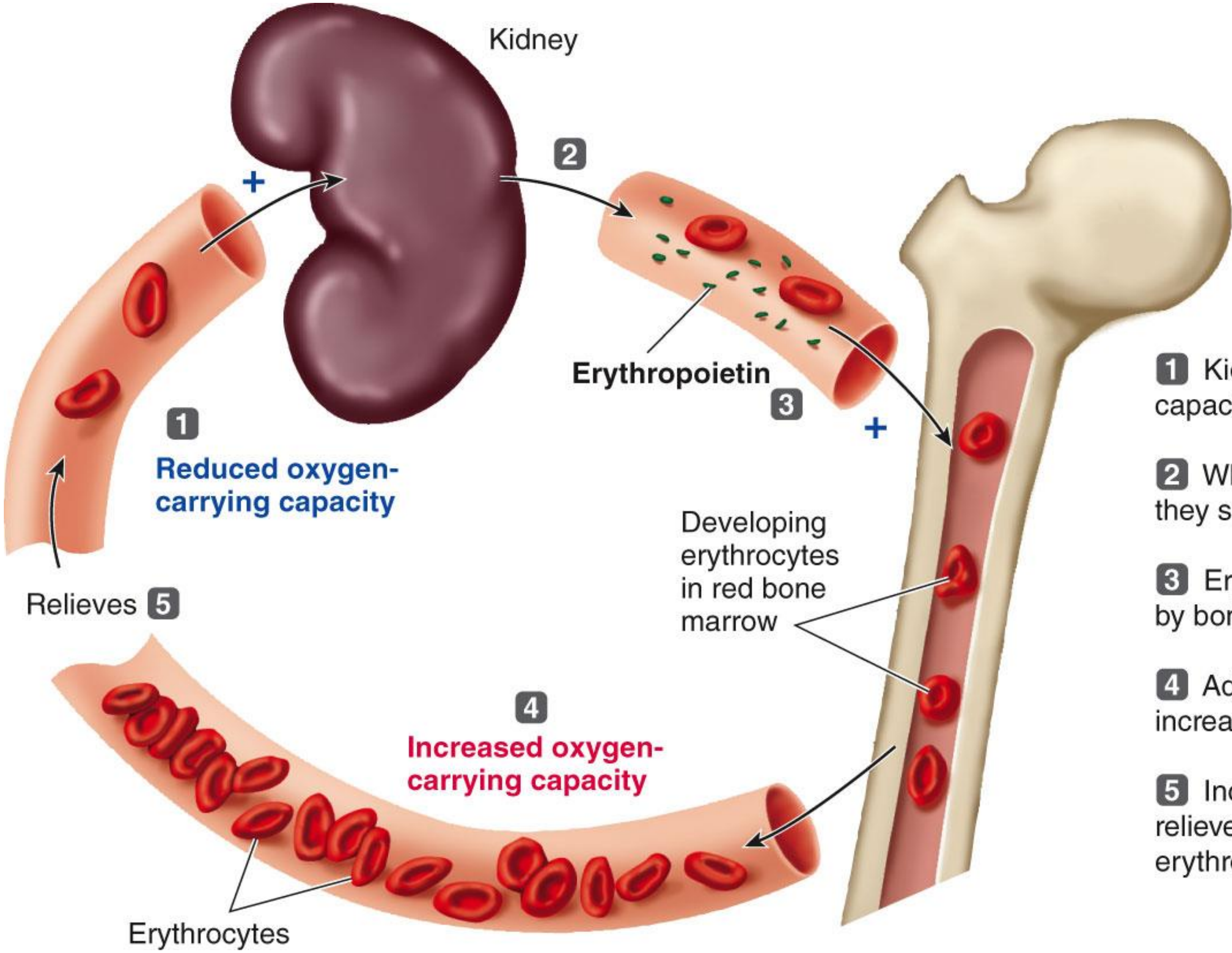
9.2 Circulatory Fluids and Cells



9.2 Circulatory Fluids and Cells

- **Erythrocytes** (red blood cells) transport oxygen from lungs or gills to tissues
  - Contain **hemoglobin**
  - Oval shaped in most vertebrates
    - **Biconcave discs** without nuclei or organelles in mammals
  - **Spleen** removes old erythrocytes and stores healthy erythrocytes, platelets and lymphocytes
  - **Hemopoietic tissues** (e.g. red bone marrow in birds and mammals) generate new erythrocytes (**erythropoiesis**)
  - **Erythropoietin** (EPO) is secreted by the kidneys to stimulate erythropoiesis . It is also **produced** in perisinusoidal cells in the **liver**.

9.2 Circulatory Fluids and Cells



1. Kidneys detect reduced O_2-carrying capacity of blood.
2. When less O_2 is delivered to the kidneys, they secrete erythropoietin into blood.
3. Erythropoietin stimulates erythropoiesis by bone marrow.
4. Additional circulating erythrocytes increase O_2-carrying capacity of blood.
5. Increased O_2-carrying capacity relieves initial stimulus that triggered erythropoietin secretion.

9.2 Circulatory Fluids and Cells

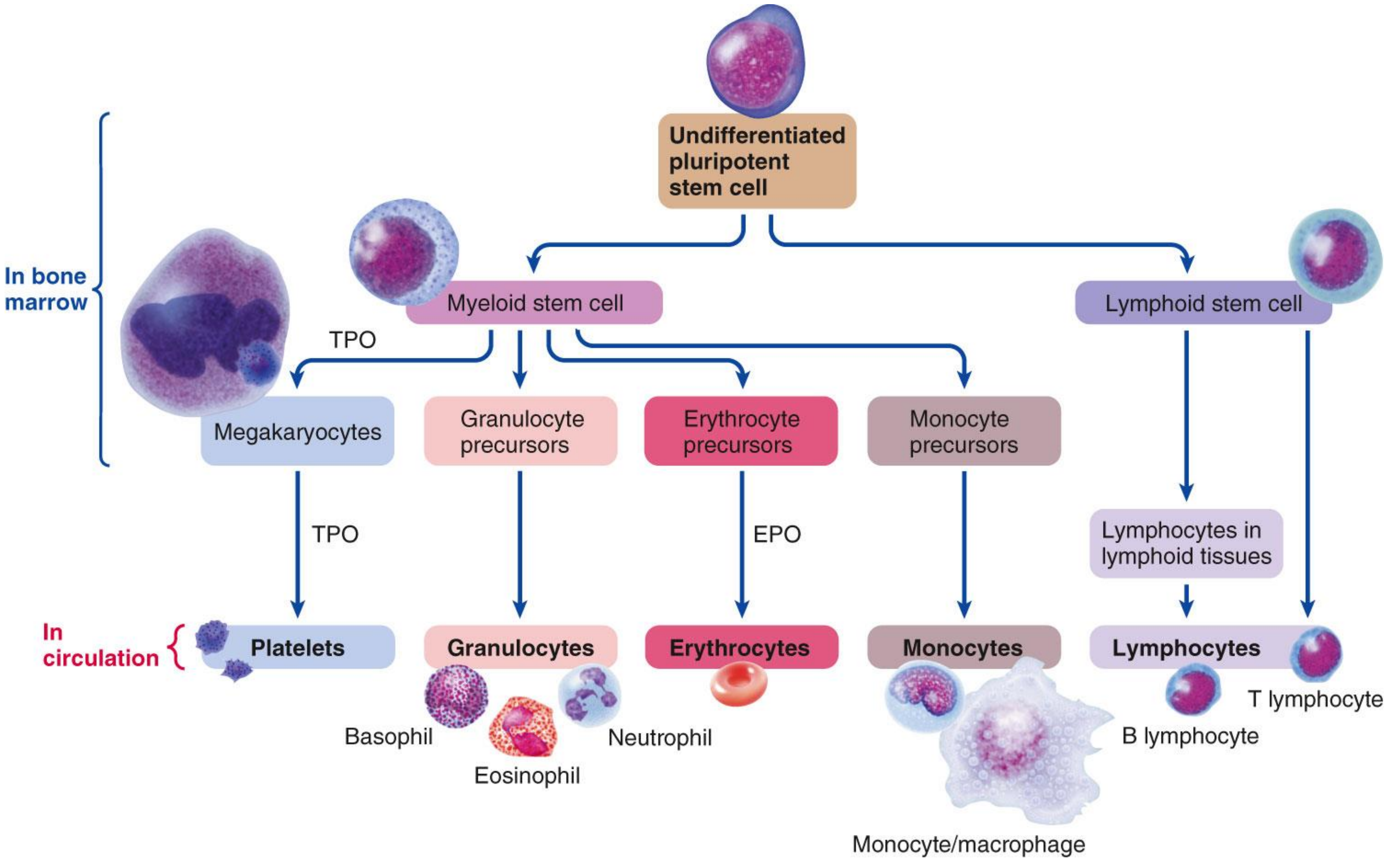
- **Leukocytes** (white blood cells) are key components of vertebrate **immune systems**

- **Thrombocytes** and **platelets** are involved in **blood clotting**

  - **Thrombocytes** are living cells found in all vertebrates except mammals
    - Break up into platelet-like fragments when activated by injury

  - **Platelets** are cell fragments circulating in mammalian blood
    - Shed from **megakaryocytes** in bone marrow

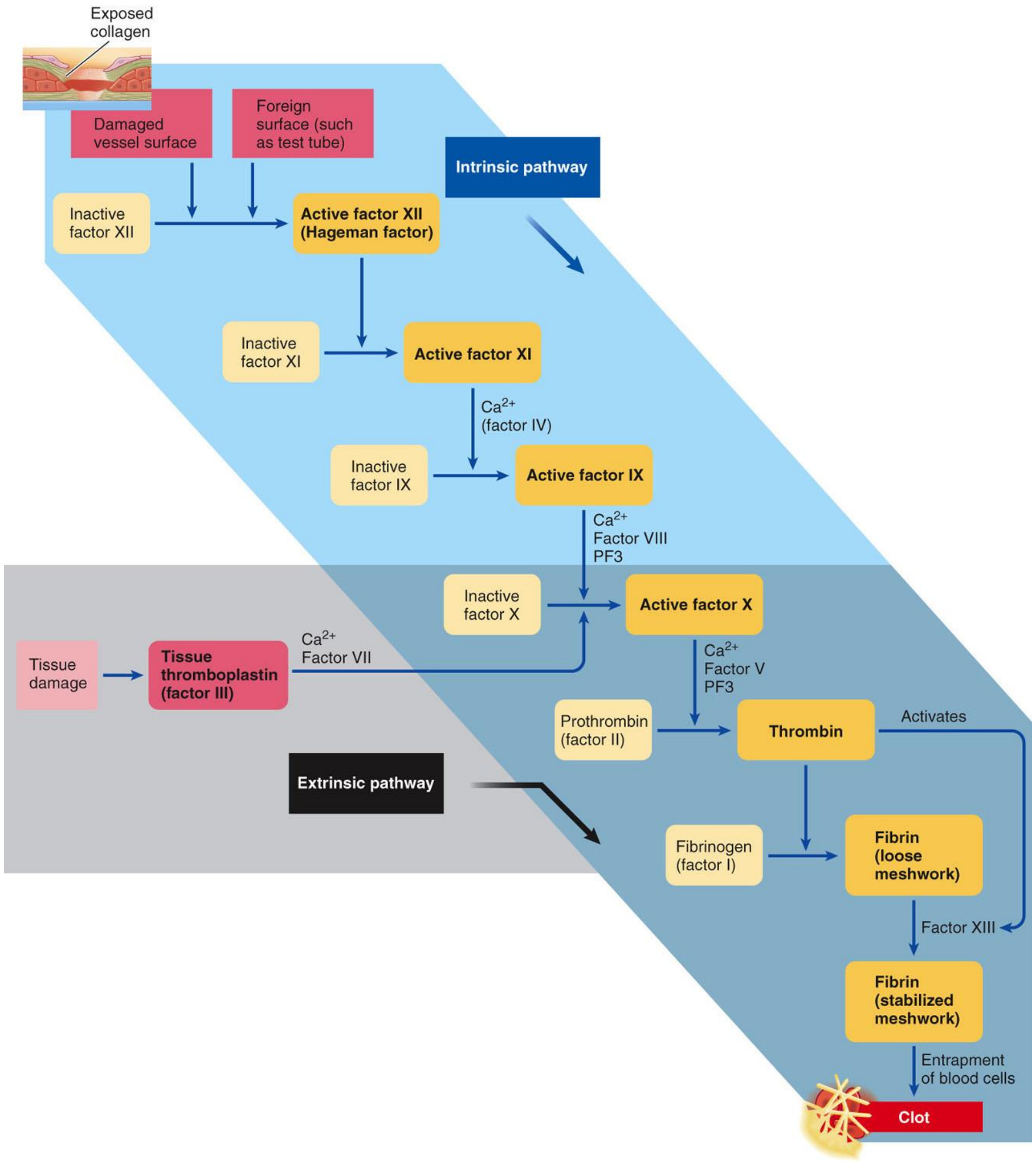
9.2 Circulatory Fluids and Cells



9.3 Circulatory Fluids: Hemostasis

- **Blood coagulation** is the transformation of blood from a liquid into a solid gel.
  - **Clotting cascade** involves **12 clotting factors**
    - Most are plasma proteins synthesized by the liver
    - Present in plasma in **inactive** forms
    - **Intrinsic pathway** involves 7 steps
    - Activation of the first factor (factor XII) triggers the **clotting cascade**
    - **Extrinsic pathway** involves 4 steps and requires contact with tissue factors outside of the blood
  - **Final steps**
    - **Thrombin** converts **fibrinogen** into **fibrin**
    - **Fibrin**, an insoluble thread-like molecule, forms a stabilized meshwork at the site of a platelet plug

9.3 Circulatory Fluids: Hemostasis



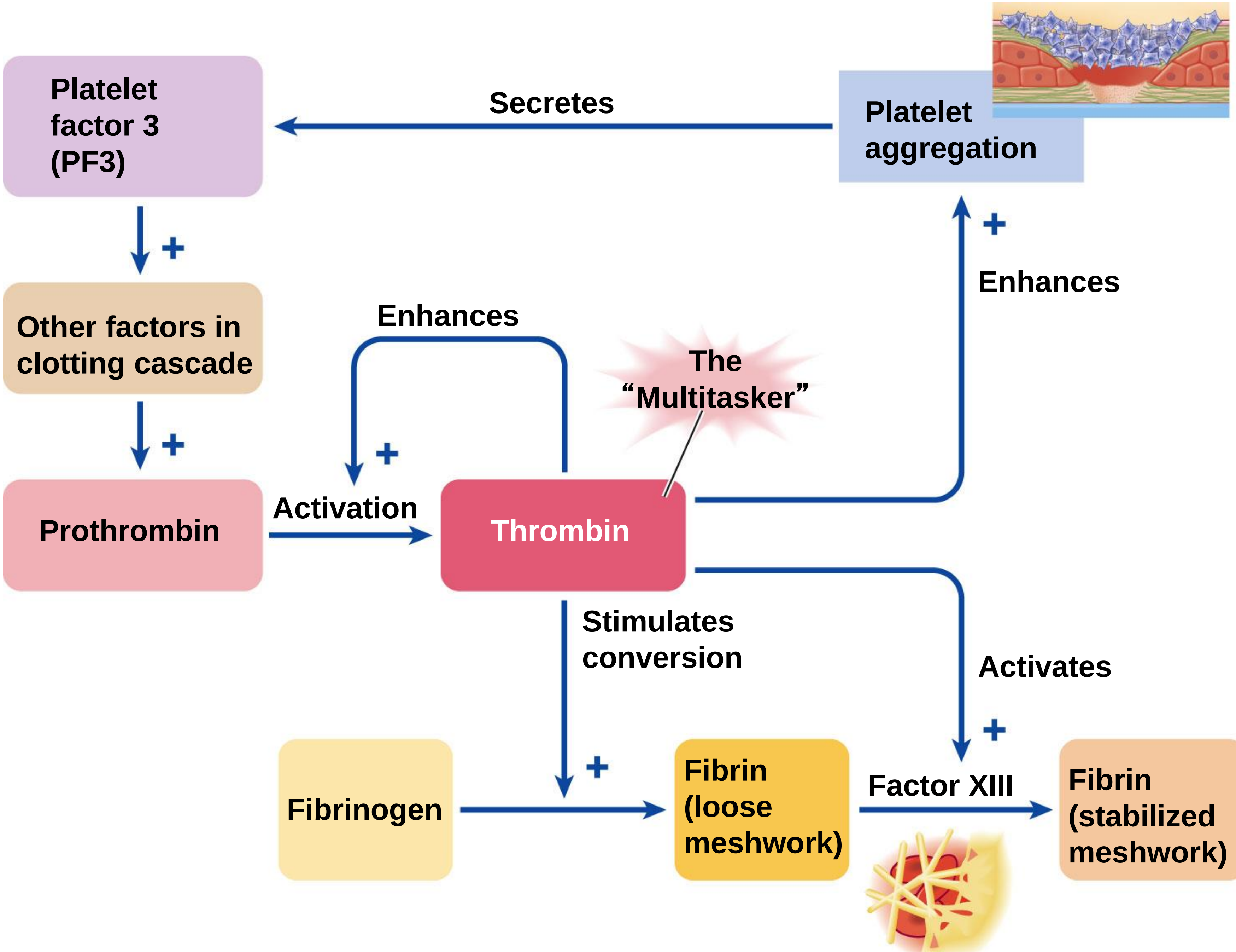


Figure 9-11 p397

9.3 Circulatory Fluids: Hemostasis

- **Plasmin** dissolves clots
  - **Plasminogen** is produced by the liver
  - **Factor XII** (which began the clotting cascade) triggers a cascade of reactions leading to activation of plasminogen to form **plasmin**
  - **Tissue plasminogen activator** (tPA) produces a low level of plasmin activation in the absence of clotting

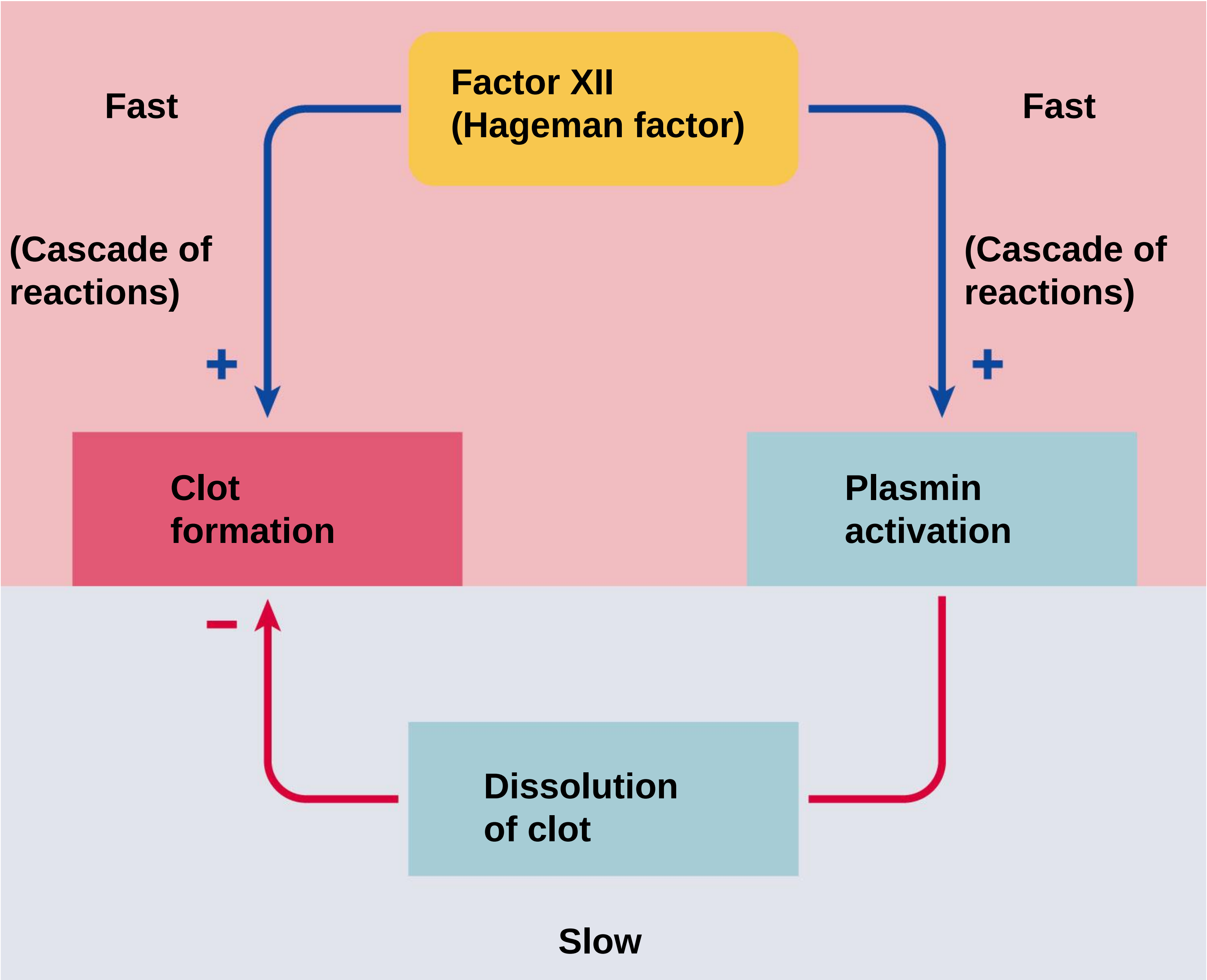
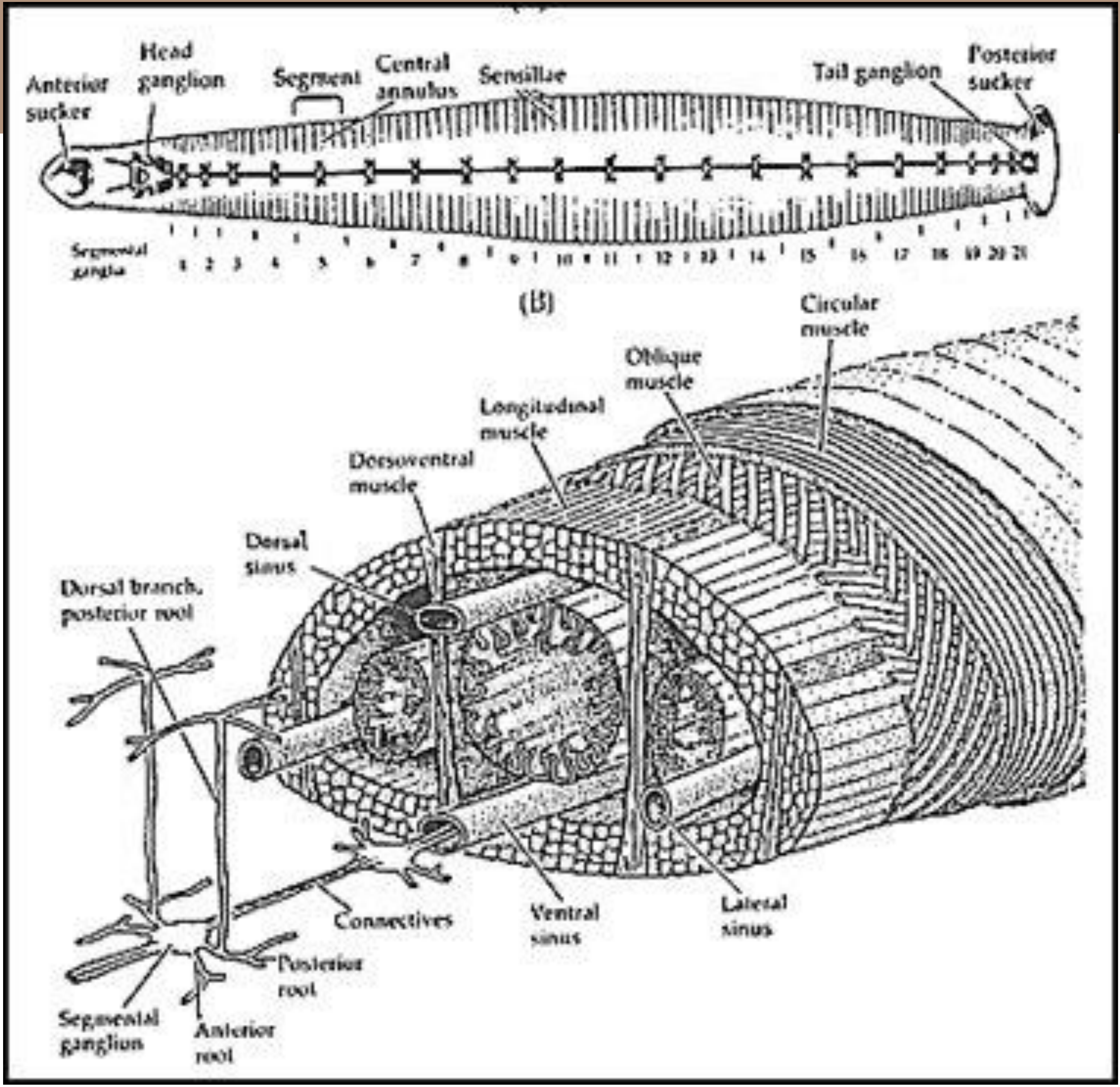


Figure 9-14 p399

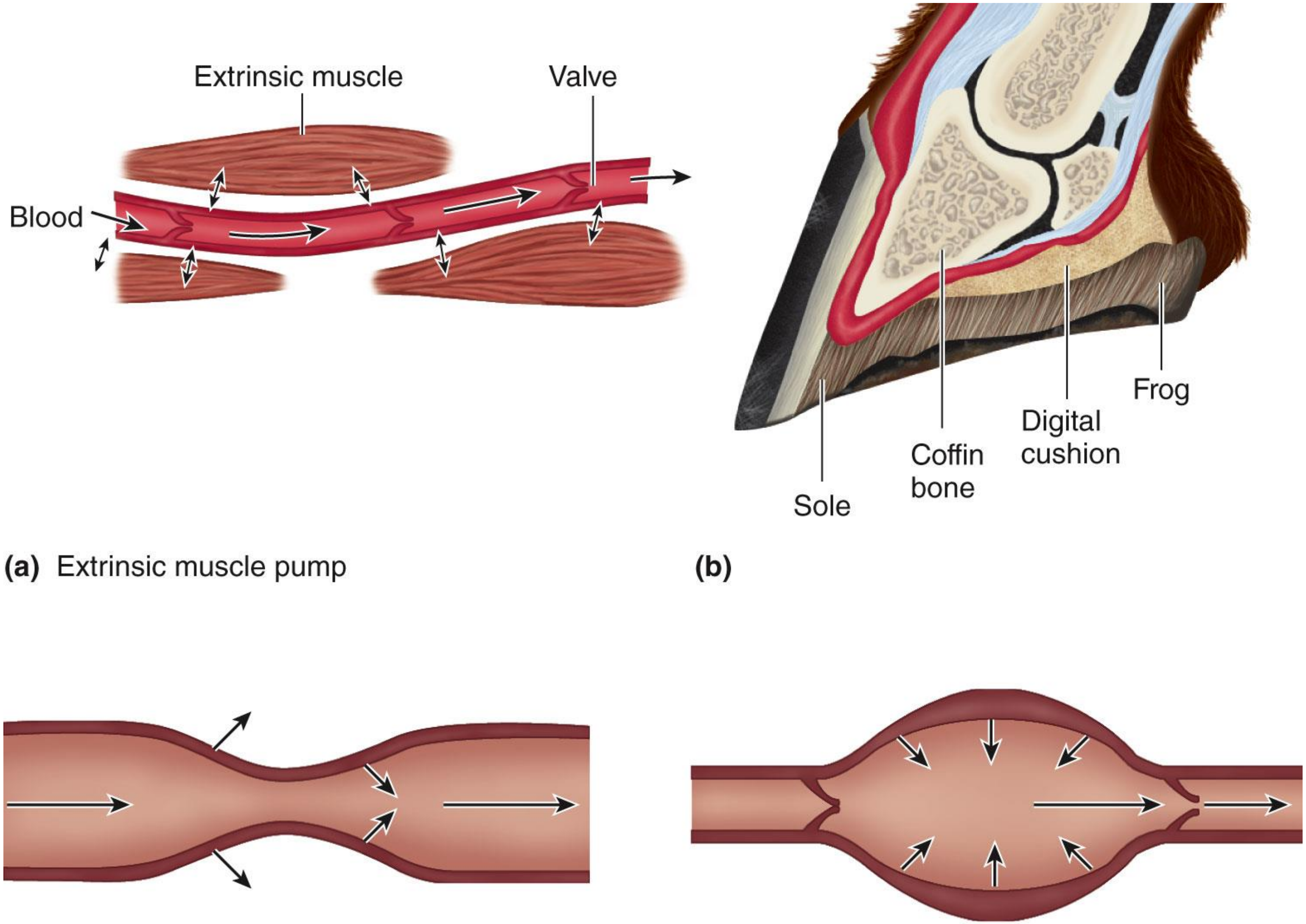


Head ganglion
Anterior sucker
Segment
Central annulus
Sensillae
Tail ganglion
Posterior sucker
Segmental ganglia
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
(B)
Circular muscle
Oblique muscle
Longitudinal muscle
Dorsoventral muscle
Dorsal sinus
Dorsal branch, posterior root
Connectives
Ventral sinus
Lateral sinus
Posterior root
Segmental ganglion
Anterior root

9.4 Circulatory Pumps: Evolution

- Pumping mechanisms
  - **Flagella**
    - Flagella of epithelial cells create slow currents
  - **Extrinsic muscle or skeletal pumps**
    - Muscle contractions associated with locomotion
  - **Peristaltic (tubular) muscle pumps**
    - Walls of vessels contract in a moving wave
  - **Chamber muscle pumps**
    - One-way valves create flow in one direction
  - **Auxiliary hearts**
    - Boost flow to certain parts (e.g. gills) in animals with primary hearts

9.4 Circulatory Pumps: Evolution



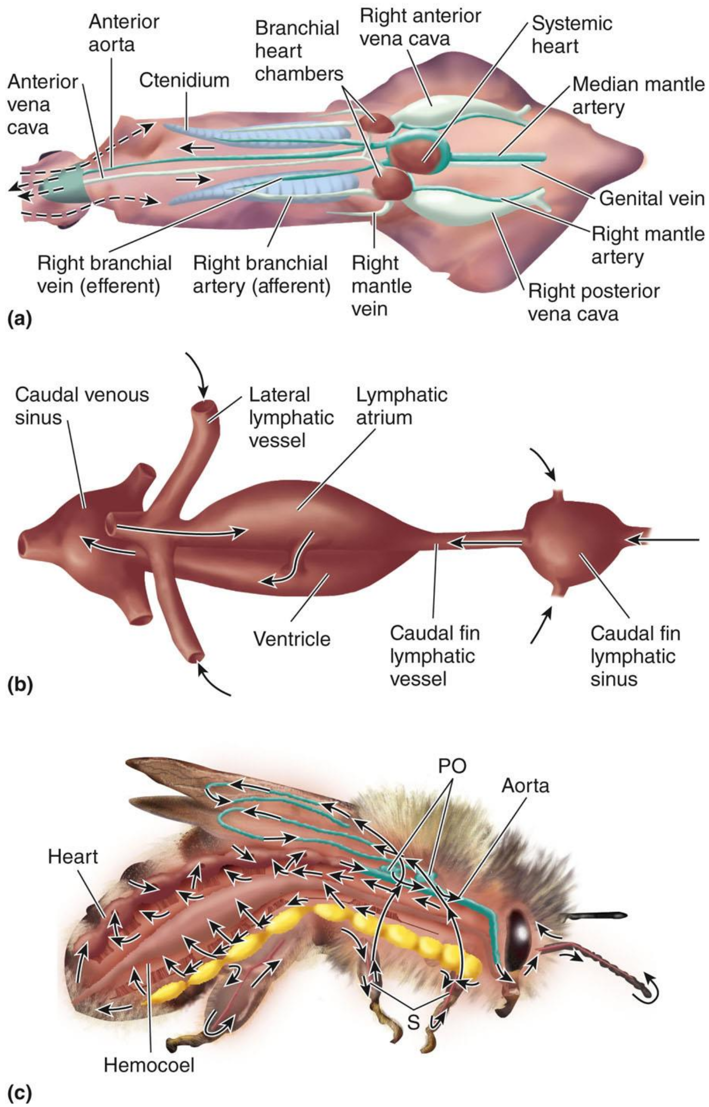
(a) Extrinsic muscle pump

(b)

(c) Peristaltic heart

(d) Chambered heart

9.4 Circulatory Pumps: Evolution

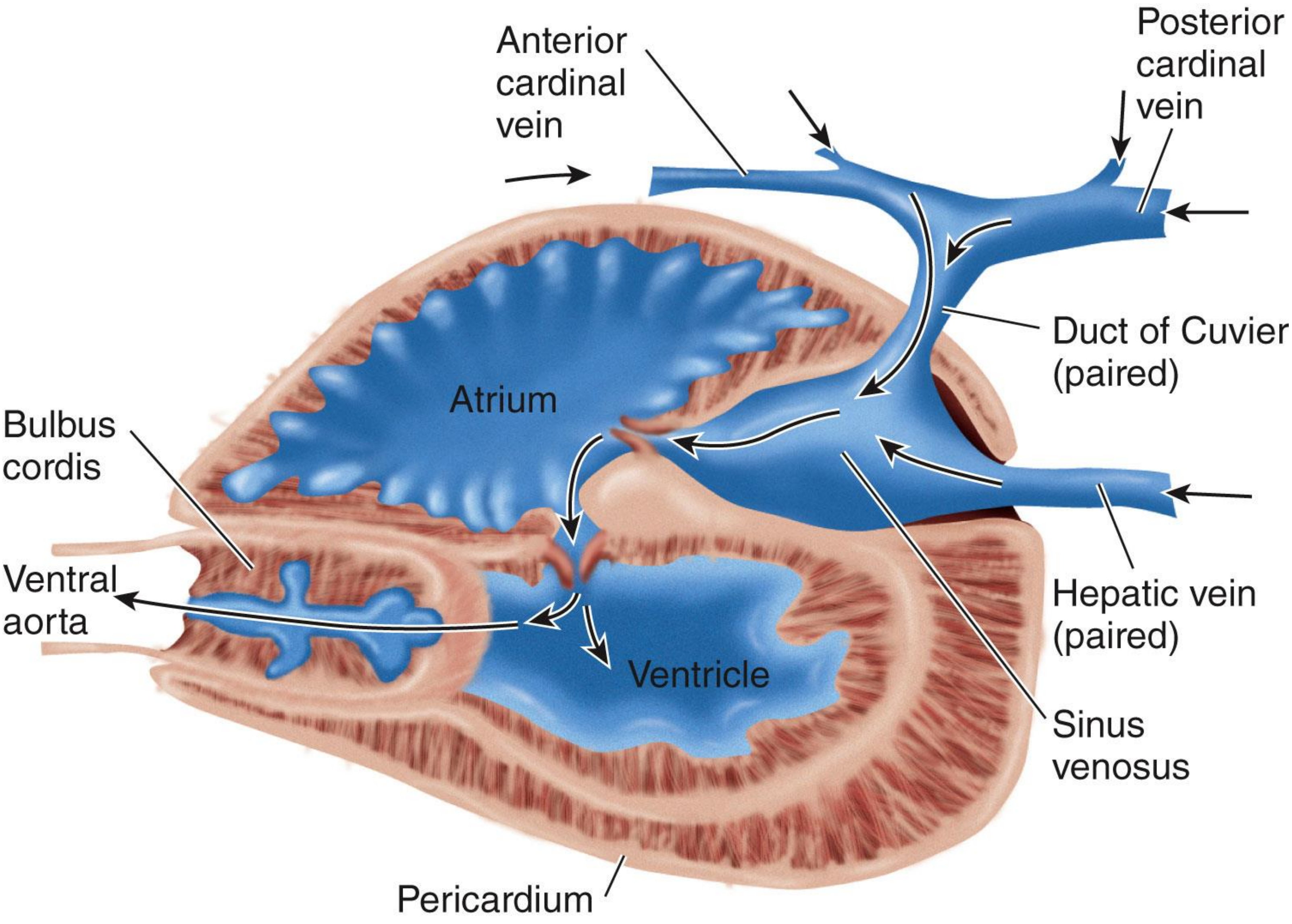


9.4 Circulatory Pumps: Evolution

- **Arthropod hearts**
  - Dorsally located
  - Single-chamber pumps (crustaceans)
  - Tubular pumps (insects and arachnids)
  - **Ostia** are pore-like openings that allow hemolymph to reenter the heart

- **Fish hearts** have **two primary chambers**
  - **Atrium** collects returning blood
  - **Ventricle** pumps to body
  - Auxiliary chambers
    - **Sinus venosus** collects blood from veins before entering atrium
    - **Conus arteriosus** (in cartilaginous fish) or **bulbus arteriosus** (in bony fish) dampen pulsatile pressure output of the ventricle

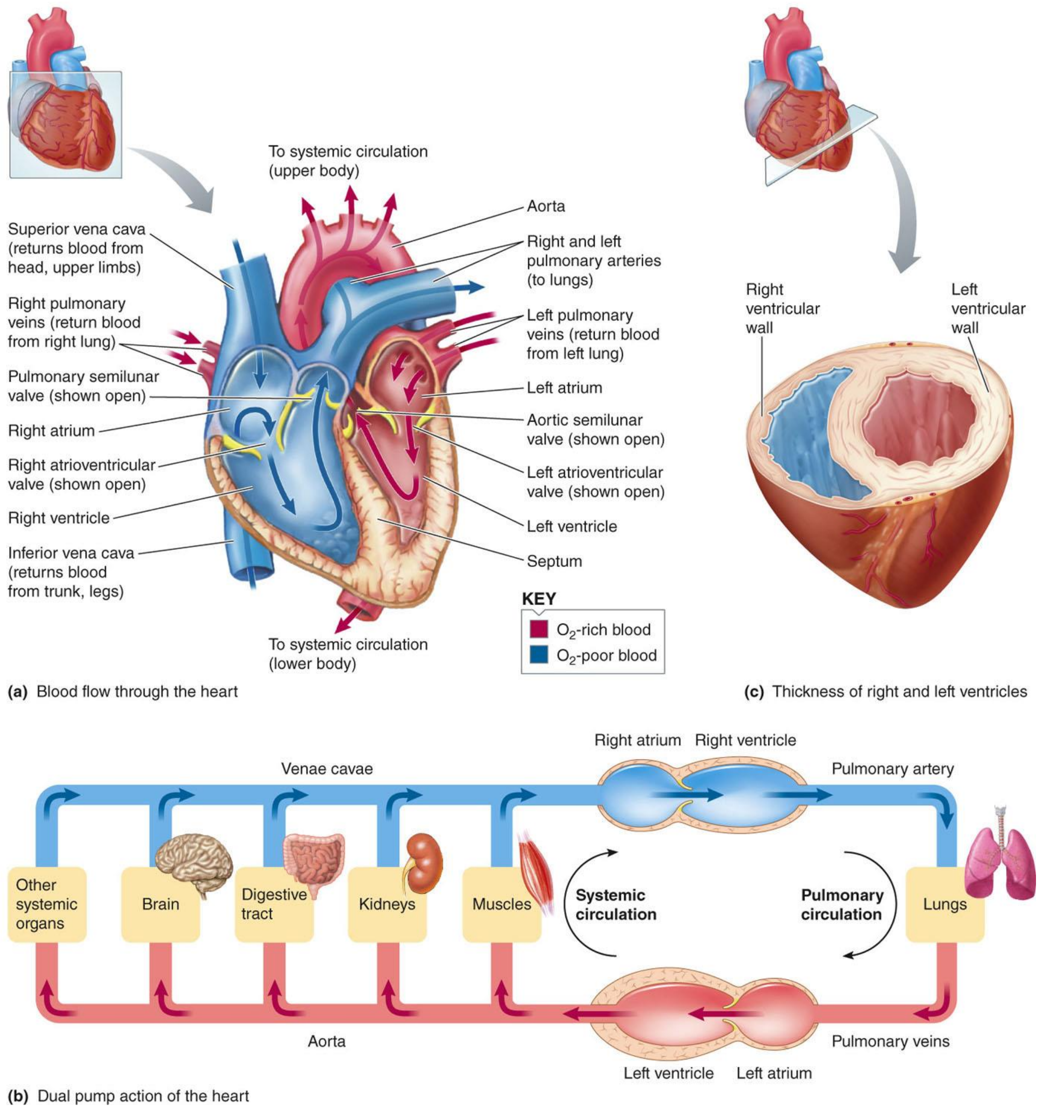
9.4 Circulatory Pumps: Evolution



9.4 Circulatory Pumps: Evolution

- **Avian** and **mammalian** hearts have **dual pumps**
  - Two **atria** receive blood
  - Two **ventricles** pump blood
  - Two pumps are separated by the **septum**
  - **Right** half pumps **oxygen-depleted** blood into the **pulmonary circulation**
  - **Left** half pumps **oxygen-rich** blood into the **systemic circulation**
  - Both sides of the heart pump **equal amounts** of blood

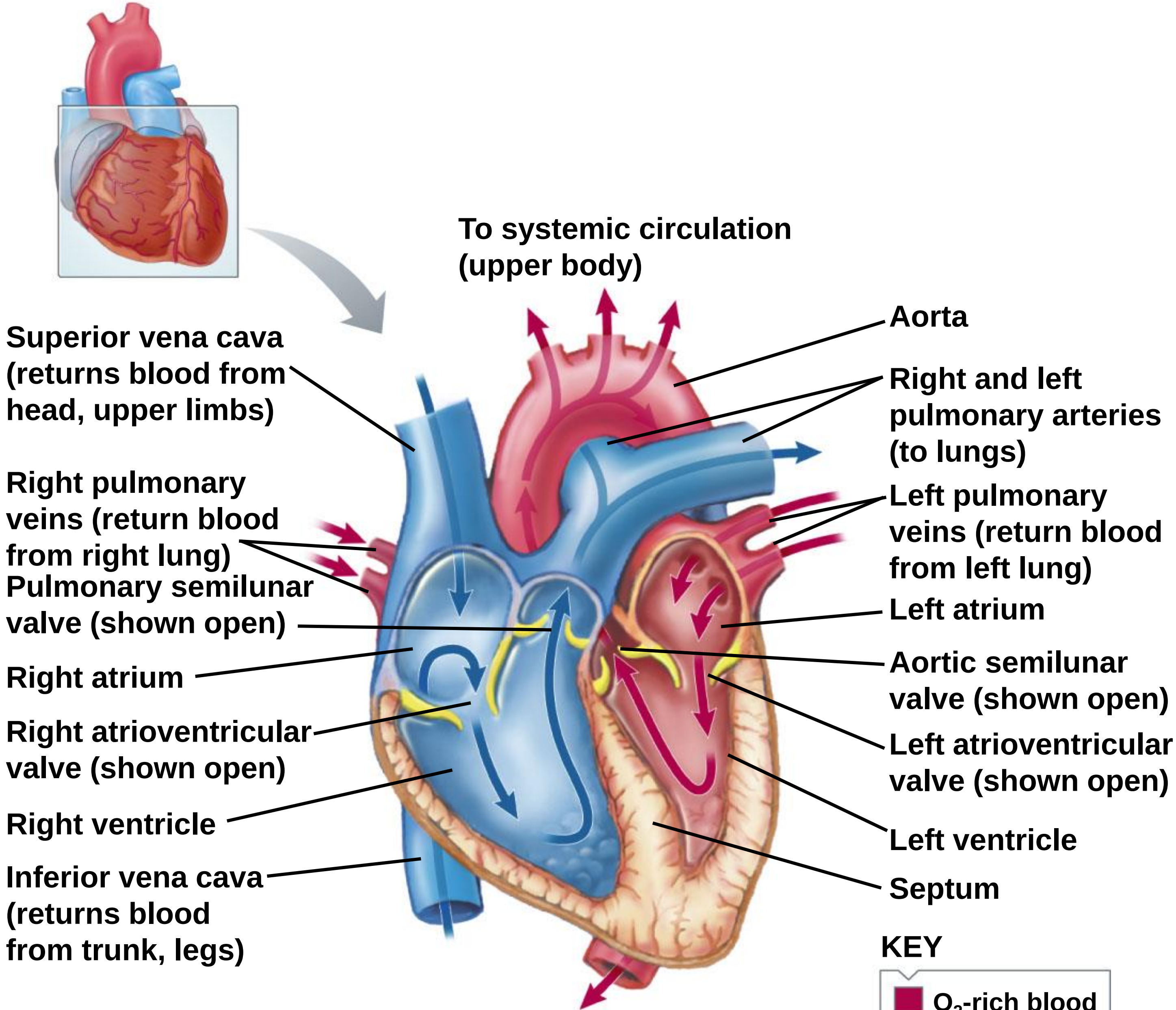
9.4 Circulatory Pumps: Evolution



(a) Blood flow through the heart

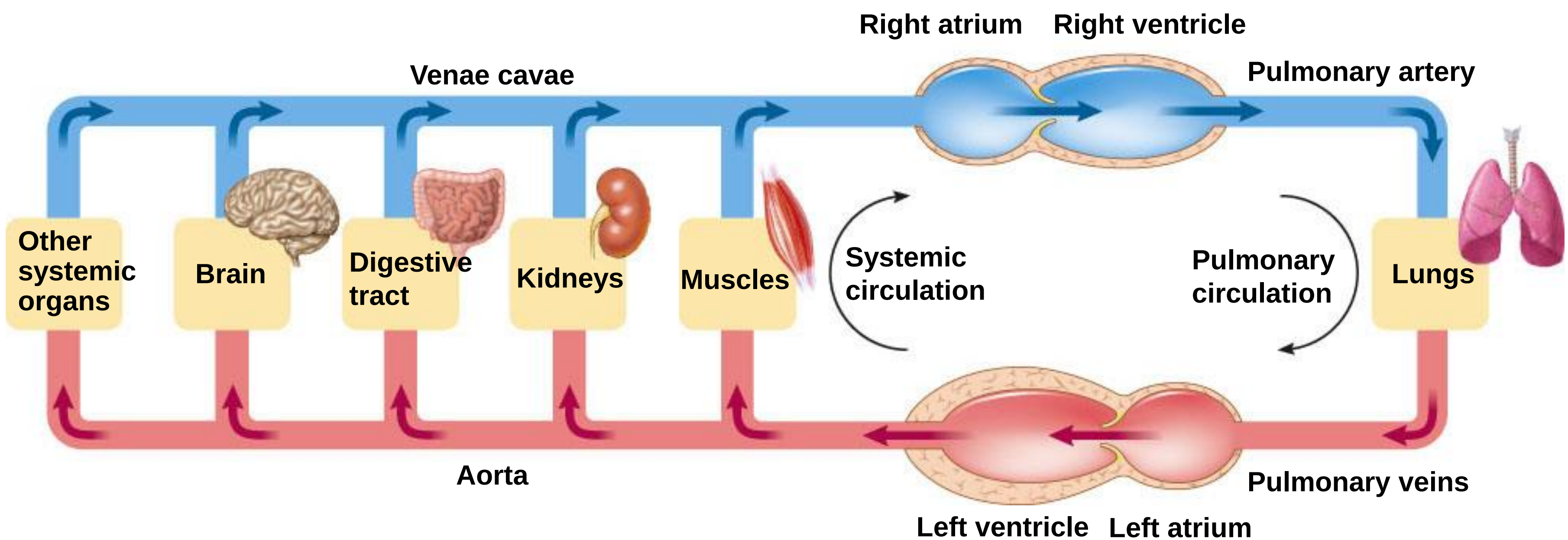
(c) Thickness of right and left ventricles

(b) Dual pump action of the heart



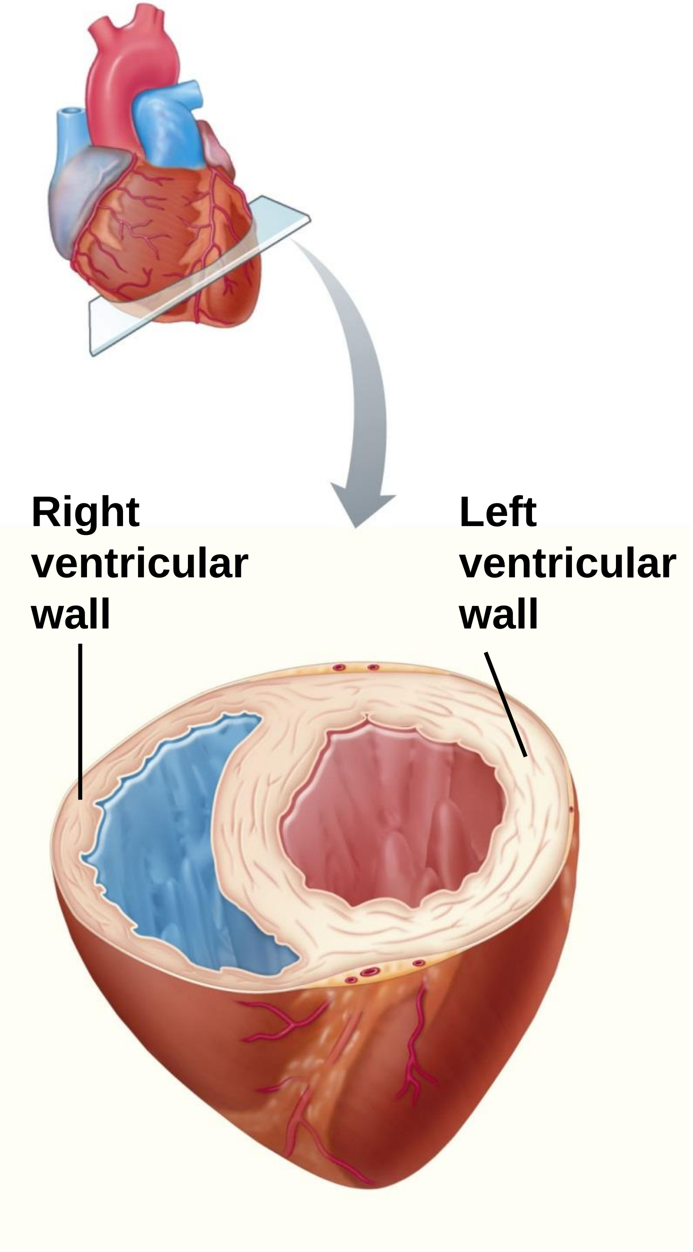
(a) Blood flow through the heart

Figure 9-18a p404



(b) Dual pump action of the heart

Figure 9-18b p404

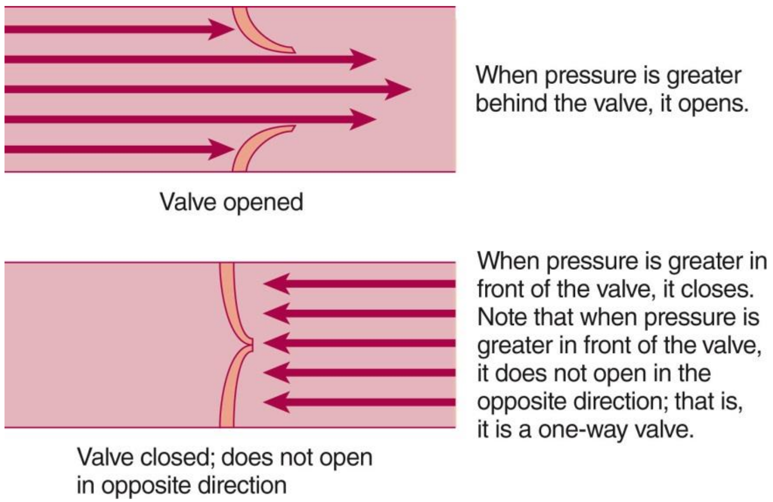


(c) Thickness of right and left ventricles

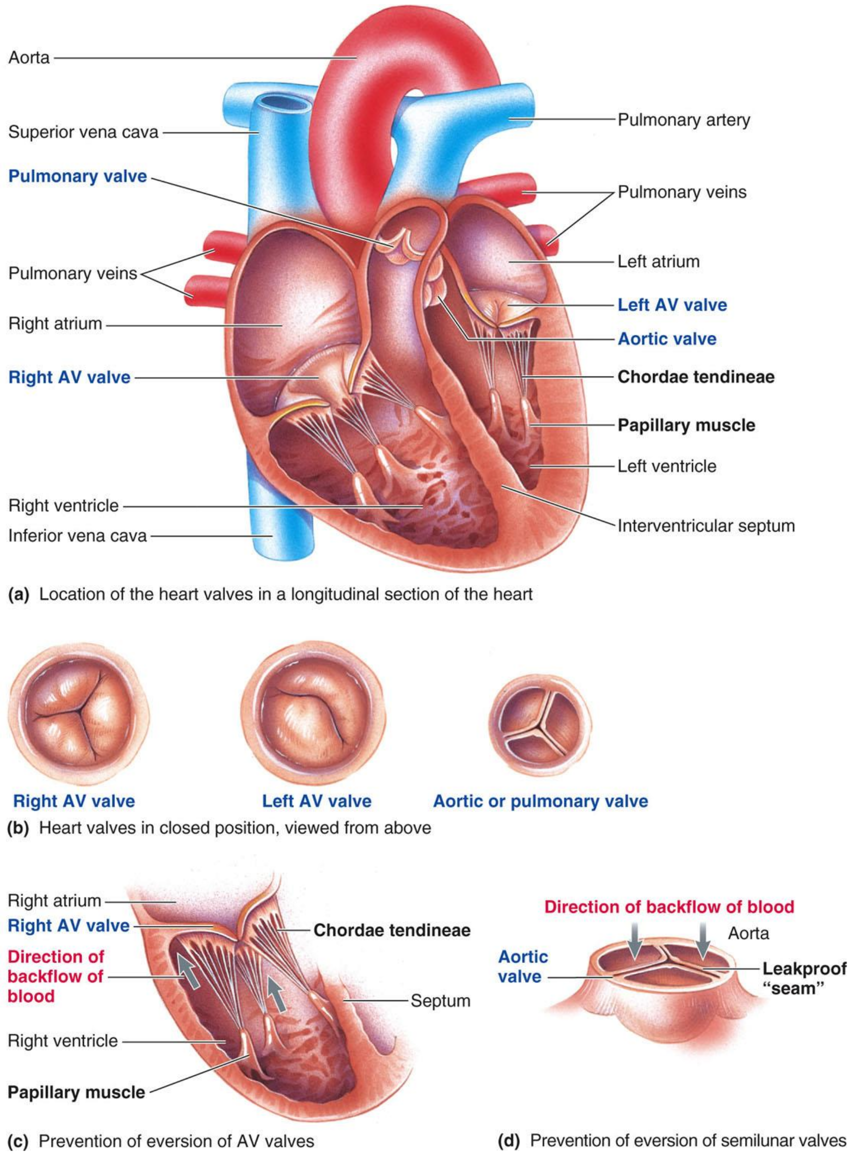
Figure 9-18c p404

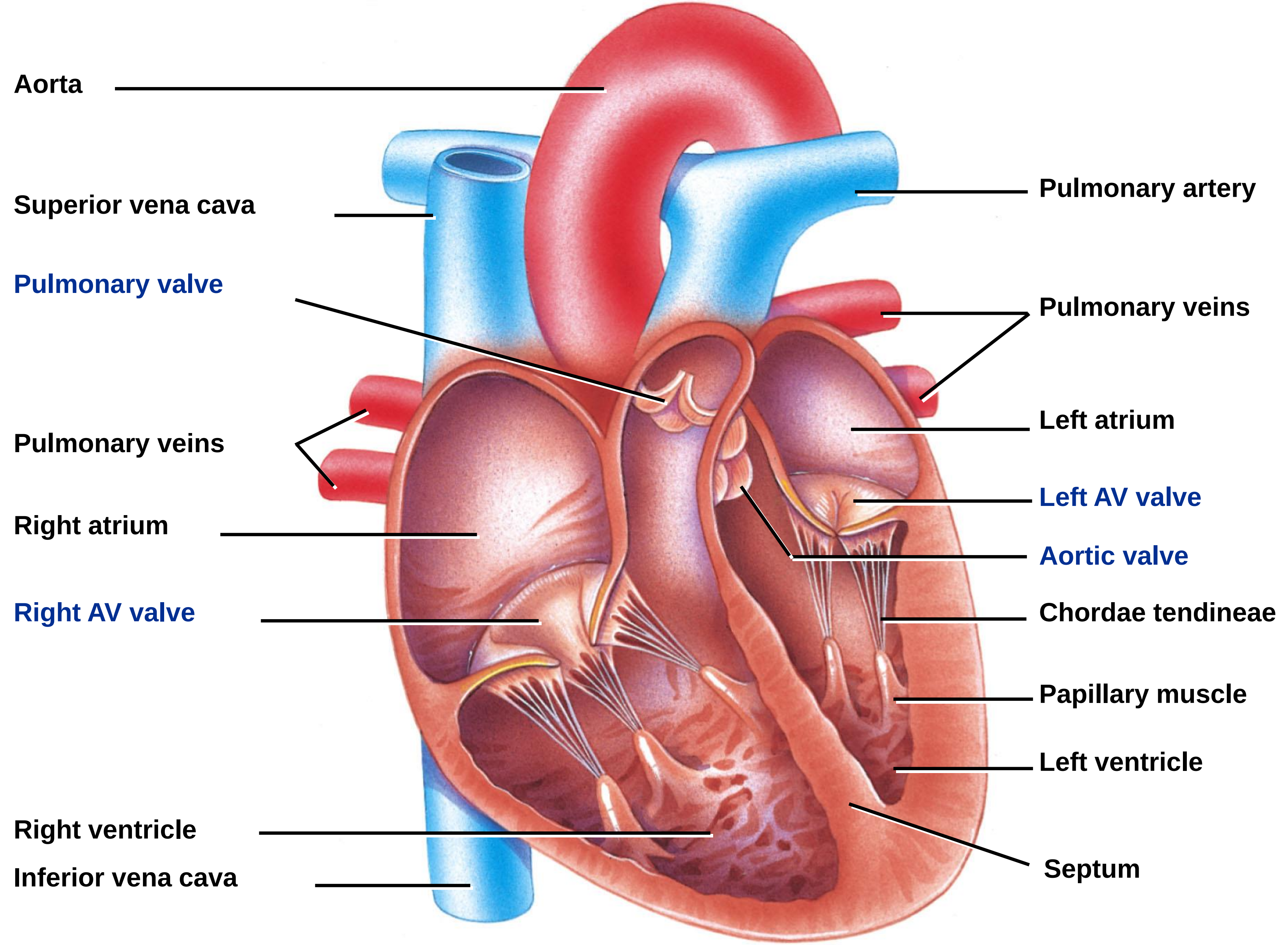
9.4 Circulatory Pumps: Evolution

- **Heart valves** ensure unidirectional flow of blood
  - **Right** and **left atrioventricular** (AV) **valves** allow flow from atria into ventricles during ventricular filling
  - **Aortic** and **pulmonary** (**semilunar**) **valves** allow flow from ventricles into arteries during ventricular contraction
  - Backflow from atria into veins does not occur because atrial pressures are not much higher than venous pressures



9.4 Circulatory Pumps: Evolution



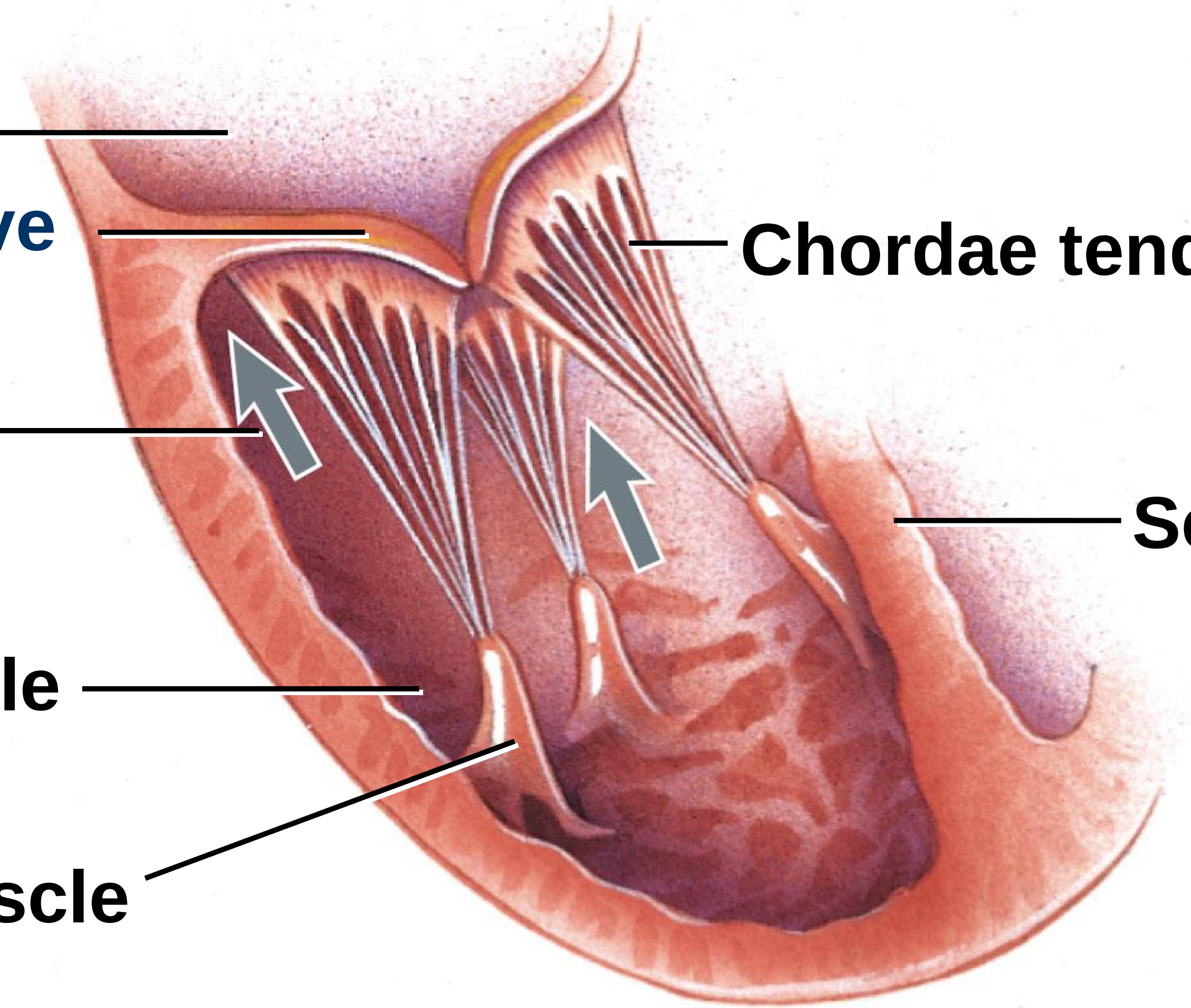


(a) Location of the heart valves in a longitudinal section of the heart

Figure 9-20a p405

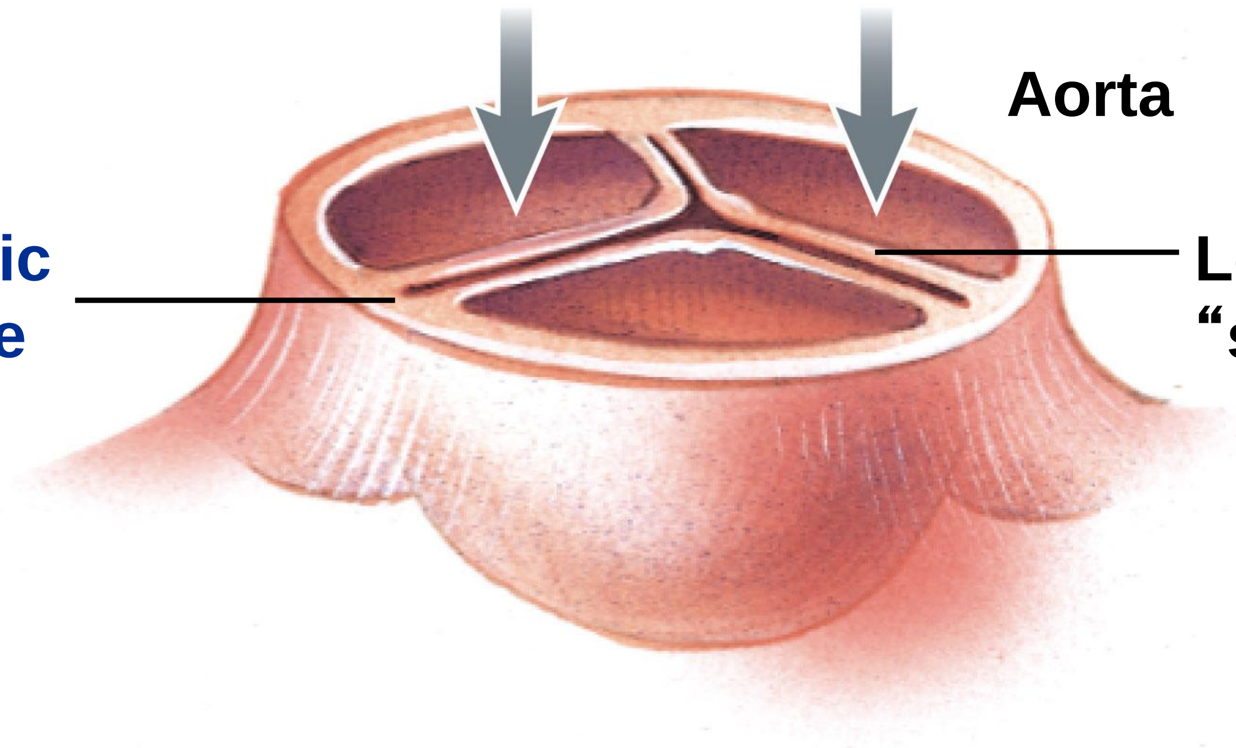
(b) Heart valves in closed position, viewed from above

Figure 9-20b p405



(c) Prevention of eversion of AV valves

Figure 9-20c p405



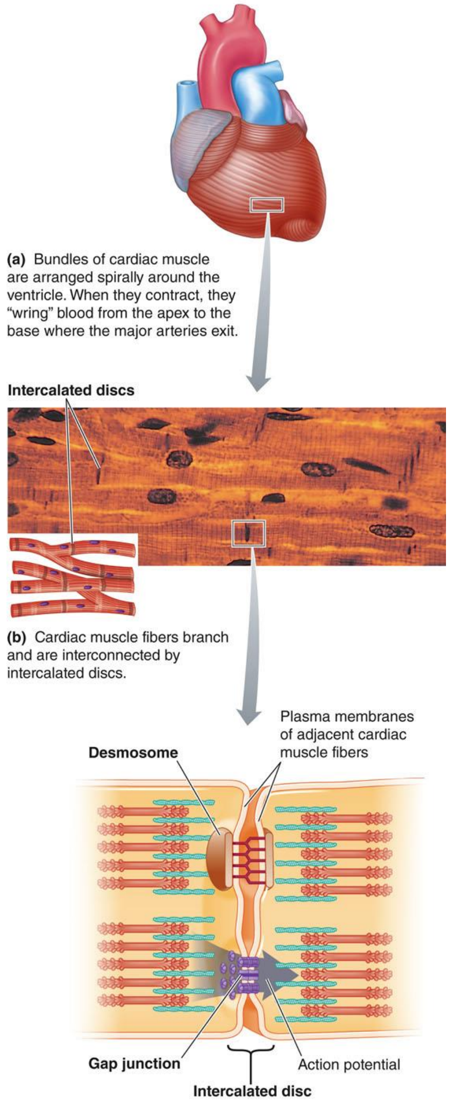
(d) Prevention of eversion of semilunar valves

Figure 9-20d p405

9.4 Circulatory Pumps: Evolution

- **Vertebrate heart walls**
  - Thick, muscular **myocardium** is sandwiched between **endocardium** and **epicardium**
  - Myocardium consists of interlacing bundles of **cardiac muscle fibers** arranged spirally
    - Cardiac muscle cells form **branching fibers** with adjacent cells joined end-to-end at **intercalated discs**
    - Intercalated discs contain **desmosomes** and **gap junctions**
    - Impulses spread to all cells joined by **gap junctions** to form a **functional syncytium**

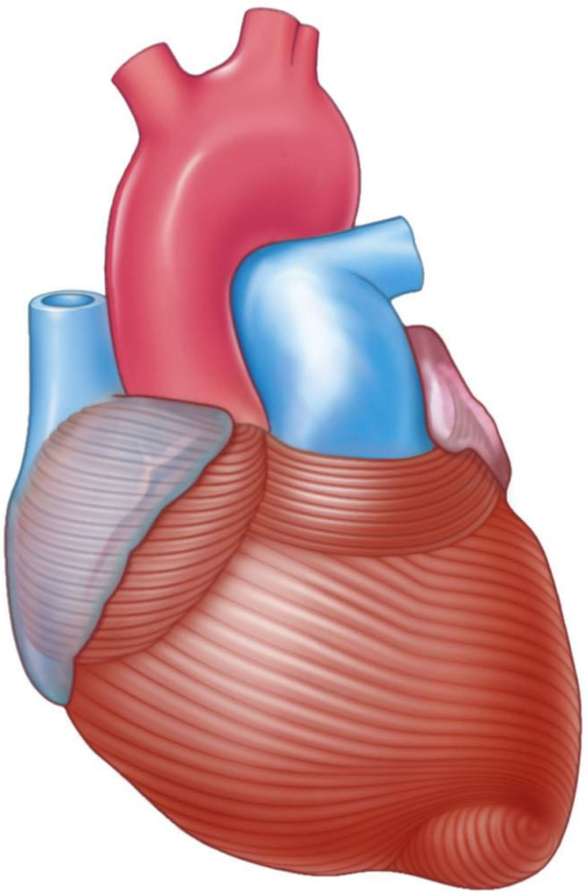
9.4 Circulatory Pumps: Evolution



(a) Bundles of cardiac muscle are arranged spirally around the ventricle. When they contract, they "wring" blood from the apex to the base where the major arteries exit.

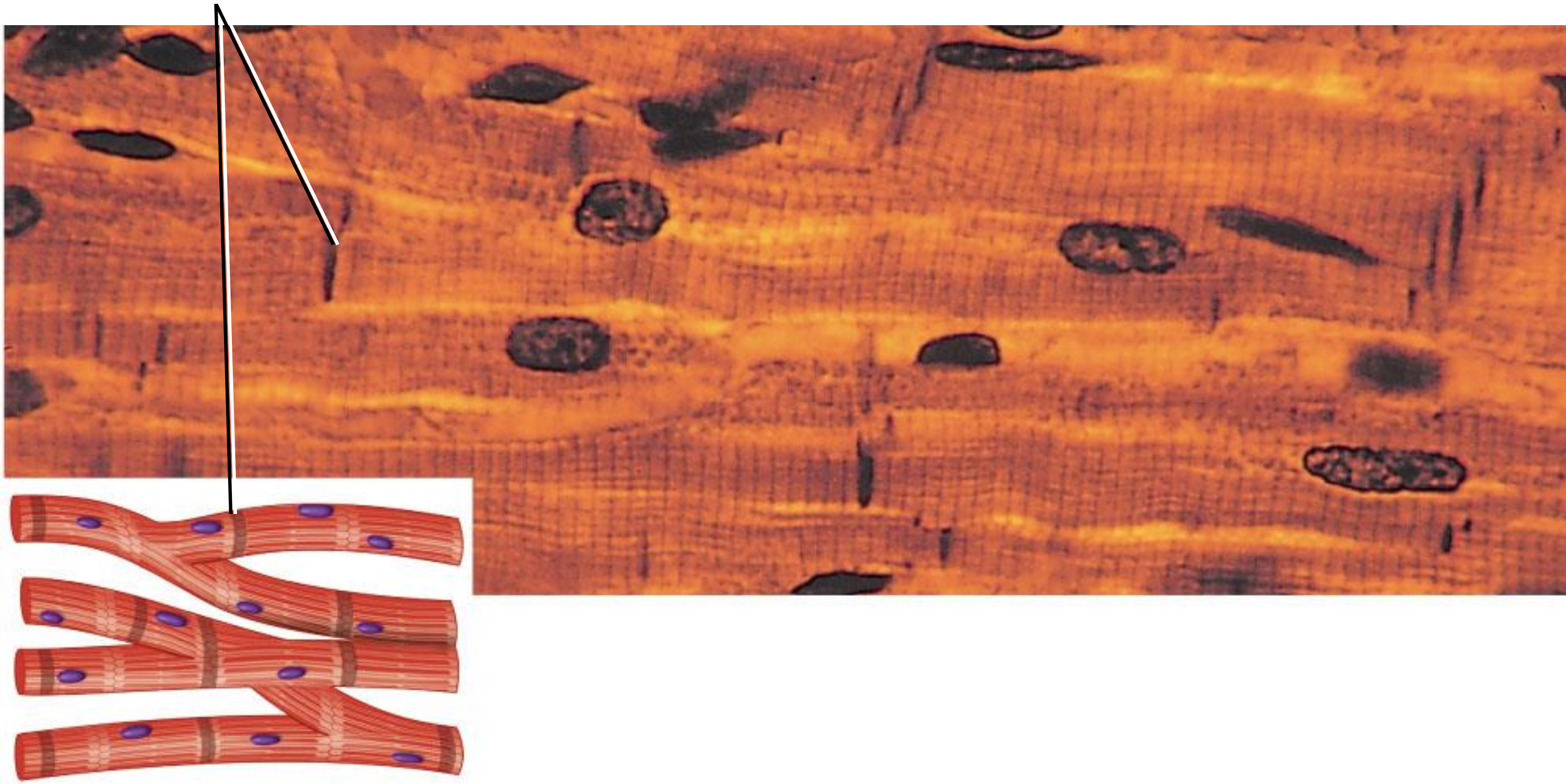
(b) Cardiac muscle fibers branch and are interconnected by intercalated discs.

(c) Intercalated discs contain two types of membrane junctions: mechanically important desmosomes and electrically important gap junctions.



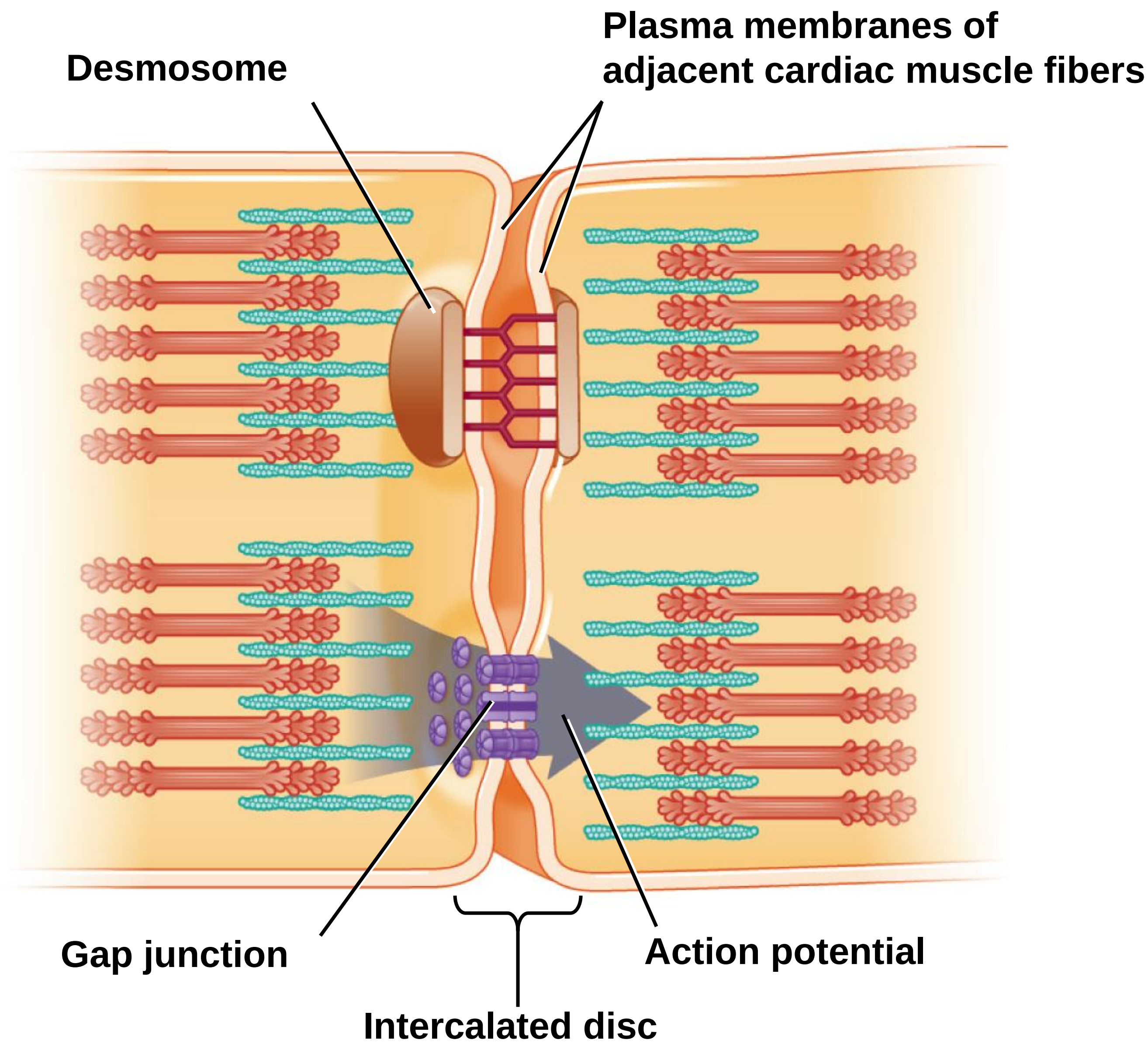
(a) Bundles of cardiac muscle are arranged spirally around the ventricle. When they contract, they “wring” blood from the apex to the base where the major arteries exit.

Figure 9-21a p406



(a) Cardiac muscle fibers branch and are interconnected by intercalated discs.

Figure 9-21b p406



(c) Intercalated discs contain two types of membrane junctions: mechanically important desmosomes and electrically important gap junctions.

Figure 9-21c p406

9.5 Circulatory Pumps: Heart Electrical Activity

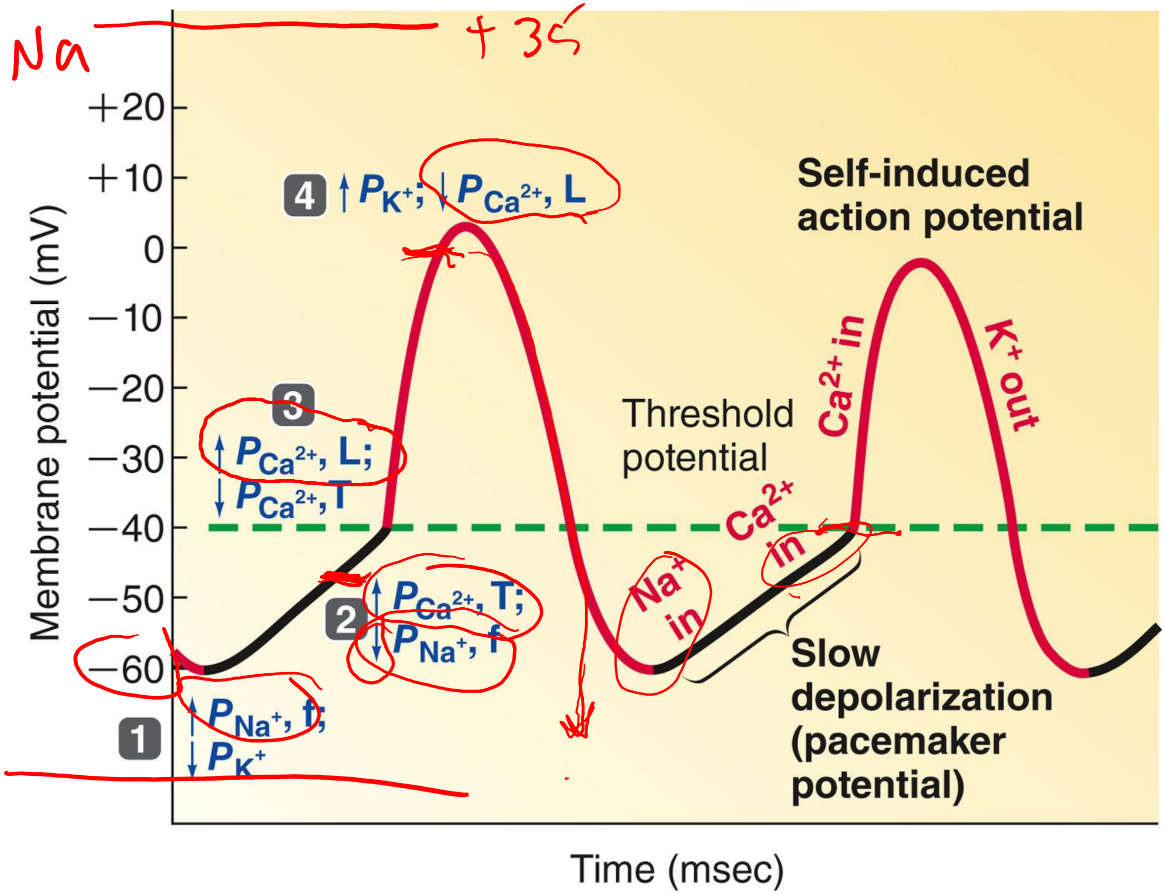
- **Neurogenic hearts** require external neural stimulus to beat (e.g. decapod crustaceans)

- **Myogenic hearts** have **pacemaker cells**

  - Membrane potential of pacemaker cells slowly **depolarizes** due to:
    - Increased inward Na^+ current
    - Decreased outward K^+ current
    - Increased inward Ca^{2+} current

  - **Action potential** is produced when L-type **Ca^{2+} channels** open at **threshold**
    - Large influx of Ca^{2+} causes rapid depolarization

9.5 Circulatory Pumps: Heart Electrical Activity



KEY

f = Funny channels
T = Transient-type channels
L = Long-lasting channels

9.5 Circulatory Pumps: Heart Electrical Activity

- Conduction pathway

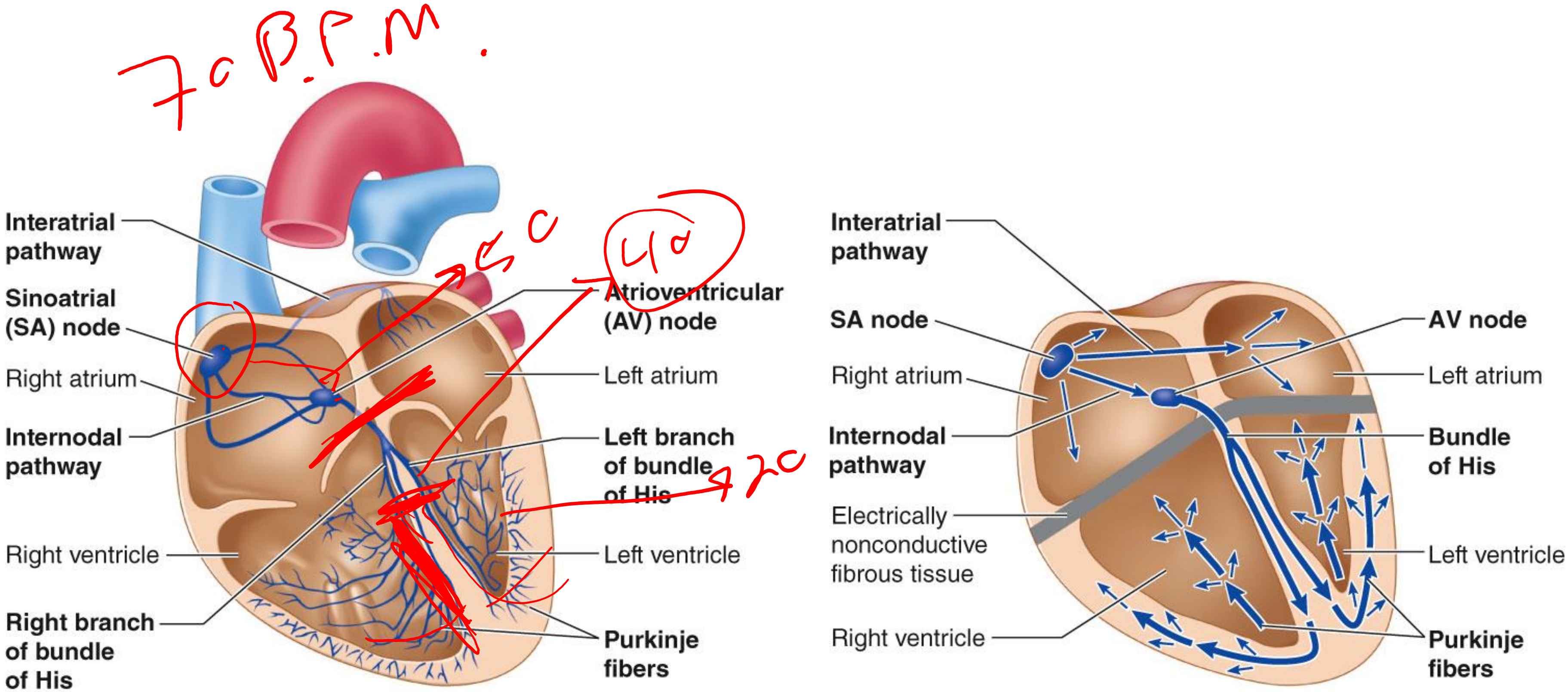
1. **Sinoatrial (SA) node** is the normal pacemaker
   - Fastest rate of autorhythmicity

2. **Atrial excitation**

3. **Atrioventricular (AV) node** transmits impulses between the atria and the ventricles
   - Slower rate of autorhythmicity
   - Impulse is delayed about 0.1 sec

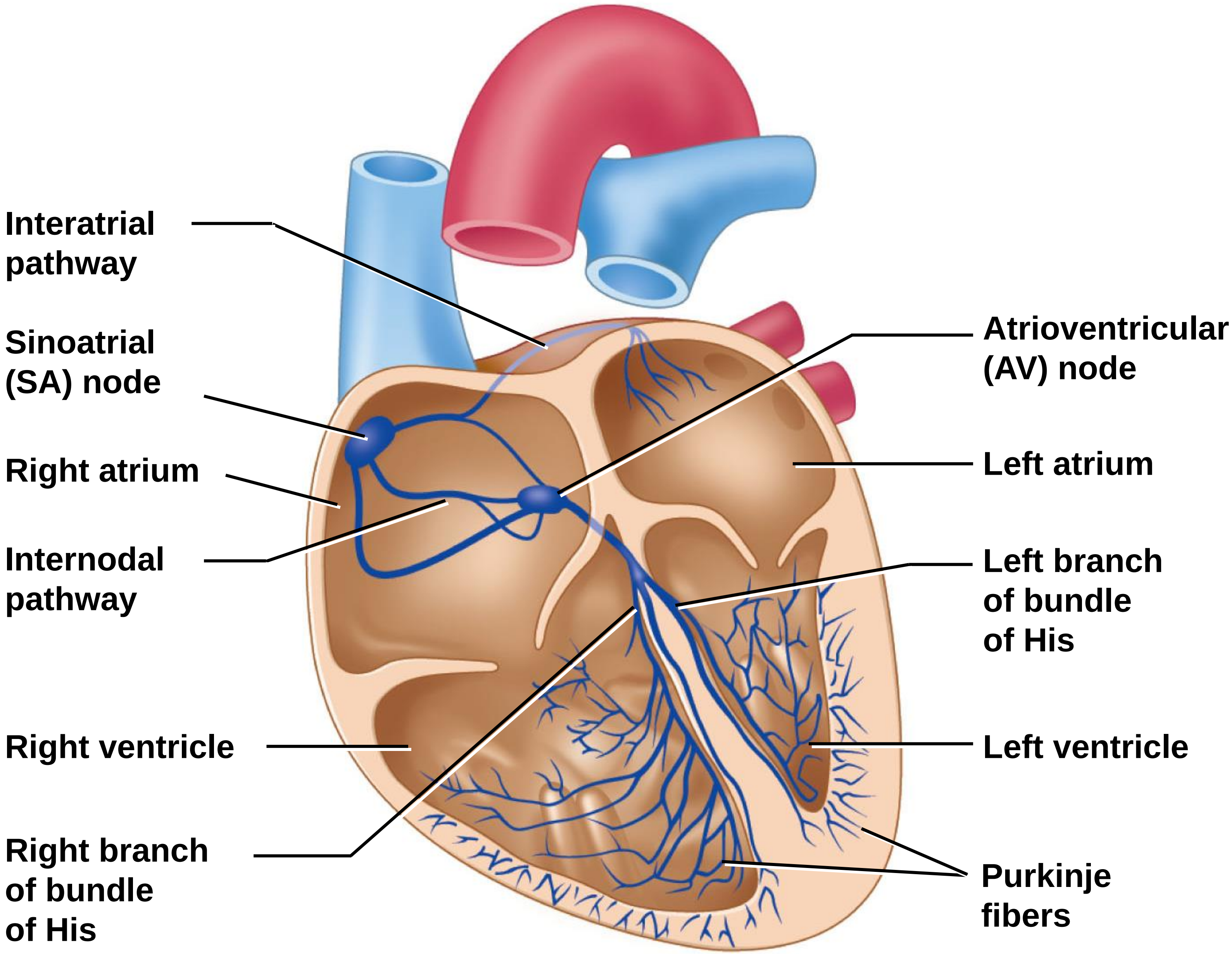
4. **Ventricular excitation**
   - Impulse passes down the **bundle of His** in the interventricular septum
   - **Purkinje fibers** extend through the ventricular myocardium

9.5 Circulatory Pumps: Heart Electrical Activity



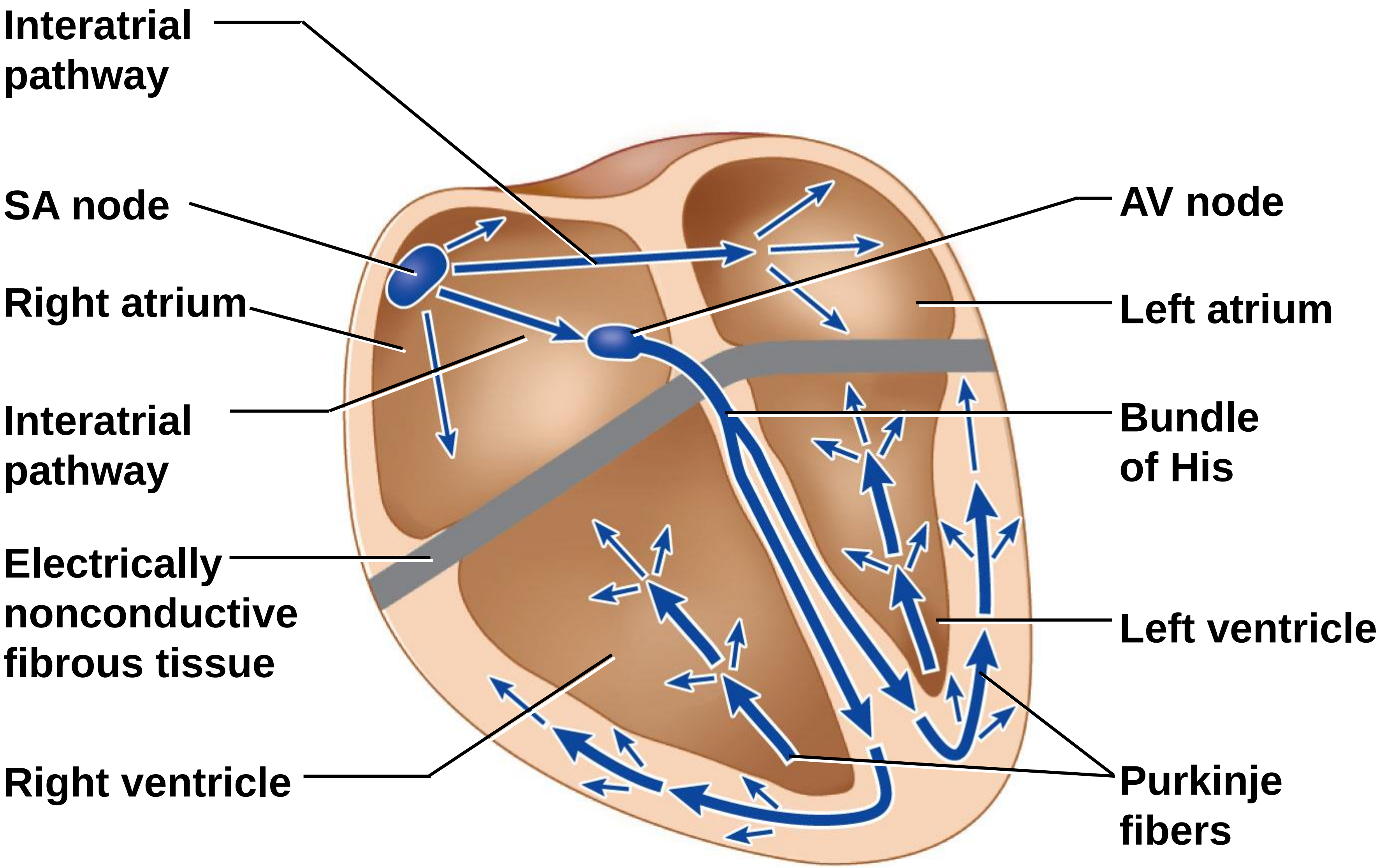
(a) Specialized conduction system of the heart

(b) Spread of cardiac excitation



(a) Specialized conduction system of the heart

Figure 9-23a p408



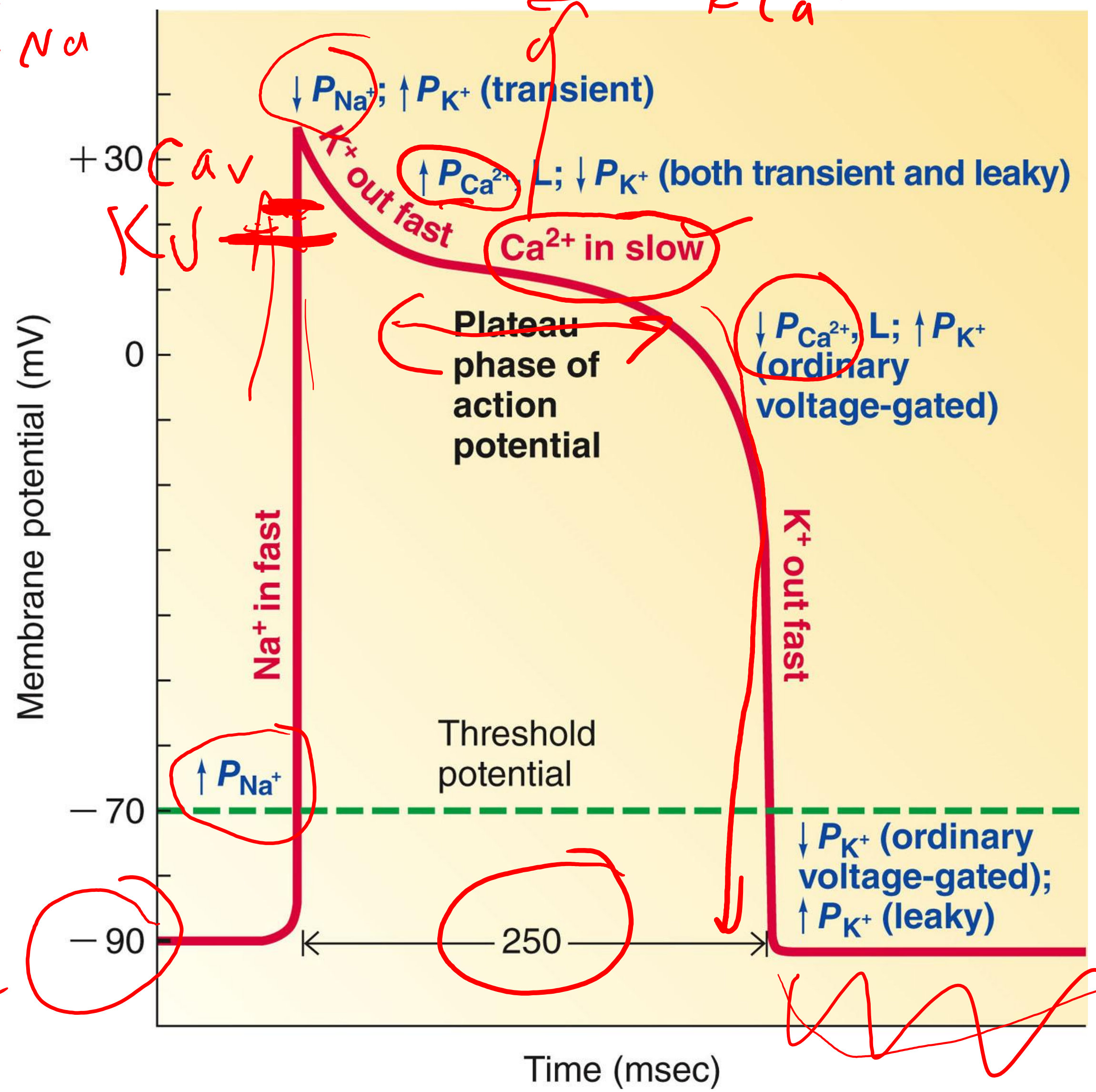
(b) Spread of cardiac excitation

Figure 9-23b p408

9.5 Circulatory Pumps: Heart Electrical Activity

- The action potential of contractile cardiac muscle cells shows a **plateau phase.**
  - Contractile cells remain **relaxed** until excited by adjacent cells through **gap junctions**
  - **Rising phase**
    - Opening of voltage-gated Na^+ channels
    - K^+ channels transiently open at the peak of action potential, producing slight repolarization
  - **Plateau phase**
    - Opening of "slow" L-type Ca^{2+} channels
    - Closing of K^+ channels
    - Prolongs contraction to aid pumping
  - **Falling phase**
    - Opening of voltage-gated K^+ channels

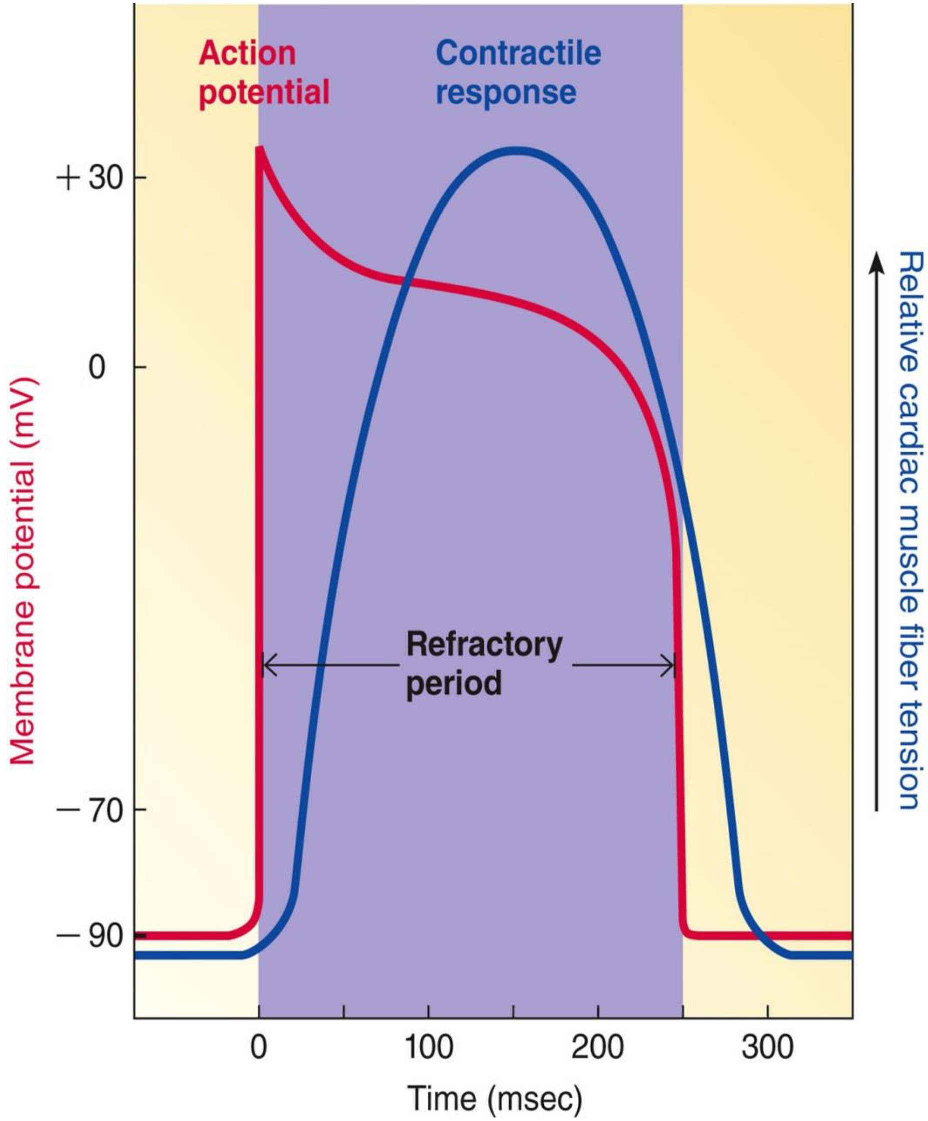
9.5 Circulatory Pumps: Heart Electrical Activity



9.5 Circulatory Pumps: Heart Electrical Activity

- Excitation-contraction coupling in the heart
  - **Ca^{2+} enters the cytosol** through L-type channels in T tubules
  - Triggers **release of Ca^{2+}** from sarcoplasmic reticulum
  - **Ca^{2+} binds** to troponin-tropomyosin complex to allow cross-bridge cycling
  - **Extent** and **duration** of cross-bridge activity varies with the **amount of cytosolic Ca^{2+}**
  - Cardiac muscle has a **long refractory period** (250 msec)
    - Na^{+} channels remain inactivated during the plateau phase
    - Prevents summation and tetanus

9.5 Circulatory Pumps: Heart Electrical Activity



9.5 Circulatory Pumps: Heart Electrical Activity

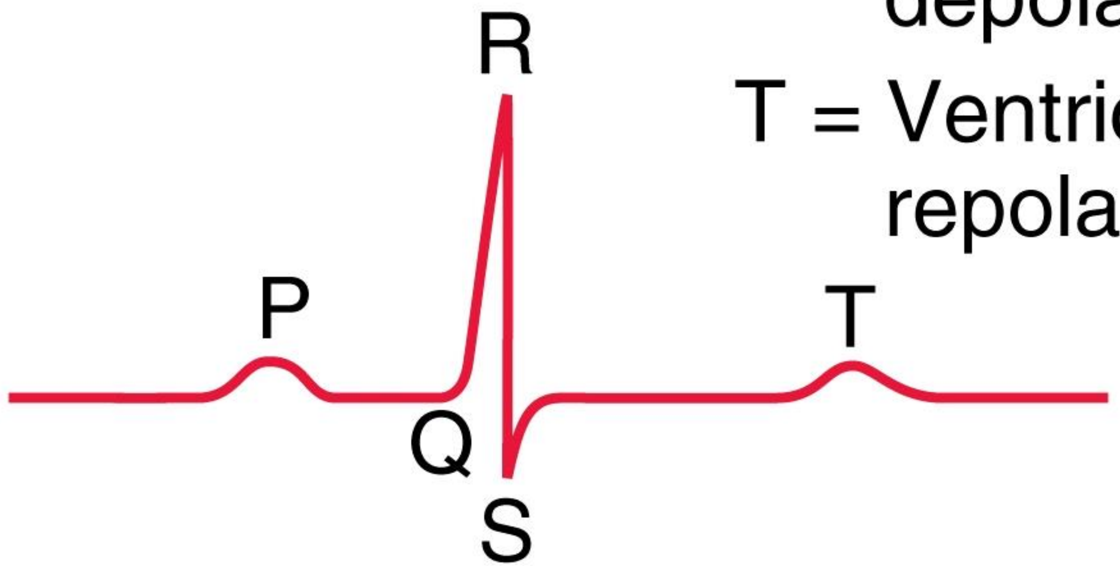
- Electrocardiogram (ECG)
  - Electrical currents generated by cardiac muscle can be detected using **recording electrodes** on the skin.
  - **Interpretation** of the ECG
    - **P wave** represents **atrial depolarization**
    - **QRS complex** represents **ventricular depolarization**
    - **T wave** represents **ventricular repolarization**
  - Periods of **no current flow**
    - **PR segment** represents **AV nodal delay**
    - **ST segment** represents **plateau phase**
    - **TP interval** represents **passive ventricular filling** while all chambers are at rest

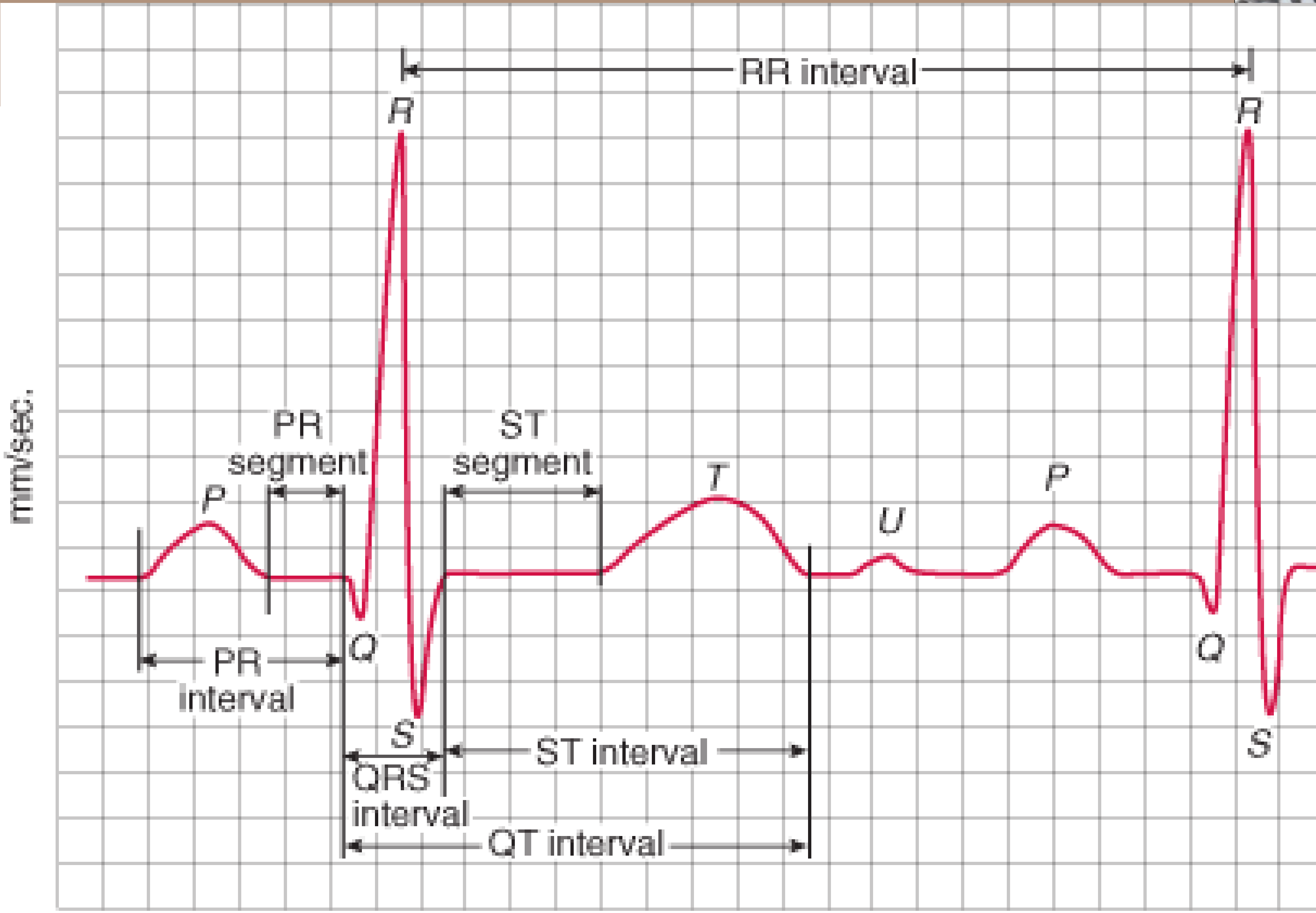
(a) Electrocardiogram

P = Atrial depolarization

Q,R,S = Ventricular depolarization

T = Ventricular repolarization



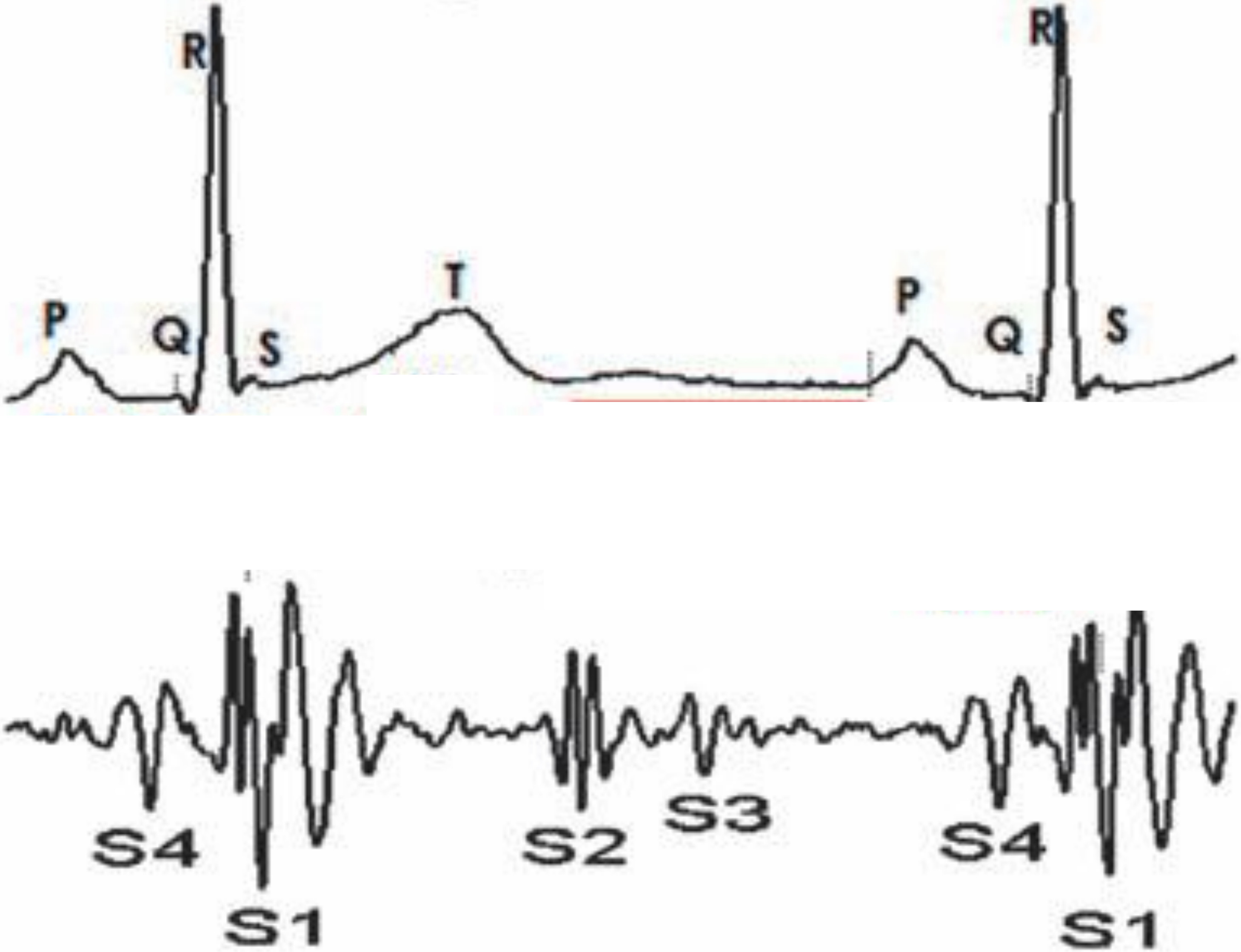


mm/mV 1 square = 0.04 sec/0.1mV

Table 5.1 Components of the ECG & Typical Lead II Values*

ECG COMPONENT		*Measurement area…*	*Represent…*	Duration *(seconds)*	*Amplitude (millivolts)*
Waves	**P**	begin and end on isoelectric line (baseline); normally upright in standard limb leads	depolarization of the right and left atria.	0.07 – 0.18	< 0.25
	QRS complex	begin and end on isoelectric line (baseline) from start of Q wave to end of S wave	depolarization of the right and left ventricles. Atrial repolarization is also part of this segment, but the electrical signal for atrial repolarization is masked by the larger QRS complex (see Fig 5.2)	0.06 – 0.12	0.10 – 1.50
	T	begin and end on isoelectric line (baseline)	repolarization of the right and left ventricles.	0.10 – 0.25	< 0.5
Intervals	**P-R**	from start of P wave to start of QRS complex	time from the onset of atrial depolarization to the onset of ventricular depolarization.	0.12-0.20	
	Q-T	from start of QRS complex to end of T wave	time from onset of ventricular depolarization to the end of ventricular repolarization. It represents the refractory period of the ventricles.	0.32-0.36	
	R-R	from peak of R wave to peak of succeeding R wave	time between two successive ventricular depolarizations.	0.80	
Segments	**P-R**	from end of P wave to start of QRS complex	time of impulse conduction from the AV node to the ventricular myocardium.	0.02 – 0.10	
	S-T	between end of S wave and start of T wave	period of time representing the early part of ventricular repolarization during which ventricles are more or less uniformly excited.	< 0.20	
	T-P	from end of T wave to start of successive P wave	time from the end of ventricular repolarization to the onset of atrial depolarization.	0.0 – 0.40	

ECG Tracing



Normal heart sounds are associated with heart valves closing, causing changes in blood flow

Common:

S_1 : The first heart tone, or **S_1**, forms the "lubb" of "lubb-dub“
Closure of the atrioventricular valves

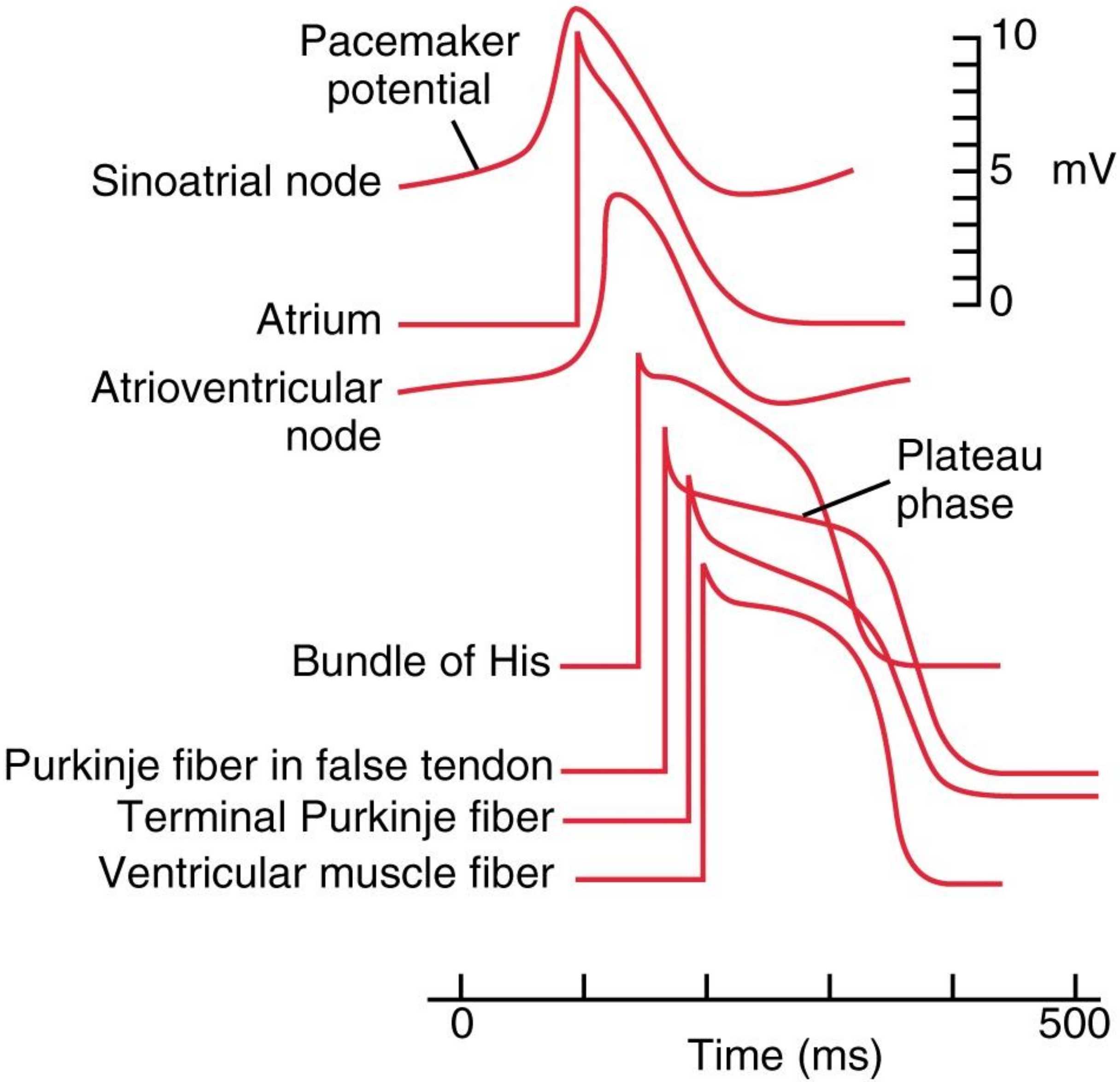
S_2: The second heart tone, or **S_2**, forms the "dub" of "lubb-dub“
Closure of the aortic valve and pulmonary valve at the end of ventricular systole

Not so common: In young children can hear. IN NEONATES ONE SHOULD NOT HEAR S3 AND S4.

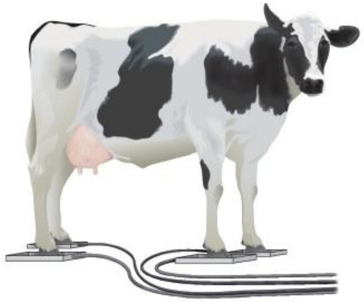
S_3: Also called a **protodiastolic gallop**, **ventricular gallop**, or informally the "Kentucky" gallop (**S1=ken; S2=tuc; S3=ky**). It is not of valvular origin.
S3 is thought to be caused by the oscillation inrushing blood from the atria.

S_4: The rare fourth heart sound is sometimes audible in healthy children and again in trained athletes. Is called a **presystolic gallop** or **atrial gallop**. Producing a rhythm sometimes referred to as the "**Tennessee**" gallop where S4 represents the "tenn-" syllable.

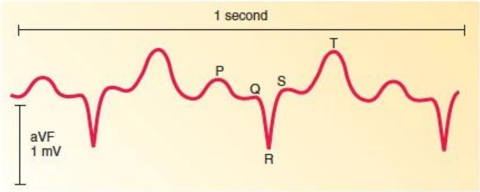
(b) Cardiac action potentials



9.5 Circulatory Pumps: Heart Electrical Activity



(a)



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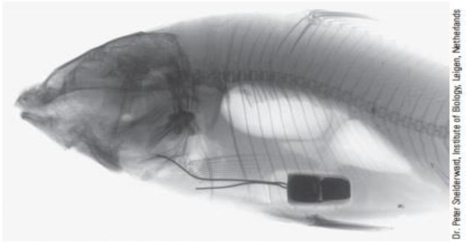
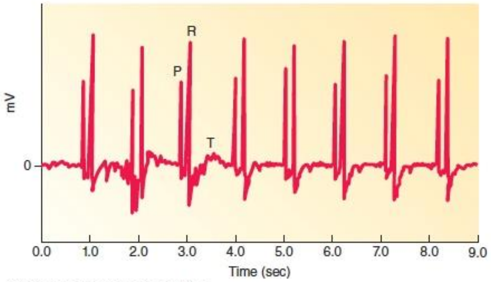
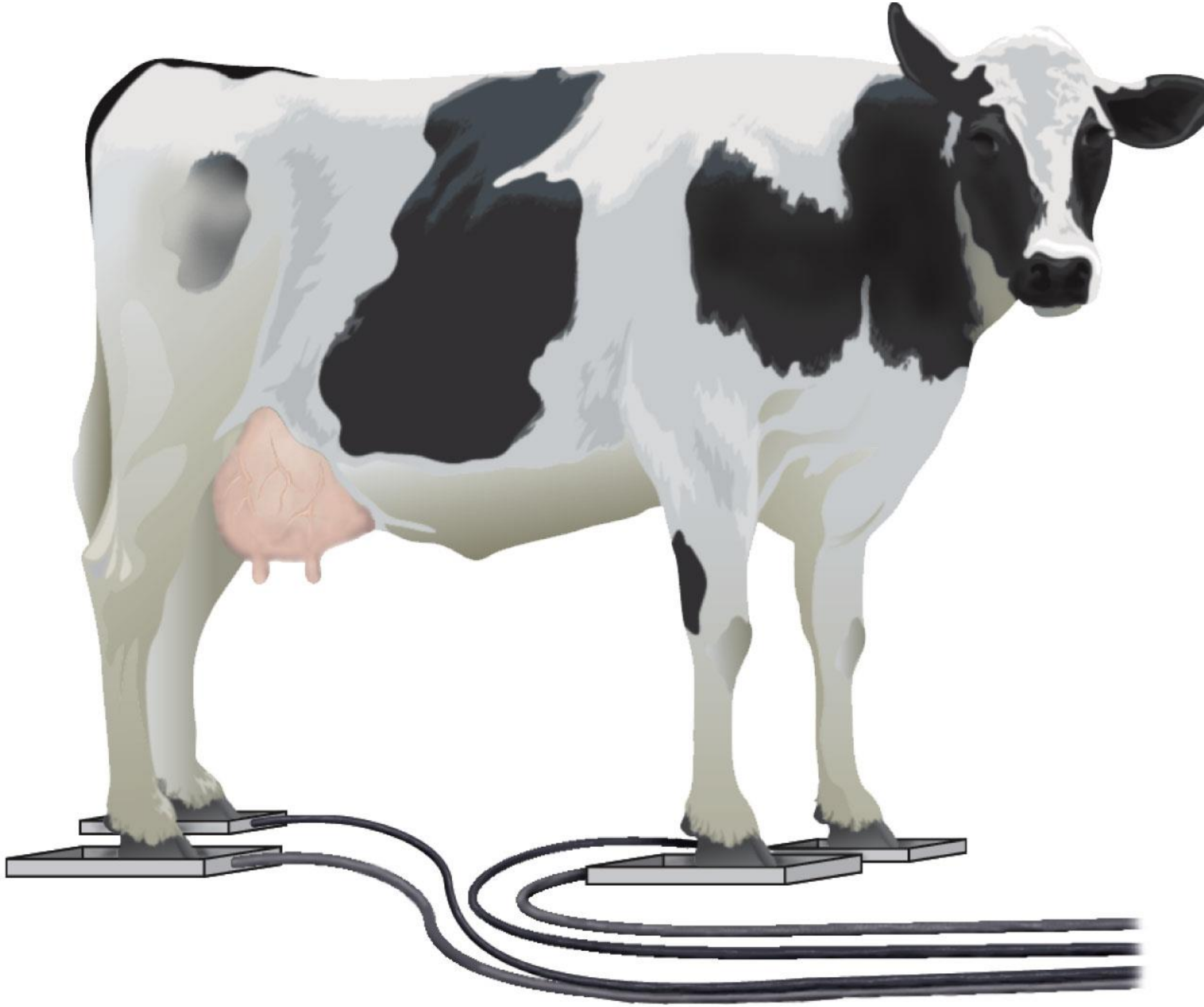


(c) Goldfish with implanted sensor for ECG

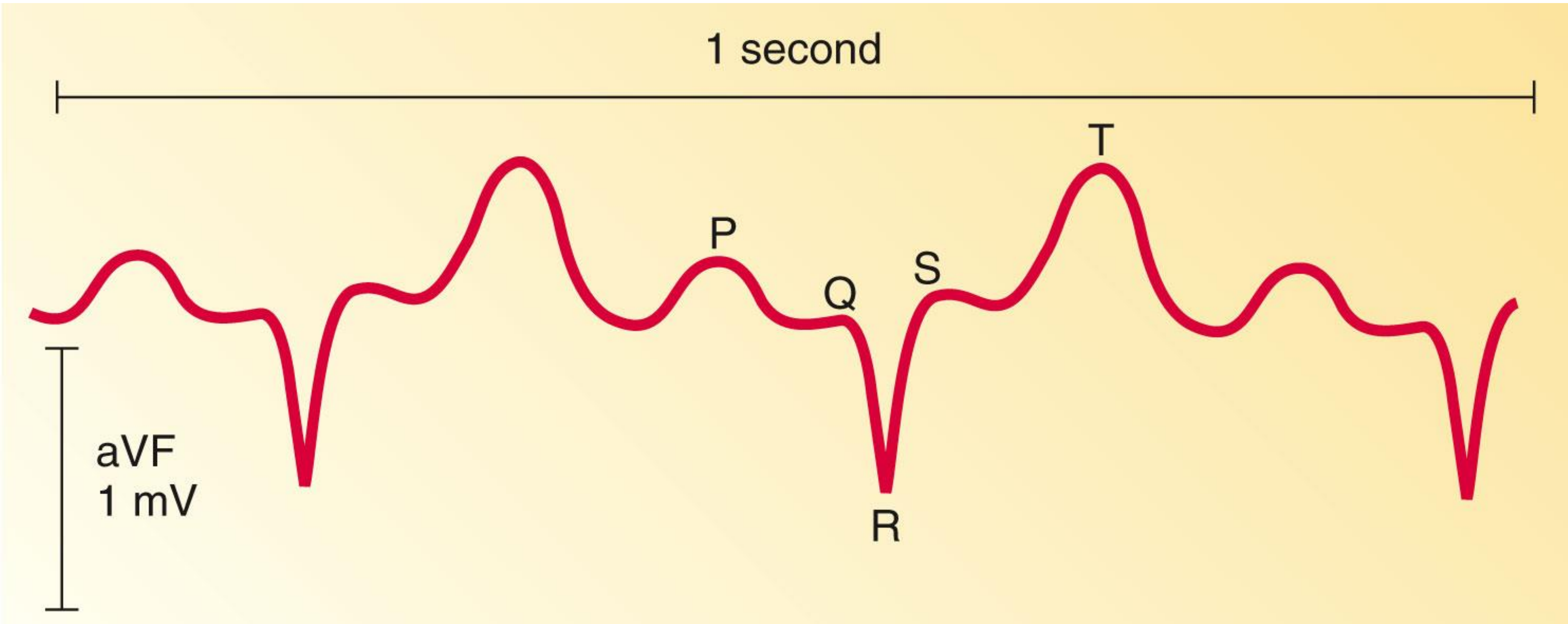


(d) Electrocardiogram of a goldfish



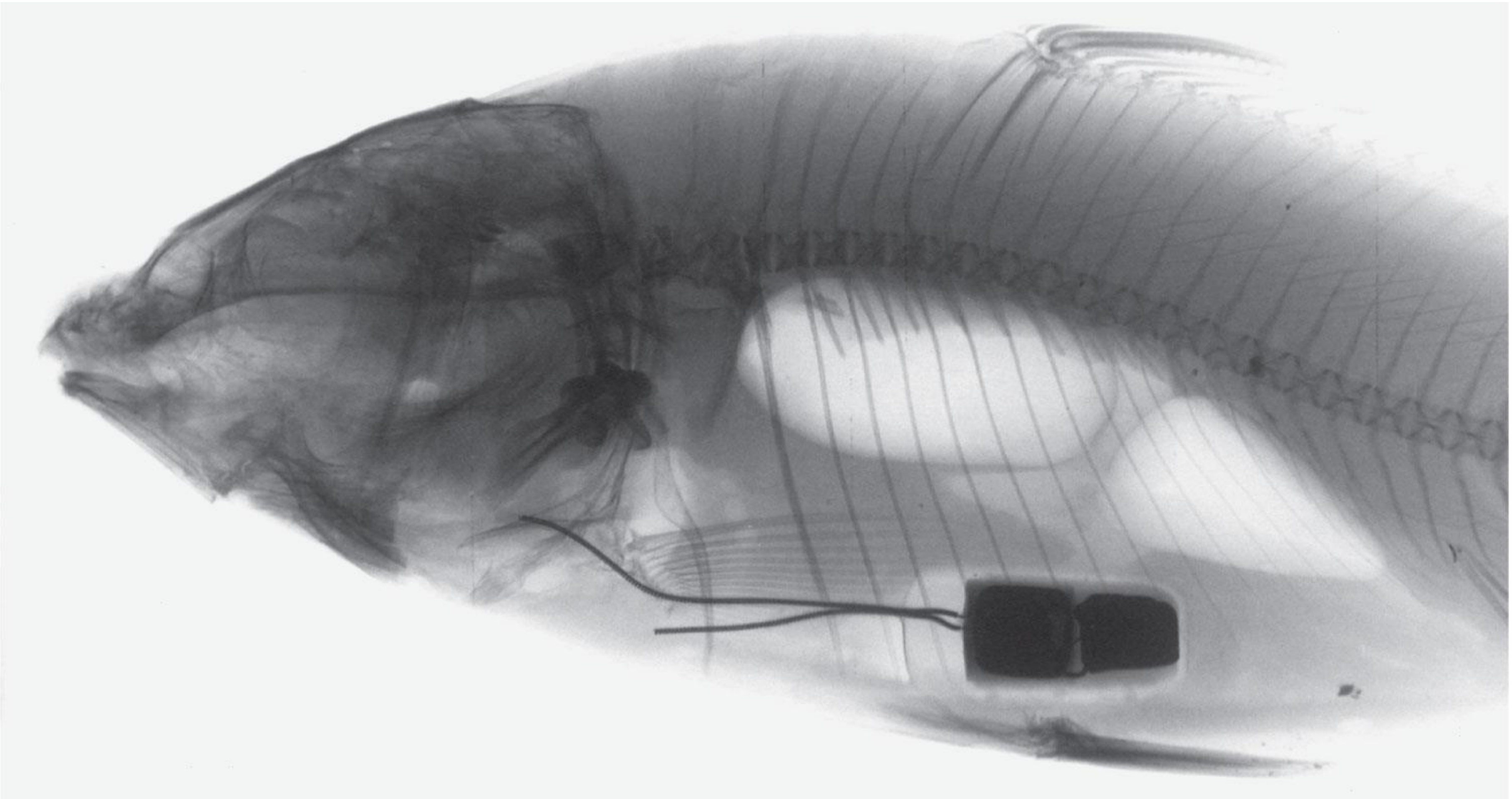
(a)

Figure 9-26a p411

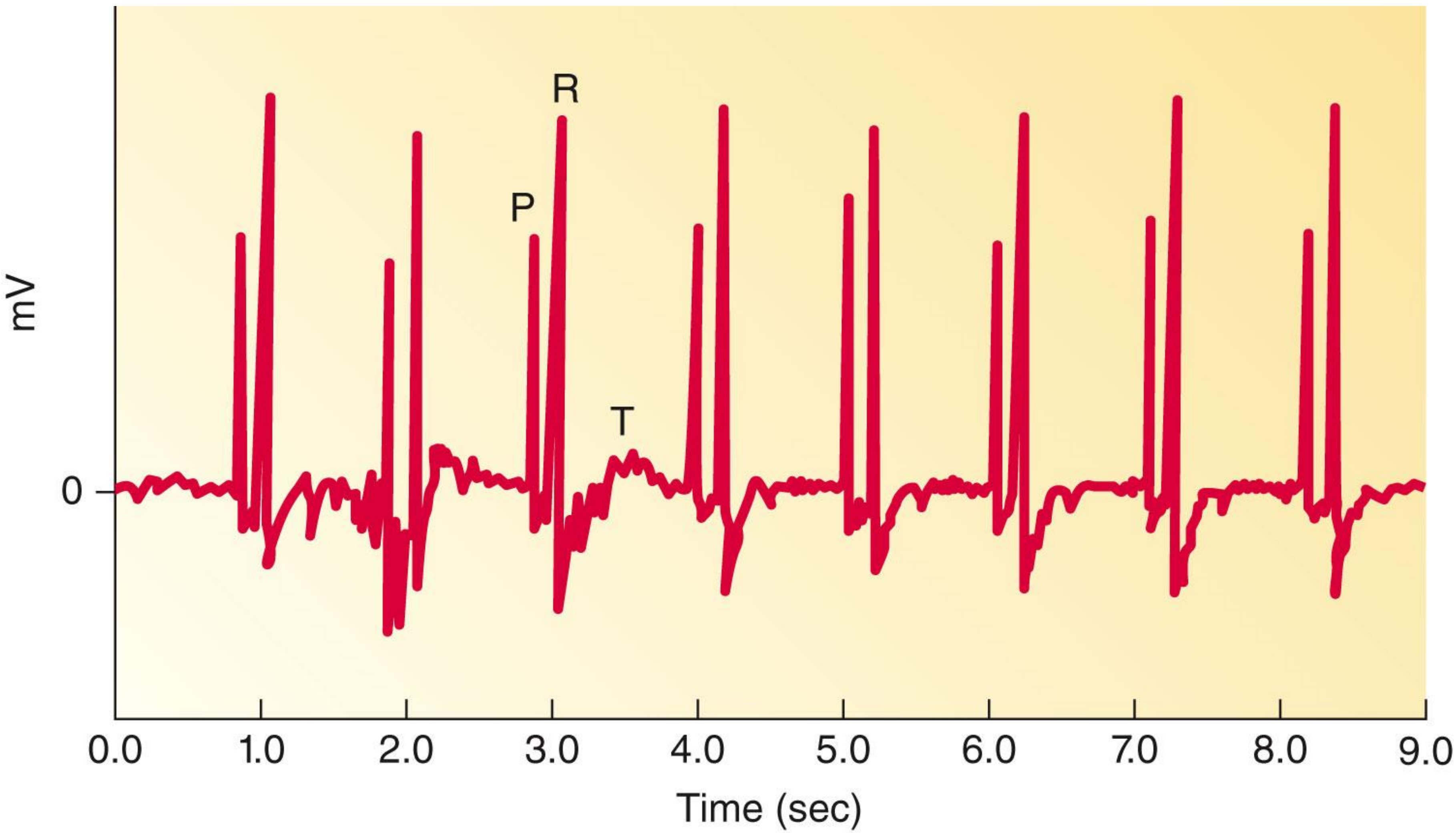


(b)

Figure 9-26b p411



(c) Goldfish with implanted sensor for ECG



(d) Electrocardiogram of a goldfish

Figure 9-26d p411

9.6 Circulatory Pumps: Heart Mechanics and the Cardiac Cycle

- The **cardiac cycle** consists of alternating periods of **systole** and **diastole**
  - **Systole** is the period of contraction and emptying
  - **Diastole** is the period of relaxation and filling
  - Events are the **same** on the left and right sides of the heart
  - **Pressures are lower** on the right

9.6 Circulatory Pumps: Heart Mechanics and the Cardiac Cycle

- Events of the cardiac cycle

1. **Early ventricular diastole**
   - AV valves are open; aortic and pulmonary valves are closed
   - Blood flows from veins into atria into ventricles
2. **Late ventricular diastole**
   - SA node fires and atria depolarize (P wave)
   - Atria contract, increasing atrial pressure
3. **End of ventricular diastole**
   - Ventricular filling is completed (end-diastolic volume)
4. **Ventricular excitation and onset of ventricular systole**
   - Impulse passes through AV node and bundle of His and depolarizes ventricles (QRS complex)
   - Ventricles contract, increasing ventricular pressure
   - AV valves close

9.6 Circulatory Pumps: Heart Mechanics and the Cardiac Cycle

- Events of the cardiac cycle

5. **Isovolumetric ventricular contraction**
   - Ventricles are closed chambers while ventricular pressure rapidly rises

6. **Ventricular ejection**
   - When ventricular pressure exceeds arterial blood pressure, aortic and pulmonary valves open
   - Blood is forced into arteries and ventricular volume decreases substantially

7. **End of ventricular systole**
   - Ventricular emptying is completed (end-systolic volume)
   - Amount of blood pumped out is the stroke volume

8. **Ventricular repolarization and onset of ventricular diastole**
   - Ventricles repolarize (T wave)
   - Ventricular pressure falls below arterial blood pressure and aortic and pulmonary valves close (dichrotic notch)

9.6 Circulatory Pumps: Heart Mechanics and the Cardiac Cycle

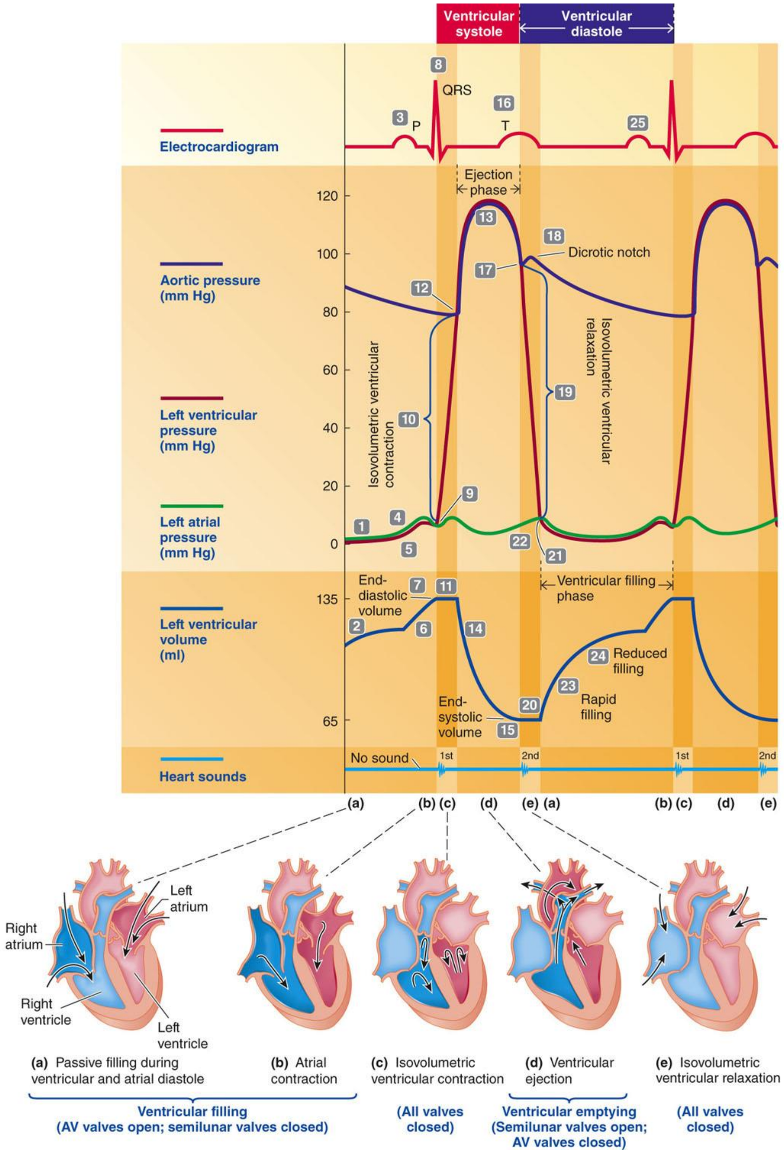
- Events of the cardiac cycle

9. **Isovolumetric ventricular relaxation**
   - Ventricles are closed chambers while ventricular pressure rapidly falls

10. **Ventricular filling**
    - When ventricular pressure falls below atrial pressure, AV valves open
    - Ventricles begin to fill with blood

- **Cardiac cycle repeats** beginning with firing of SA node

9.6 Circulatory Pumps: Heart Mechanics and the Cardiac Cycle



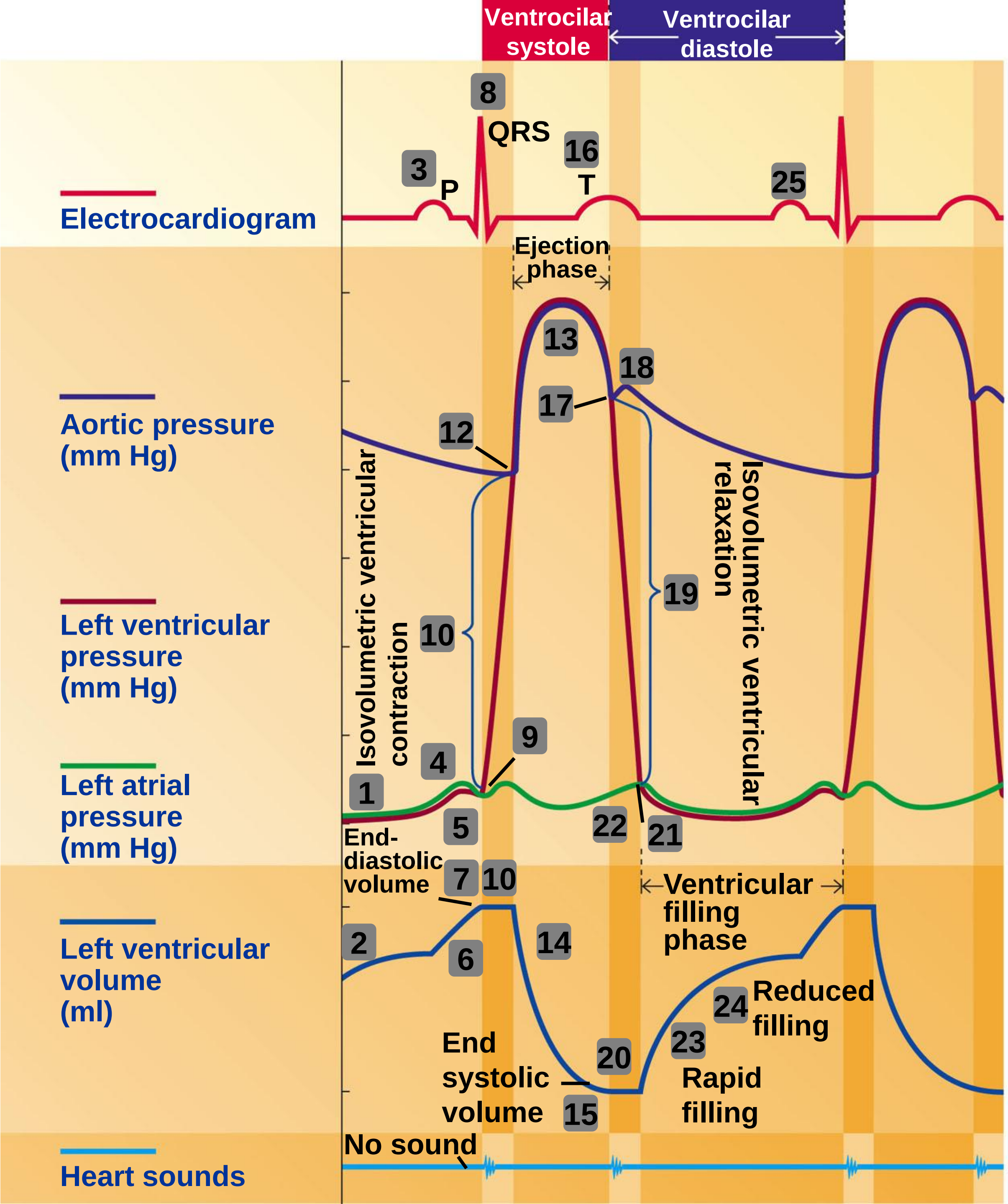


Figure 9-27 p413

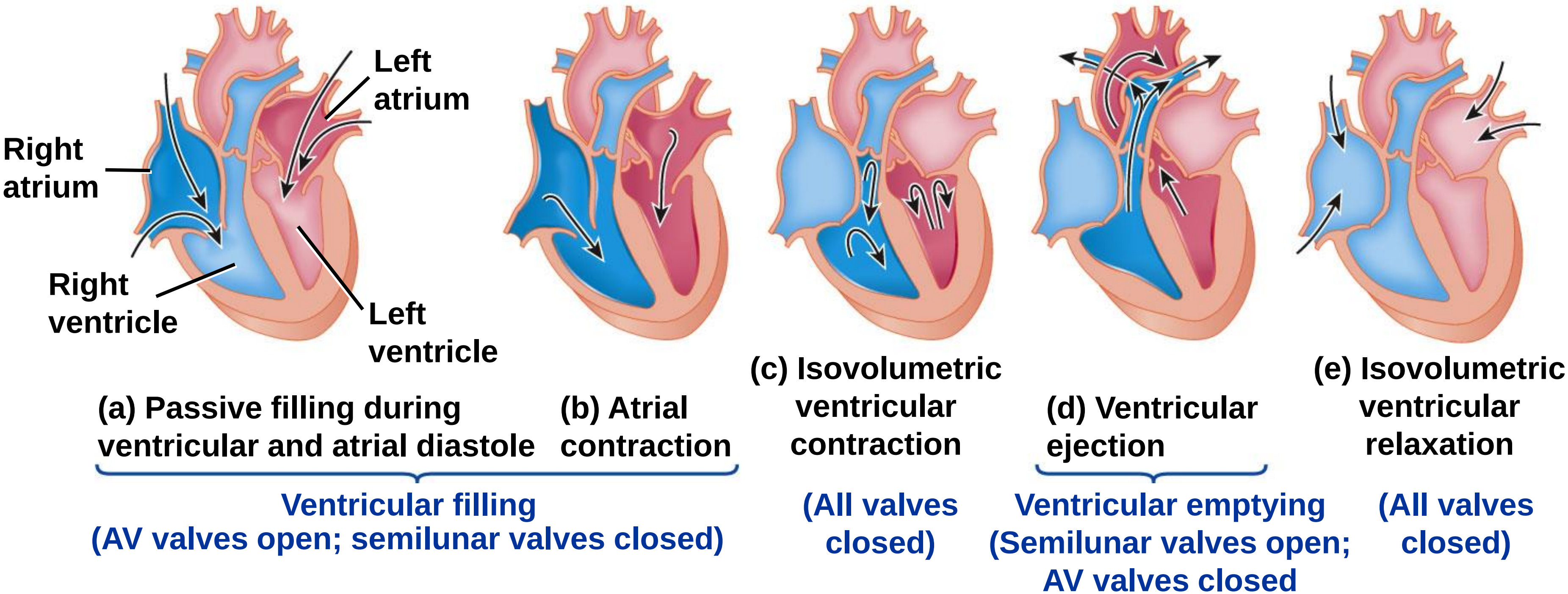


Figure 9-27 p413

9.7 Circulatory Pumps: Cardiac Output and Its Control

- **Cardiac output** (C.O.) is the volume of blood pumped per minute by a heart to the body.

Cardiac output = heart rate x stroke volume

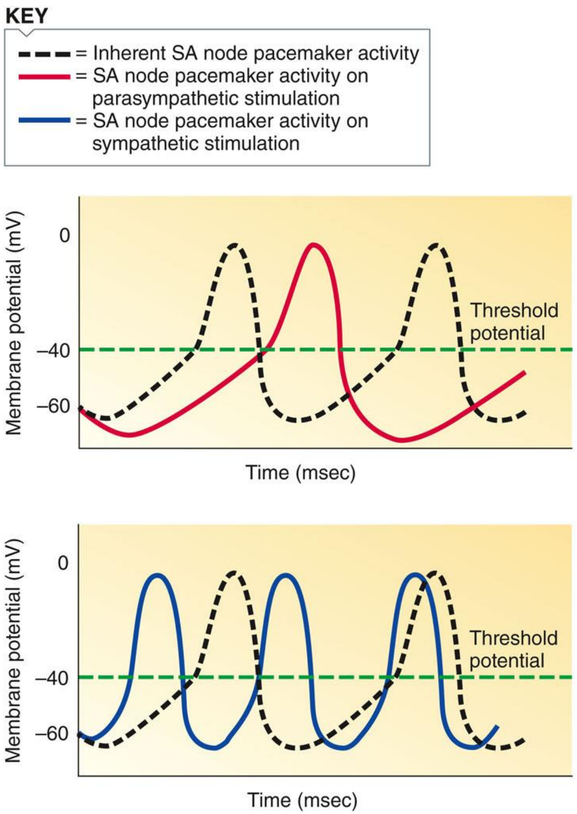
- Larger animals have slower heart rates, but larger stroke volumes

- Cardiac output **increases** with **warmer body temperature**, **age** during development, and **increased activity level.**

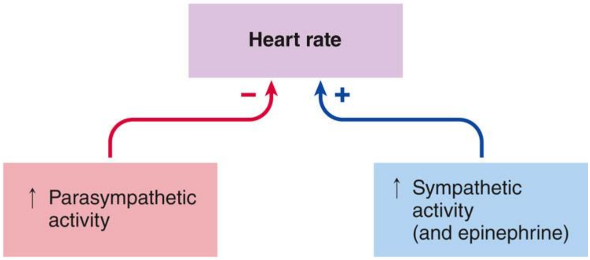
9.7 Circulatory Pumps: Cardiac Output and Its Control

- **Heart rate** is determined by **antagonistic regulation** by the **autonomic nervous system**
  - Coordinated by the **cardiovascular control center** in the **brain stem**
  - **ACh** from **vagus nerve** binds to **muscarinic receptors**
    - Decreases heart rate (SA node)
    - Decreases excitability of the AV node
    - Shortens the plateau phase of atrial contractile cells
  - **NE** from **sympathetic neurons** and **epinephrine** from the **adrenal medulla** bind to **β_1-adrenergic receptors**
    - Increases heart rate
    - Reduces AV nodal delay
    - Speeds the spread of action potentials through the conduction pathway
    - Increases contractile strength of atrial and ventricular cells

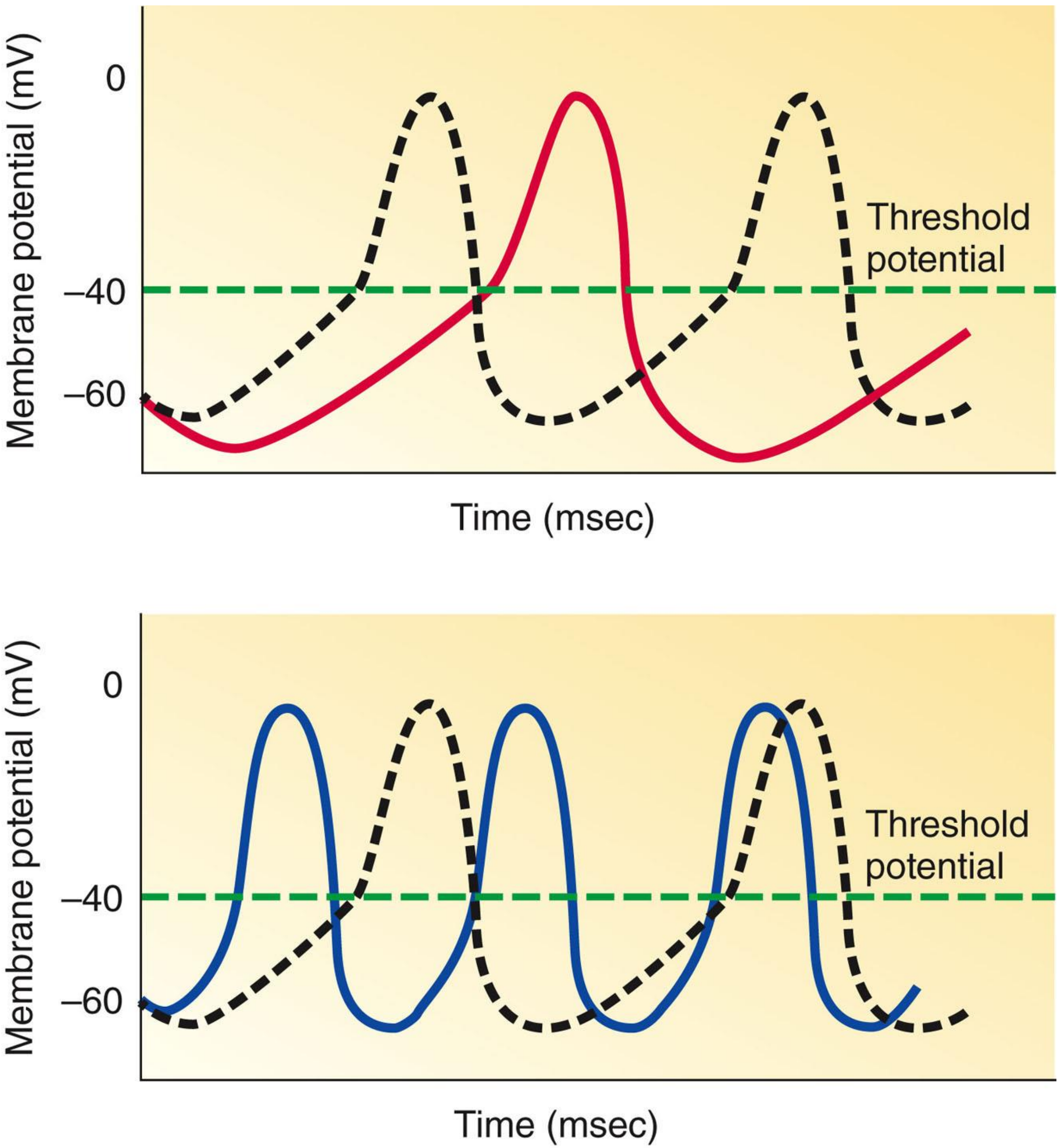
9.7 Circulatory Pumps: Cardiac Output and Its Control



(a) Autonomic influence on SA node potential

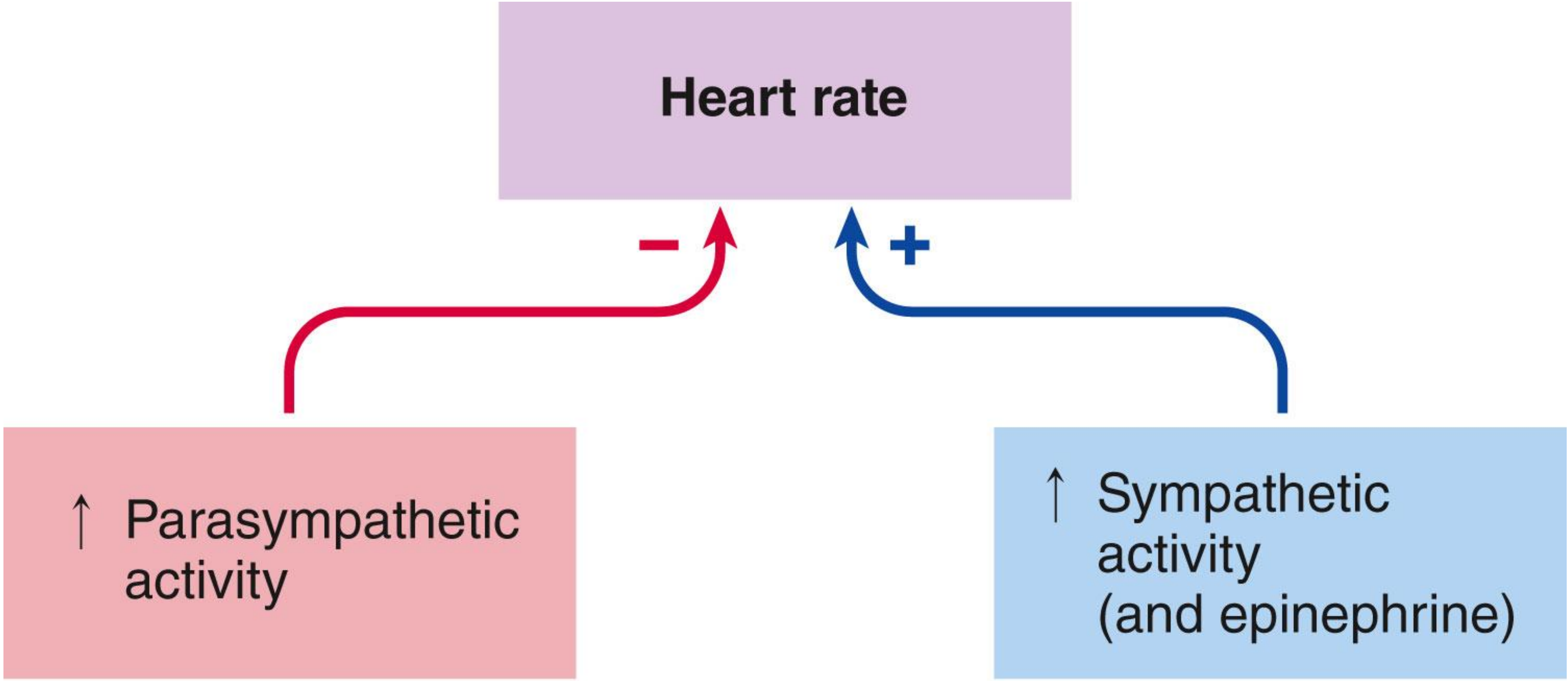


(b) Control of heart rate by autonomic nervous system



(a) Autonomic influence on SA node potential

Figure 9-28a p416



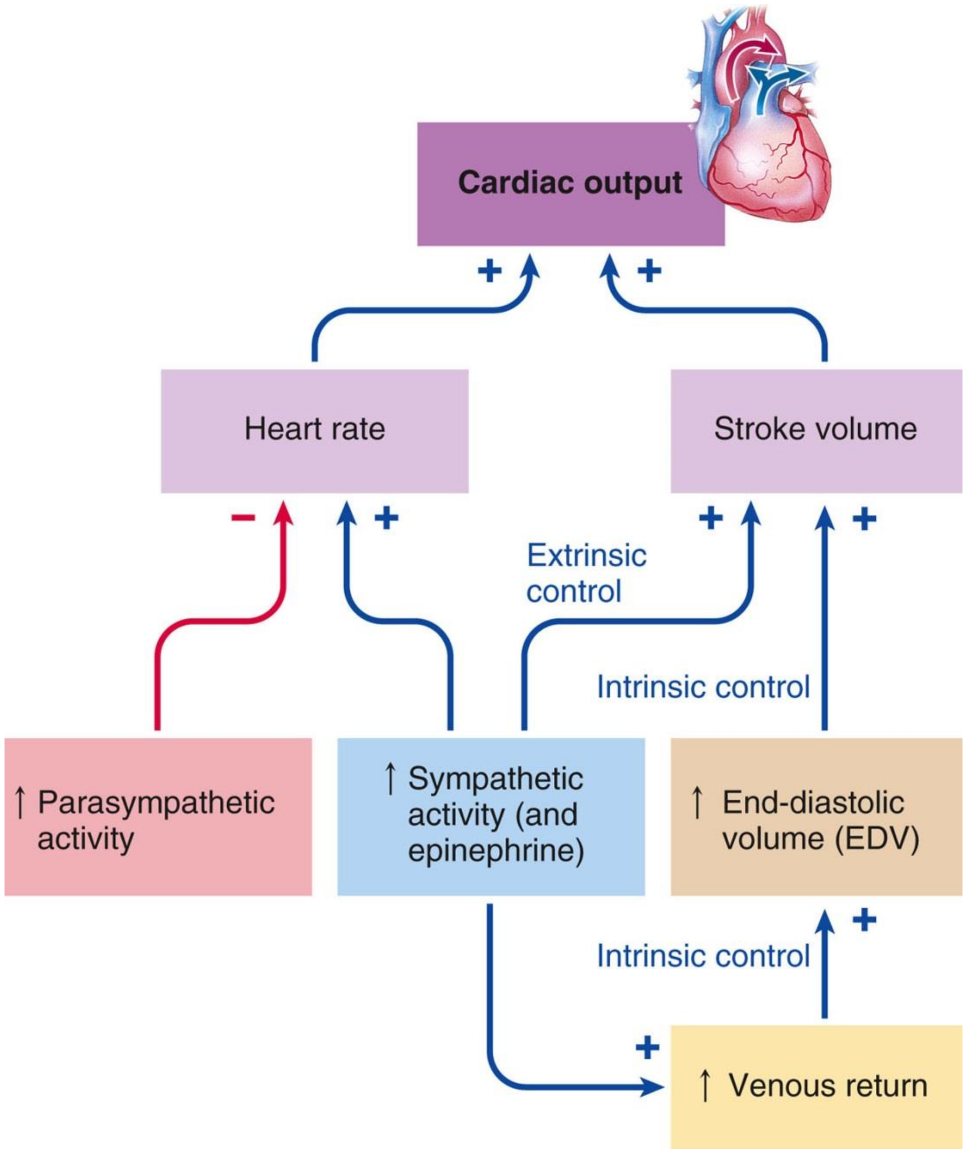
(b) Control of heart rate by autonomic nervous system

Figure 9-28b p416

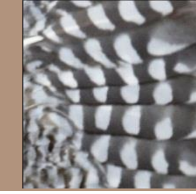
9.7 Circulatory Pumps: Cardiac Output and Its Control

- Control of stroke volume
  - **Intrinsic control**
    - Direct correlation between **end-diastolic volume (EDV)** and **stroke volume (SV)**
      - Depends on the **length-tension relationship** of cardiac muscle
      - The greater the volume of blood entering the heart, the greater the volume ejected (**Frank-Starling law of the heart**)
  - **Extrinsic control**
    - **Sympathetic stimulation** enhances contractility of the heart
    - **Sympathetic stimulation** constricts veins, enhancing venous return and increasing stroke volume

9.7 Circulatory Pumps: Cardiac Output and Its Control



9.7 Circulatory Pumps: Cardiac Output and Its Control



- The heart receives its blood supply through the **coronary circulation**

  - Heart muscle cannot extract oxygen or nutrients from blood within its chambers

  - **Coronary arteries** first evolved in active fishes
    - Branch off of brachial arteries leaving the gills
    - Coronary arteries branch off of the aorta in mammals

  - Coronary **blood flow increases** during activity
    - **Dilation** of coronary vessels is induced by **adenosine**
      - Adenosine is formed from ATP when oxygen supplies are low or cardiac activity is increased

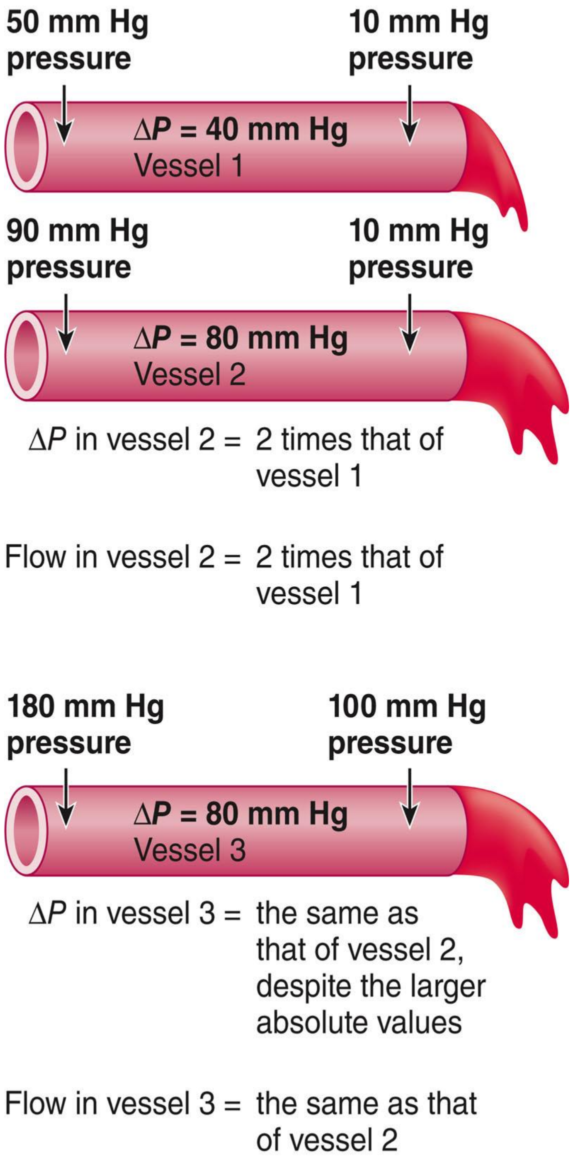
  - **Obstruction of coronary arteries** is a leading cause of death in humans

9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution

- Fluid flow obeys the **hemodynamic flow law**

$$Q = \Delta P / R$$

Q = flow rate of fluid through a vessel
ΔP = pressure gradient
R = resistance



9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution

- Fluid flow obeys the **hemodynamic flow law**
  $Q = \Delta P/R$

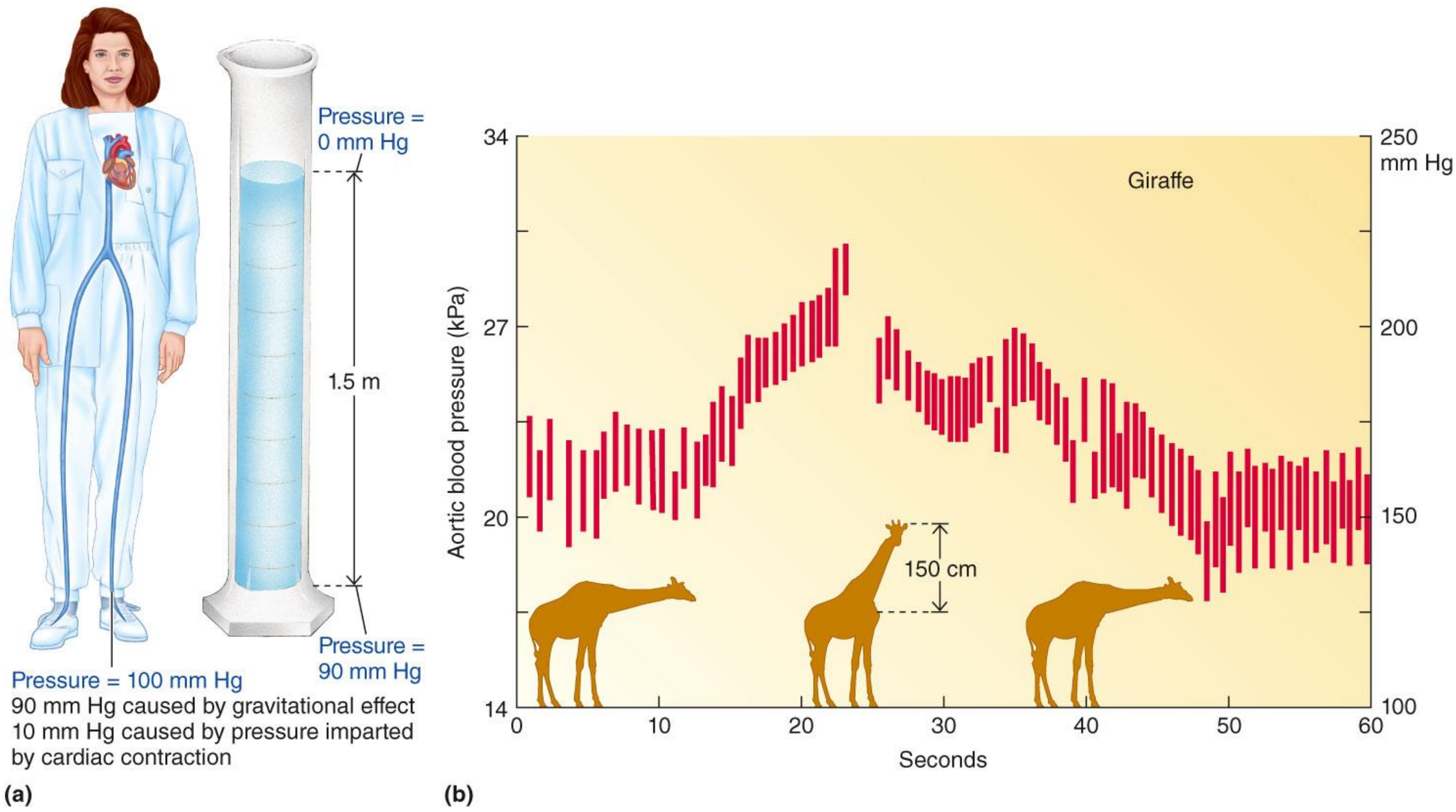
  - **Pressure gradient** is the main **driving force** for flow through a vessel

    - Blood flows from an area of **higher pressure** to an area of **lower pressure**

    - **Contraction** of the heart **generates pressure**

    - **Gravity** also contributes to the pressure gradient
      - 70 mmHg pressure is required to push a column of water up 1 meter
      - A human needs an average driving pressure of 100 mmHg to overcome gravity
      - Fishes require a much lower driving pressure (~40 mmHg)

9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution



9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution

- Fluid flow obeys the **hemodynamic flow law** $Q = \Delta P/R$

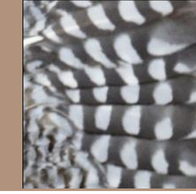
  - **Resistance** is the hindrance to blood flow through a vessel caused by friction

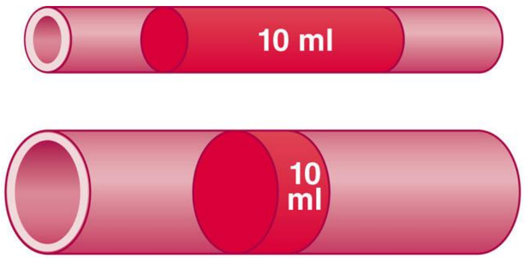
    $$R = 8\eta L/\pi r^4$$

    R = resistance
    η = viscosity of the fluid
    L = length of the vessel
    r = radius of the vessel

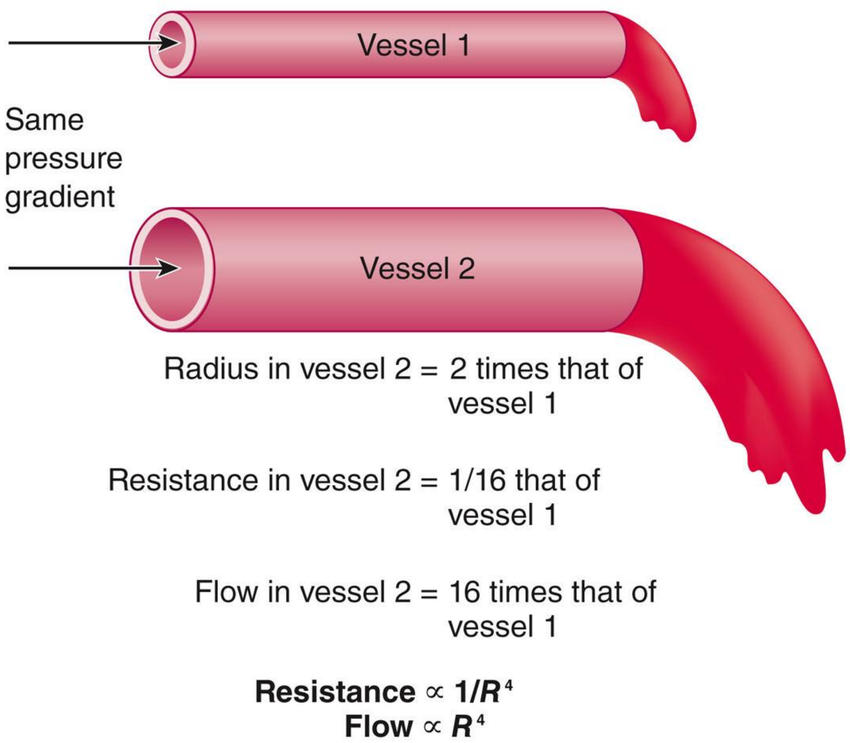
  - The major determinant of resistance to flow is the **radius** of the vessel
    - Resistance is inversely proportional to the 4th power of the radius

9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution

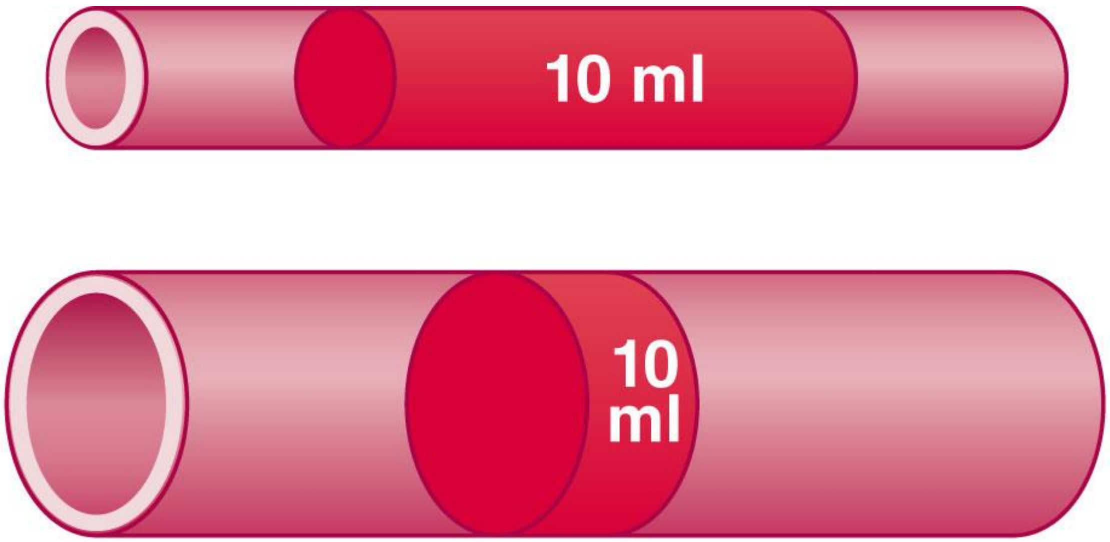




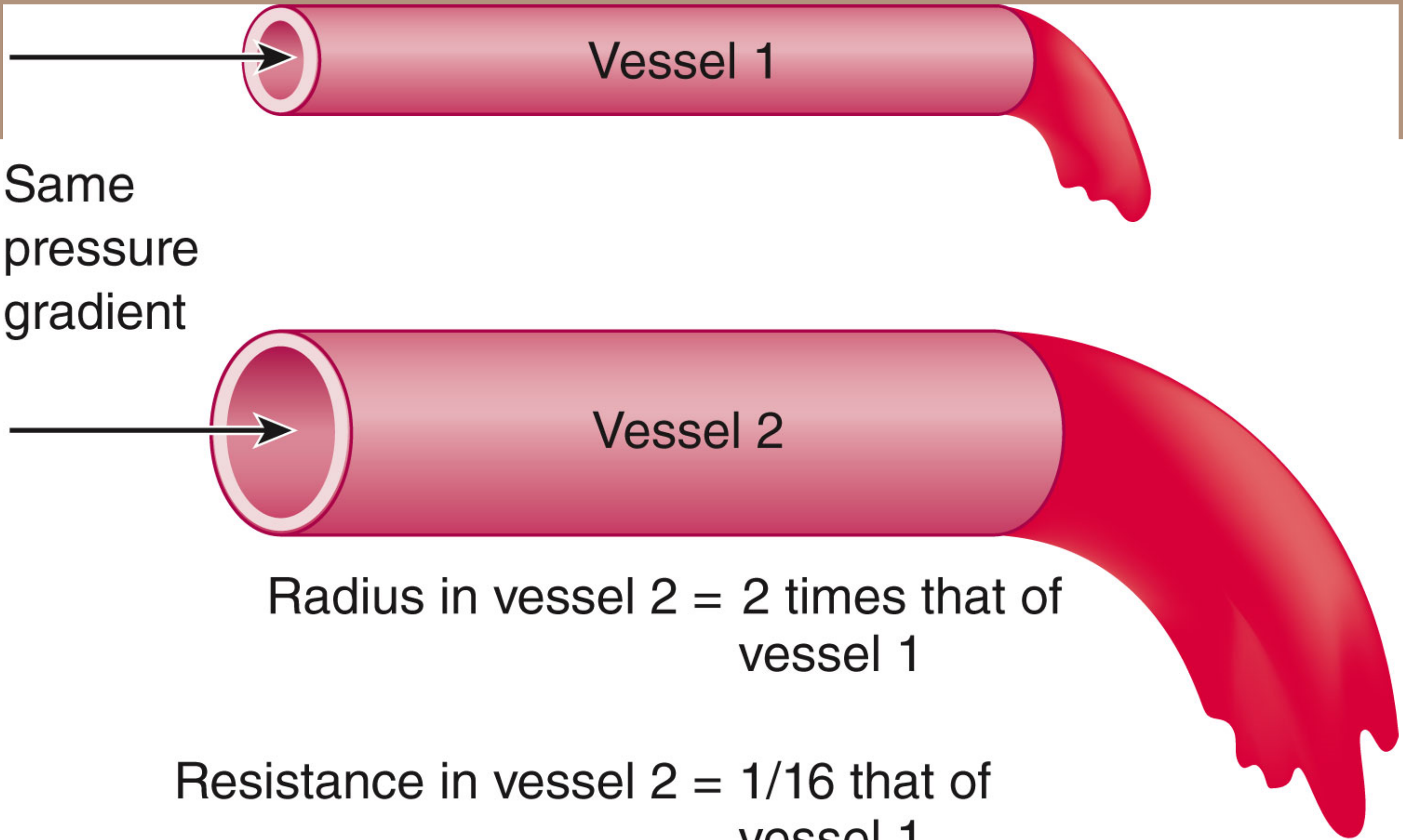
(a) Comparison of contact of a given volume of blood with the surface area of a small-radius vessel and a large-radius vessel



(b) Influence of vessel radius on resistance and flow



(a) Comparison of contact of a given volume of blood with the surface area of a small-radius vessel and a large-radius vessel



Radius in vessel 2 = 2 times that of vessel 1

Resistance in vessel 2 = 1/16 that of vessel 1

Flow in vessel 2 = 16 times that of vessel 1

Resistance $\propto$ **1/*R*^{4}**

Flow $\propto$ ***R*^{4}**

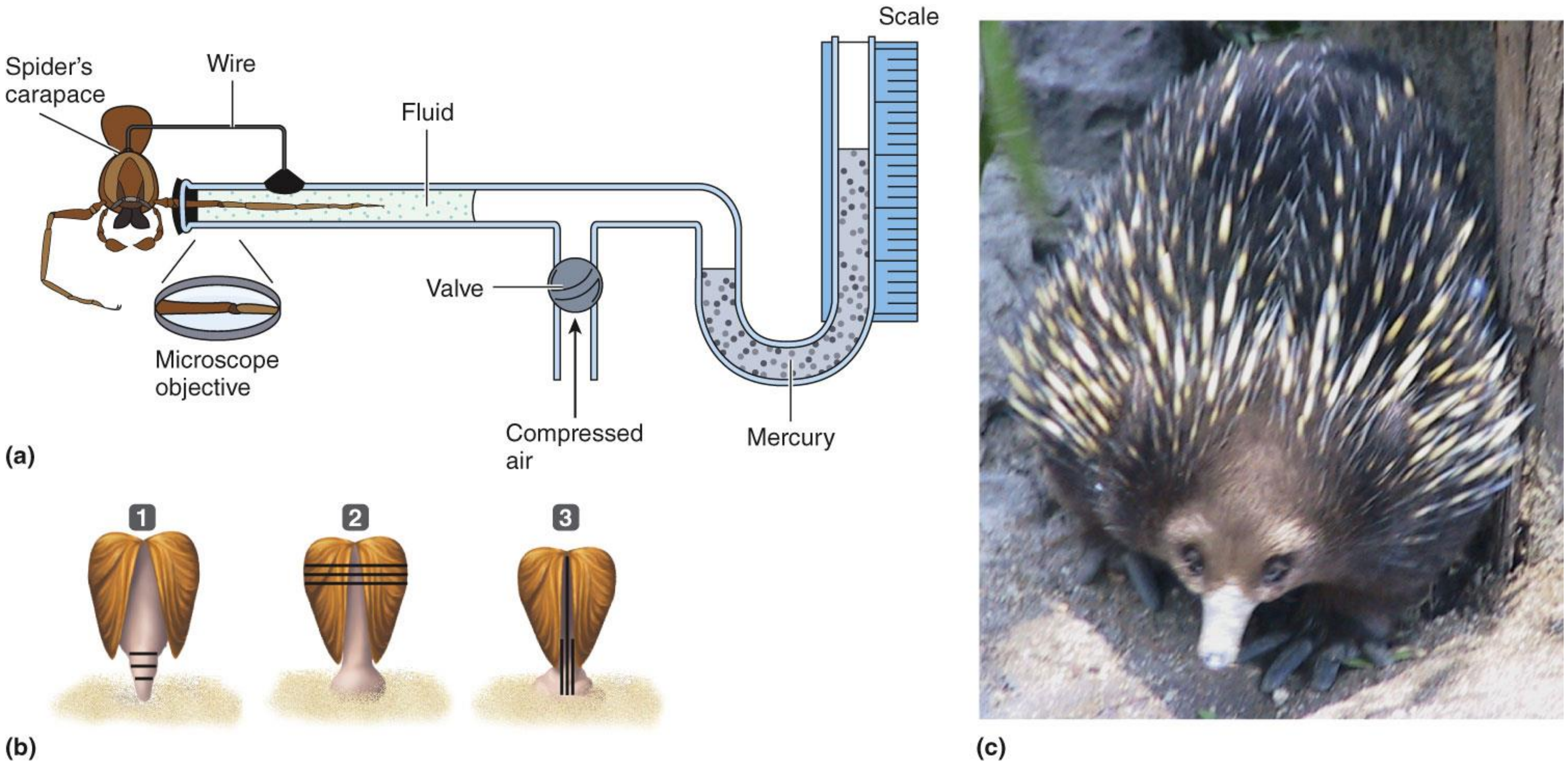
(b) Influence of vessel radius on resistance and flow

Figure 9-34b p421

9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution

- Pressure can be used to exert force for **noncirculatory** functions
  - **Movement**
    - Extending legs of arachnids
    - Extending foot in bivalves
  - **Ultrafiltration**
    - Interaction between capillary blood and ECF
    - Initial process of urine formation
  - **Erection**
    - Arousal of penis and clitoris
    - Snout of echidna

9.8 Circulatory Pathways and Vessels: Hemodynamics and Evolution



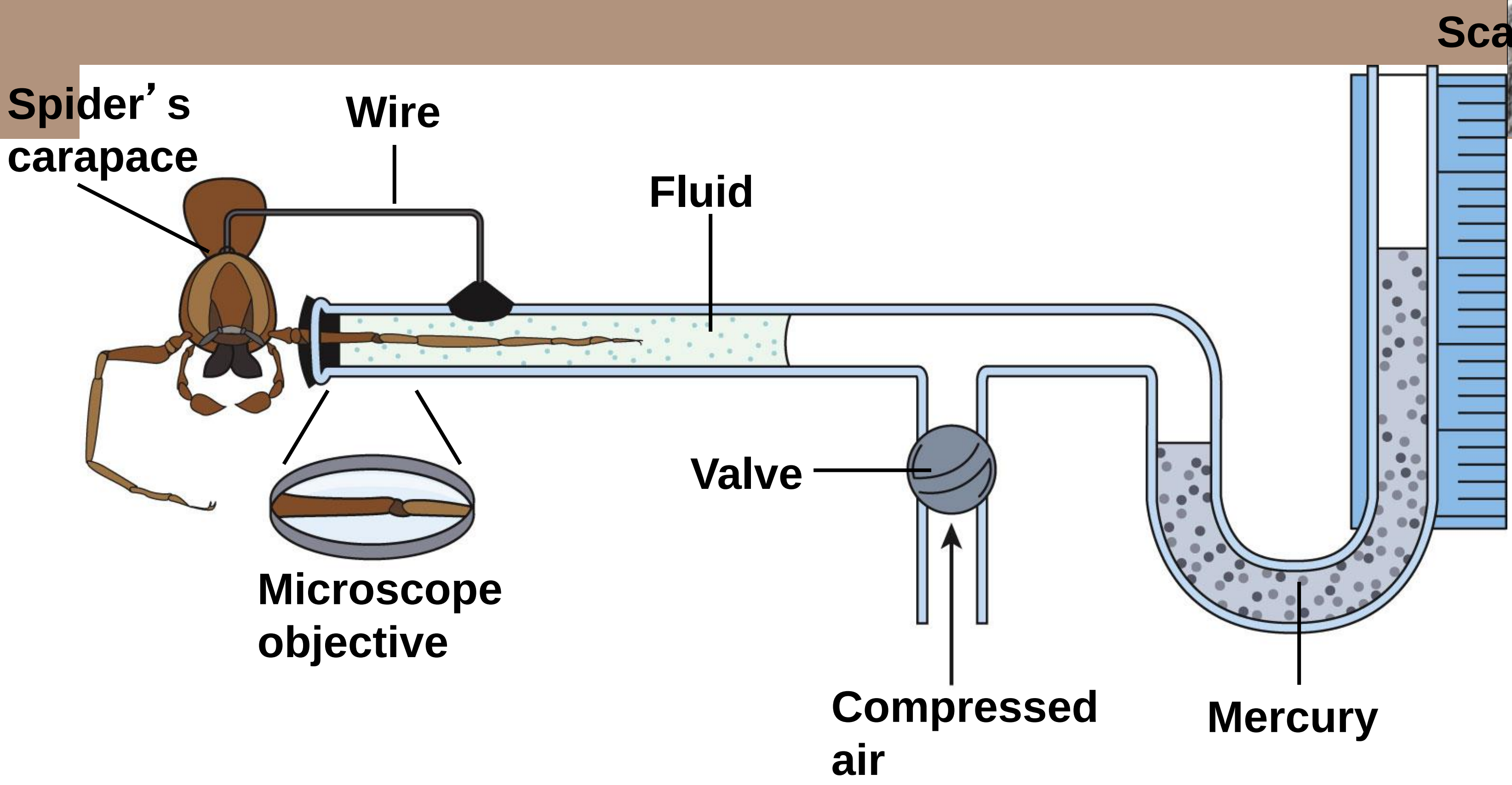
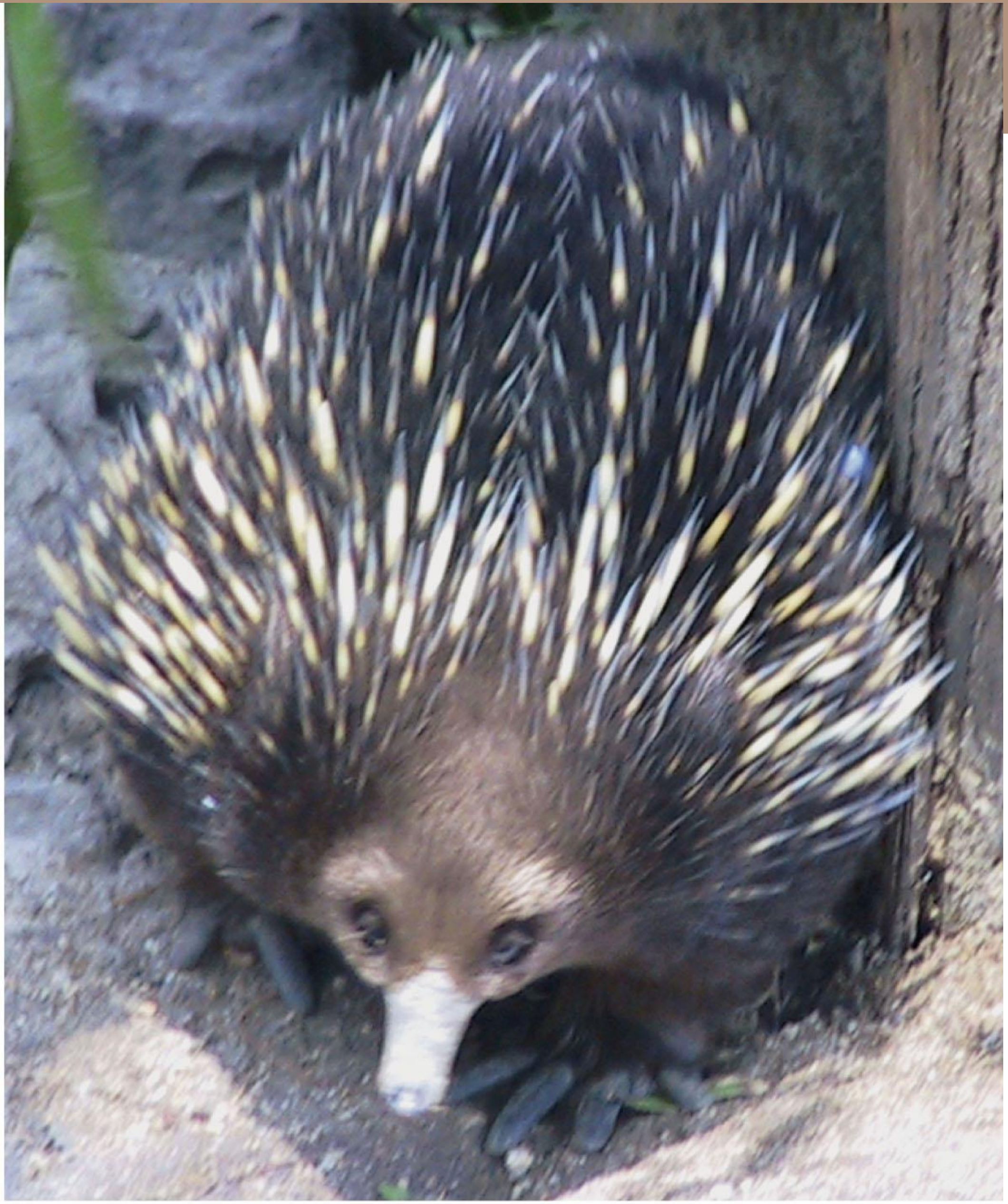


Figure 9-35a p423



(c)

Figure 9-35b p423

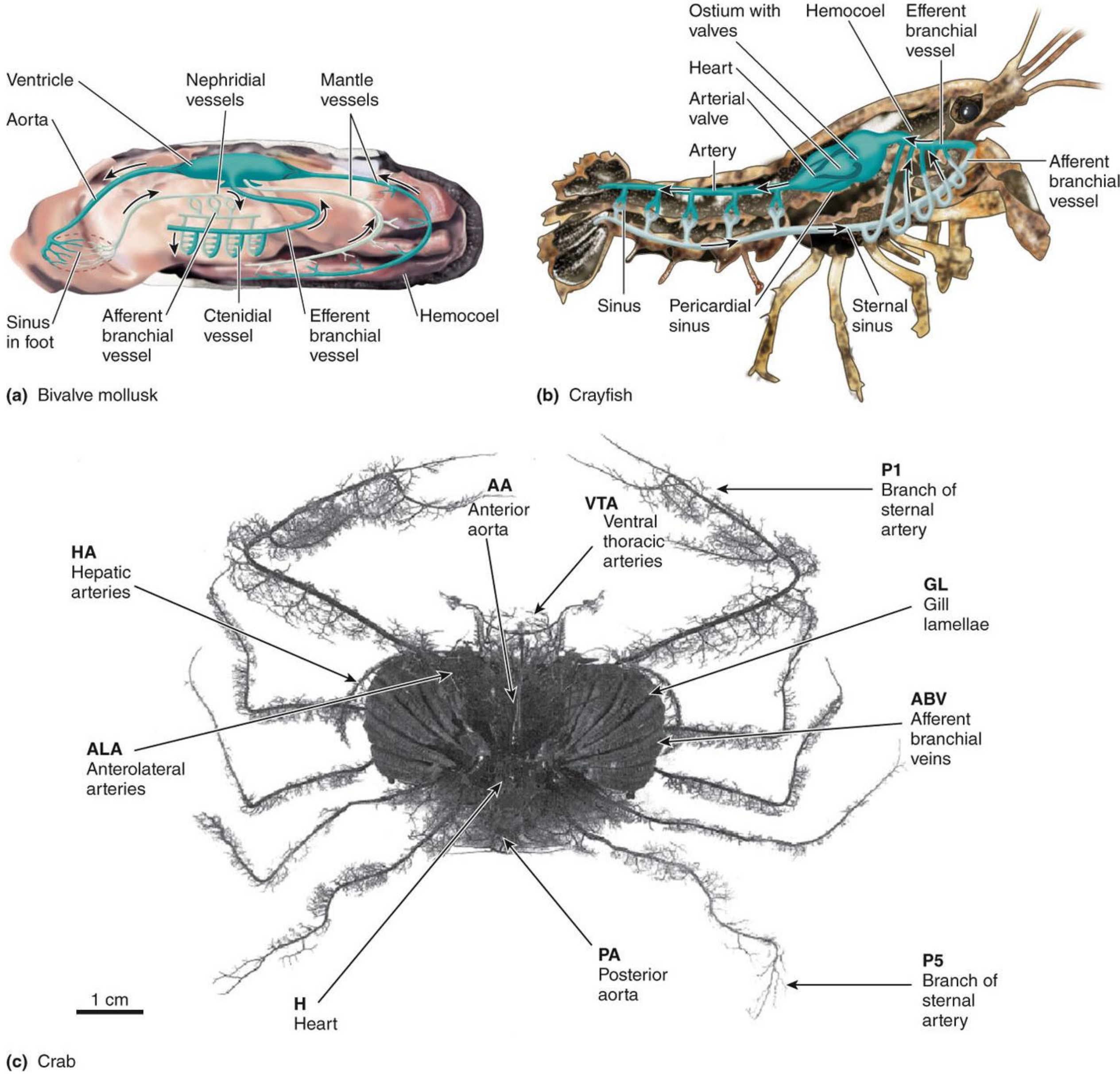
9.9 Circulatory Pathways and Vessels: Open Circulation

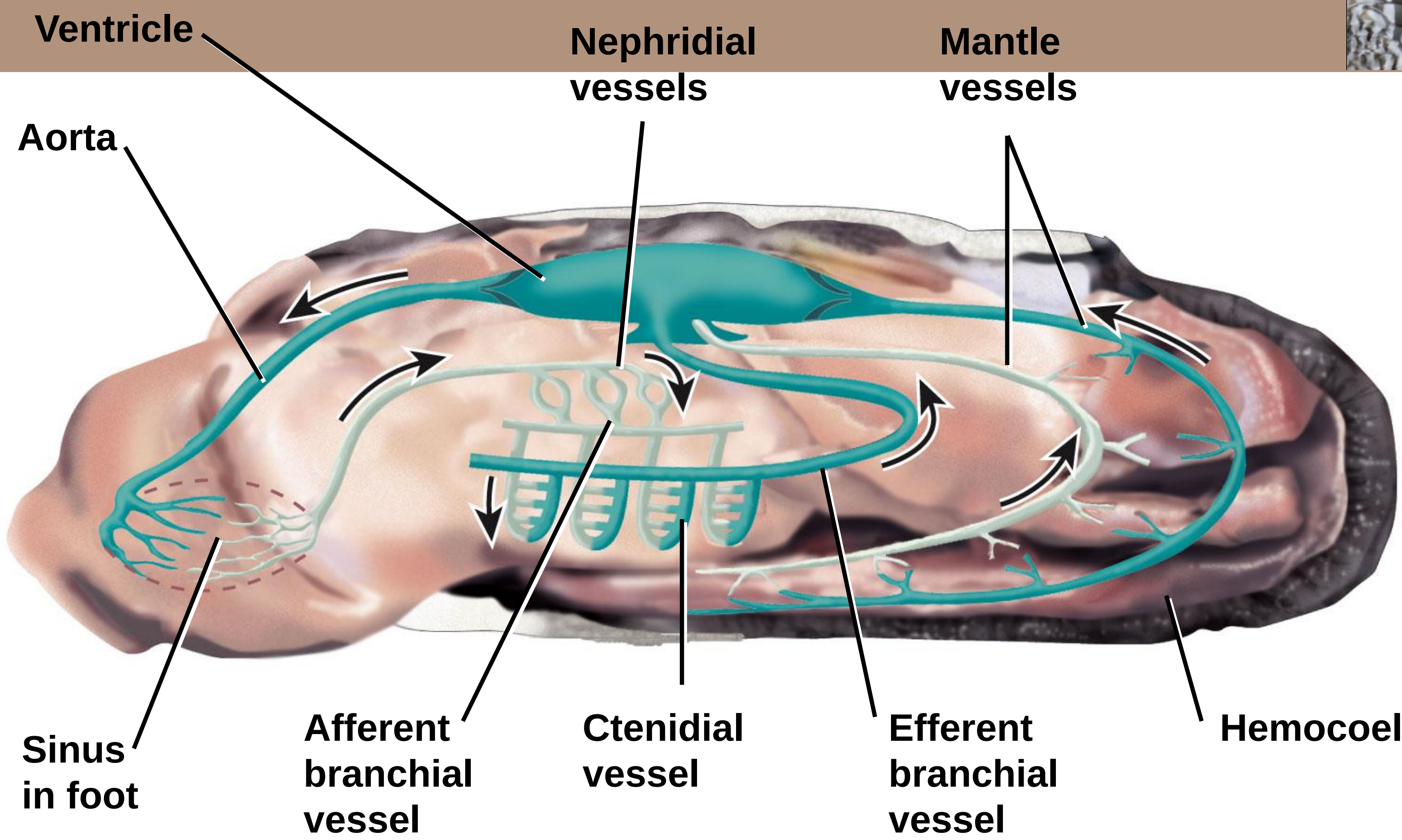
- Some nonvertebrates have **two or more separate ECF compartments**
  - Example: Echinoderms have four different ECF compartments
- **Non-cephalopod mollusks**
  - Open circulation with **myogenic chamber heart**
  - Despite having no capillaries, there is directionality to hemolymph flow (e.g. clams control hemolymph for burrowing)

9.9 Circulatory Pathways and Vessels: Open Circulation

- **Crustacea**
  - Decapods have a well-developed circulatory system with a **neurogenic chamber heart** and numerous **parallel** structures
  - Some decapods have **capillary-like arteries** opening into local **lacunae** and larger **sinuses** within target tissues
  - All hemolymph flows to the gills before returning to the heart
- **Insects**
  - **Tubular heart** and much less branching than crustaceans
  - Insects do not rely on circulation for oxygen delivery

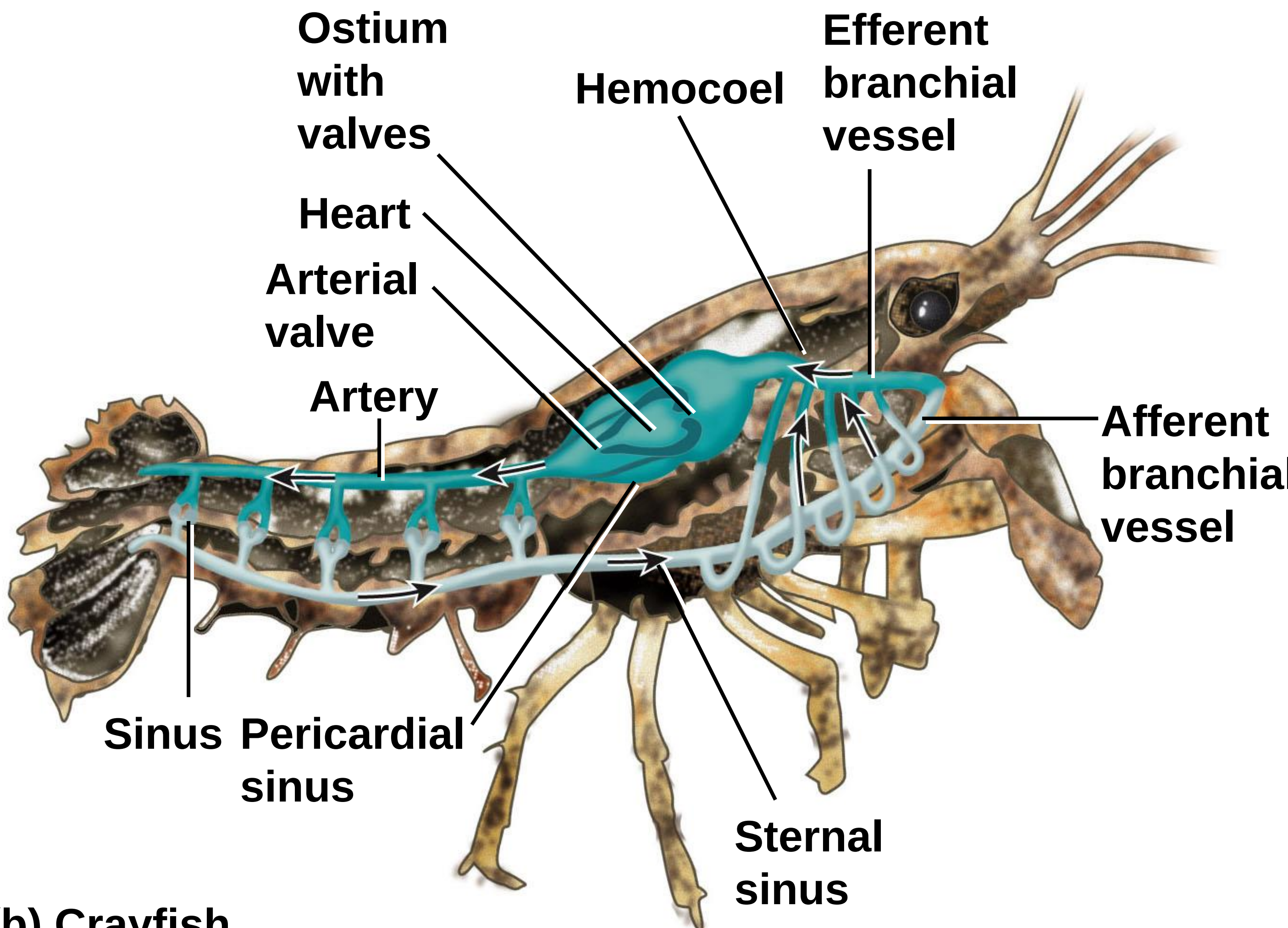
9.9 Circulatory Pathways and Vessels: Open Circulation





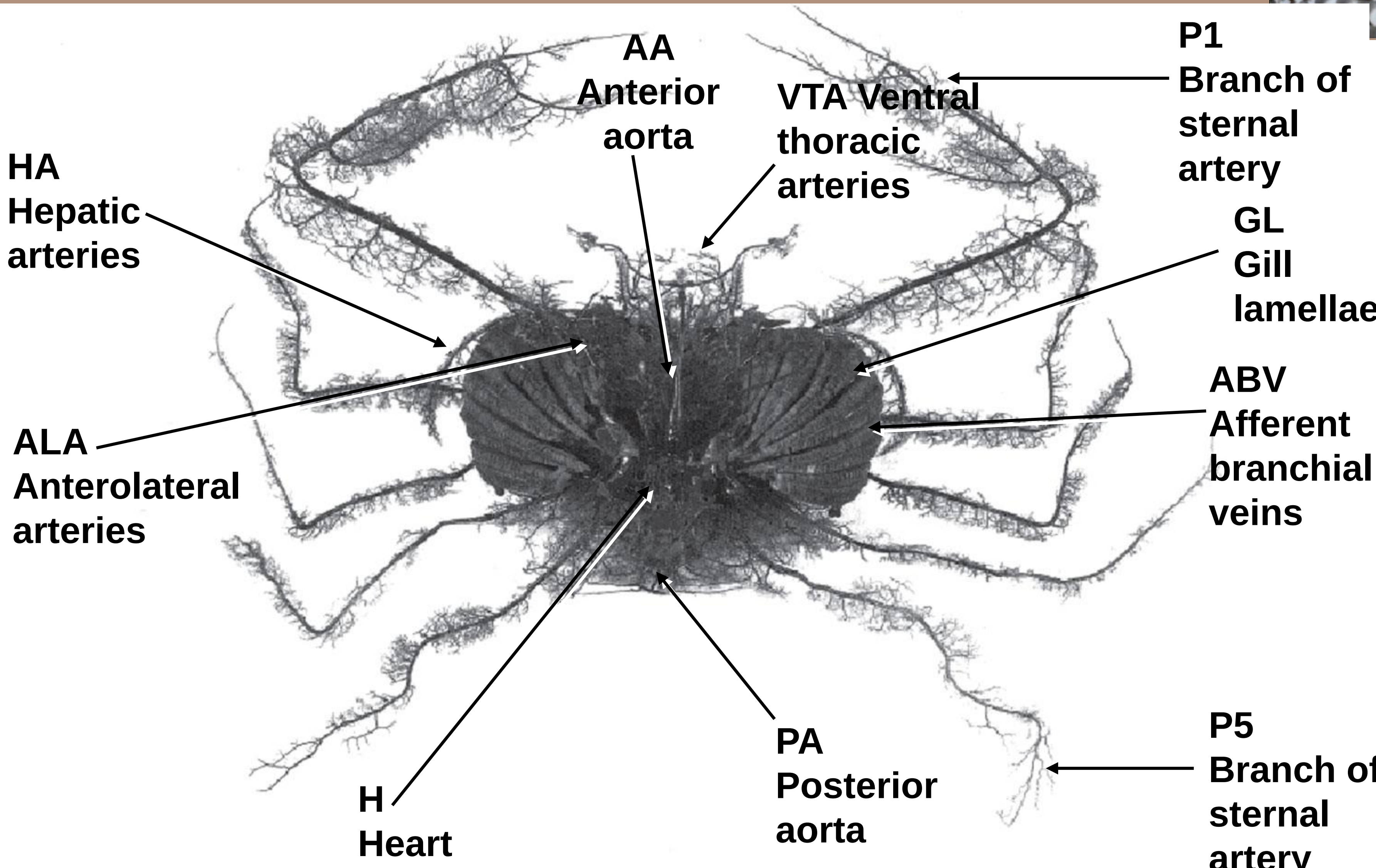
(a) Bivalve mollusk

Figure 9-36a p424



(b) Crayfish

Figure 9-36b p424



(c) Crab

Figure 9-36c p424

9.10 Circulatory Pathways and Vessels: Closed Circulation

- Vertebrate circulatory system began as a **single loop**

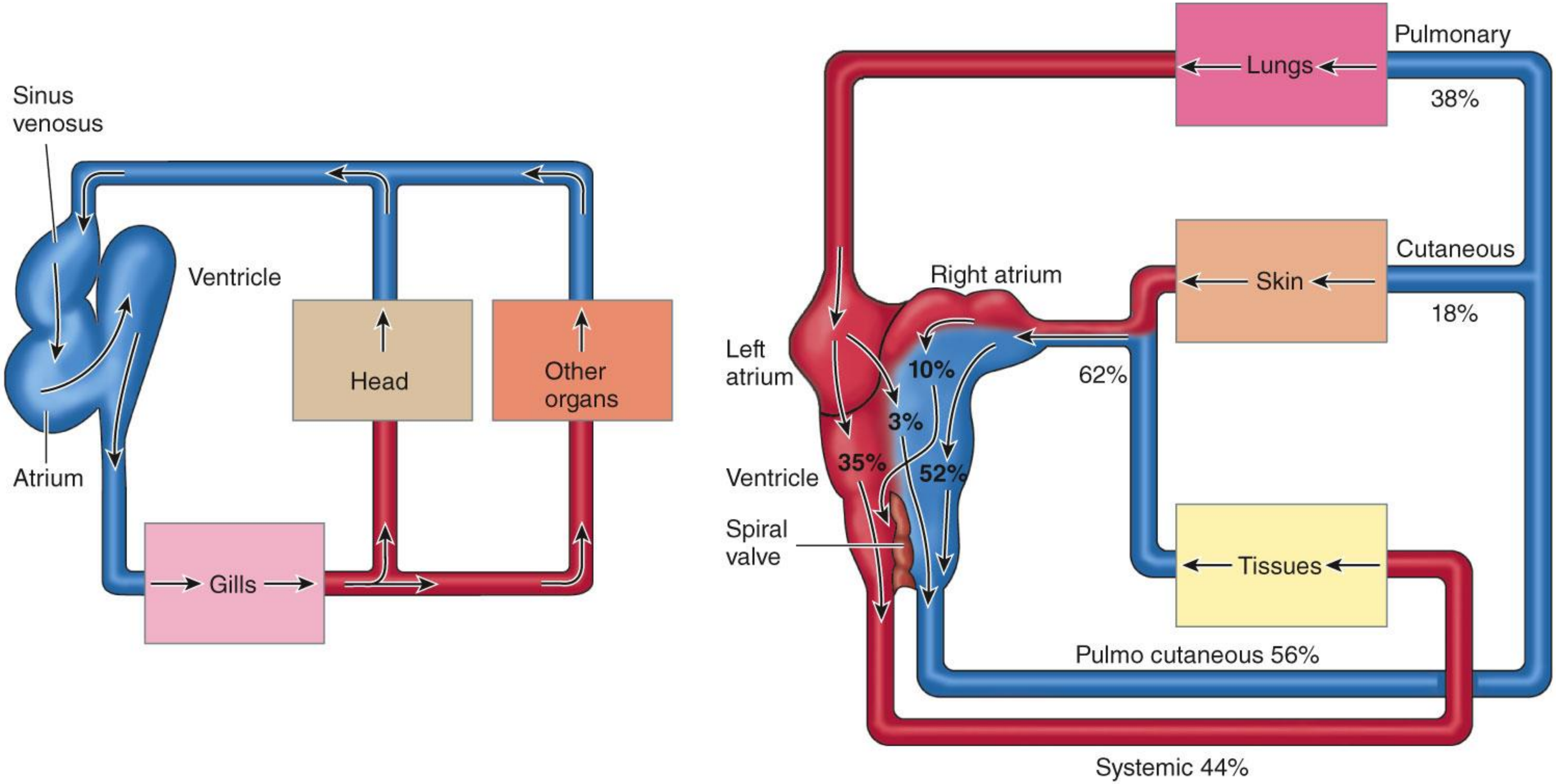
- **Fishes**

  - **Two-chambered heart** (atrium and ventricle)
  - Flow to the **gills** is in **series** with the rest of the circulation
  - **Parallel** system **distributes blood flow** amongst the other organs

- **Amphibians**

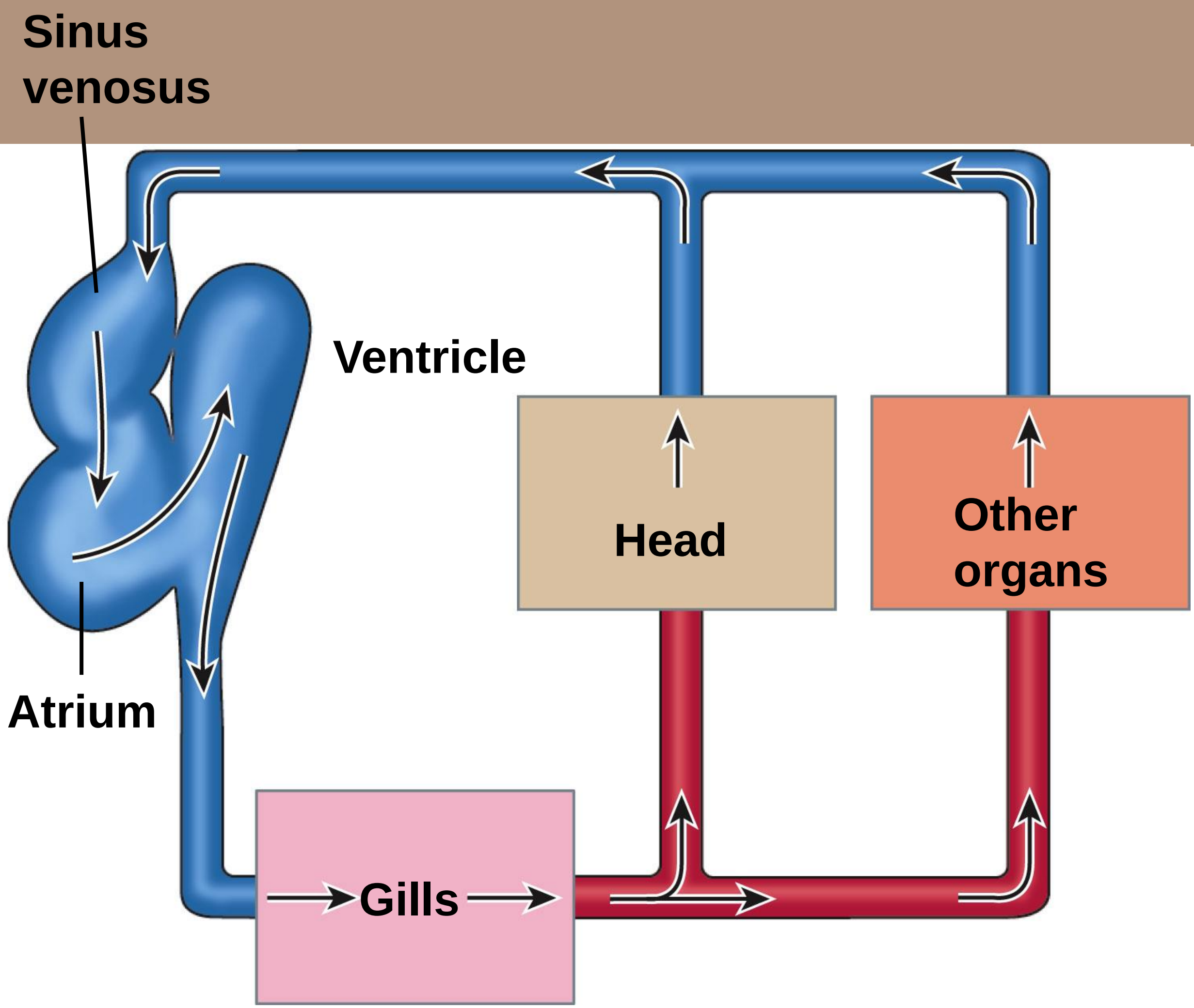
  - Separate pulmonary circuit goes to lungs and skin
  - **Three-chambered heart** with two atria
  - Oxygenated and deoxygenated blood mix in single ventricle

9.10 Circulatory Pathways and Vessels: Closed Circulation



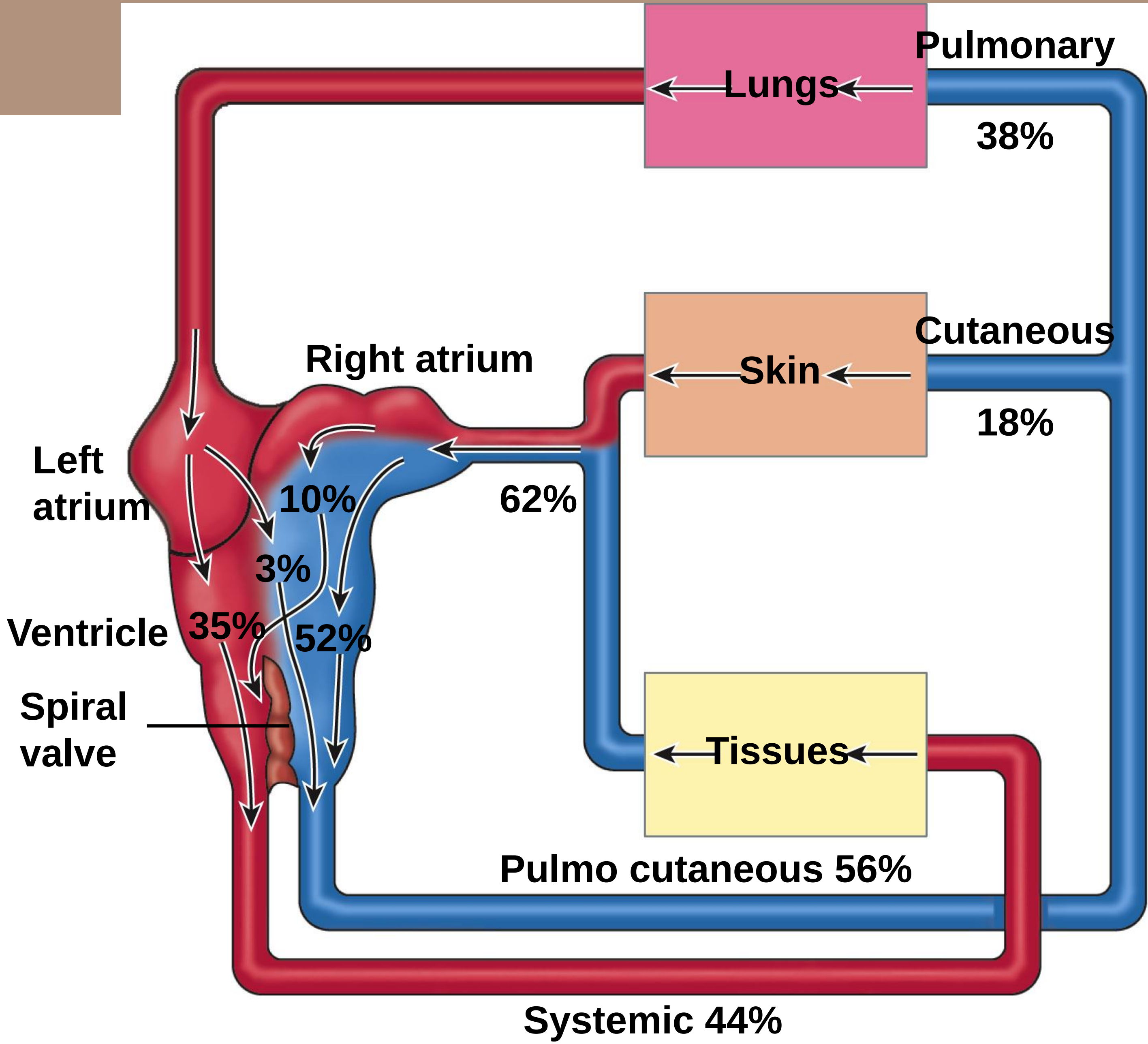
(a) Flow in a bony fish

(b) Flow in a bullfrog



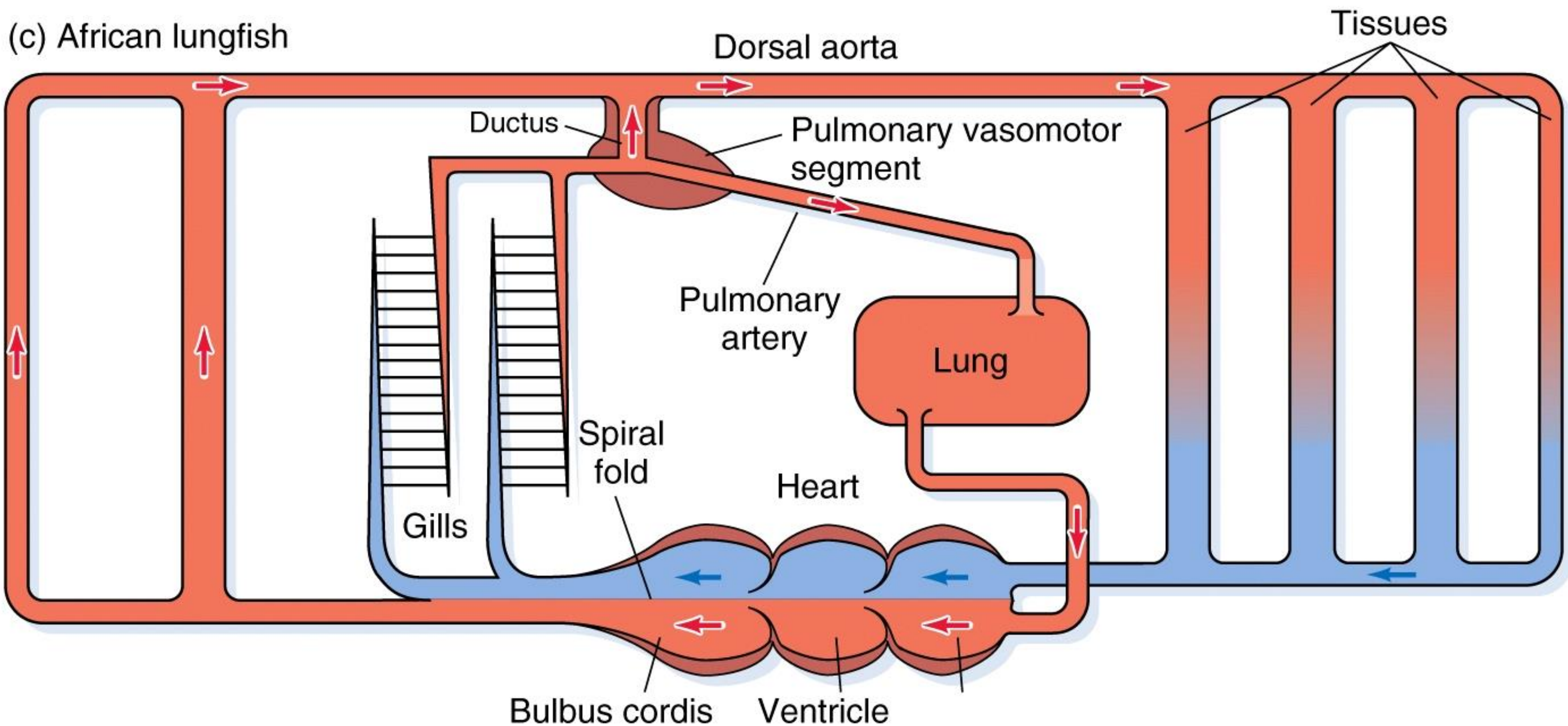
(a) Flow in a bony fish

Figure 9-38a p426



(b) Flow in a bullfrog

Figure 9-38b p426



(c) African lungfish
Dorsal aorta
Tissues
Ductus
Pulmonary vasomotor
segment
Pulmonary
artery
Lung
Spiral
fold
Heart
Gills
Bulbus cordis
Ventricle

9.10 Circulatory Pathways and Vessels: Closed Circulation

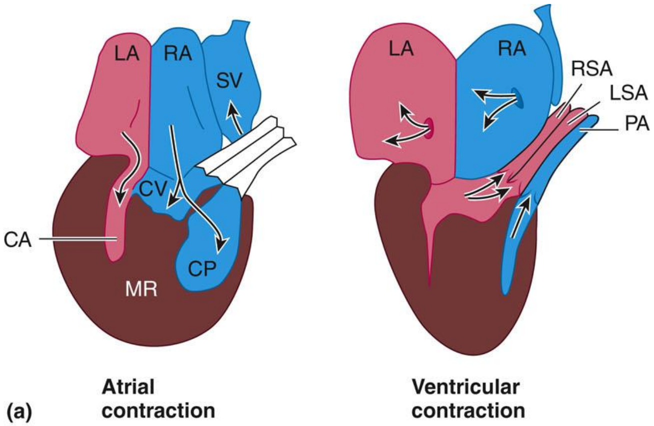
- **Reptiles**

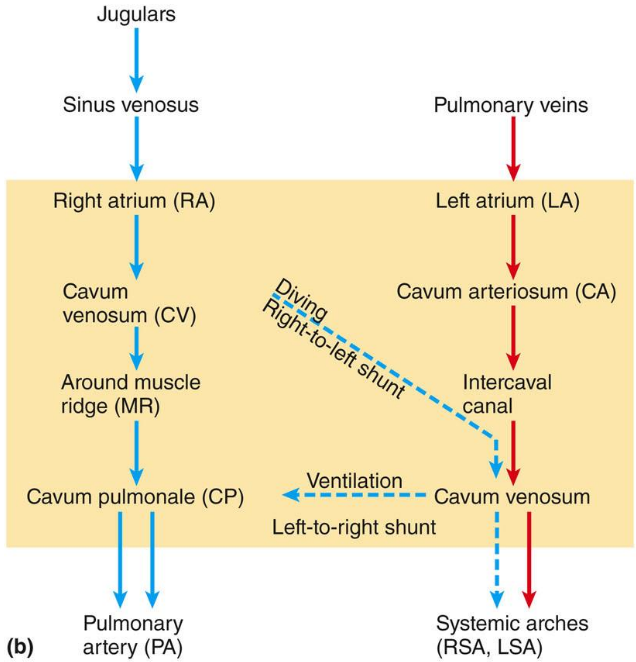
  - Three-chambered heart
  - Ventricle is divided into two large subchambers (**cavum arteriosum** and **cavum pulmonale**) by a thick muscle

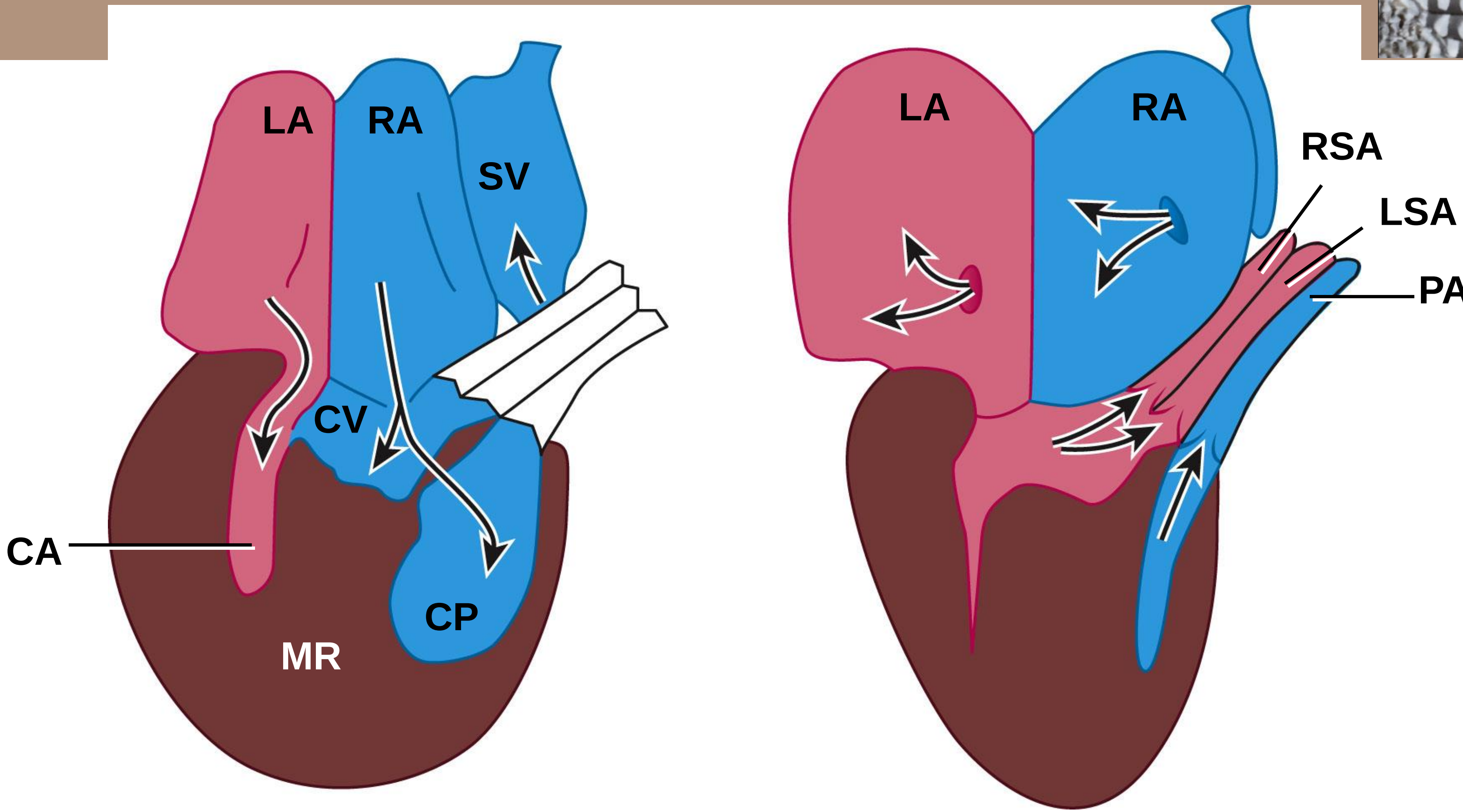
- **Crocodiles**

  - **Four-chambered heart** with two atria and two ventricles
  - Has **two aortas**, one exiting the right ventricle
  - Foramen of Panizza connects the two aorta
    - **Coglike valves** between right ventricle and pulmonary arteries control diversion of blood flow
    - Adaptation for prolonged breath-holding during a dive

9.10 Circulatory Pathways and Vessels: Closed Circulation







Atrial contraction **Ventricular contraction**

Figure 9-39a p427

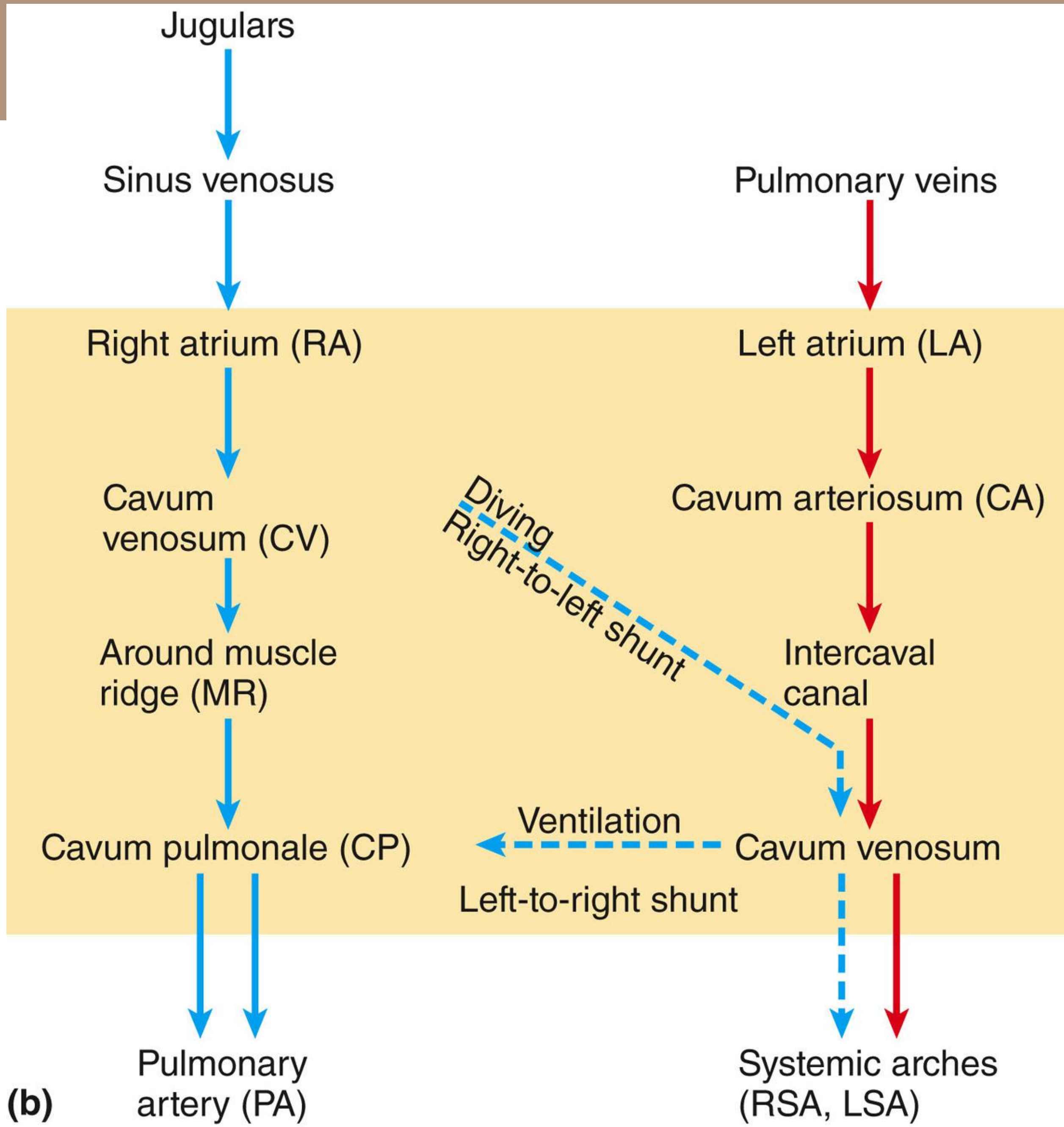
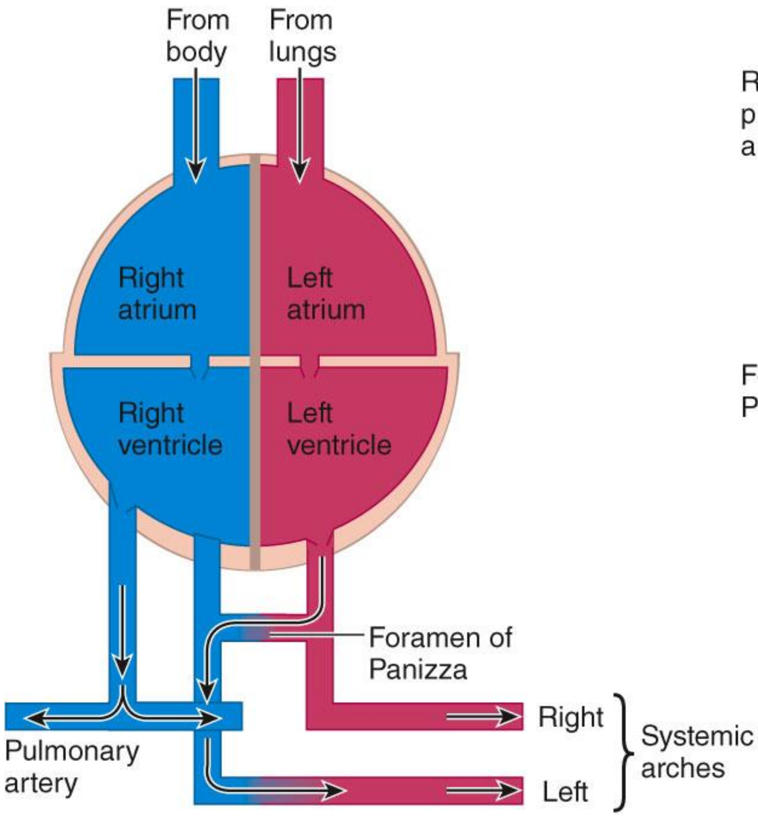
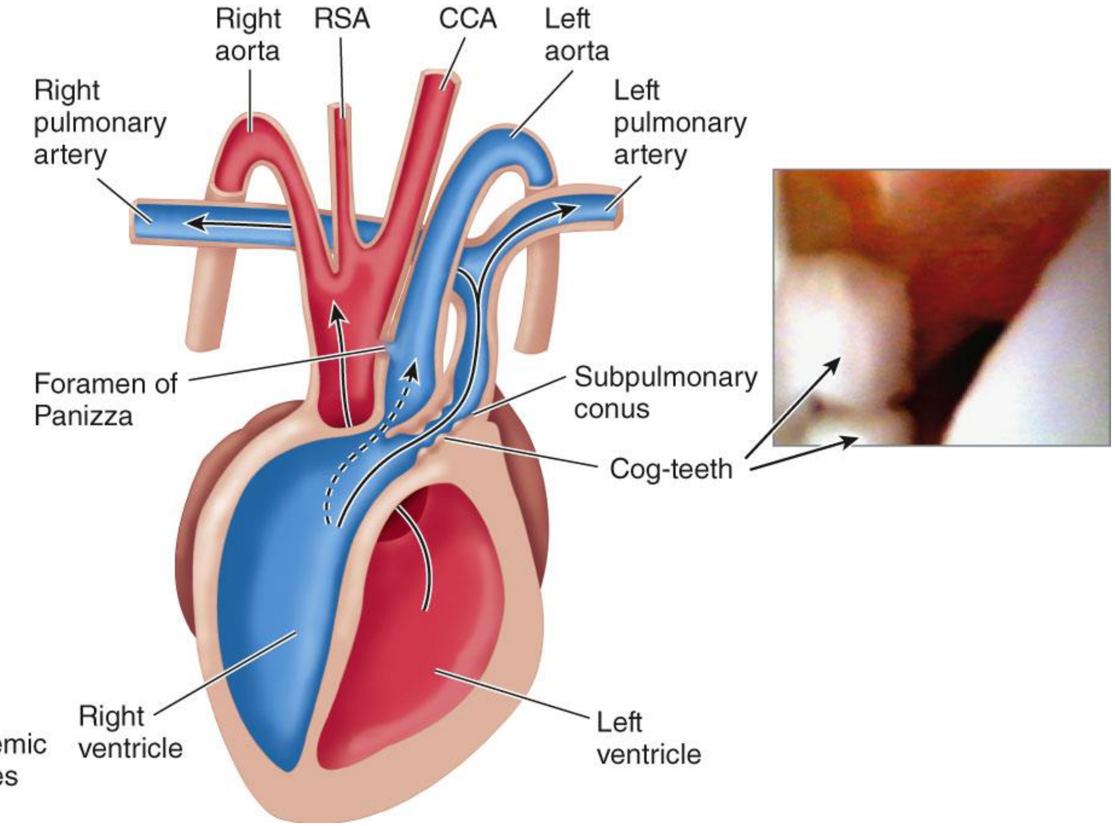


Figure 9-39b p427

9.10 Circulatory Pathways and Vessels: Closed Circulation





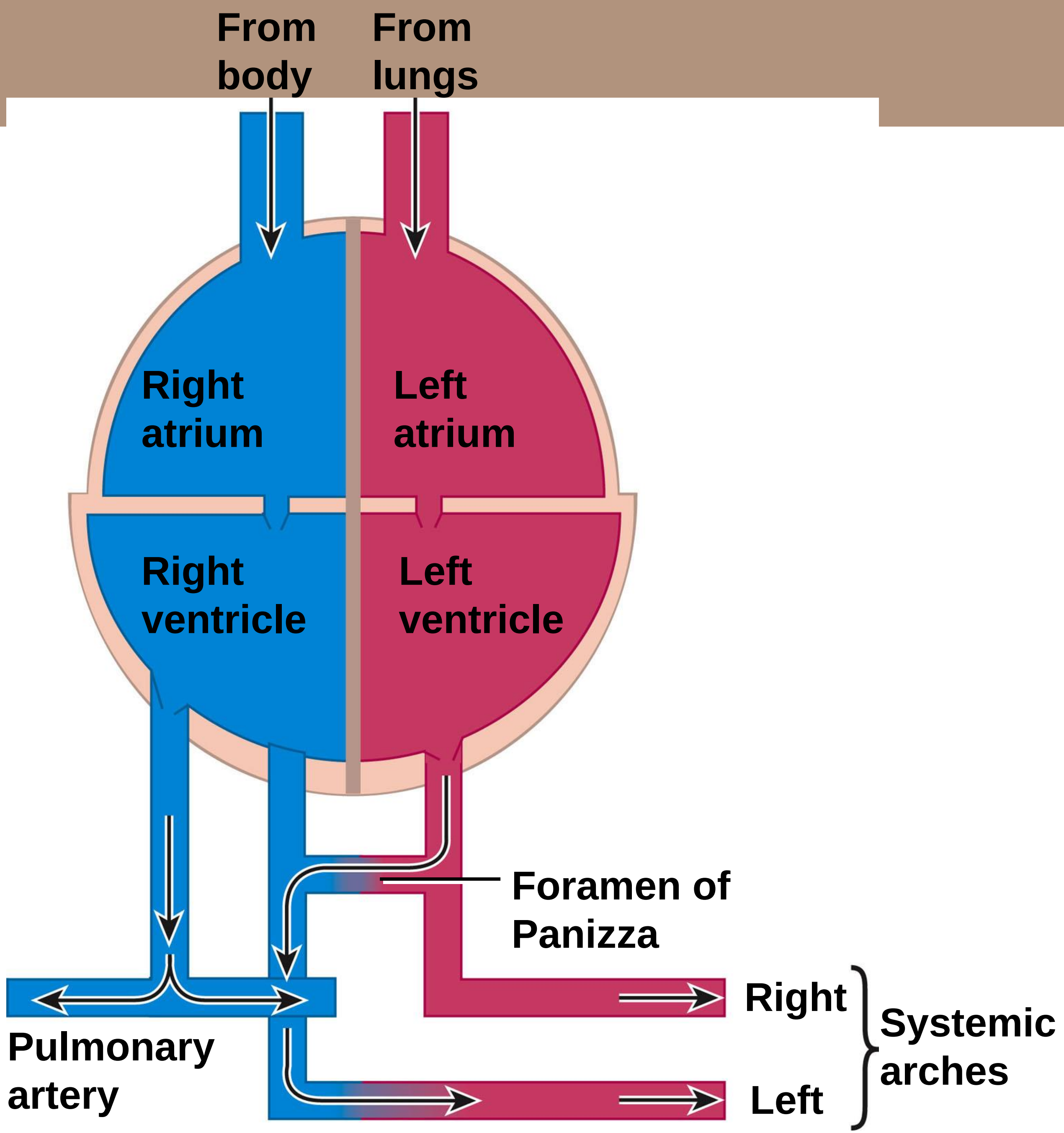


Figure 9-40a p428

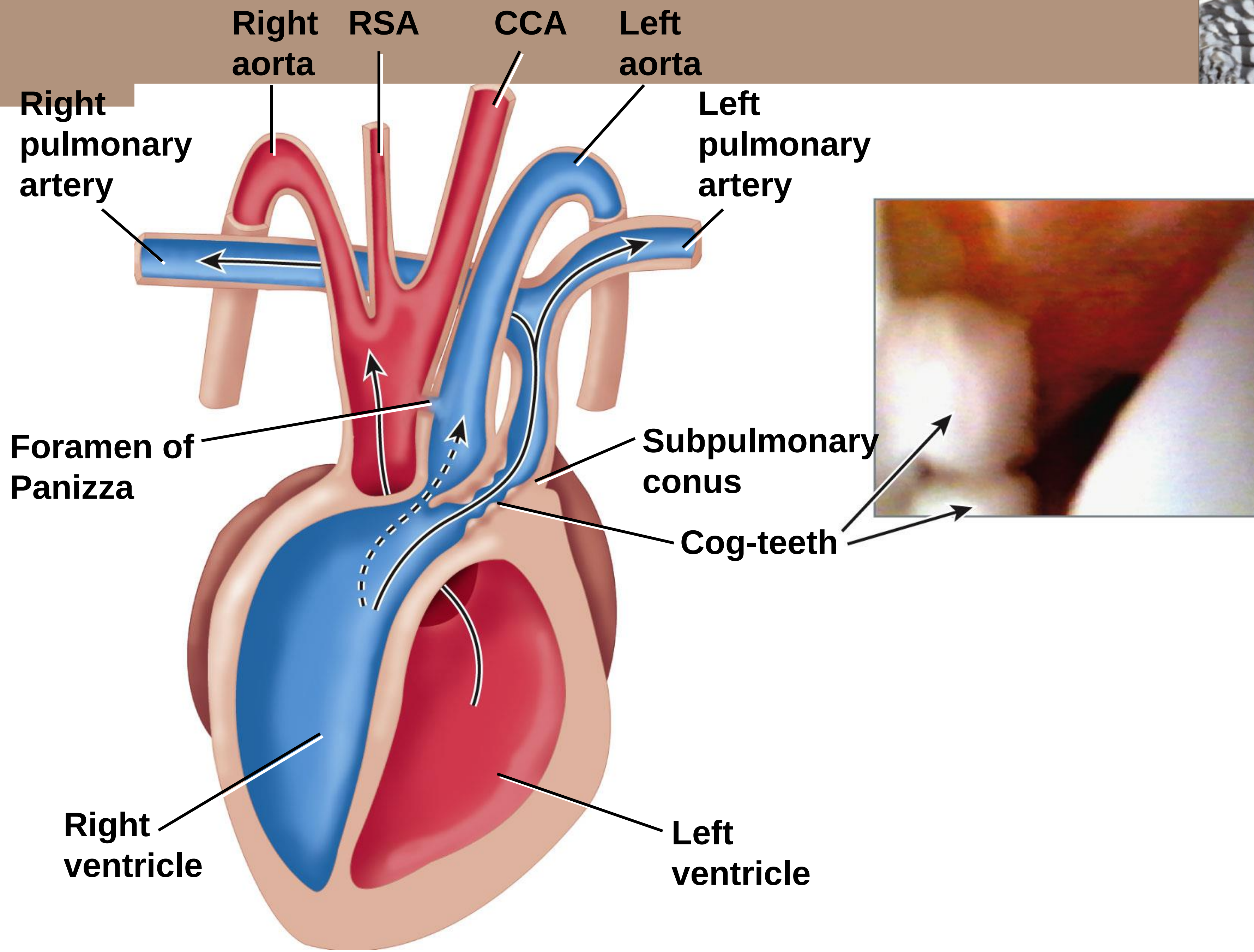
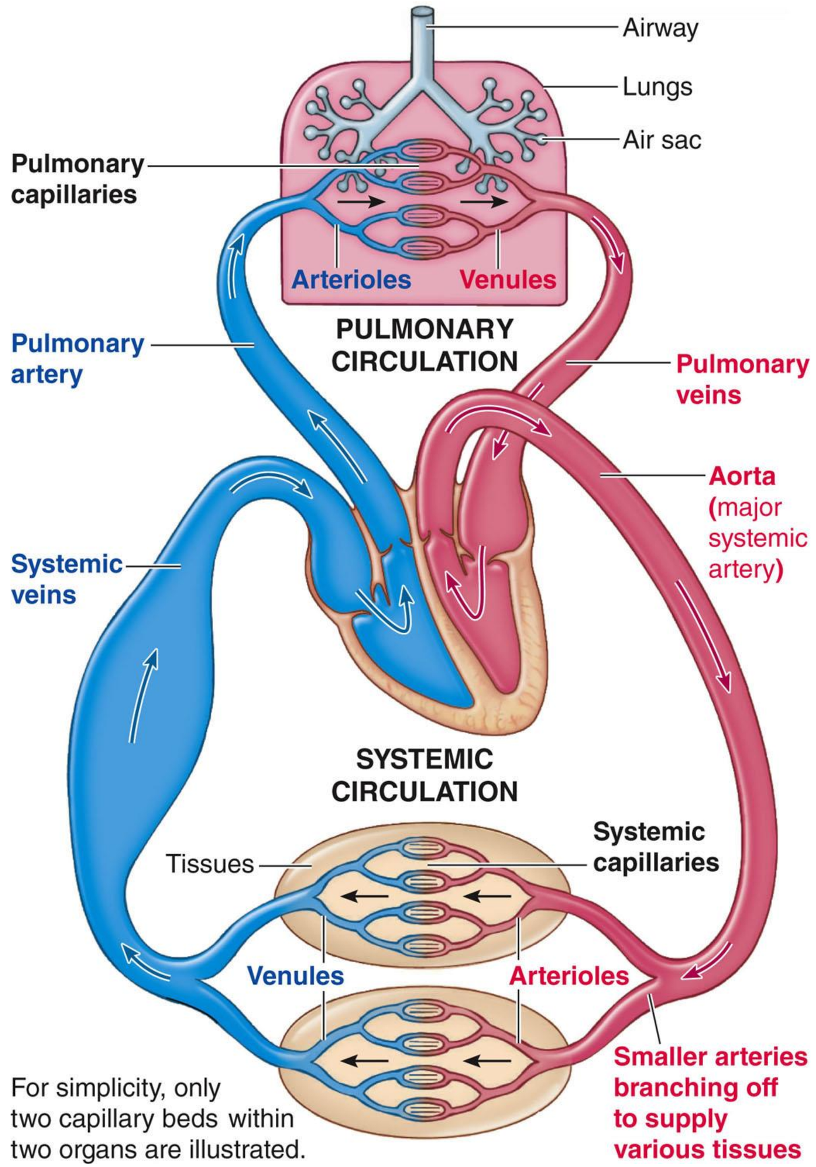


Figure 9-40b p428

9.10 Circulatory Pathways and Vessels: Closed Circulation

- **Birds** and **mammals**
  - **Four-chambered heart** with no shunts
  - **Complete separation** of pulmonary and systemic flow evolved independently in birds and mammals to support their **high endothermic metabolisms**
  - **Fetus** of placental mammals has two **bypasses** since lungs are not functional
    - **Foramen ovale** -- opening in septum between right and left atrium
    - **Ductus arteriosus** -- connects pulmonary artery and aorta

9.10 Circulatory Pathways and Vessels: Closed Circulation



4. THE AORTIC ARCHES

The first pair of aortic arches is formed by the curving of the ventral aorta into the primitive dorsal aorta. This arch is hidden in the mandibular arch and participates in formation of the *maxillary artery,* and contribute to the *external carotid artery*

II. The second pair of aortic arches make their appearance in the middle of week 4. They cross the second branchial arches and give rise to the *stapedial* and *hyoid arteries.* (It should be noted that arches I and II regress rapidly and are not seen after day 31)

III. The third pair of aortic arches make their appearance at the end of week 4. They give rise to the *common carotids* and *proximal portions of the internal carotid arteries.* The latter are the short cephalic prolongations of the primitive dorsal aortas and are associated with development and supply of the brain

A. THE INTERNAL CAROTID ARTERIES are secondarily attached to the cranial portions of the dorsal aortas, which form the remainder of the carotid artery

B. THE ORIGIN OF THE EXTERNAL CAROTID ARTERIES is controversial, but in later stages of development, they are found to sprout from aortic arch III. (Arch I, however, has been implicated in its developmental contribution)

IV. The fourth pair of aortic arches make their appearance shortly after the third arches, at the end of week 4. Their development is different for the right and left sides

A. ON THE RIGHT SIDE arch IV forms the proximal portion of the *right subclavian artery* and is continuous with the seventh segmental artery
   1. The caudal portion of the right primitive dorsal aorta disappears
   2. The distal portion of the subclavian artery forms from the right dorsal aorta and the right seventh intersegmental artery

B. ON THE LEFT SIDE arch IV persists as the *arch of the aorta,* which grows significantly and is continuous with the primitive left dorsal aorta.
   1. The *left subclavian artery* (or seventh segmental) arises directly from the aorta

C. THE SHORT PORTION of the right primitive ventral aorta, which persists between arches IV and VI, forms the *brachiocephalic arterial trunk* and the *first portion of the aortic arch*

V. The fifth pair of aortic arches: in 50% of embryos, these arches are rudimentary vessels that degenerate with no derivatives. In fact, they may never even develop

VI. The sixth pair of aortic arches make their appearance in the middle of week 5 and give rise to the *right* and *left pulmonary arteries.* After pulmonary vascularization is established, the communication with the corresponding primitive dorsal aorta regresses

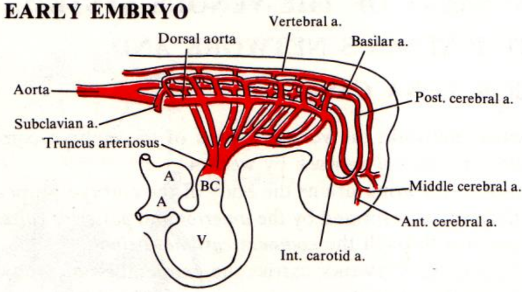
A. REGRESSION is total and complete on the right side. The proximal portion of the right arch forms the proximal part of right pulmonary artery; its distal portion degenerates

B. THE PROXIMAL PORTION OF THE LEFT ARCH persists as the proximal part of the left pulmonary artery
   1. The distal portion of the left arch, in which communication persists with the dorsal aorta until birth, forms the *ductus arteriosus* and diverts blood from the pulmonary artery to the aorta. Closure of the ductus arteriosus takes place in the neonatal period, and the functional duct becomes the anatomic *ligamentum arteriosum*

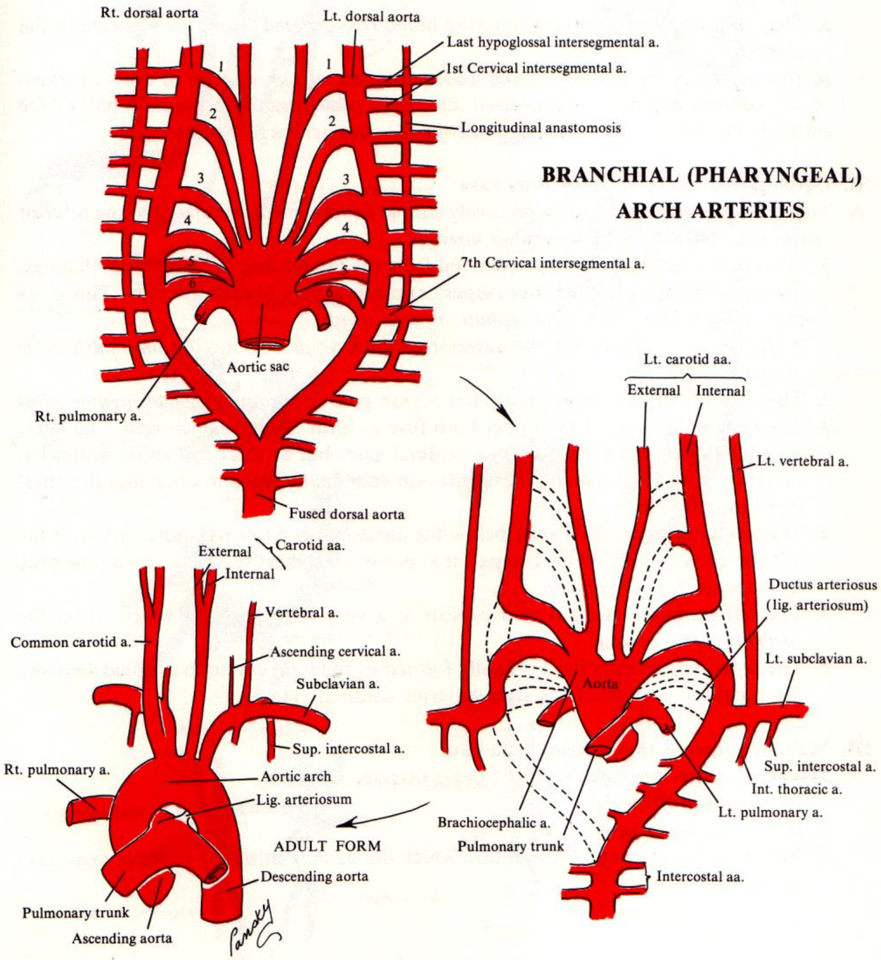
C. THE DISTAL PORTIONS OF THE PULMONARY ARTERIES are derived from buds of the sixth aortic arches that grow into the developing lungs. After partitioning of the truncus arteriosus, the pulmonary arteries arise from the pulmonary trunk

VII. Summary of the aortic arches: arch I regresses; arch II regresses; arch III forms the carotid system; arch IV forms the aortic arch (on the left) and the subclavian (on the right); arch V disappears; and arch VI forms the pulmonary arteries and the ductus arteriosus (on the left)

DEVELOPING ARTERIES IN HEAD REGION OF EARLY EMBRYO



BRANCHIAL (PHARYNGEAL) ARCH ARTERIES

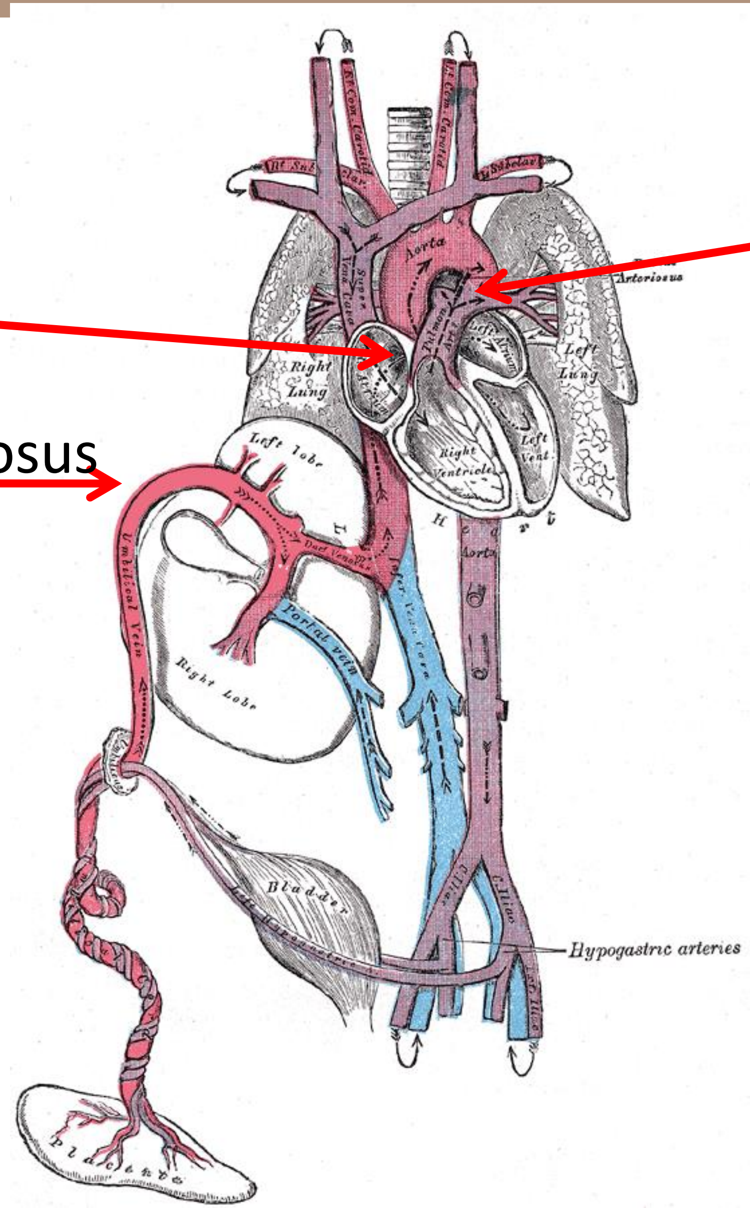


Fetus Heart: Before & After Birth

Foramen ovale

Ductus venosus

Ductus arteriosus



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.

Pediatrics in Review Vol.28 No.4 April

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.

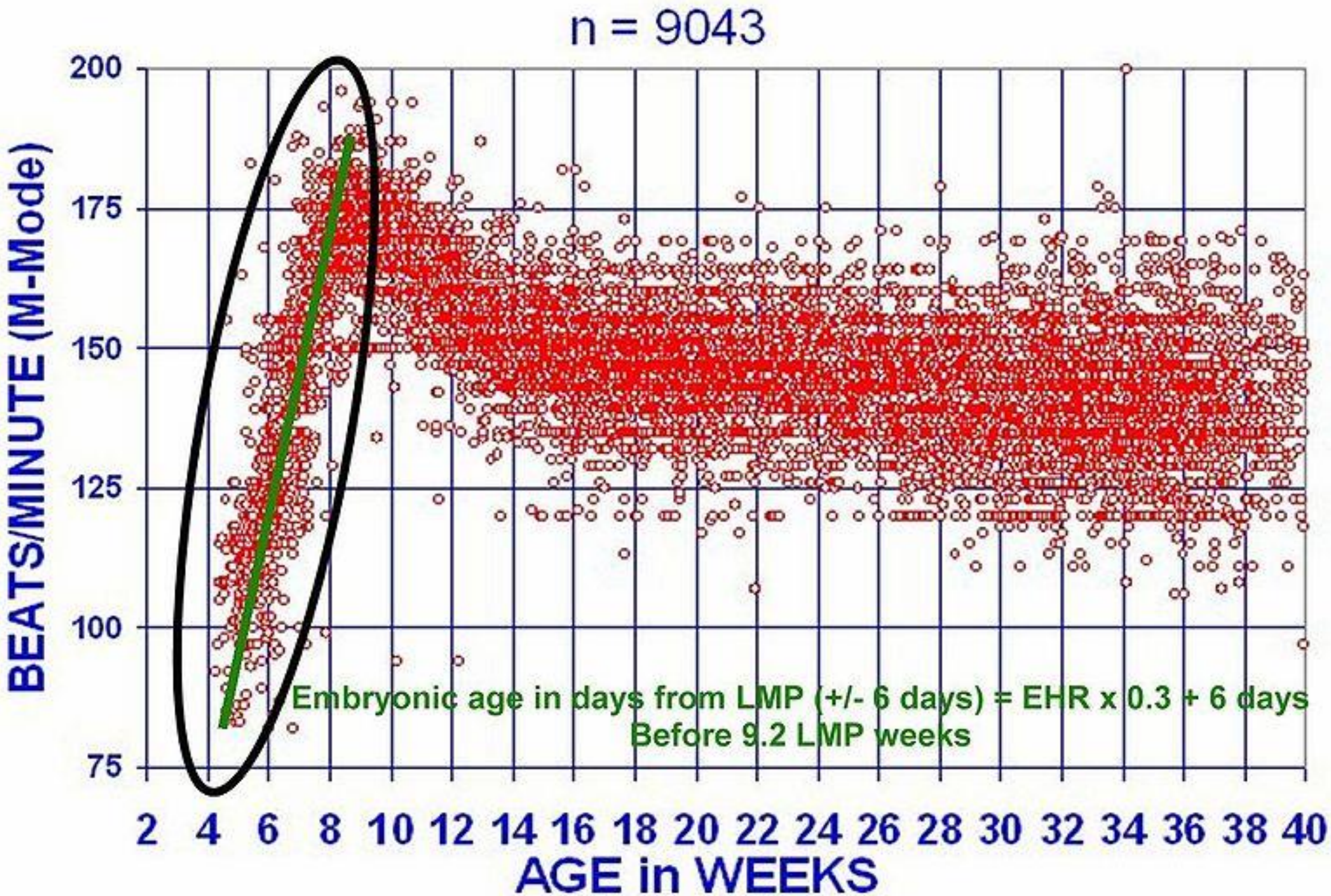
The human heart begins beating at a rate near the mother's, about 75-80 BPM.

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.

HEART RATE & AGE



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*

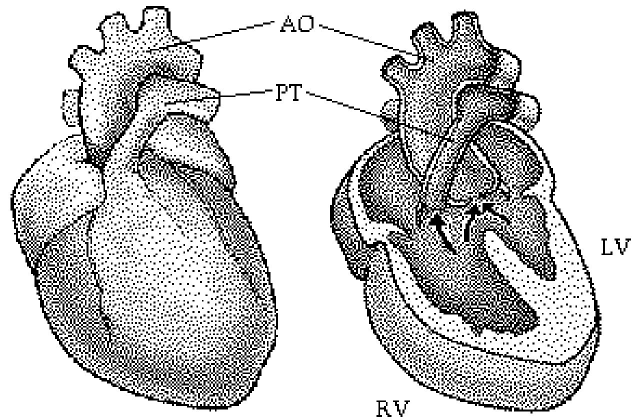
Etiologic Syndrome	Frequency of Cardiac Anomalies† All (%)	Frequency of Cardiac Anomalies† Distinctive or Most Common	Distinguishing Features
Autosomal Dominant			
Adams-Oliver syndrome	20	Left-sided obstruction (eg, COA, parachute MVP), TOF	Scalp cutis aplasia, terminal transverse limb defects
Alagille 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

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

Malformation	Prevalence (per 10,000 Births): Metropolitan Atlanta Congenital Defect Program, 1995–1997	Prevalence (per 10,000 Births): 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.

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%

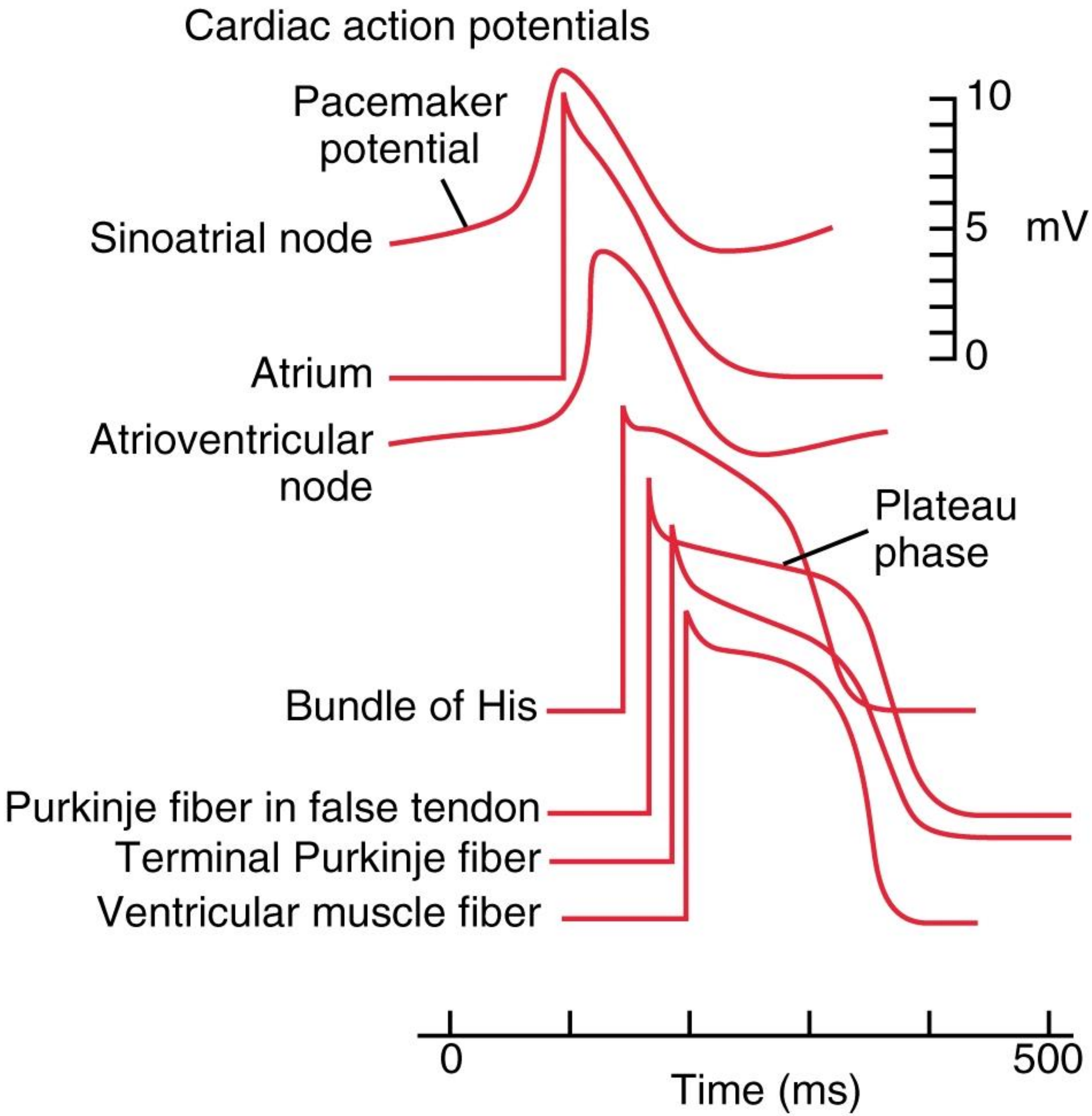
Hypoplastic left heart syndrome

Mortality ~10%

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

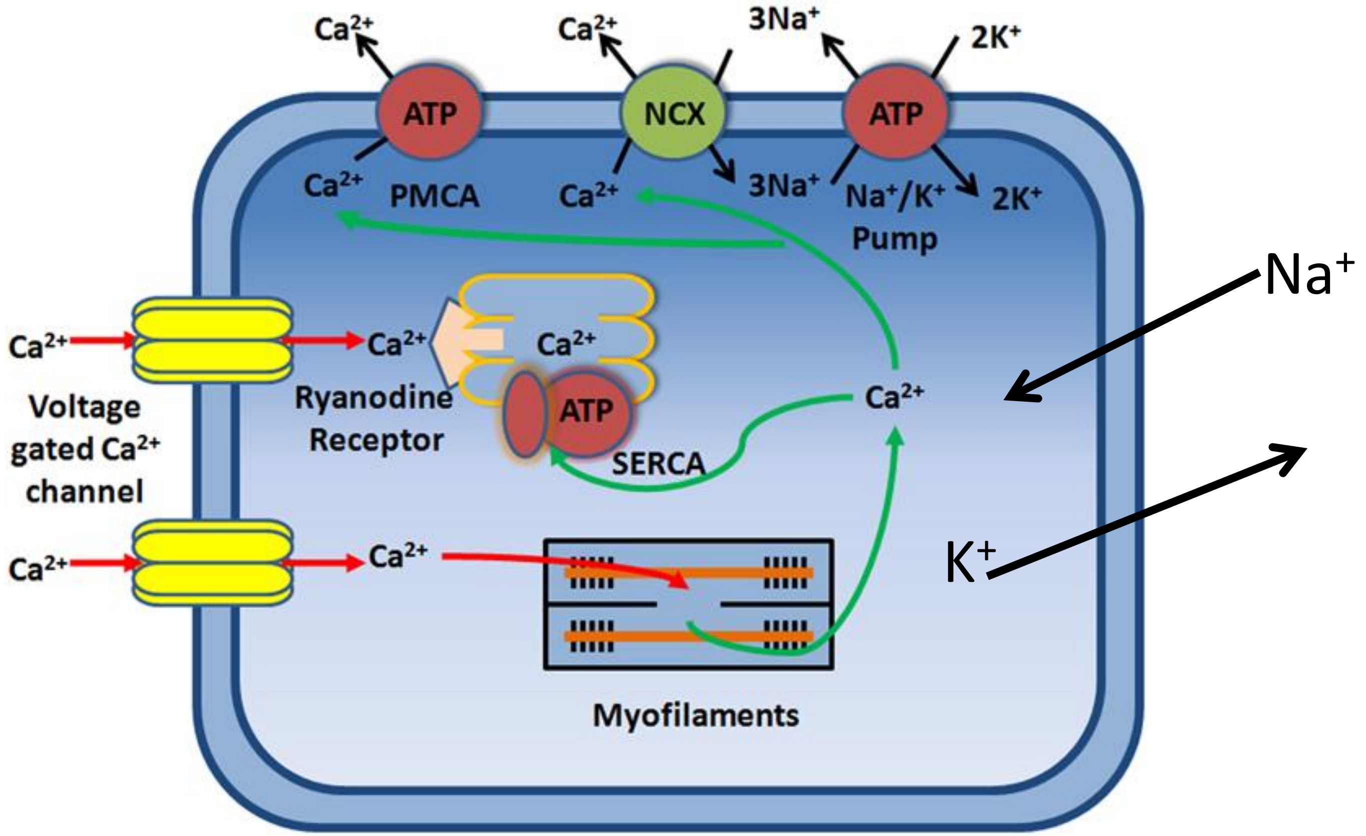
ECG: Neonate

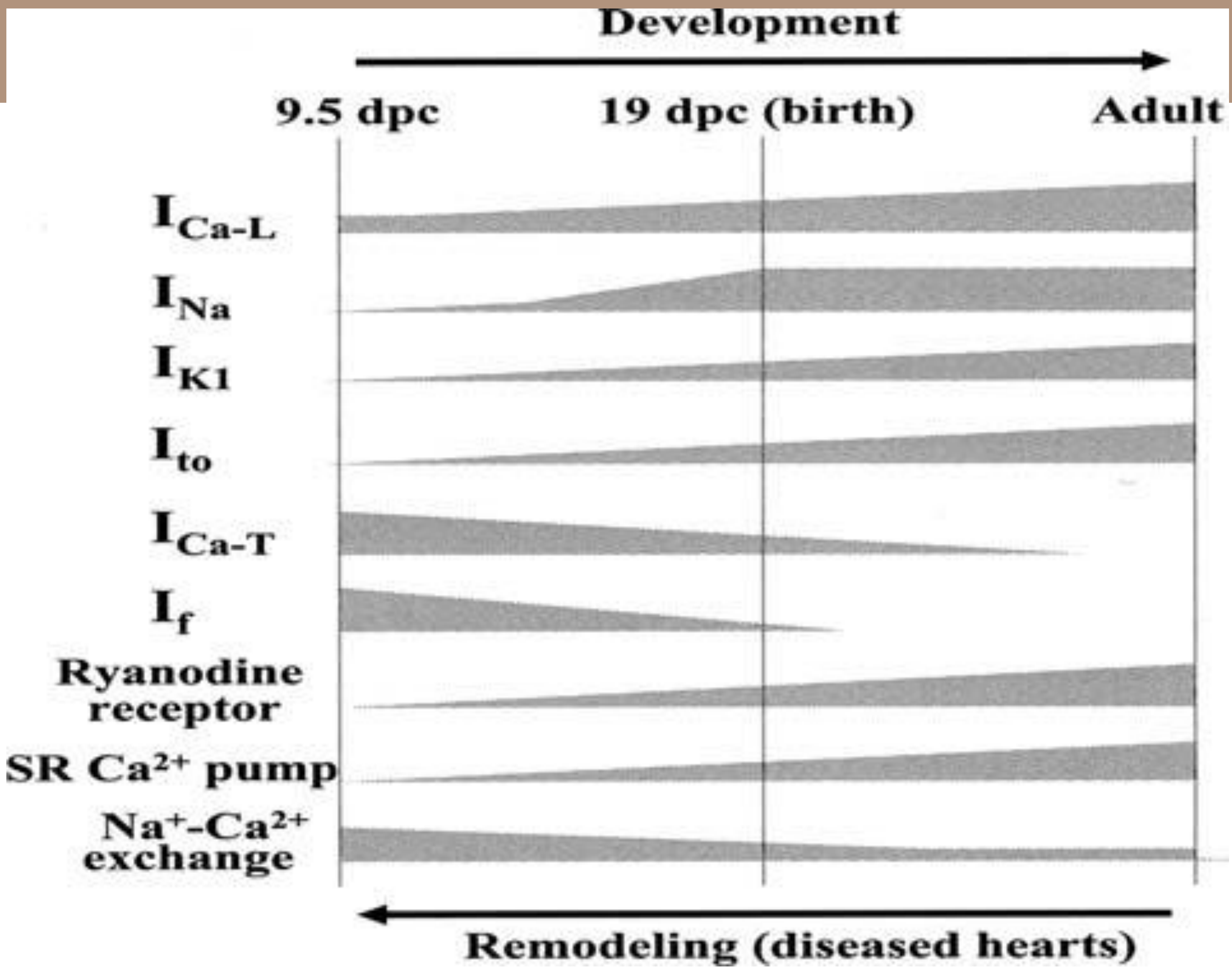
- Arrhythmias in fetuses and newborns are relatively common, occurring in up to 90% of newborns and in 1% to 3% of pregnancies *(NeoReviews Vol.9 No.6 2008 e242,2008 American Academy of Pediatrics, Fetal and Neonatal Arrhythmias)*
- Weak arterial pulses and right heart overload in the **electrocardigram** 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
Plateau phase
Bundle of His
Purkinje fiber in false tendon
Terminal Purkinje fiber
Ventricular muscle fiber
10
5
0
mV
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**.

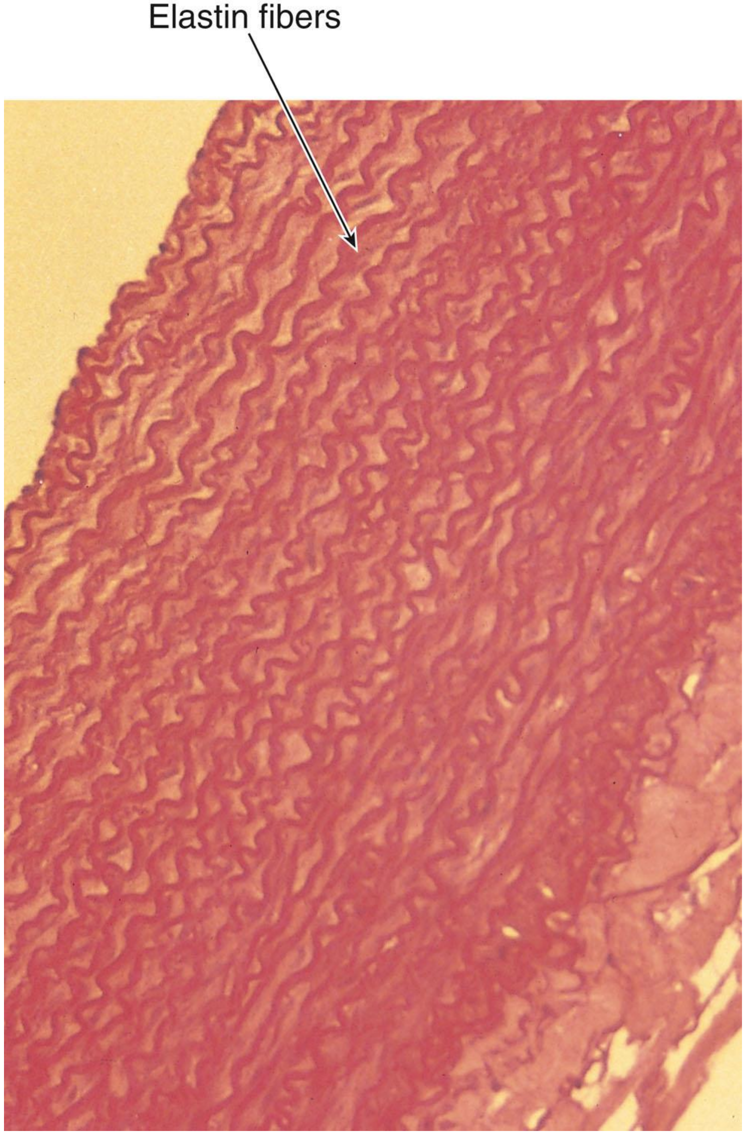
(J Bioenerg Biomembr. 2009 Apr;41(2):133-6. Uncouple my heart: the benefits of inefficiency.

Sonographer: Pediatric echocardiography

9.11 Circulatory Vessels: Arteries

- Arteries provide **rapid passage** of blood from the heart to the tissues
  - **Large radii** offer **little resistance** to flow
  - Blood flows at **high velocity**
- Arteries serve as **pressure reservoirs**
  - Arteries' **elasticity** enables them to **expand** during ventricular systole
  - **Elastic recoil** is the **driving force** for continued flow of blood during diastole

9.11 Circulatory Vessels: Arteries



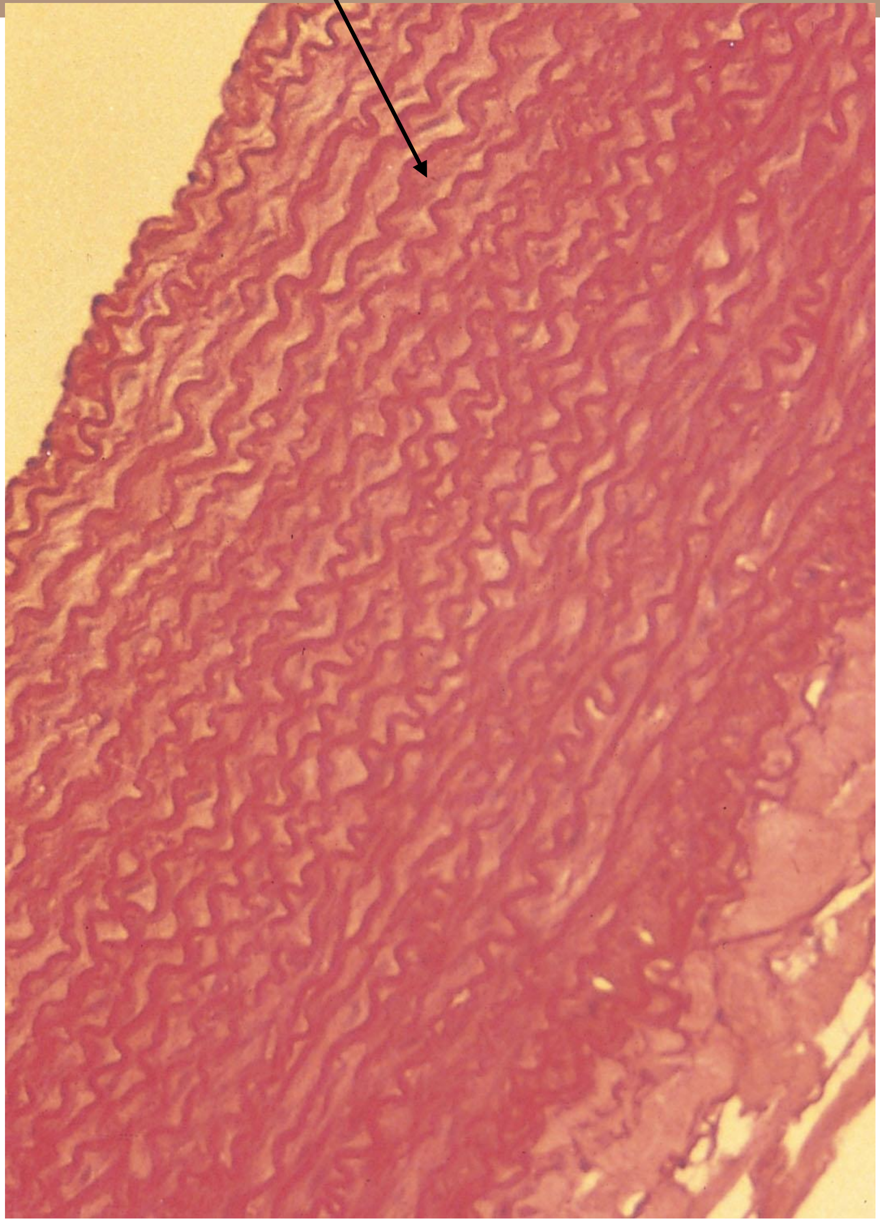
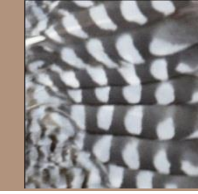
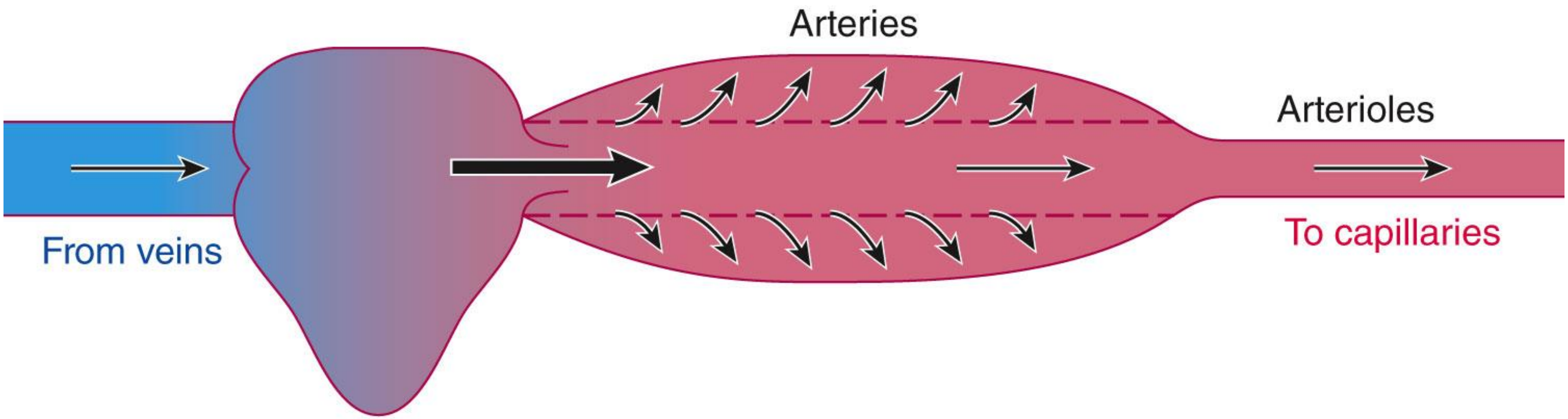


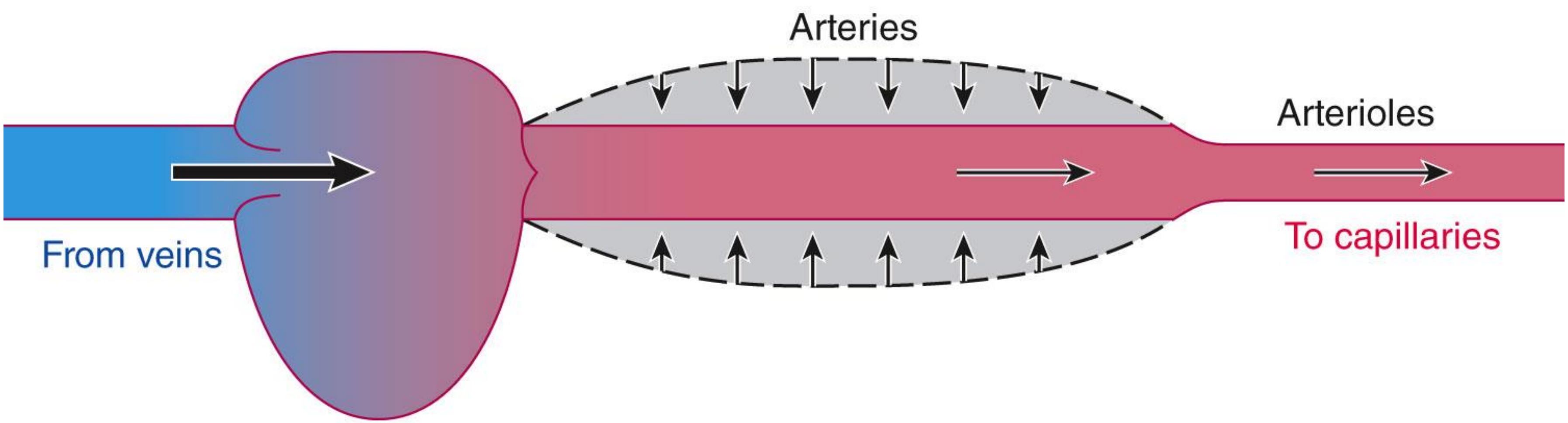
Figure 9-42 p430

9.11 Circulatory Vessels: Arteries

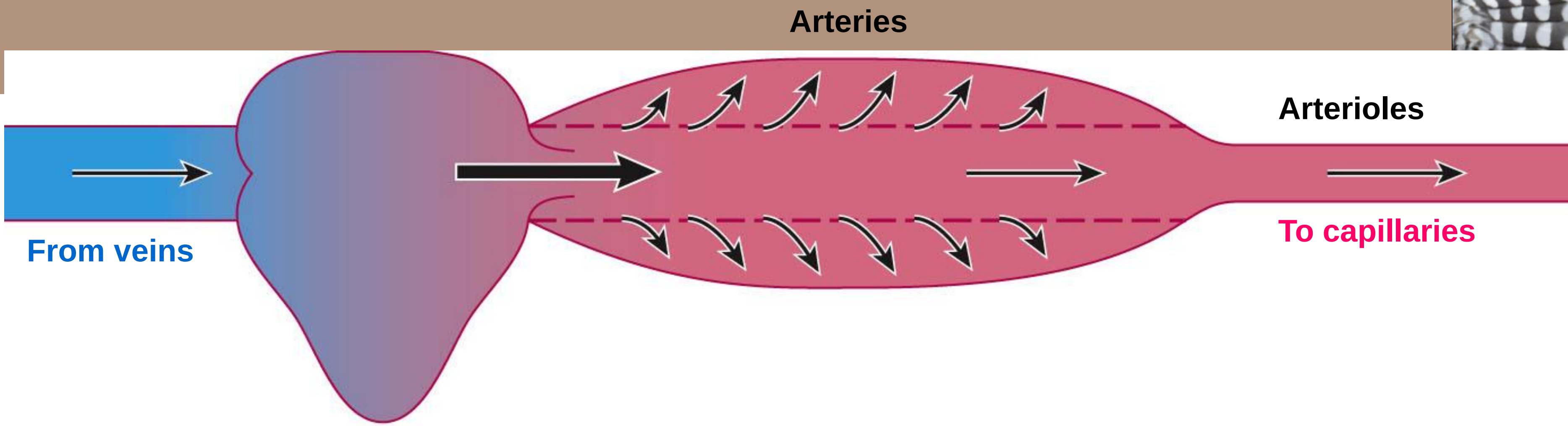




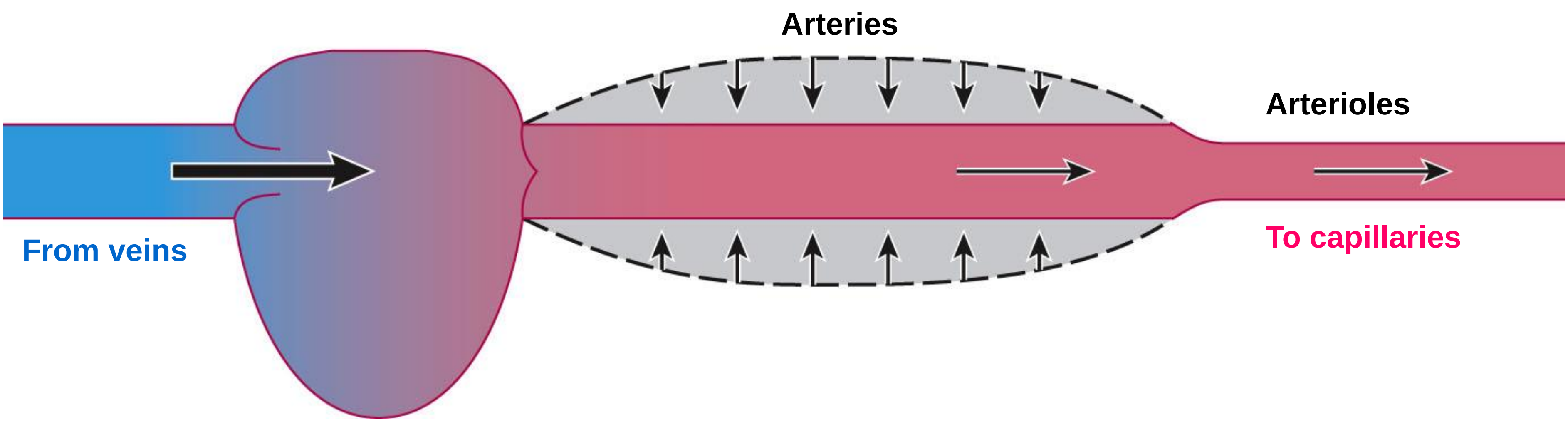
(a) Heart contracting and emptying



(b) Heart relaxing and filling



(a) Heart contracting and emptying



(b) Heart relaxing and filling

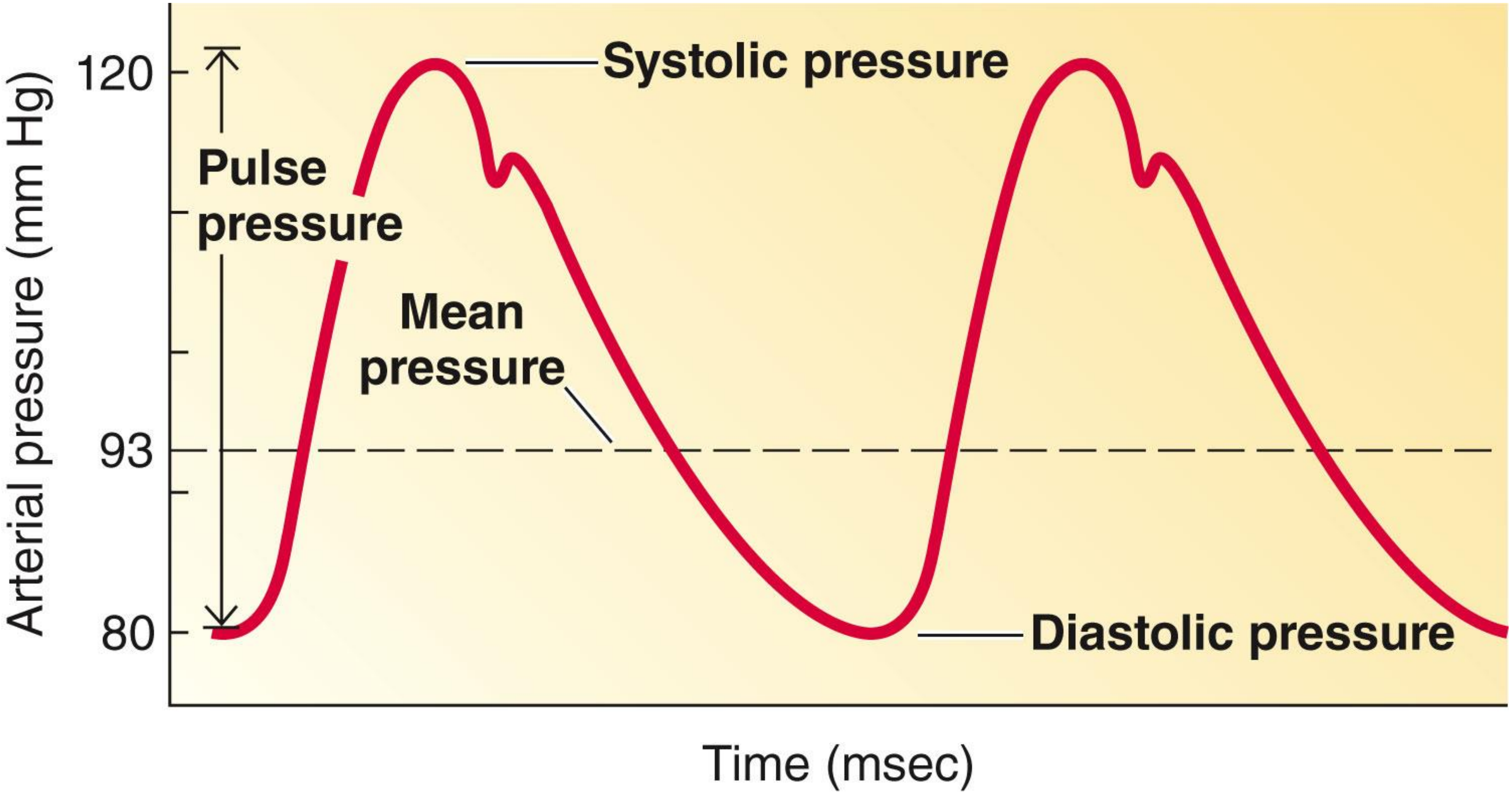
Figure 9-43 p430

9.11 Circulatory Vessels: Arteries

- **Arterial blood pressure**
  - Maximum pressure exerted on the arteries (**systolic blood pressure**) averages 120 mmHg in humans
  - Minimum pressure (**diastolic blood pressure**) averages 80 mmHg
  - Arterial blood pressure is expressed as a fraction (e.g. 120/80 mmHg)
  - **Mean arterial pressure** is the main **driving force** of blood flow

Mean arterial pressure = diastolic pressure + 1/3 (systolic pressure - diastolic pressure)

9.11 Circulatory Vessels: Arteries



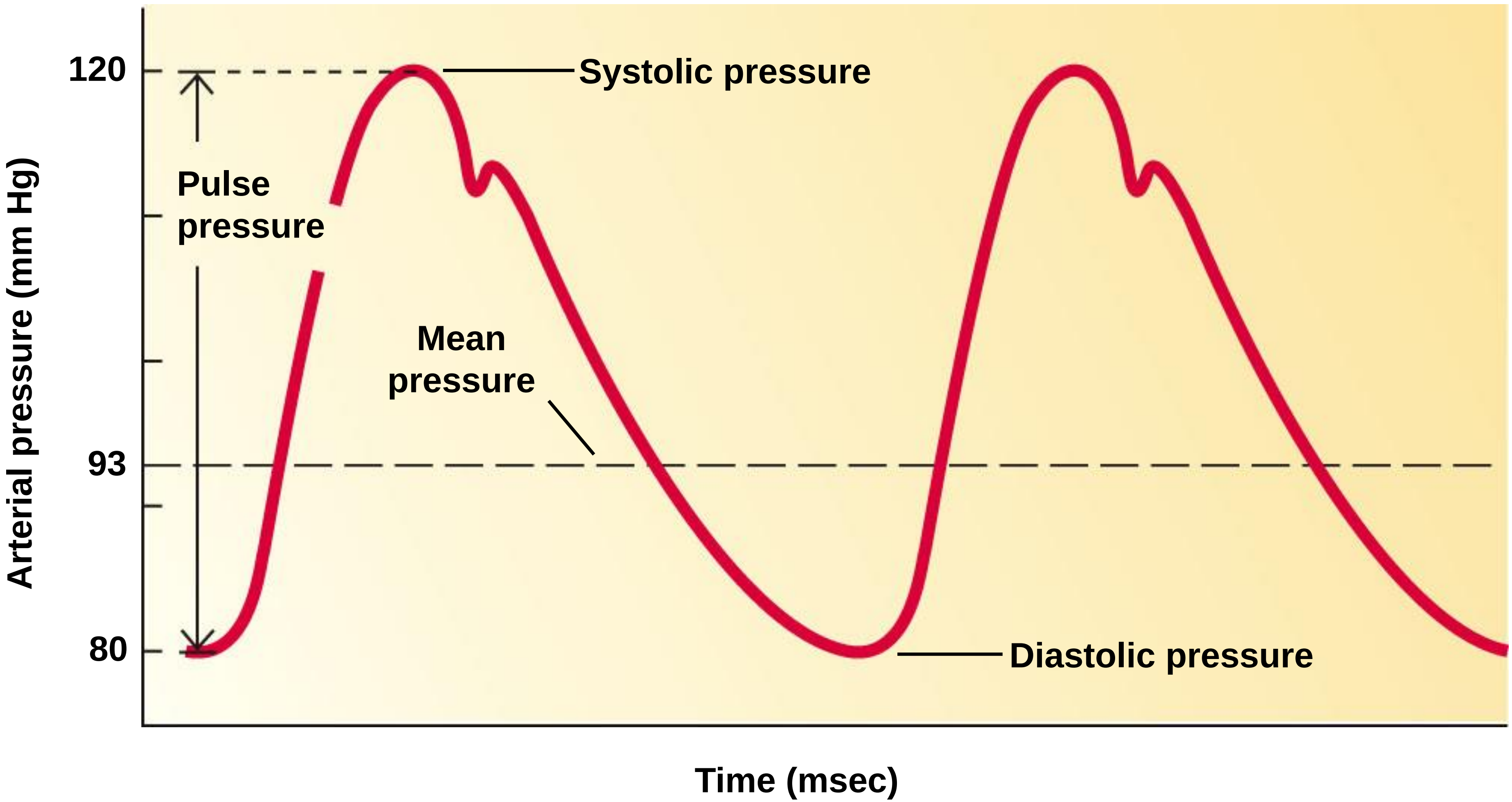
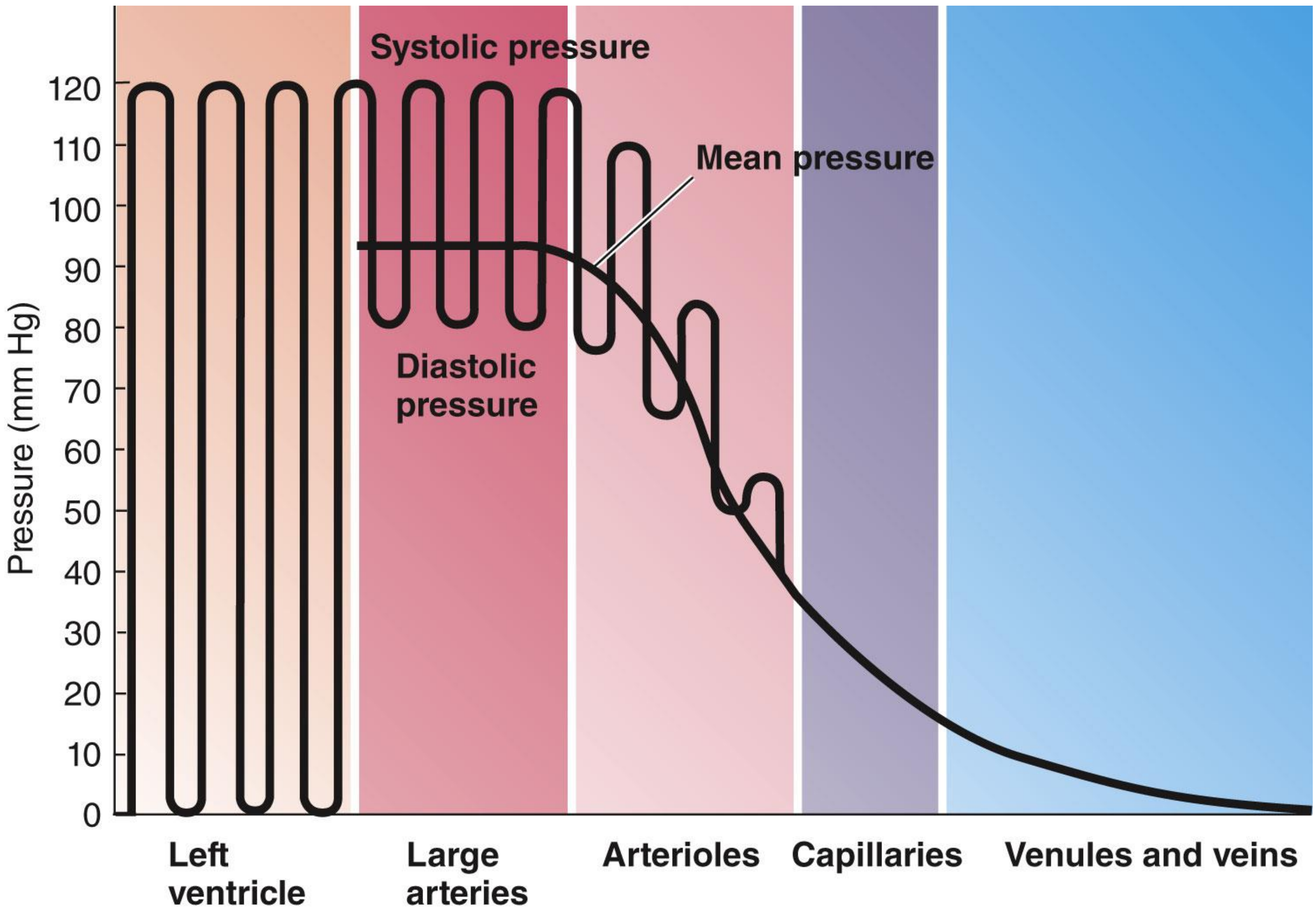


Figure 9-44 p432

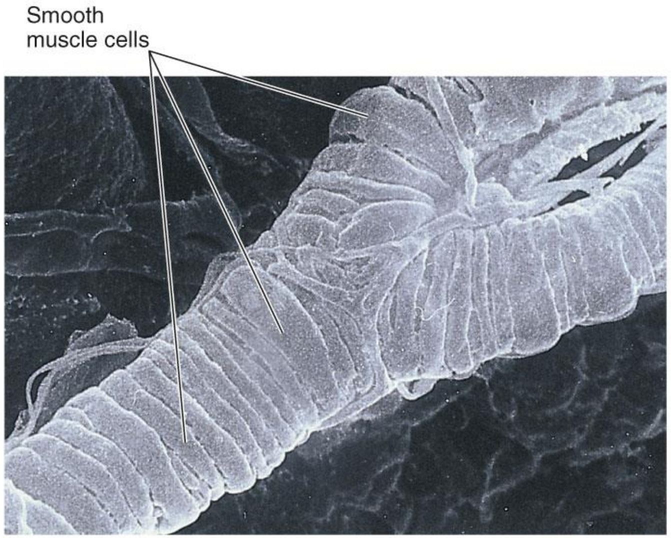
9.12 Circulatory Vessels: Arterioles

- Arterioles are the **major resistance vessels**
  - Radii of arterioles are small enough to offer considerable **resistance to flow**
  - Large **drop in blood pressure** through the arterioles
    - Mean arterial pressure of 93 mmHg drops to 37 mmHg where blood enters the capillaries
  - Eliminates pulsatile pressure swings
  - Thick layer of **smooth muscle** is innervated by **sympathetic** nerve fibers
    - **Vasoconstriction** results from smooth muscle contraction —-> decreased radius, increased resistance
    - **Vasodilation** results from smooth muscle relaxation —-> increased radius, decreased resistance

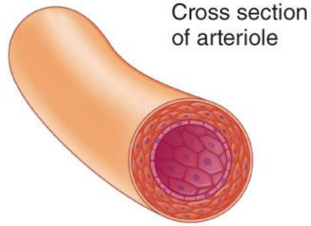
9.12 Circulatory Vessels: Arterioles



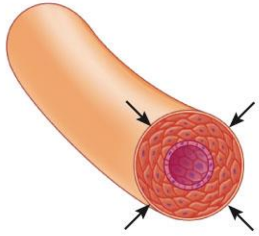
9.12 Circulatory Vessels: Arterioles



(a) Scanning electron micrograph of an arteriole showing how the smooth muscle cells run circularly around the vessel wall

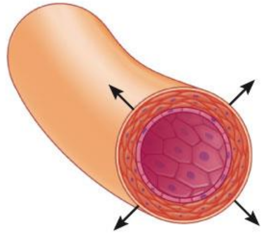


(b) Normal arteriolar tone



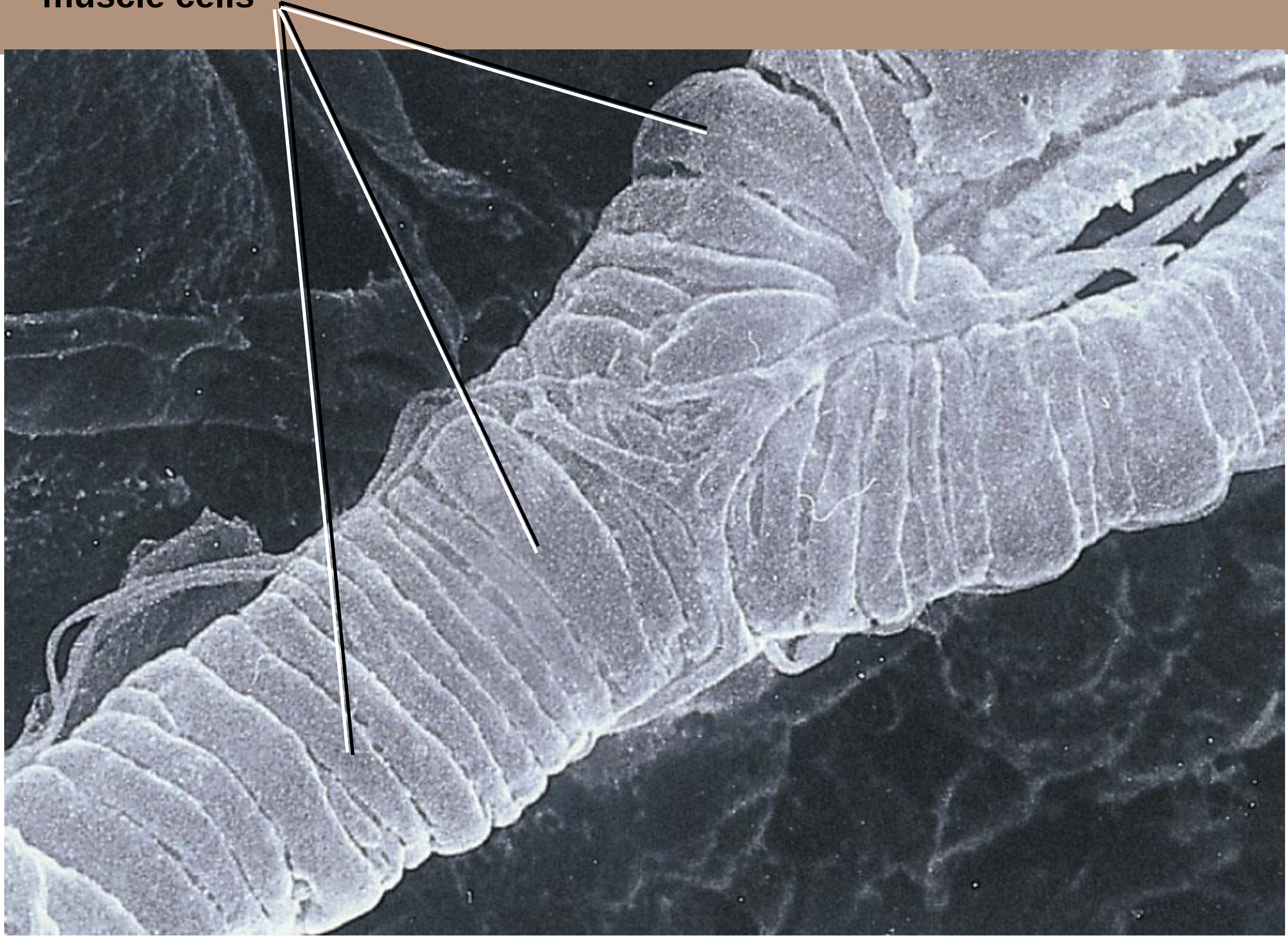
Caused by:
- ↑ Myogenic activity
- ↑ Oxygen (O_2)
- ↓ Carbon dioxide (CO_2) and other metabolites
- ↑ Endothelin
- ↑ Sympathetic stimulation
- Vasopressin; angiotensin II
- Cold

(c) Vasoconstriction (increased contraction of circular smooth muscle in the arteriolar wall, which leads to increased resistance and decreased flow through the vessel)



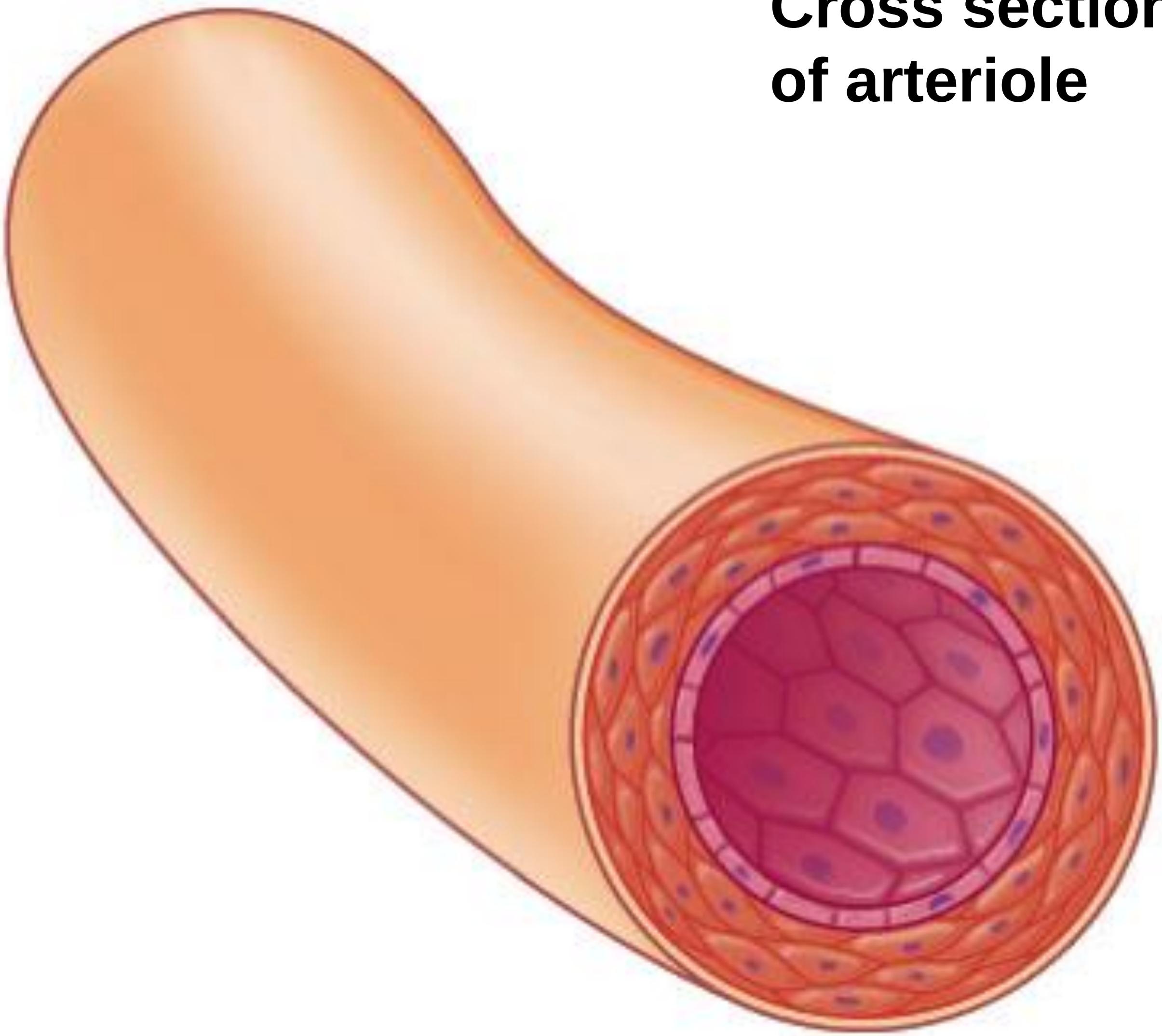
Caused by:
- ↓ Myogenic activity
- ↓ O_2
- ↑ CO_2 and other metabolites
- ↑ Nitric oxide
- ↓ Sympathetic stimulation
- Histamine release
- Heat

(d) Vasodilation (decreased contraction of circular smooth muscle in the arteriolar wall, which leads to decreased resistance and increased flow through the vessel)



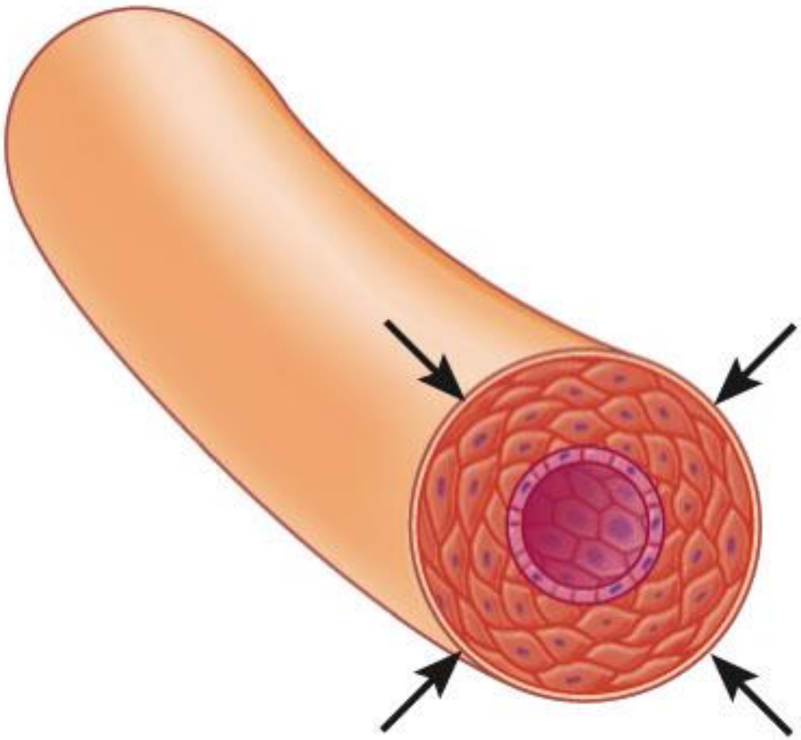
(a) Scanning electron micrograph of an arteriole showing how the smooth muscle cells run circularly around the vessel wall

Figure 9-46a p433



(b) Normal arteriolar tone

Figure 9-46b p433

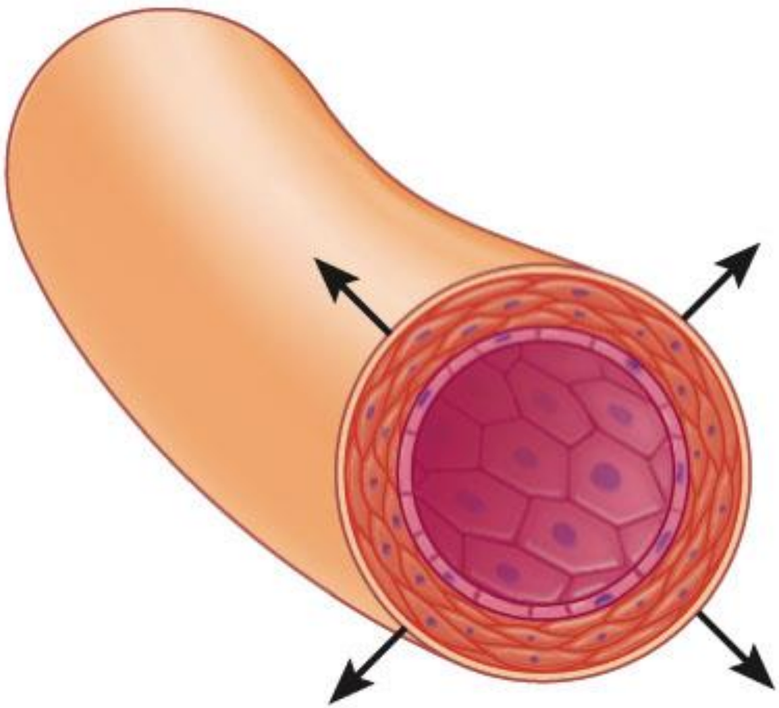


Caused by:

- ↑ Myogenic activity
- ↑ Oxygen (O_2)
- ↓ Carbon dioxide (CO_2) and other metabolites
- ↑ Endothelin
- ↑ Sympathetic stimulation
- Vasopressin; angiotensin II
- Cold

(c) Vasoconstriction (increased contraction of circular smooth muscle in the arteriolar wall, which leads to increased resistance and decreased flow through the vessel)

Figure 9-46c p433



Caused by:

- ↓ Myogenic activity
- ↓ O_2
- ↑ CO_2 and other metabolites
- ↑ Nitric oxide
- ↓ Sympathetic stimulation
- Histamine release
- Heat

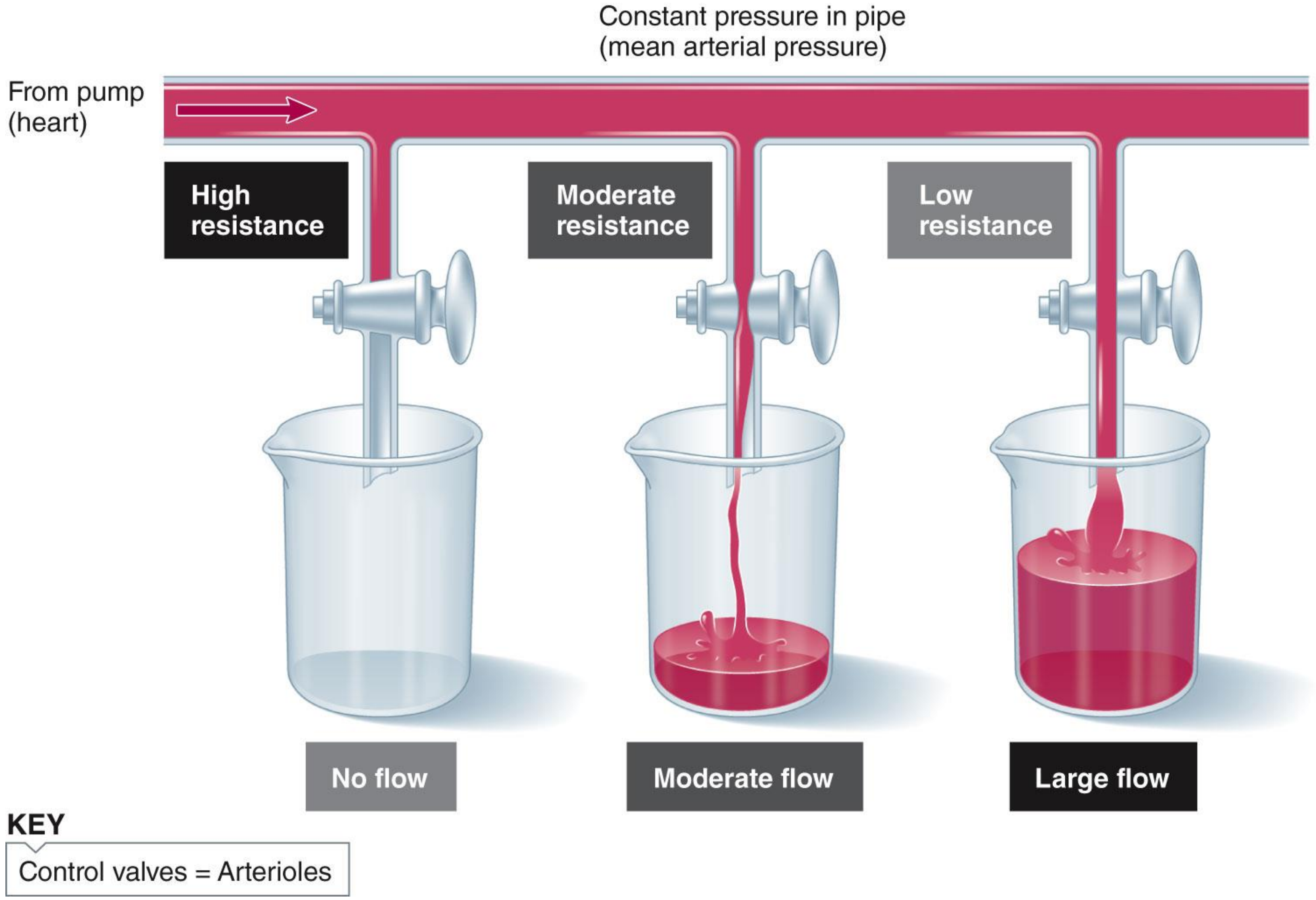
(d) Vasodilation (decreased contraction of circular smooth muscle in the arteriolar wall, which leads to decreased resistance and increased flow through the vessel)

Figure 9-46d p433

9.12 Circulatory Vessels: Arterioles

- Distribution of blood flow
  - **Amount of blood flow** received by each organ is determined by the **number** and **diameter** of its arterioles
  - **Local (intrinsic) controls** are changes within a tissue that alter the radii of arterioles
    - **Local metabolic changes** produce relaxation of arteriolar smooth muscle to increase blood flow to the organ (**active hyperemia**)
    - **Histamine** release causes vasodilation as an inflammatory response
    - Exposure to **heat** or **cold**
    - **Stretch** produces myogenic vasoconstriction

9.12 Circulatory Vessels: Arterioles



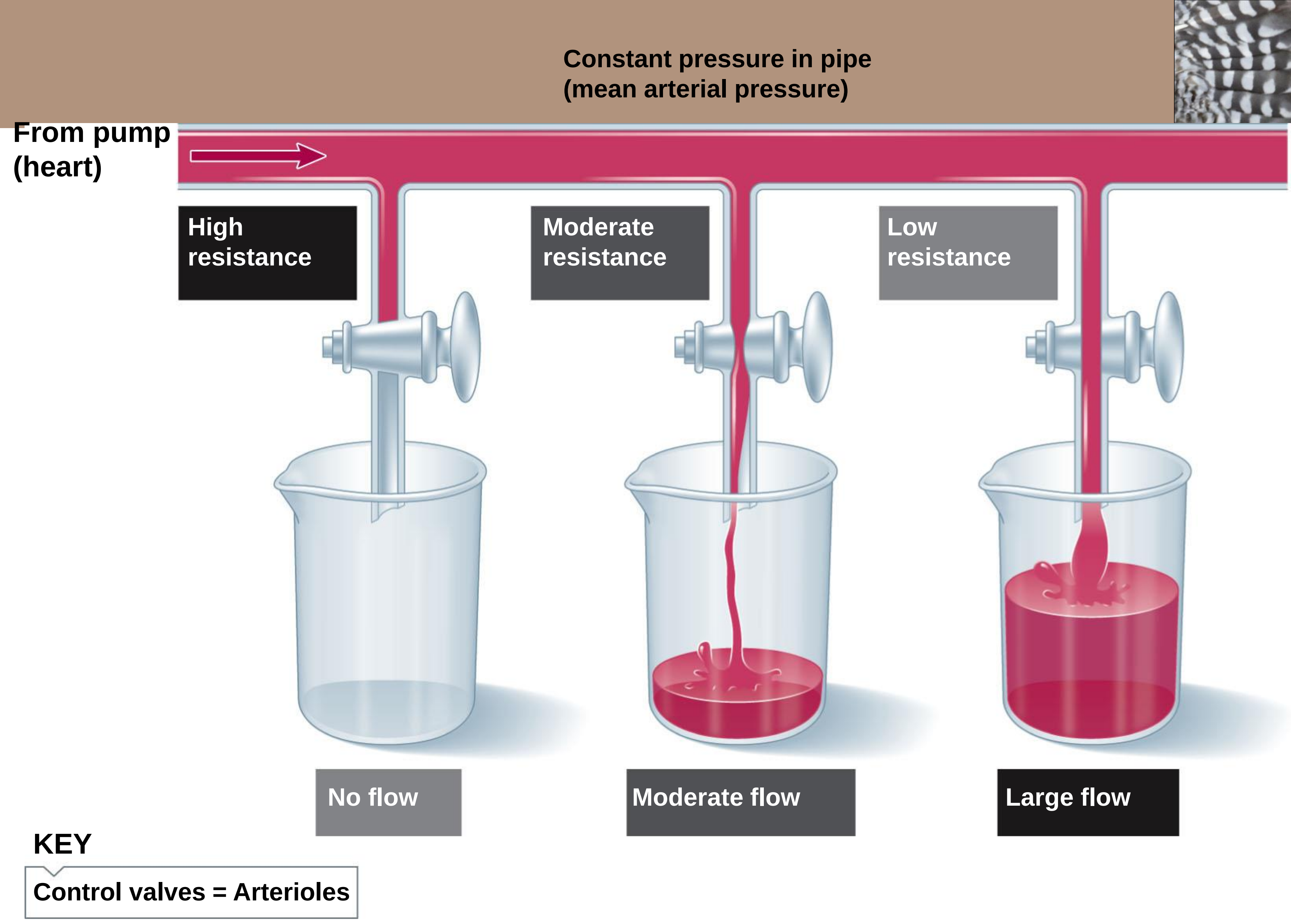
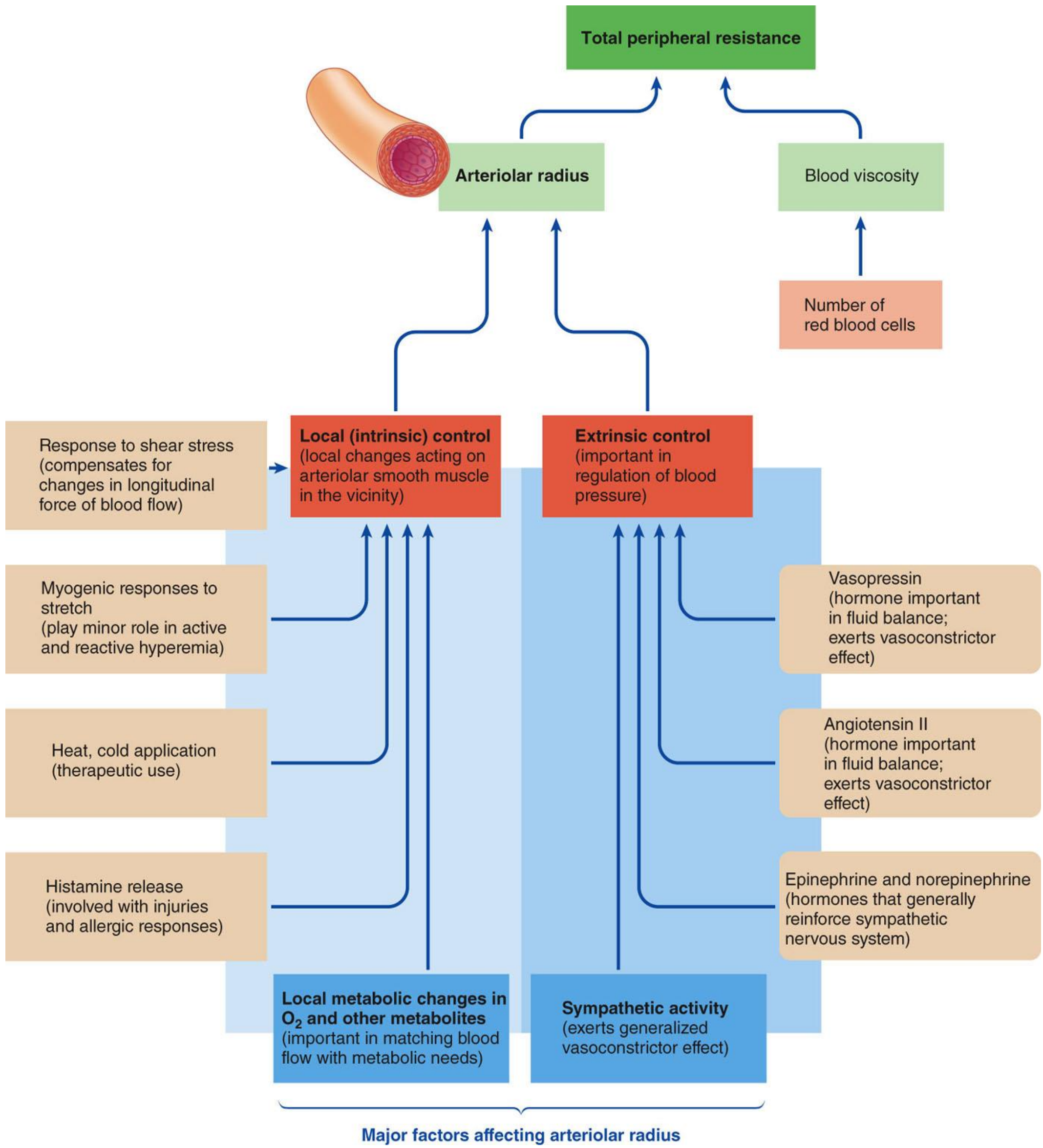


Figure 9-47 p434

9.12 Circulatory Vessels: Arterioles

- **Extrinsic control** includes **neural** and **hormonal** influences with the **sympathetic nervous system** dominating
  - Sympathetic stimulation **redistributes blood flow** to the heart and skeletal muscle at the expense of other organs
    - **Vasoconstriction** of most arterioles due to activation of **α_1-adrenergic receptors** by NE or epinephrine
    - **Lung** and **brain** arterioles do not have α_1-receptors and do not constrict
    - Epinephrine activates **β_2-adrenergic receptors** in arterioles of **skeletal muscle** and **heart** to cause **vasodilation**
  - **Mean arterial pressure** is the product of **cardiac output** and **total peripheral resistance**

9.12 Circulatory Vessels: Arterioles



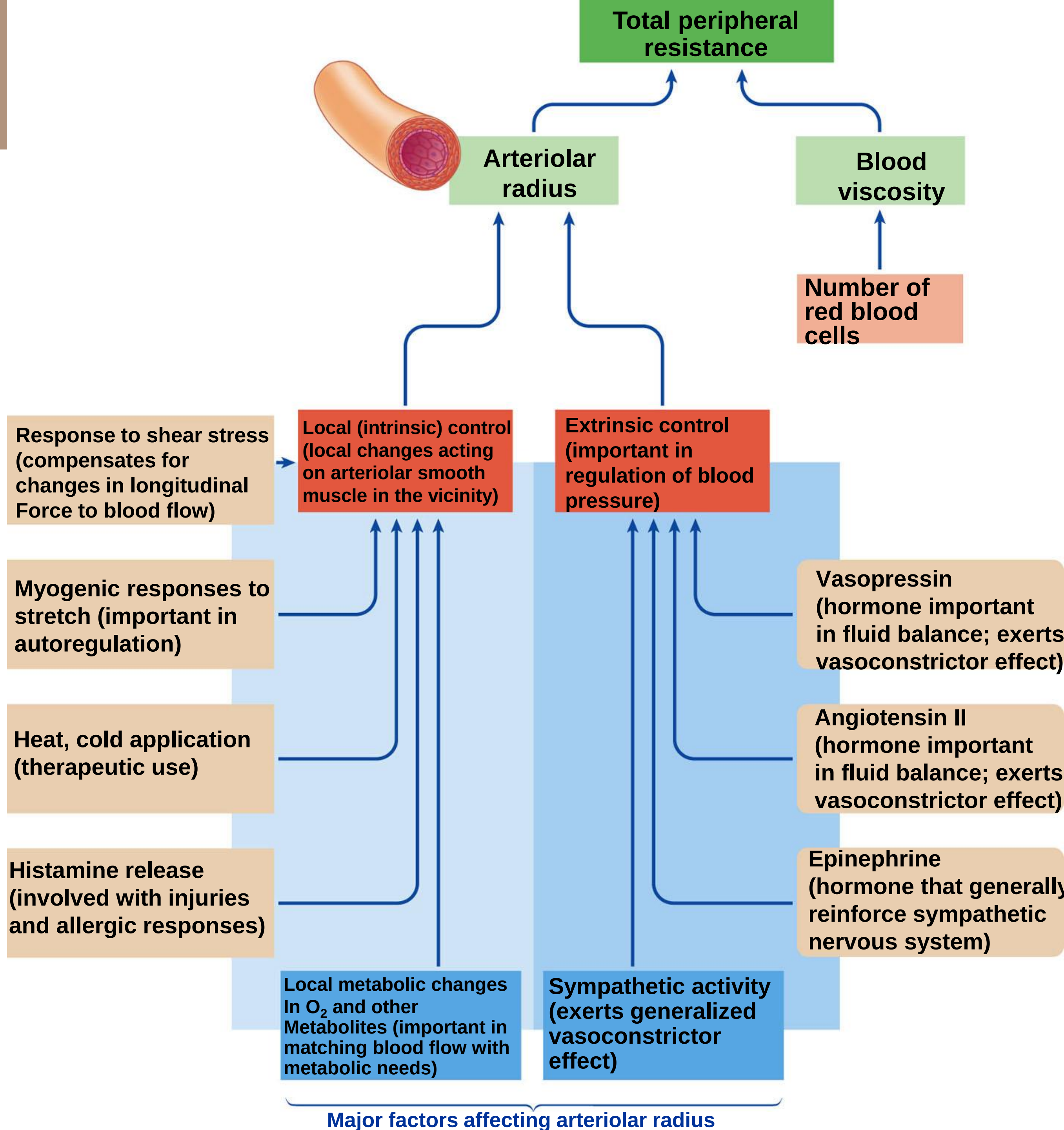
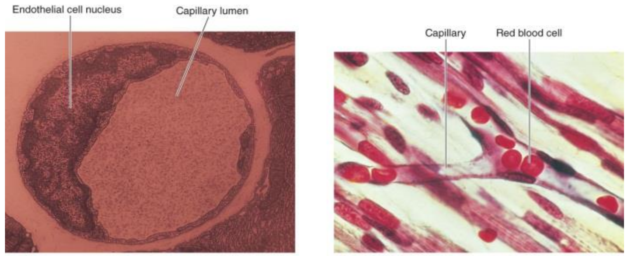


Figure 9-48 p438

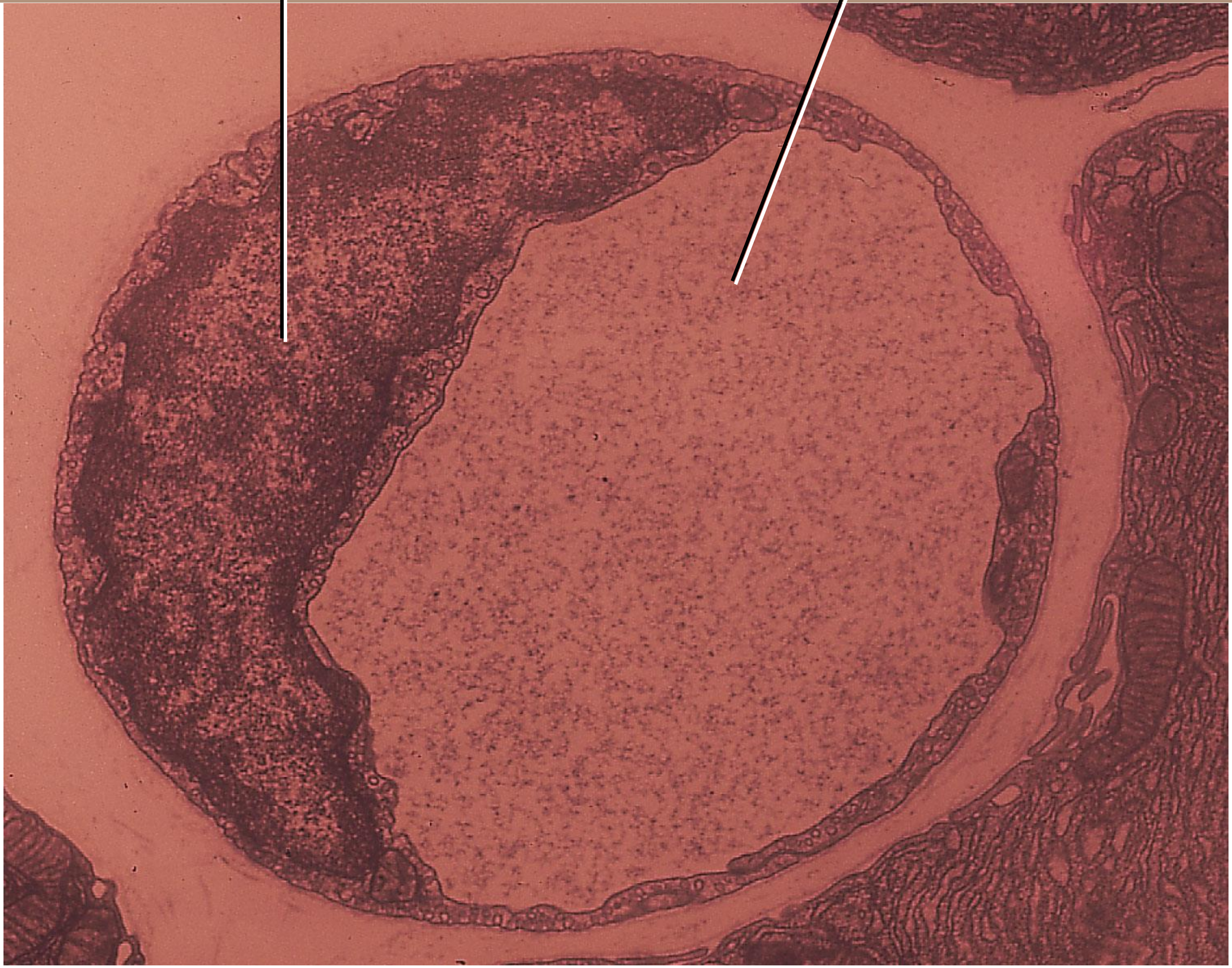
9.13 Circulatory Vessels: Capillaries

- **Capillaries** maximize diffusion rates for **exchange of materials**
  - **Diffusion distance** is short
    - Capillary walls are thin
    - Capillaries are narrow
    - Capillaries branch extensively

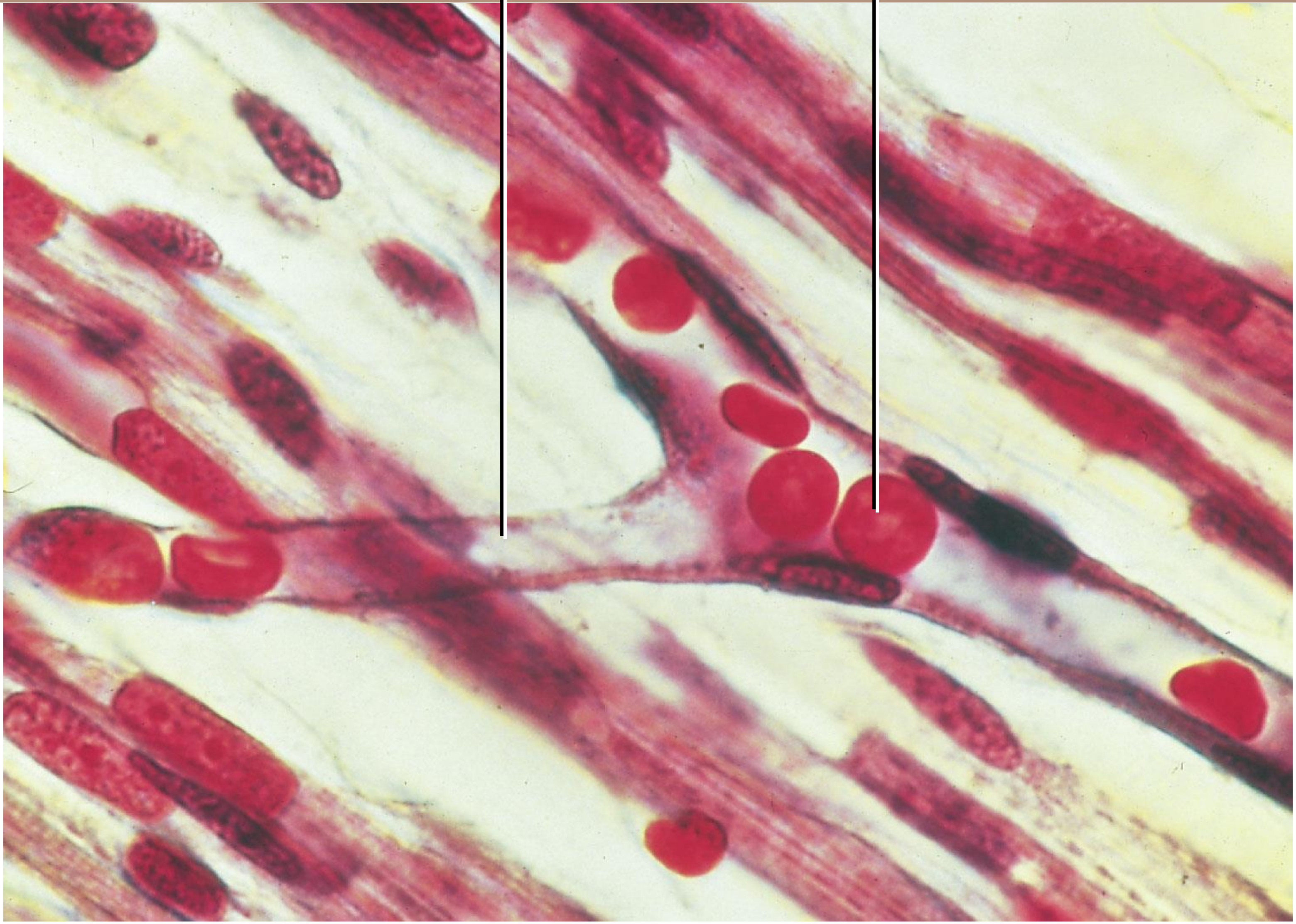


(a) Cross section of a capillary
(b) Capillary bed

  - **Surface area** is large
  - **Permeability** is high
    - **Pores** between capillary endothelial cells
      - **Tight junctions** in **brain** capillaries form protective **blood-brain barrier**
      - **Large pores** in **liver** capillaries allow passage of **proteins**



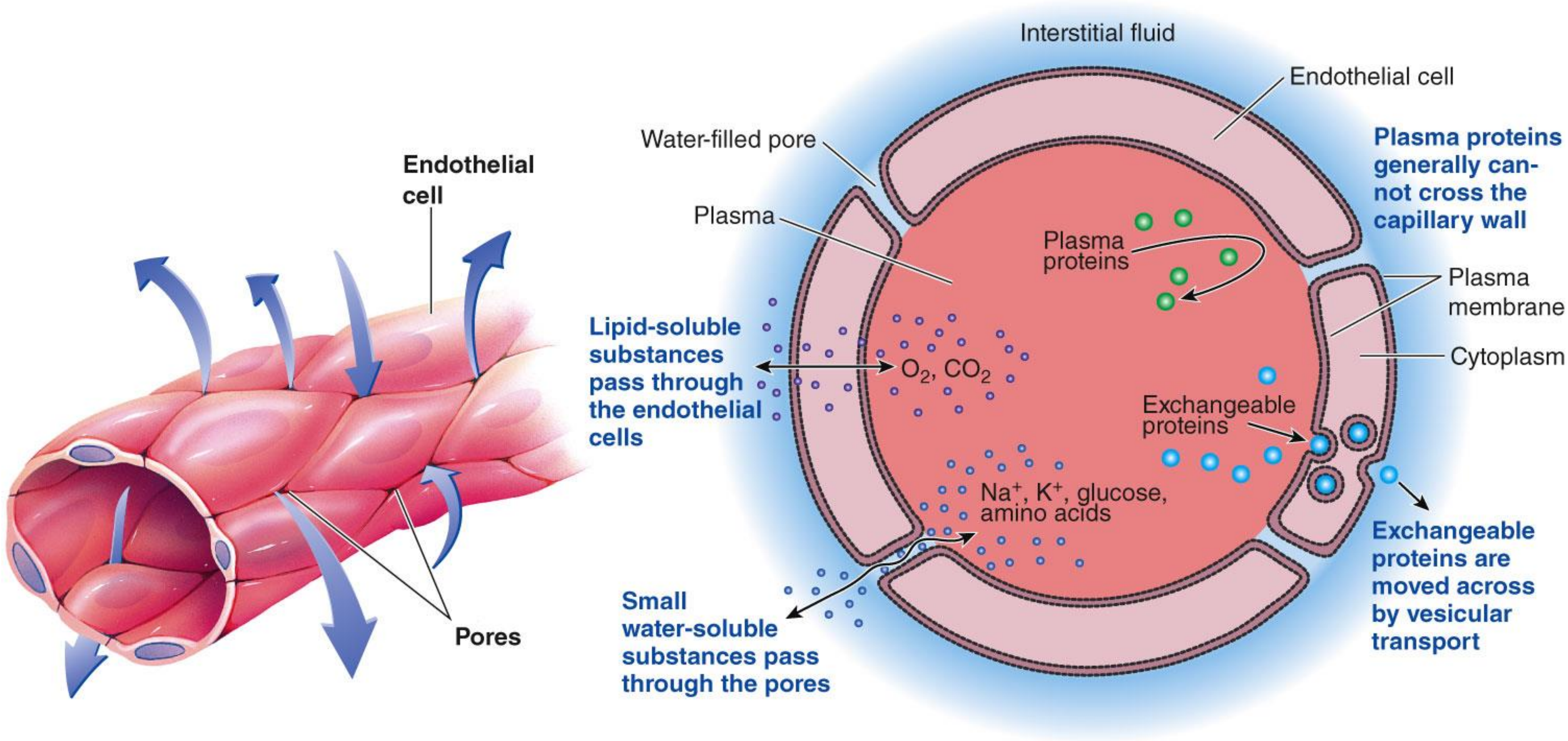
(a) Cross section of a capillary



(b) Capillary bed

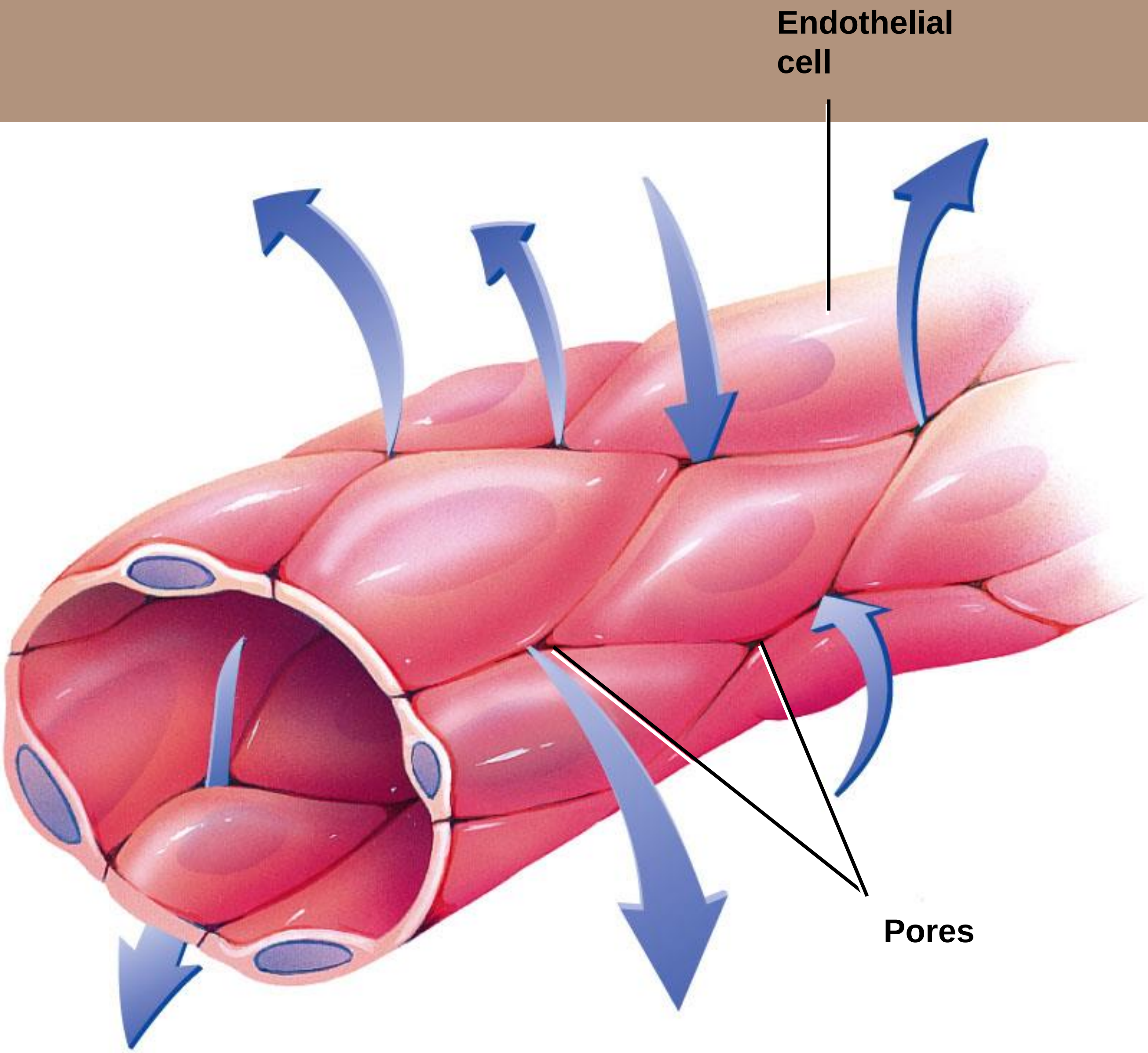
Figure 9-49b p439

9.13 Circulatory Vessels: Capillaries



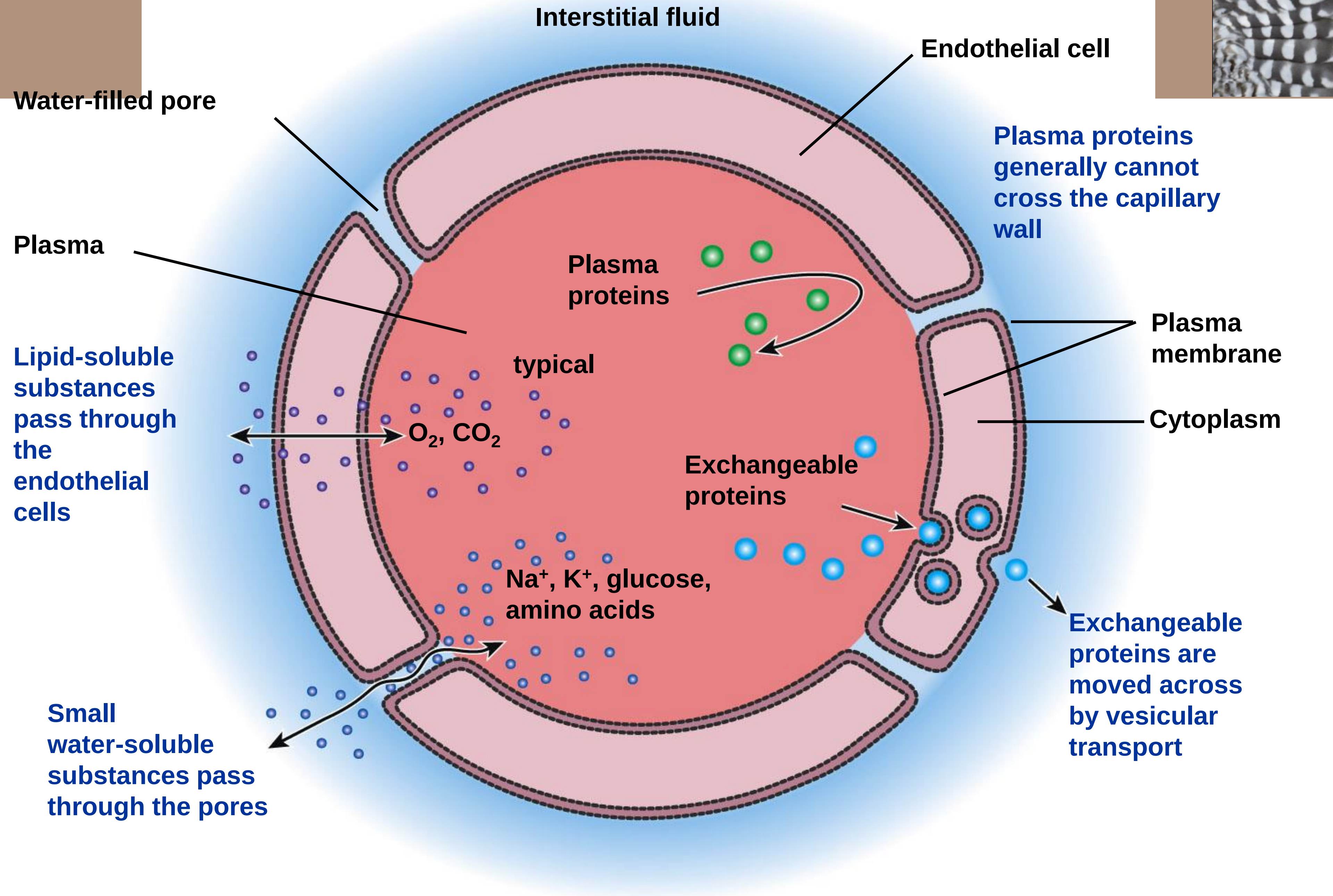
(a) Continuous capillary

(b) Transport across a continuous capillary wall



(a) Typical capillary

Figure 9-50 p439



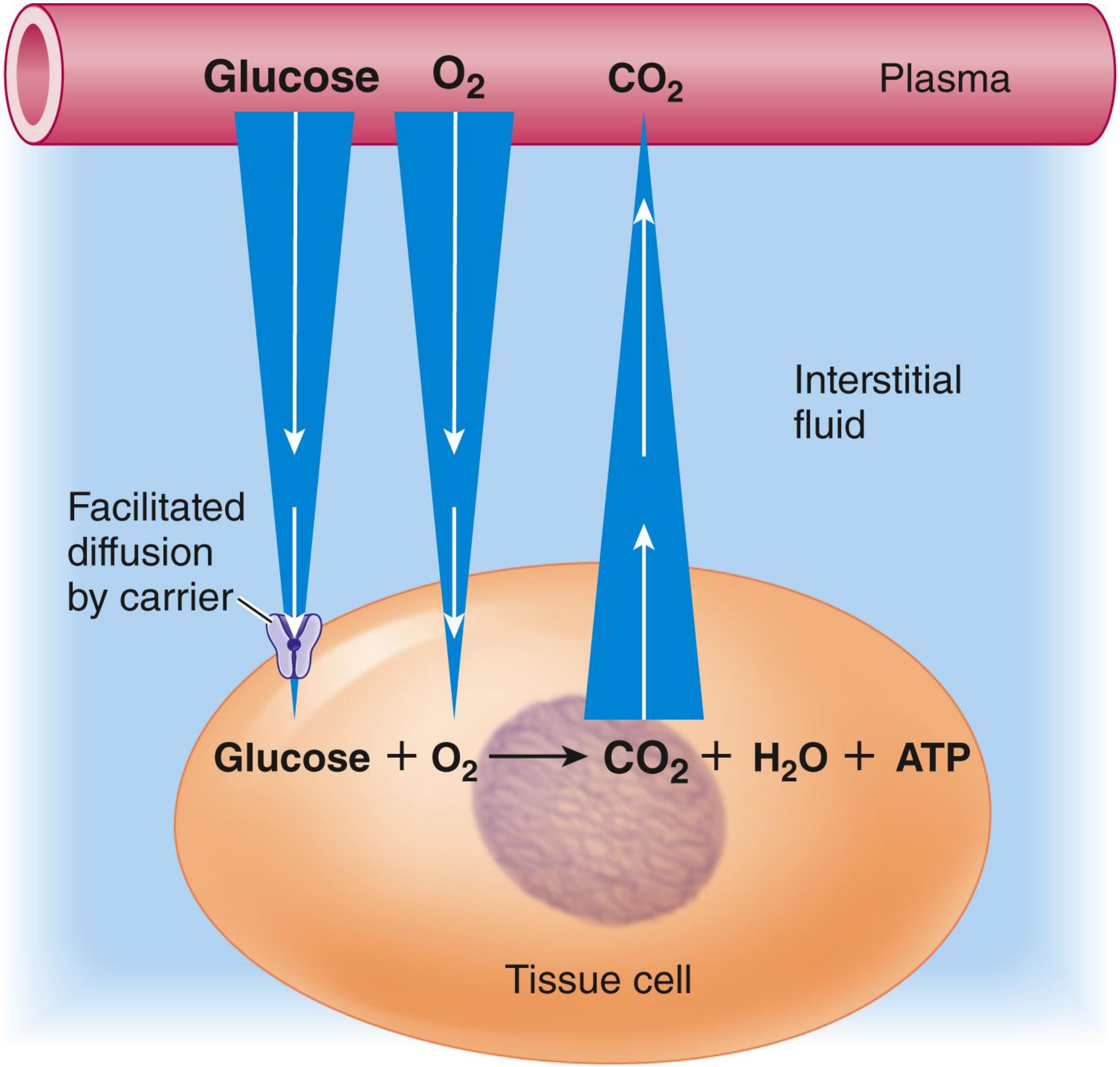
(b) Transport across a typical capillary wall

Figure 9-50 p439

9.13 Circulatory Vessels: Capillaries

- Diffusion follows **concentration gradients** between blood and the surrounding cells
  - Cells **consume** glucose and oxygen
  - Cells **produce** CO_2 and other metabolic wastes
  - **Precapillary sphincters** controlling flow through individual capillaries are sensitive to **local metabolic changes**
  - **Blood velocity** is **slowest** due to the very **large cross-sectional area** of the capillaries

9.13 Circulatory Vessels: Capillaries



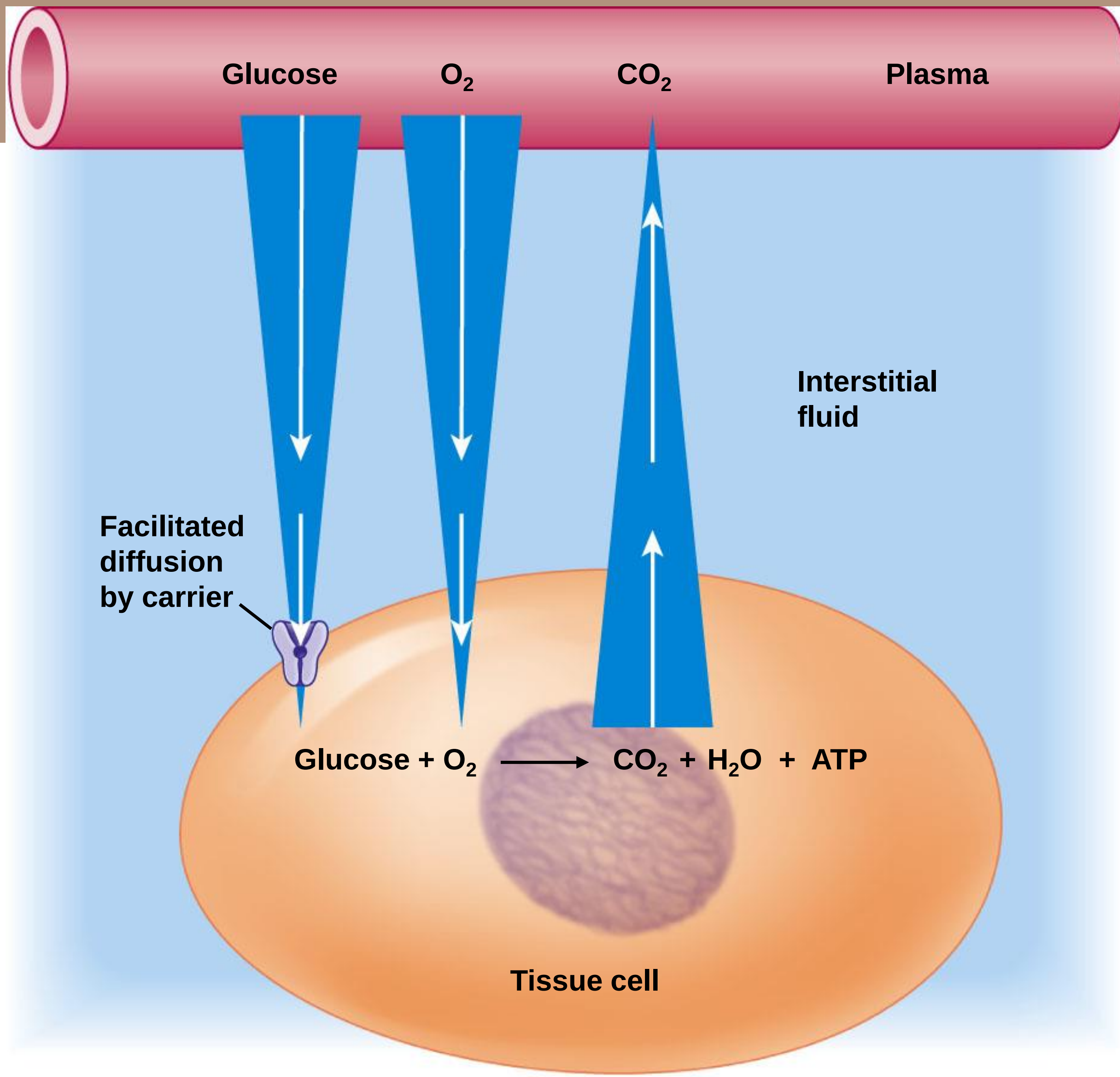
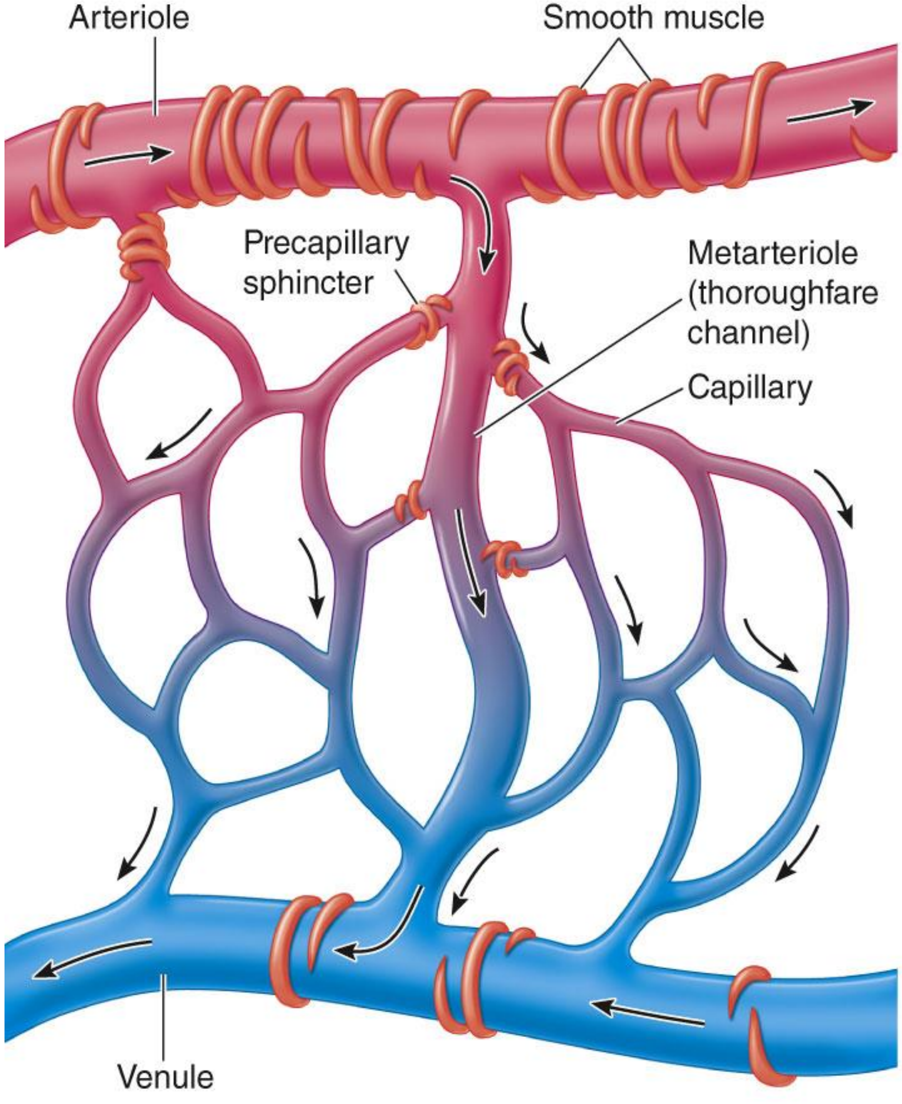
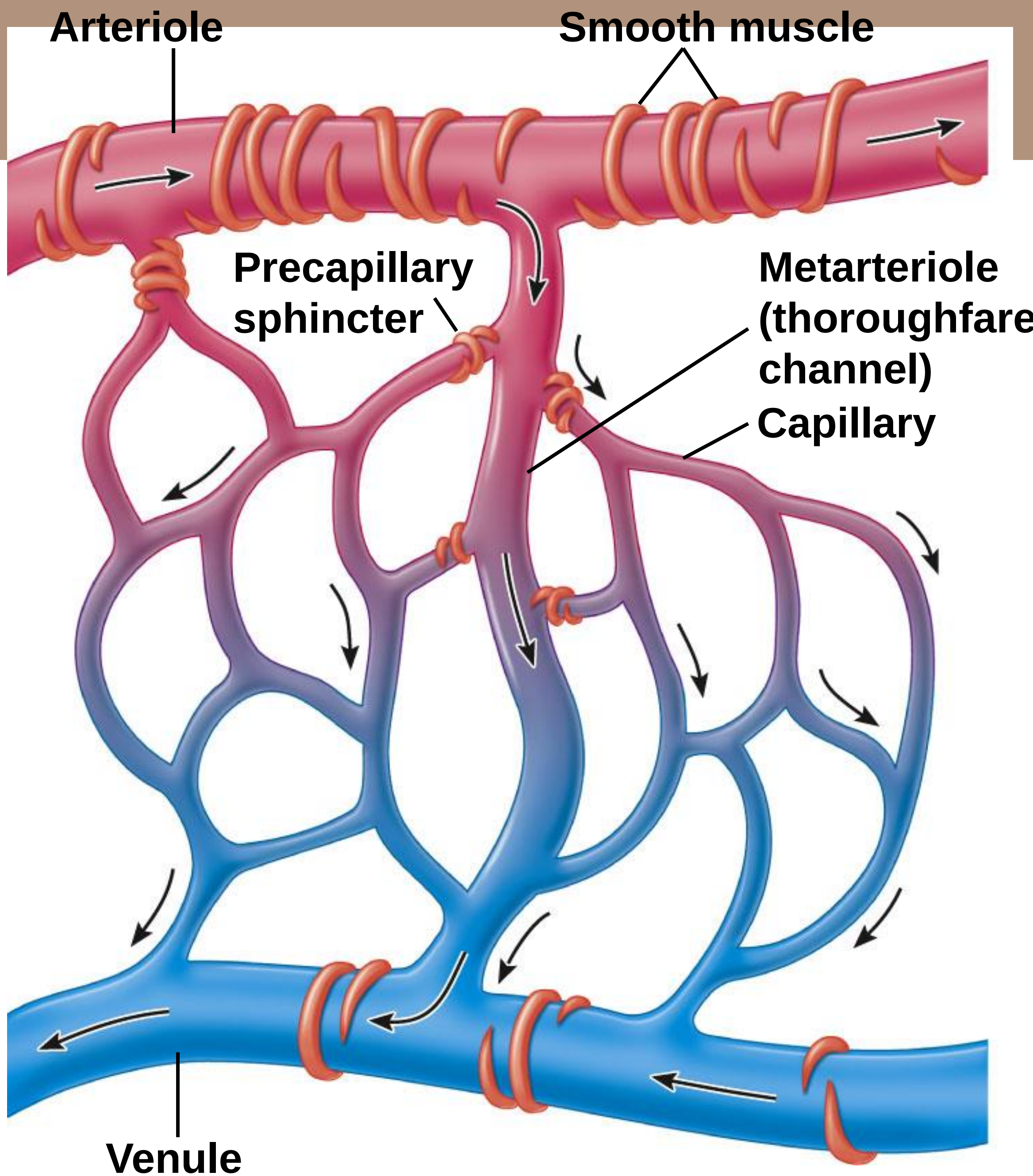


Figure 9-51 p440

9.13 Circulatory Vessels: Capillaries



(a) Sphincters relaxed



(a) Sphincters relaxed

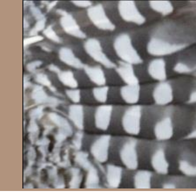
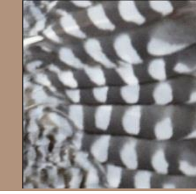
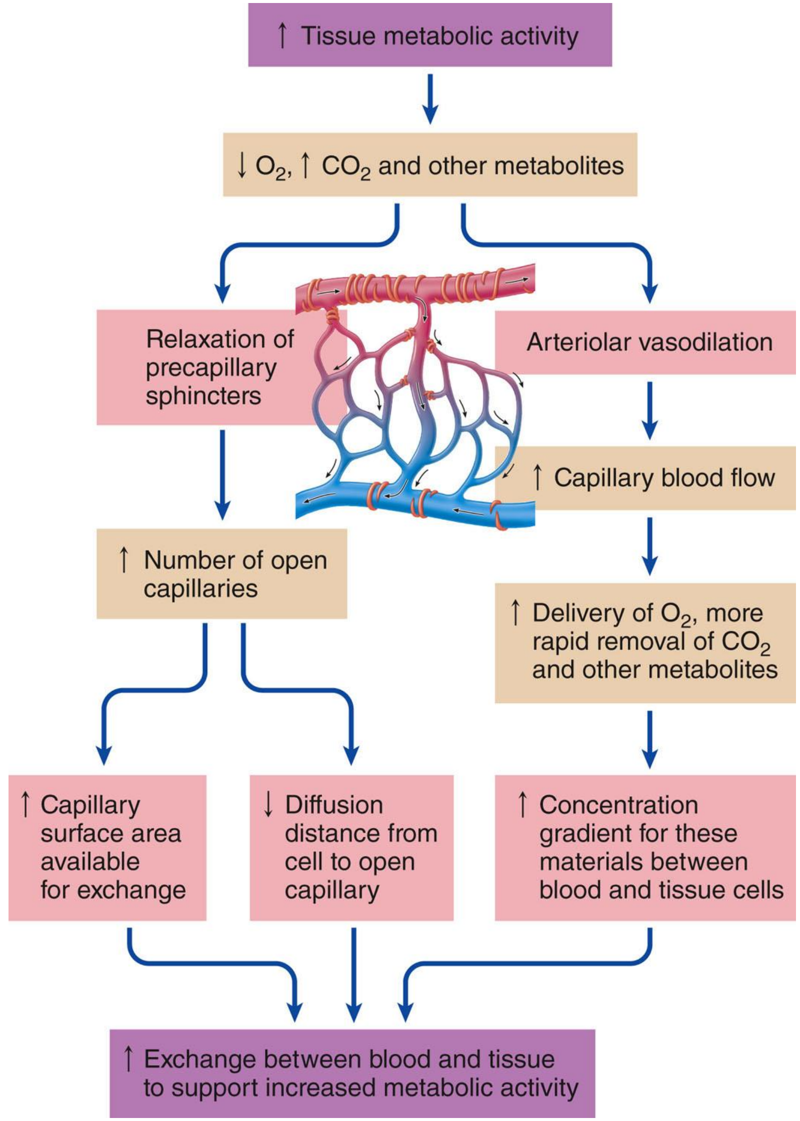


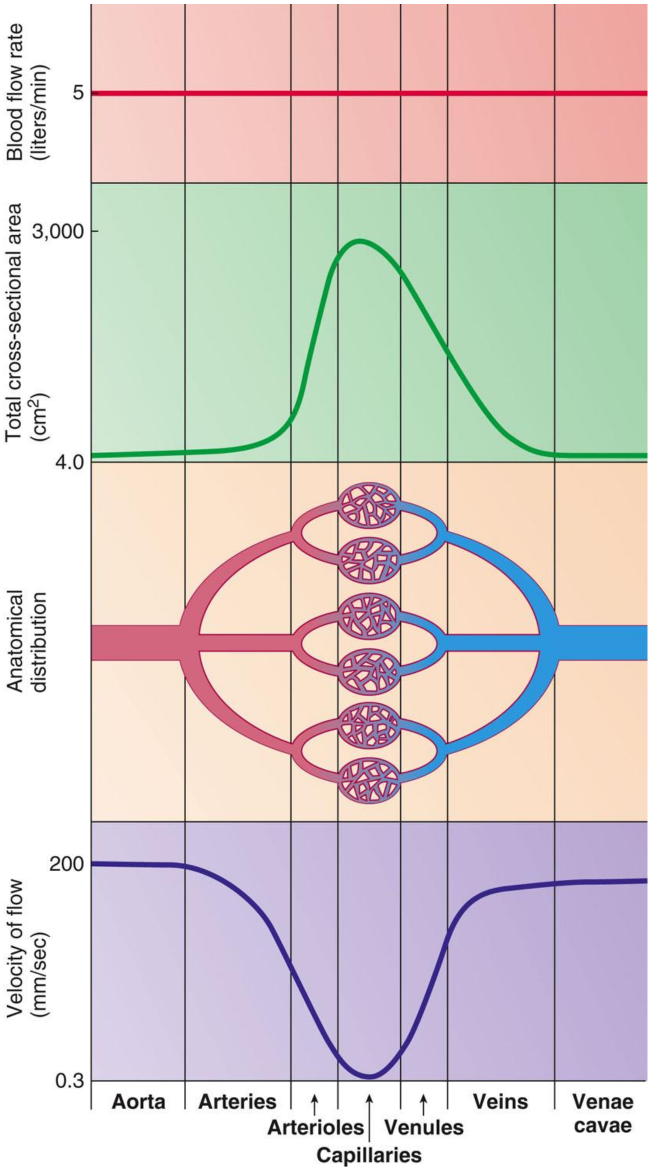
Figure 9-52 p440

9.13 Circulatory Vessels: Capillaries





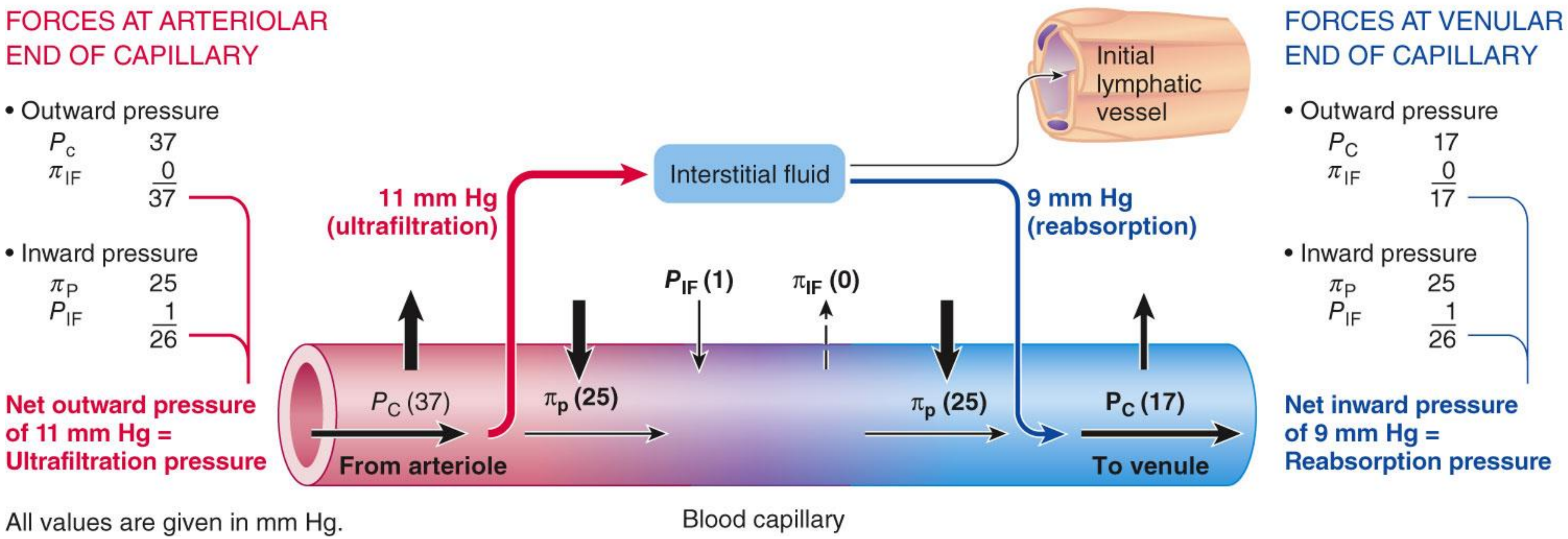
9.13 Circulatory Vessels: Capillaries



9.13 Circulatory Vessels: Capillaries

- Bulk flow across the capillary wall
  - Bulk flow occurs because of differences in the **hydrostatic** and **colloid osmotic pressures** between plasma and interstitial fluid
    - **Capillary blood pressure (P_C)** is the hydrostatic pressure exerted on the capillary walls
    - **Plasma colloid osmotic pressure (π_p)** is caused by plasma proteins
    - **Interstitial fluid hydrostatic pressure (P_{IF})** is low
    - **Interstitial fluid colloid osmotic pressure (π_{IF})** is very low
  - Protein-free plasma flows out of the capillary (**ultrafiltration**), mixes with interstitial fluid and then reenters the capillary (**reabsorption**)

9.13 Circulatory Vessels: Capillaries



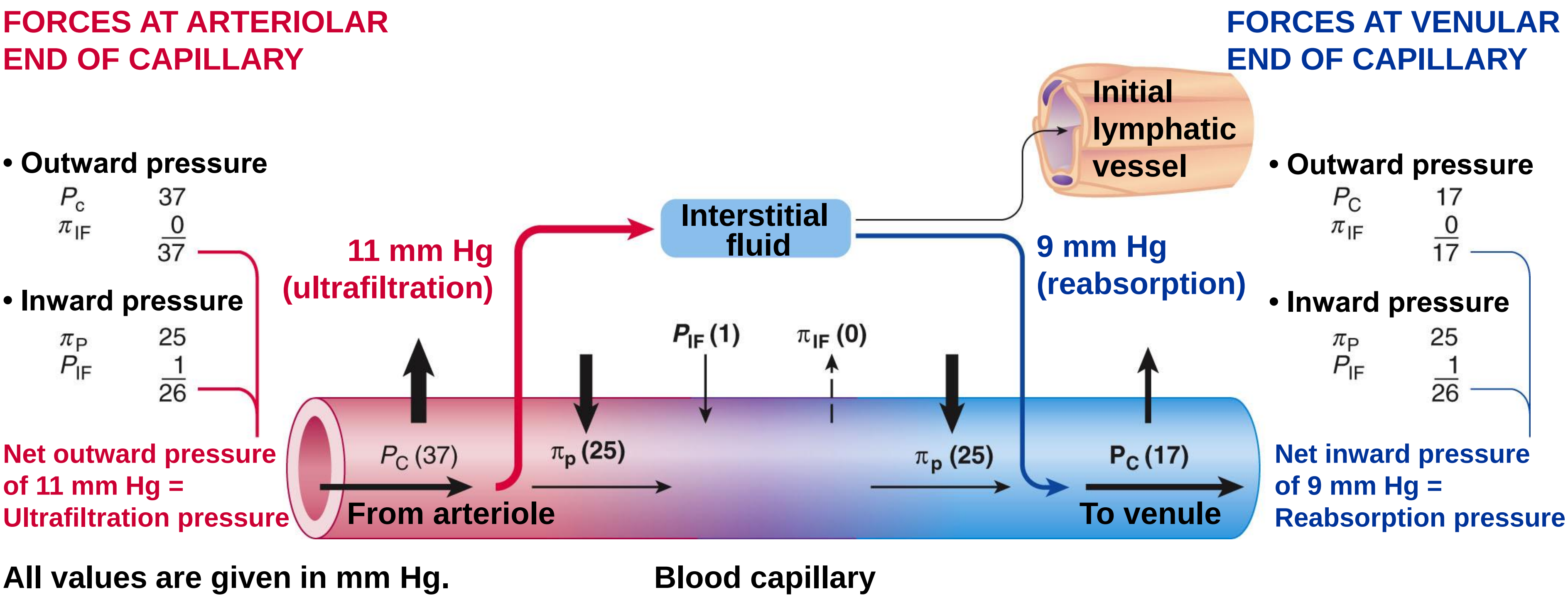
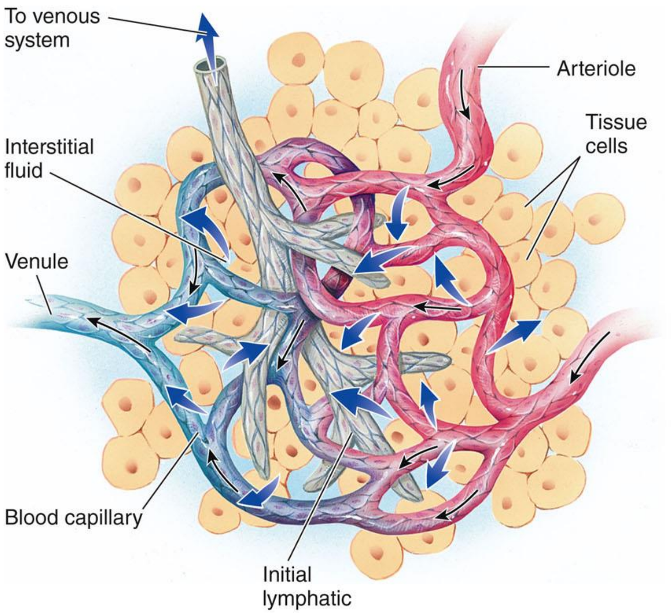


Figure 9-57 p443

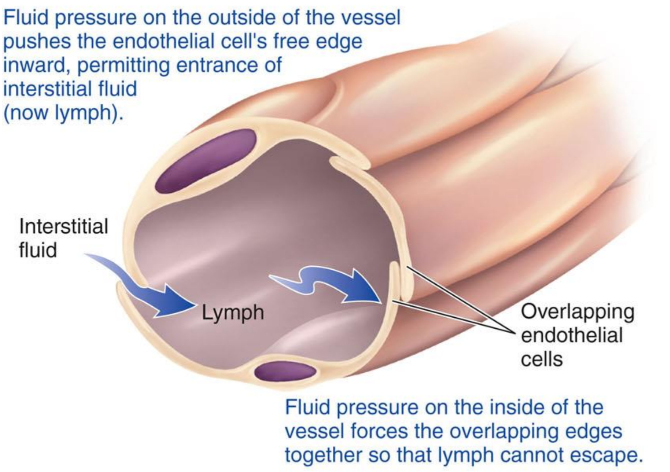
9.14 Circulatory Vessels: Lymphatic System

- The **lymphatic system** returns excess filtered fluid to the blood.
    - Network of **one-way vessels**
    - Interstitial fluid enters small blind-ended terminal vessels (**initial lymphatics**) to form **lymph**
    - Lymph vessels flow into the **venous system** near the heart
    - Passage of lymph through the **lymph nodes** aids in immune defense against disease
    - Lymph vessels **absorb fat** form the digestive tract

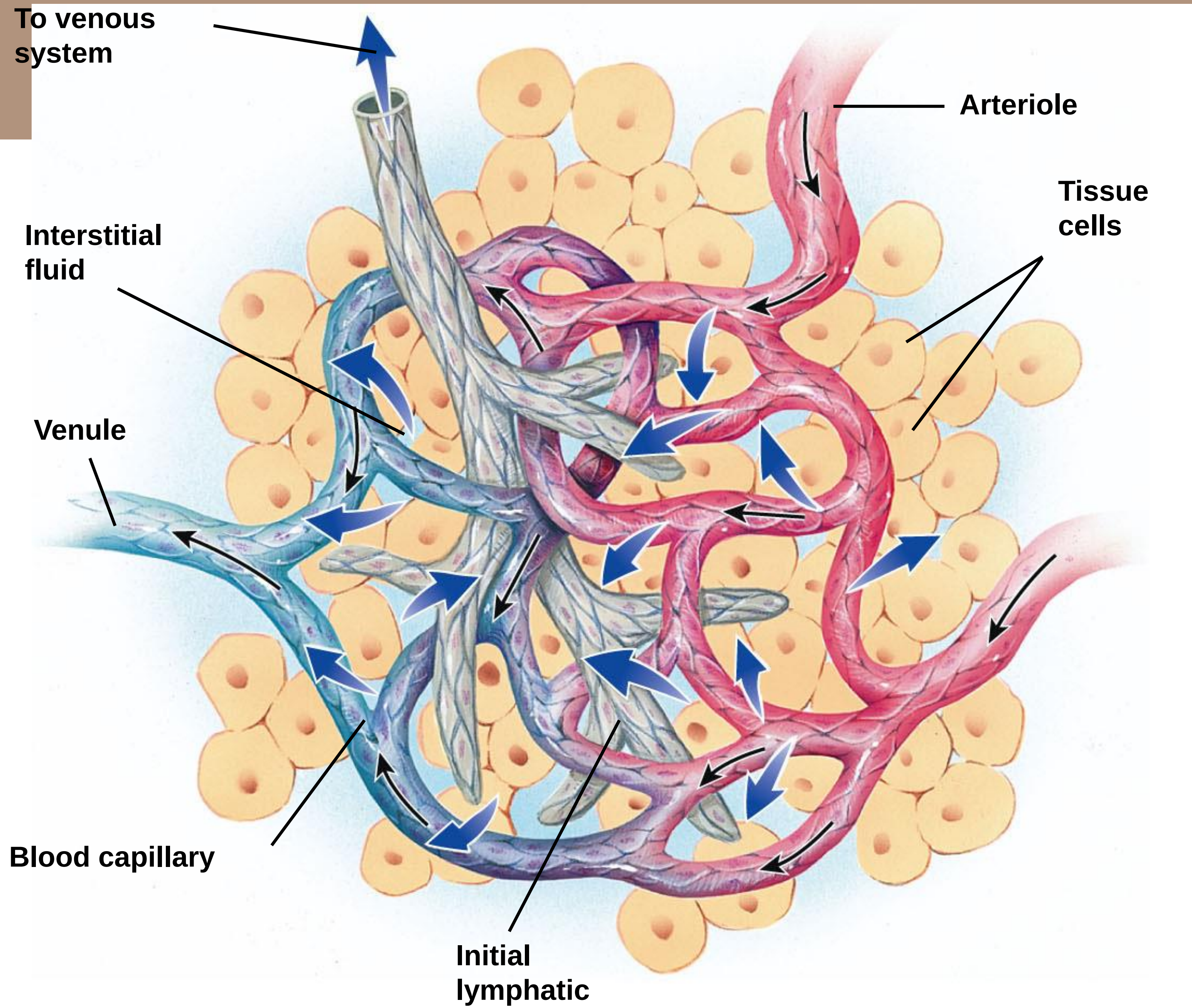
9.14 Circulatory Vessels: Lymphatic System



(a) Relationship between initial lymphatics and blood capillaries



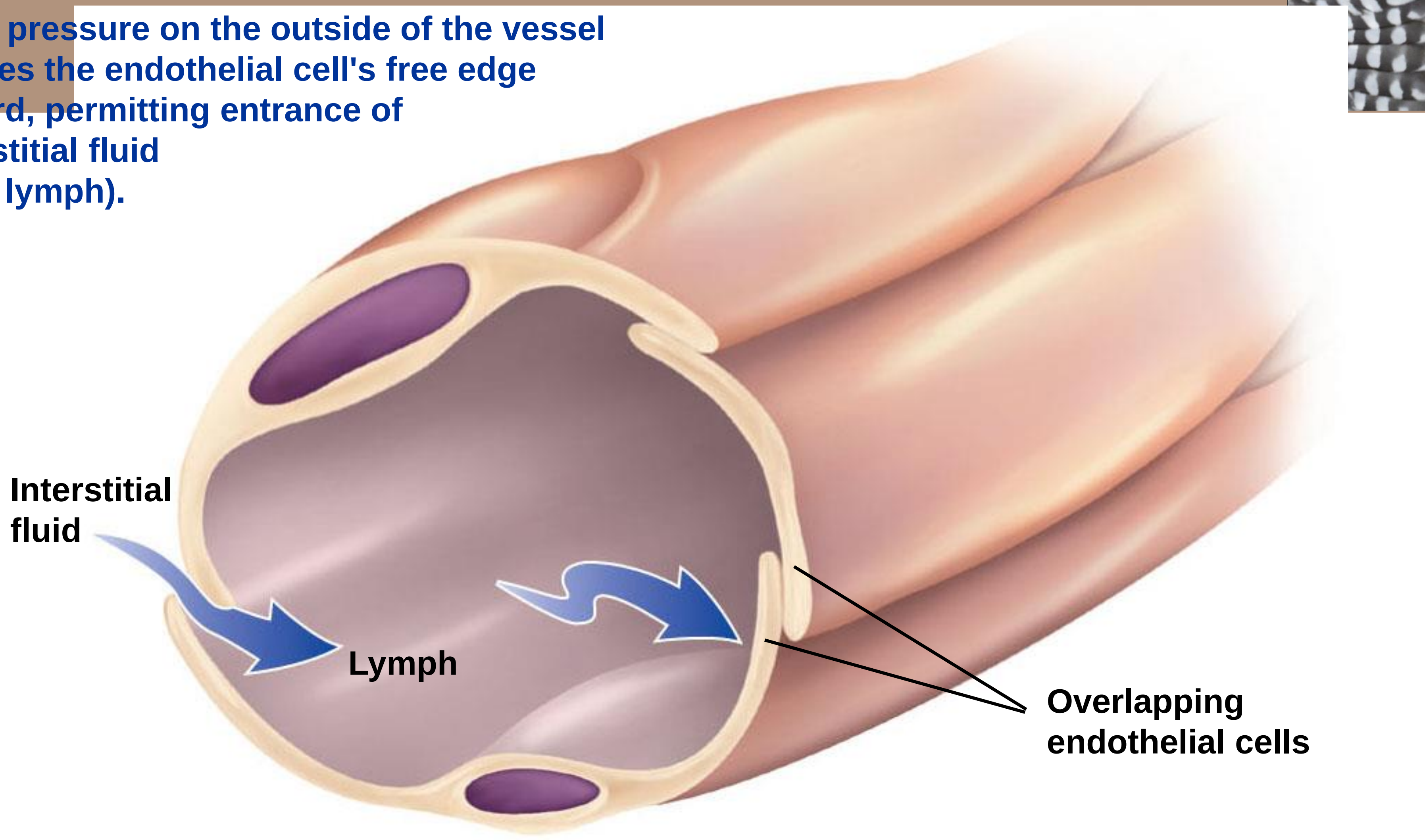
(b) Arrangement of endothelial cells in an initial lymphatic



(a) Relationship between initial lymphatics and blood capillaries

Figure 9-58a p445

Fluid pressure on the outside of the vessel pushes the endothelial cell's free edge inward, permitting entrance of interstitial fluid (now lymph).

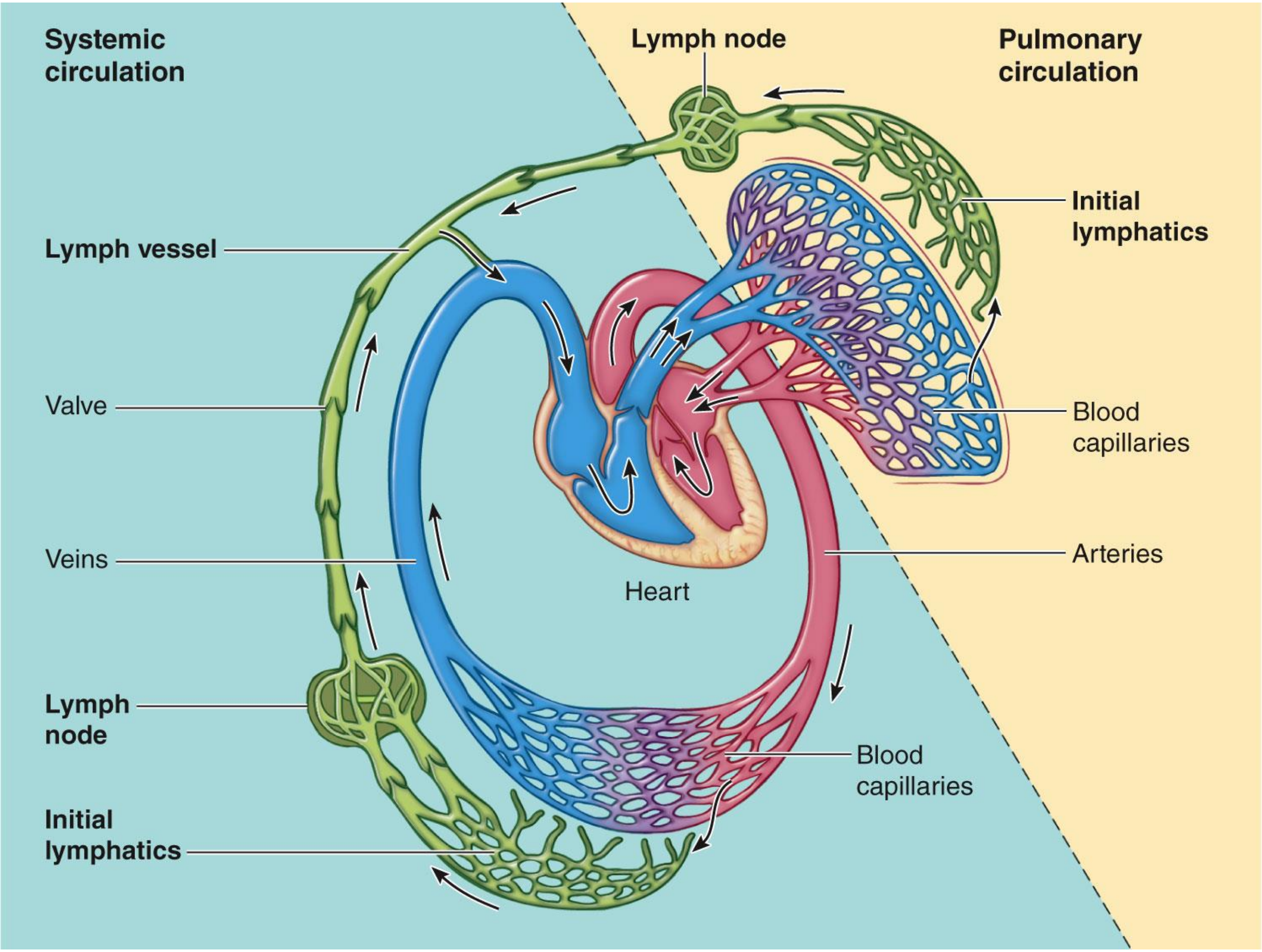


Fluid pressure on the inside of the vessel forces the overlapping edges together so that lymph cannot escape.

(b) Arrangement of endothelial cells in an initial lymphatic

Figure 9-58b p445

9.14 Circulatory Vessels: Lymphatic System



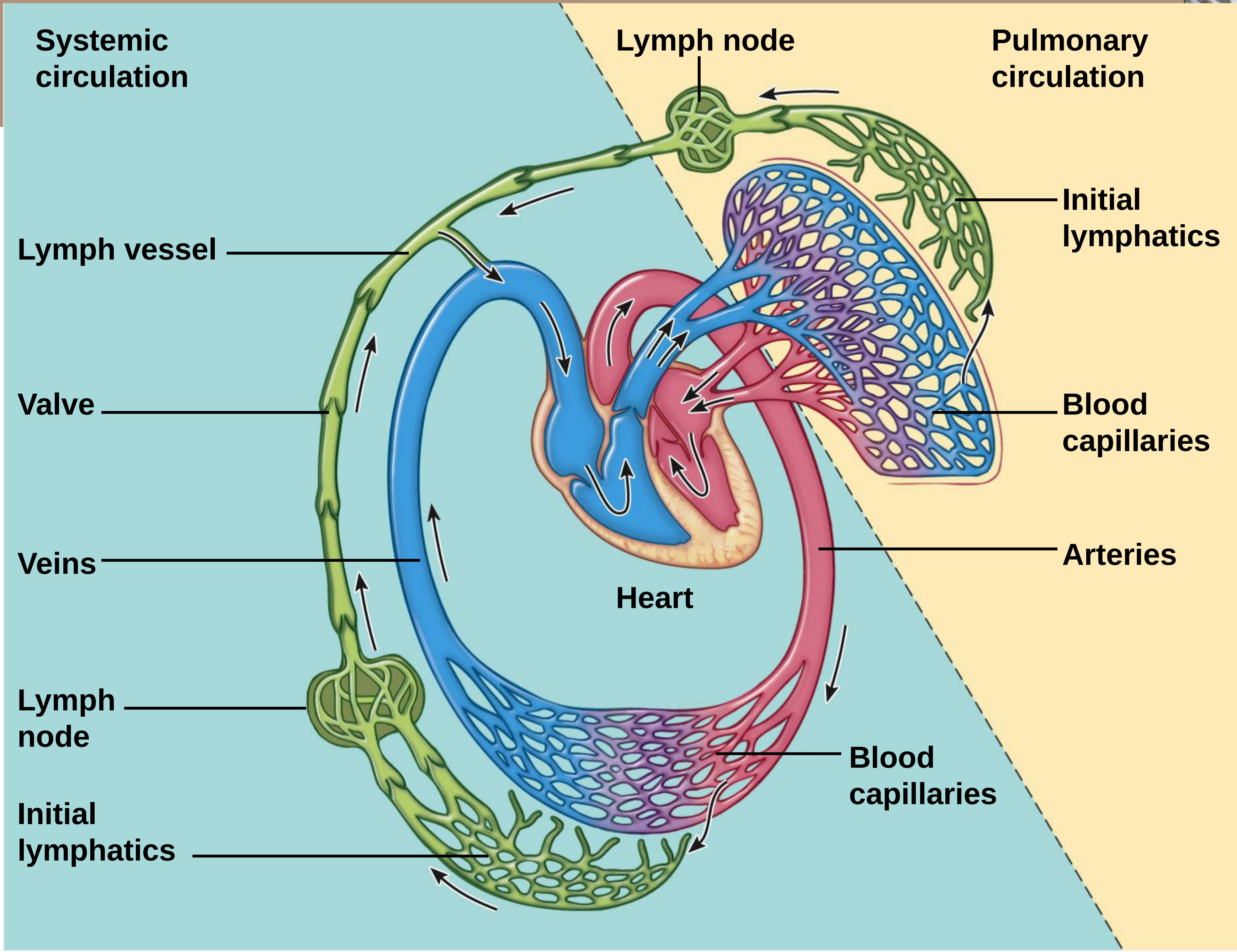
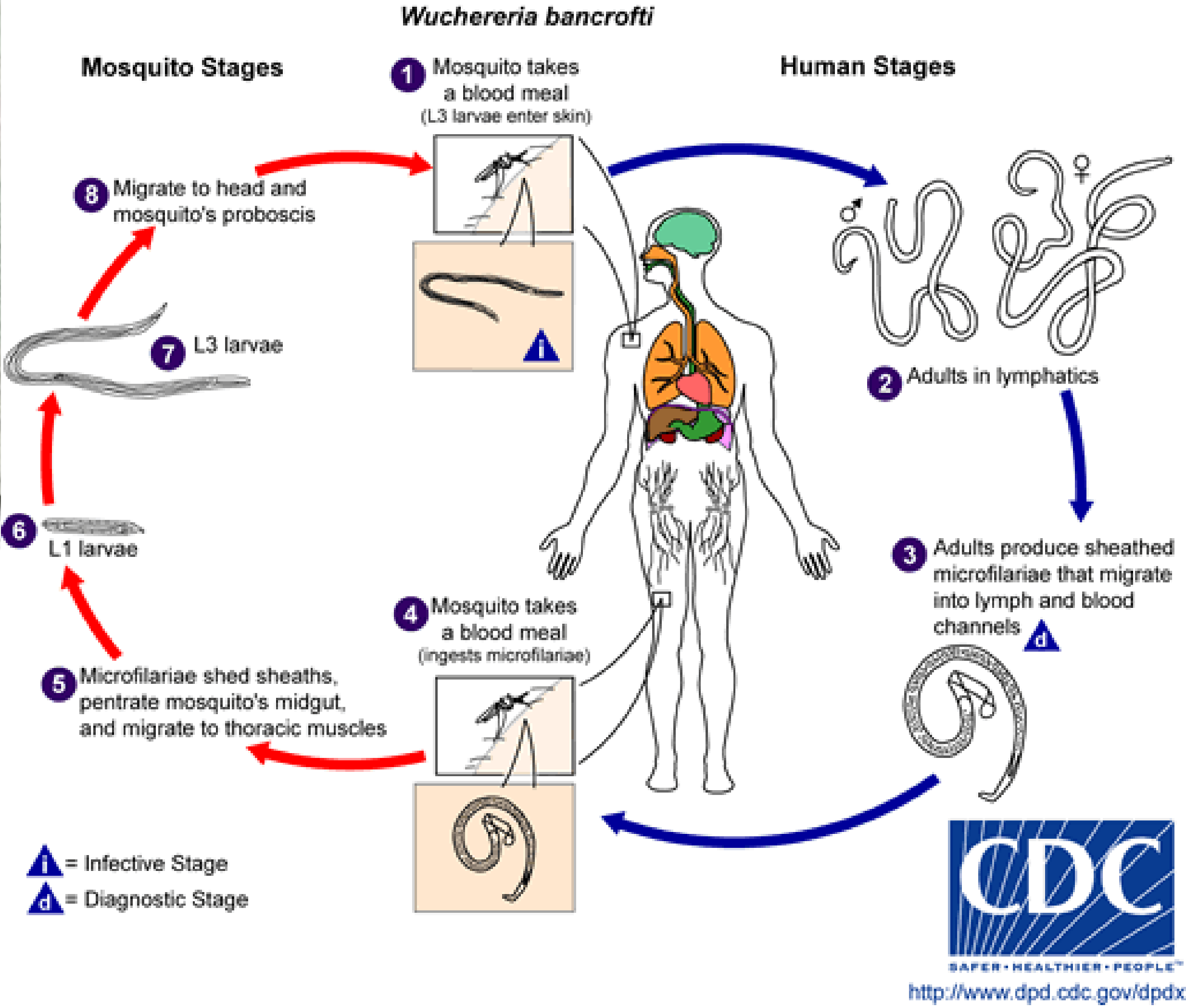


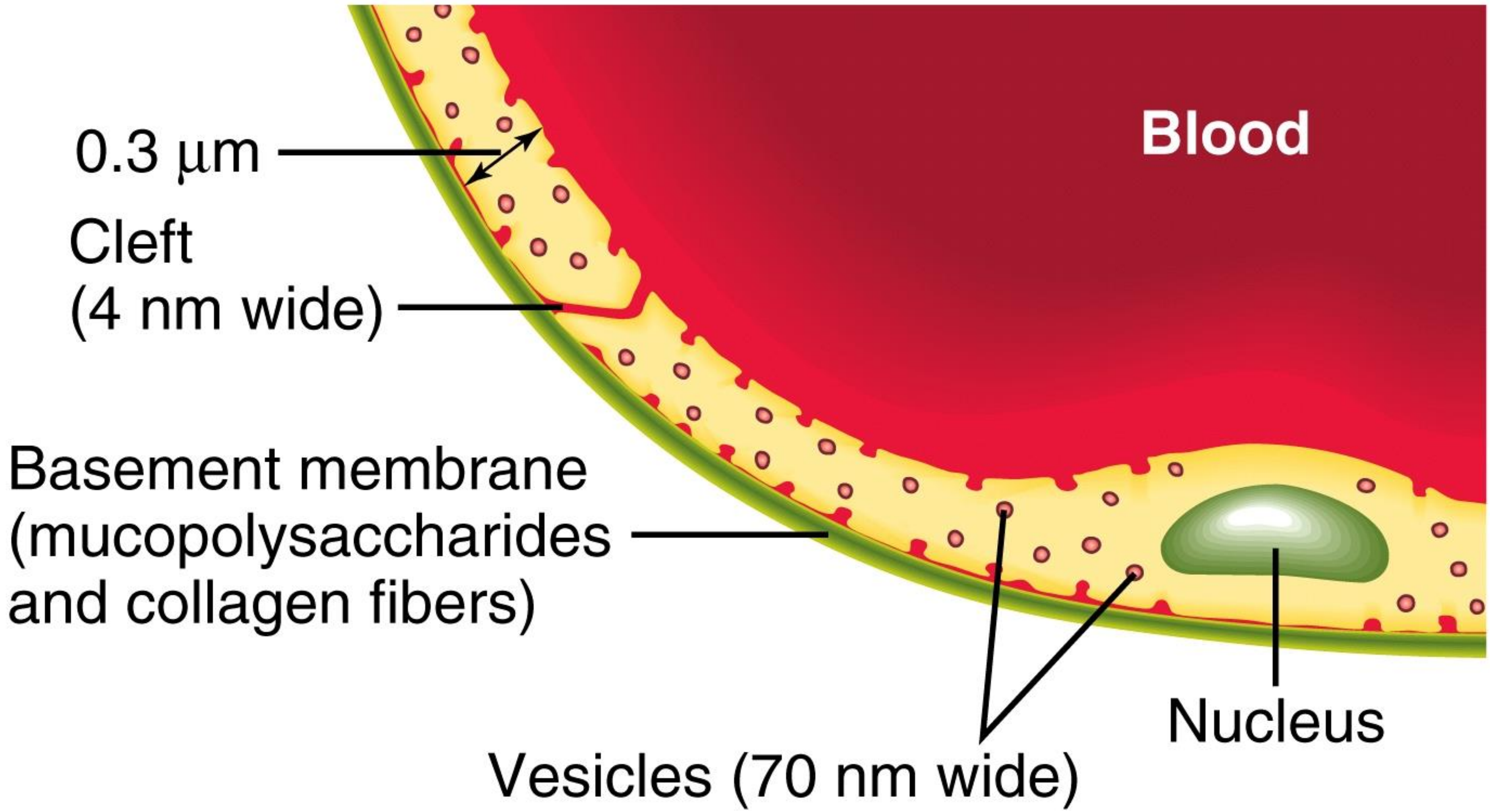
Figure 9-59 p446

Elephantiasis of the legs due to filariasis (CDC).

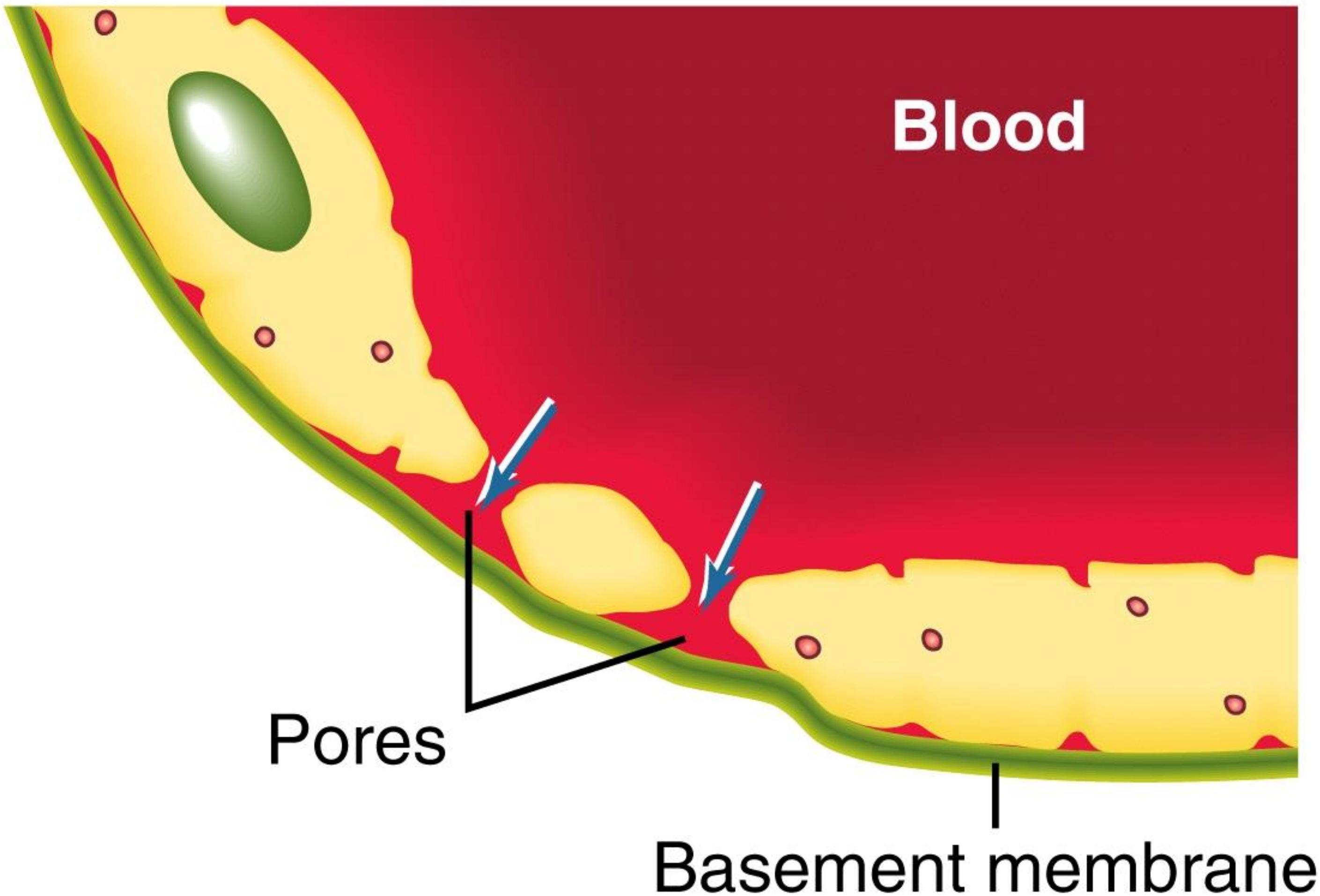


Wucheria bancrofti in blood
WHO/TDR/Stammers

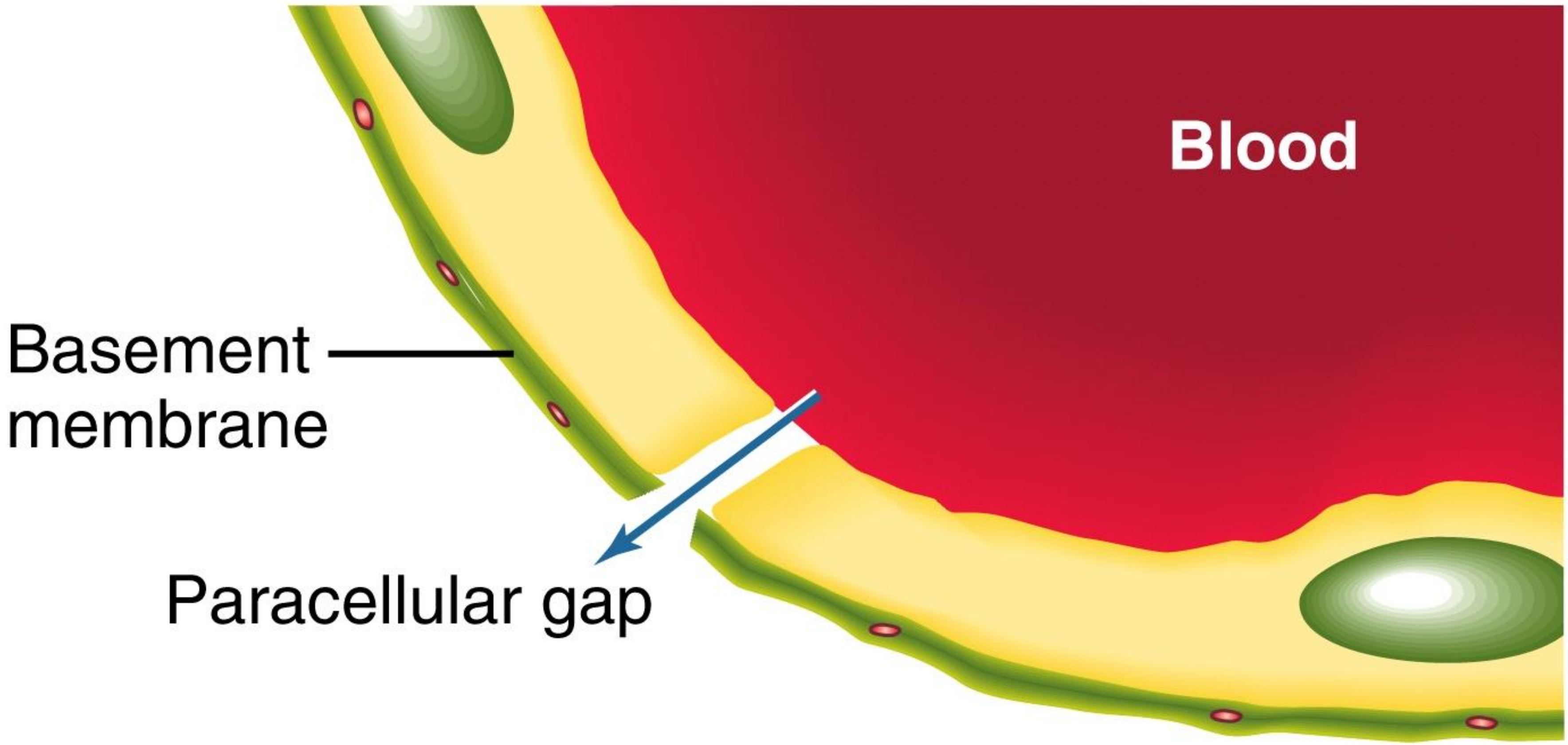
(a) Continuous capillary



(b) Fenestrated capillary



(c) Sinusoidal capillary



9.15 Circulatory Vessels: Venules and Veins

- **Veins** serve as a **blood reservoir** and **return blood** to the heart.
  - Veins are **large** in diameter with little elasticity and low myogenic tone.
    - Easily **distend** to accommodate additional volumes of blood with no recoil (**capacitance vessels**)
    - Mammalian veins contain more than 60% of the total blood volume
  - Blood velocity accelerates on return from capillaries

9.15 Circulatory Vessels: Venules and Veins

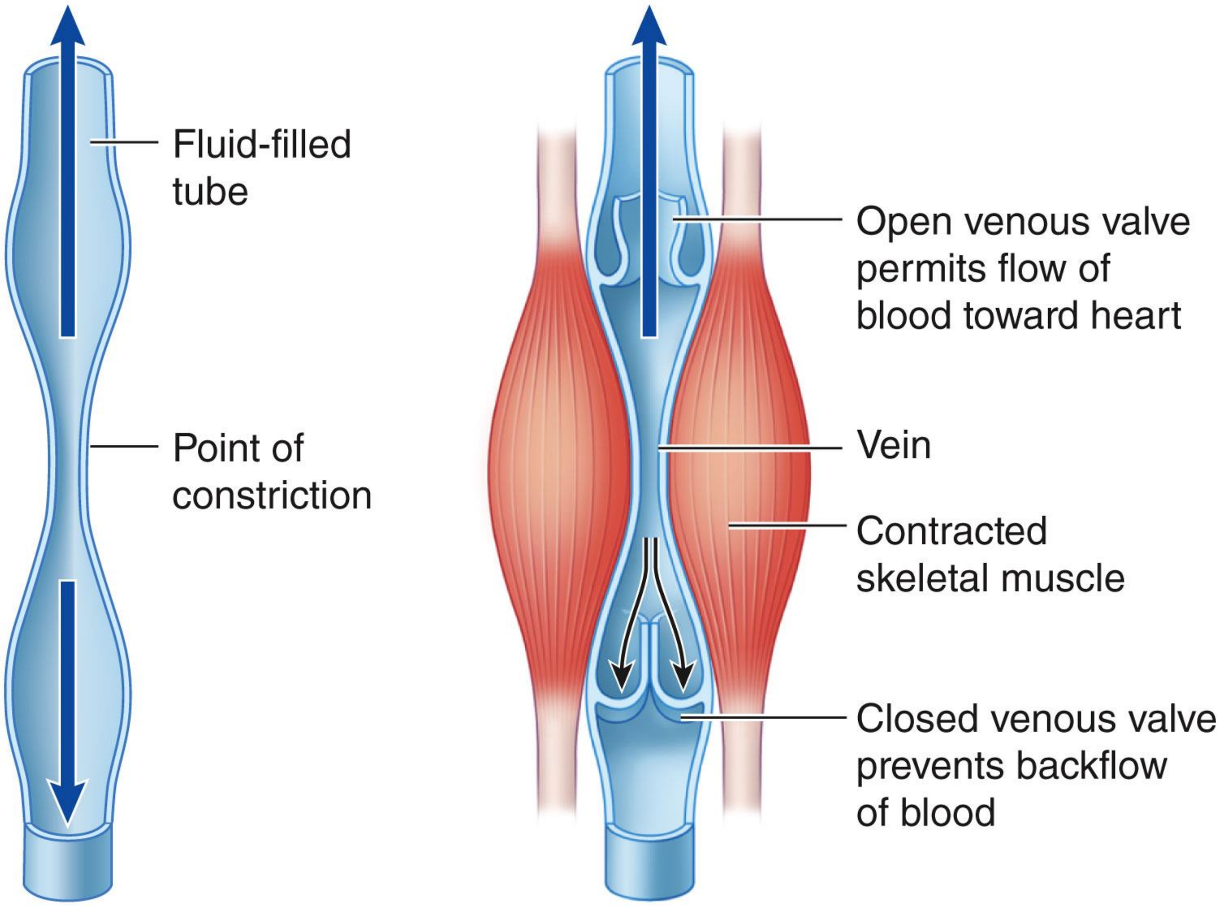
TABLE 9-2 Features of Blood Vessels (with Human Values)

	VESSEL TYPE			
Feature	**Arteries**	**Arterioles**	**Capillaries**	**Veins**
Number	Several hundred*	Half a million	Ten billion	Several hundred*
Special Features	Thick, highly elastic, walls; large radii*	Highly muscular, well-innervated walls; small radii	Very thin walled; large total cross-sectional area	Thin walled compared to arteries; highly distensible; large radii*
Functions	Passageway from heart to organs; serve as pressure reservoir	Primary resistance vessels; determine distribution of cardiac output	Site of exchange; determine distribution of extracellular fluid between plasma and interstitial fluid	Passageway to heart from organs; serve as blood reservoir

9.15 Circulatory Vessels: Venules and Veins

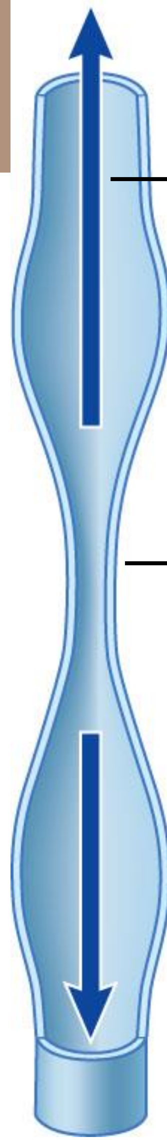
- Factors **enhancing venous return to the heart**
  - Driving pressure of **cardiac contraction**
  - **Sympathetic activity** produces venous vasoconstriction
  - Repeated **contraction of skeletal muscles** compresses veins (**skeletal muscle pump**)
  - **Venous valves** prevent blood from flowing backward (away from the heart)
  - **Respiratory activity** reduces pressure near the heart (**respiratory pump**)
  - **Cardiac suction** due to low ventricular pressures during diastole

9.15 Circulatory Vessels: Venules and Veins



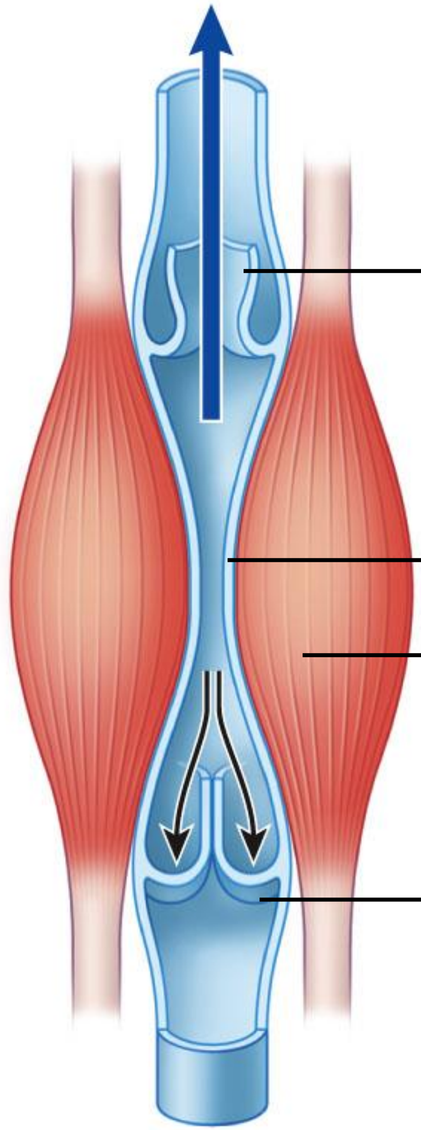
(a) Fluid moving in both directions on squeezing a fluid-filled tube

(b) Action of venous valves, permitting flow of blood toward heart and preventing backflow of blood



(a) Fluid moving in both directions on squeezing a fluid-filled tube

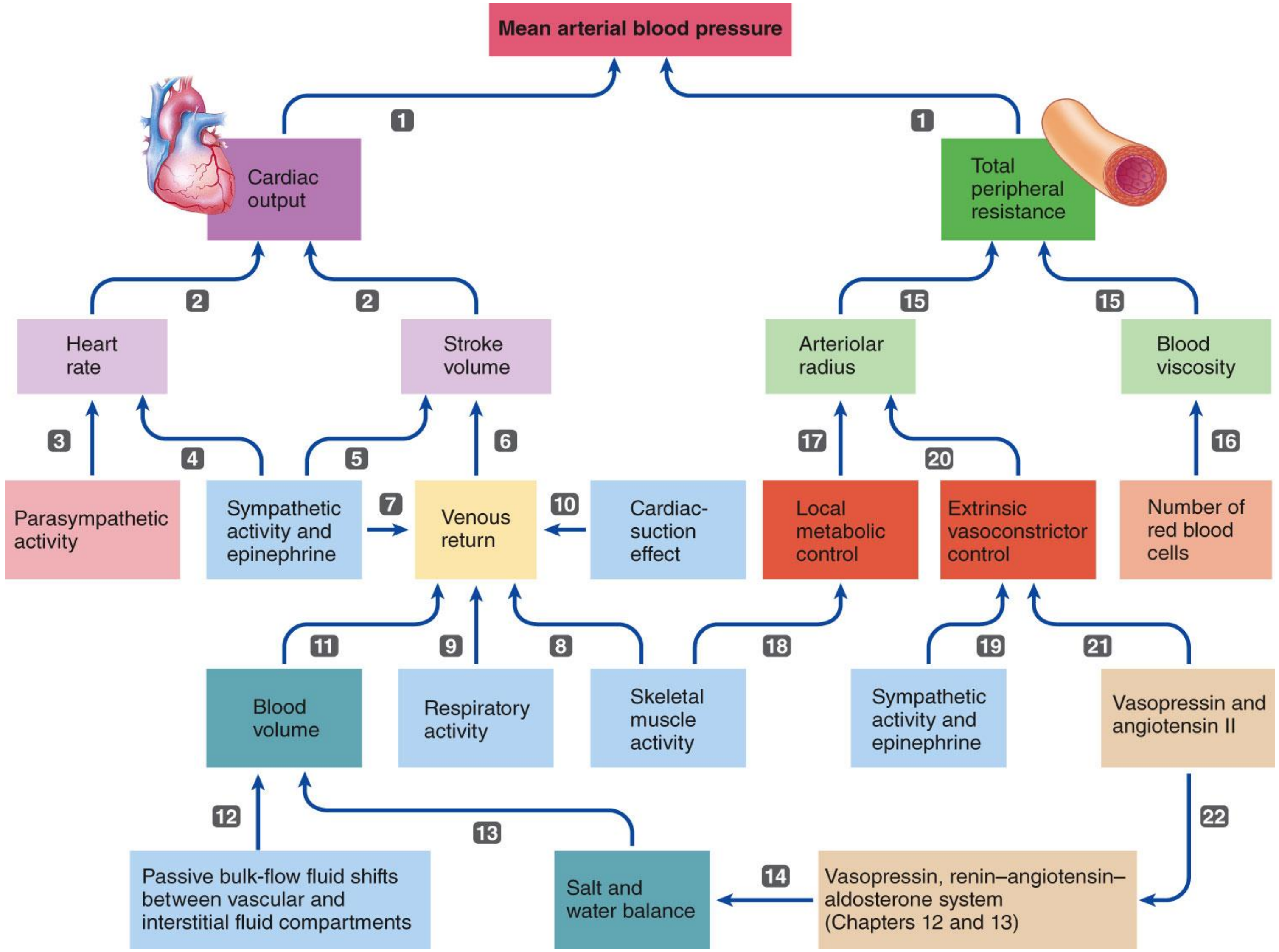
Figure 9-61a p448



(b) Action of venous valves, permitting flow of blood toward heart and preventing backflow of blood

Figure 9-61b p448

9.15 Circulatory Vessels: Venules and Veins



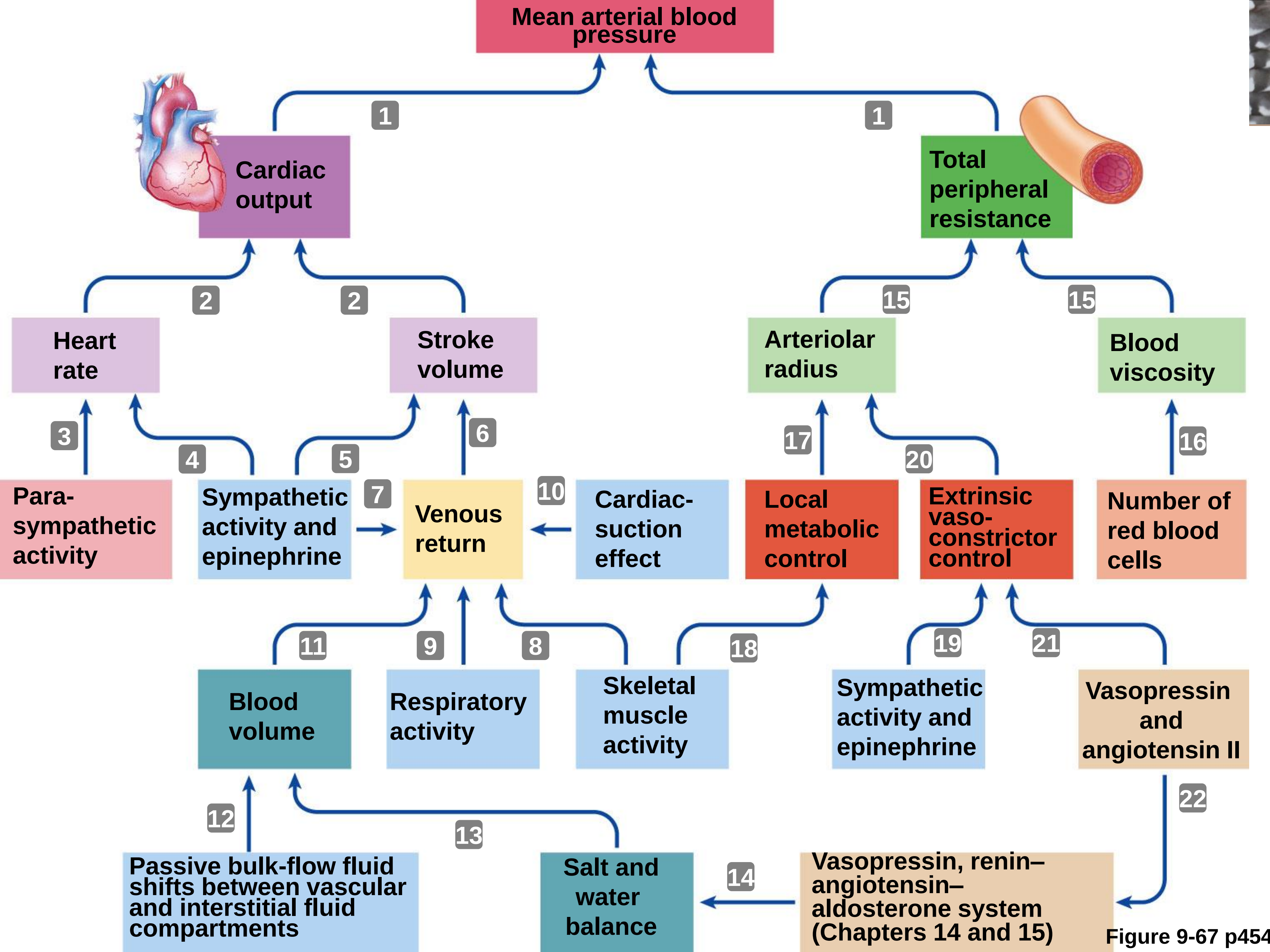


Figure 9-67 p454

9.16 Integrated Cardiovascular Function

- Cardiovascular function is regulated in an **integrated** fashion
  - Two major goals:
    - Proper gas and heat transport
    - Maintaining arterial blood pressure
  - Cardiac output is **homeostatically regulated** when an animal is at rest to maintain consistent delivery of oxygen to key organs.
  - During activity, **cardiac output** is **reset higher** to boost blood flow to skeletal muscles and skin.

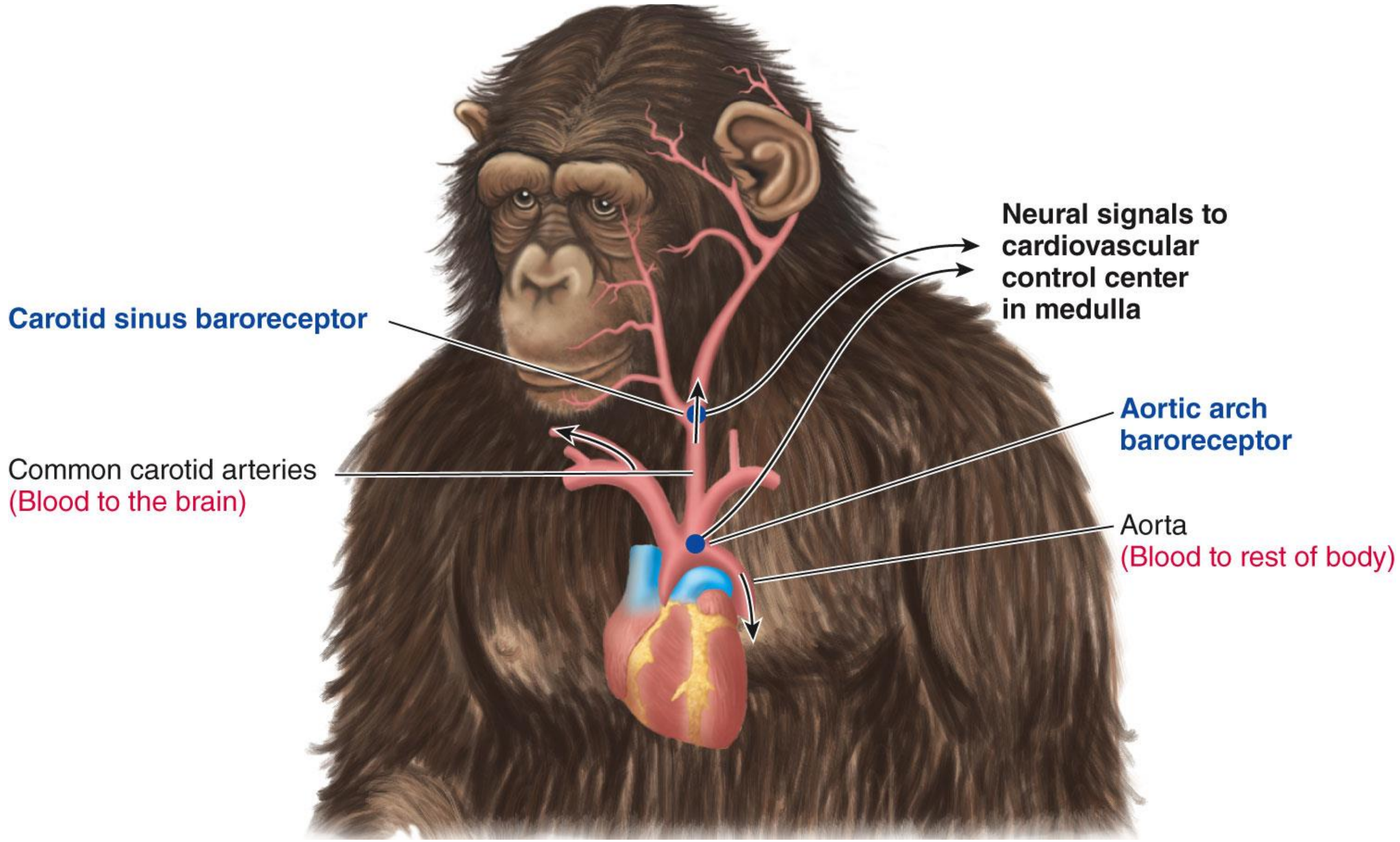
9.16 Integrated Cardiovascular Function

- Regulation of arterial blood pressure
  - Pressure must be high enough to overcome **gravity**, **friction** and other resistance factors
  - Pressure must be high enough for **ultrafiltration** in the kidneys
  - Pressure must not be so high that it creates **extra work** for the heart and increases the **risk of vascular damage.**
    - Chronic high blood pressure (**hypertension**) is due to **excess resistance**

9.16 Integrated Cardiovascular Function

- Baroreflex regulation of arterial blood pressure
  - **Arterial baroreceptors** monitor blood pressure
  - The medullary **cardiovascular control center** alters **sympathetic** and **parasympathetic** activity
  - The **heart** and **blood vessels** respond to changing autonomic output
  - Example: **Cardiovascular control center** restores falling arterial blood pressure after **standing** by increasing **sympathetic** output, thus **increasing heart rate** and **stroke volume** and **constricting blood vessels**

9.16 Integrated Cardiovascular Function



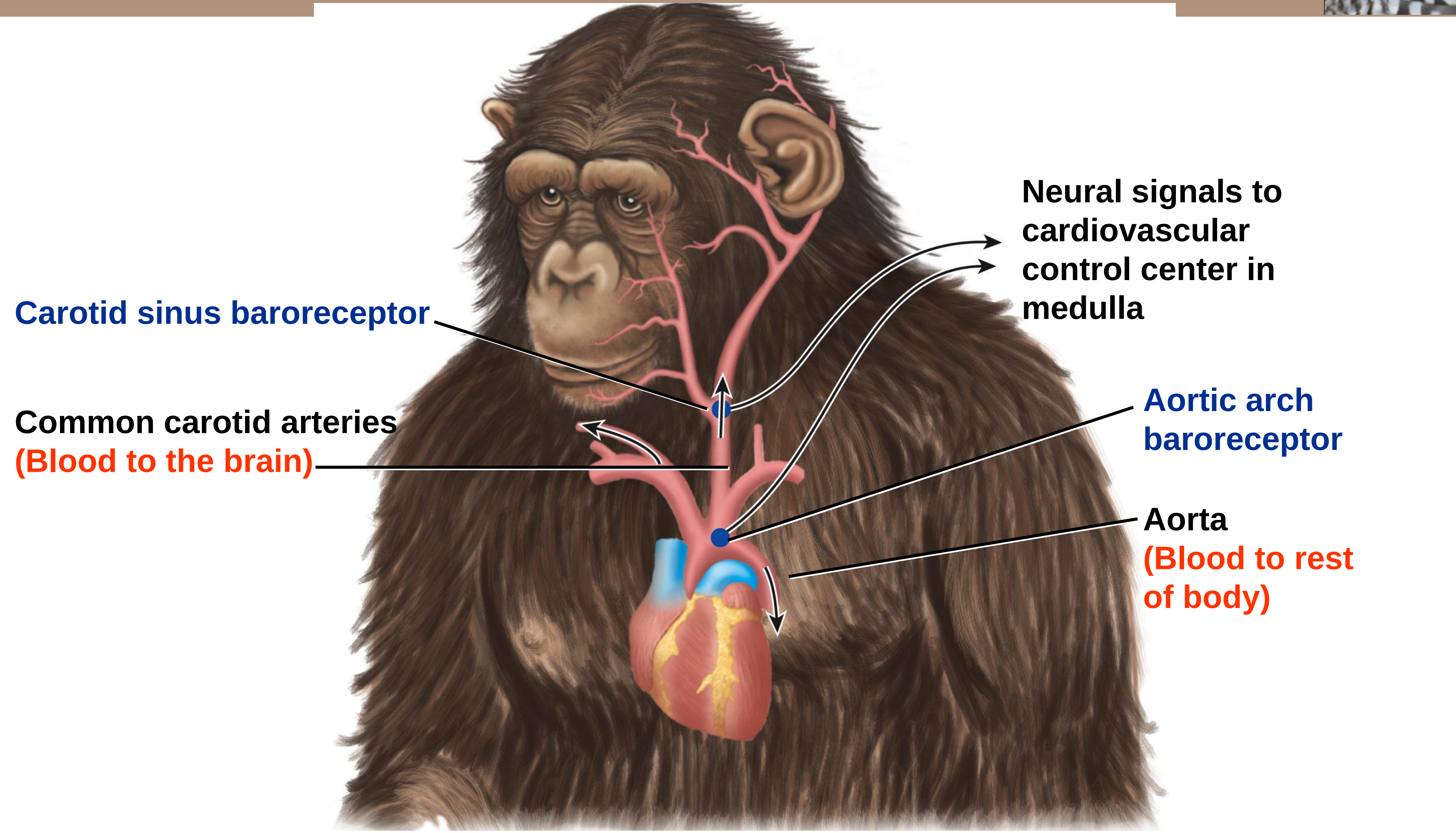
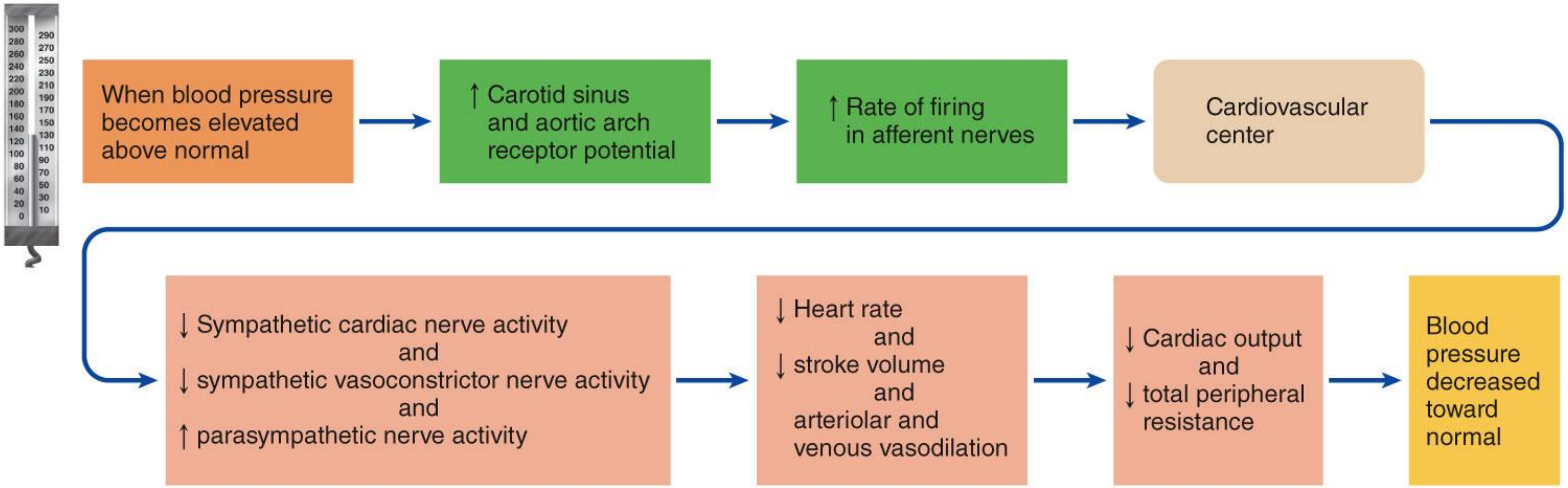
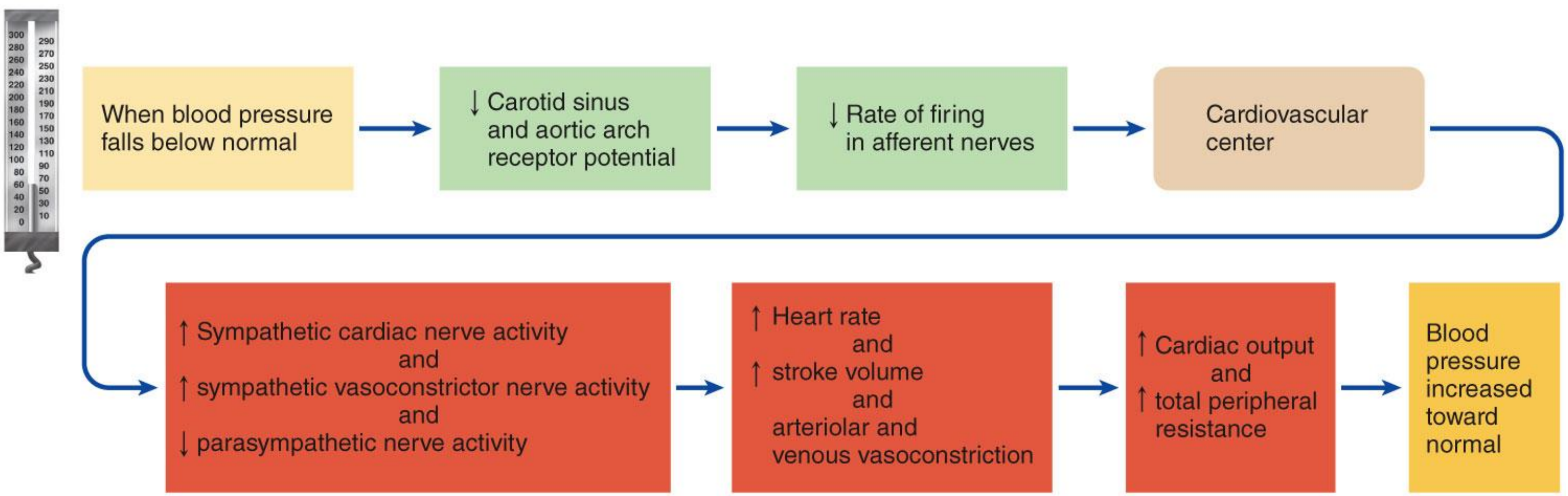


Figure 9-62 p450

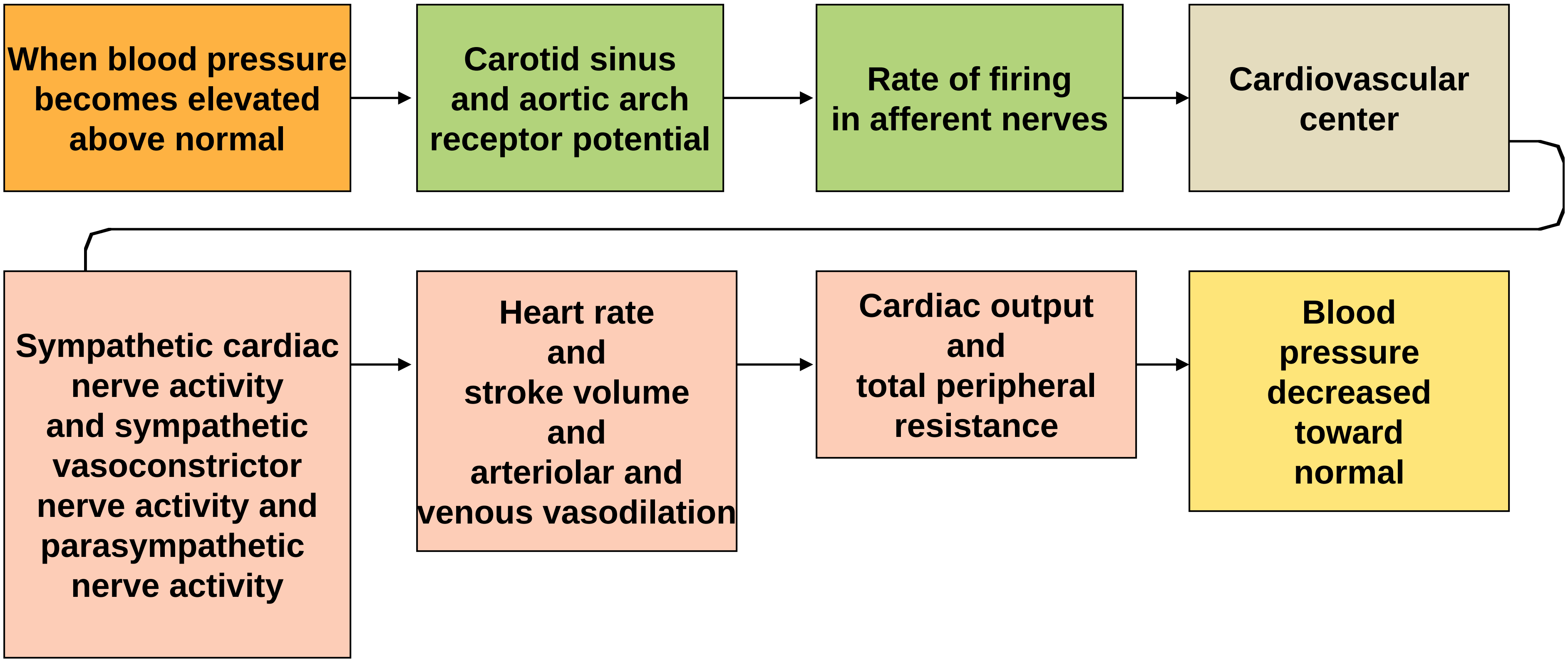
9.16 Integrated Cardiovascular Function



(a) Baroreceptor reflex in response to an elevation in blood pressure

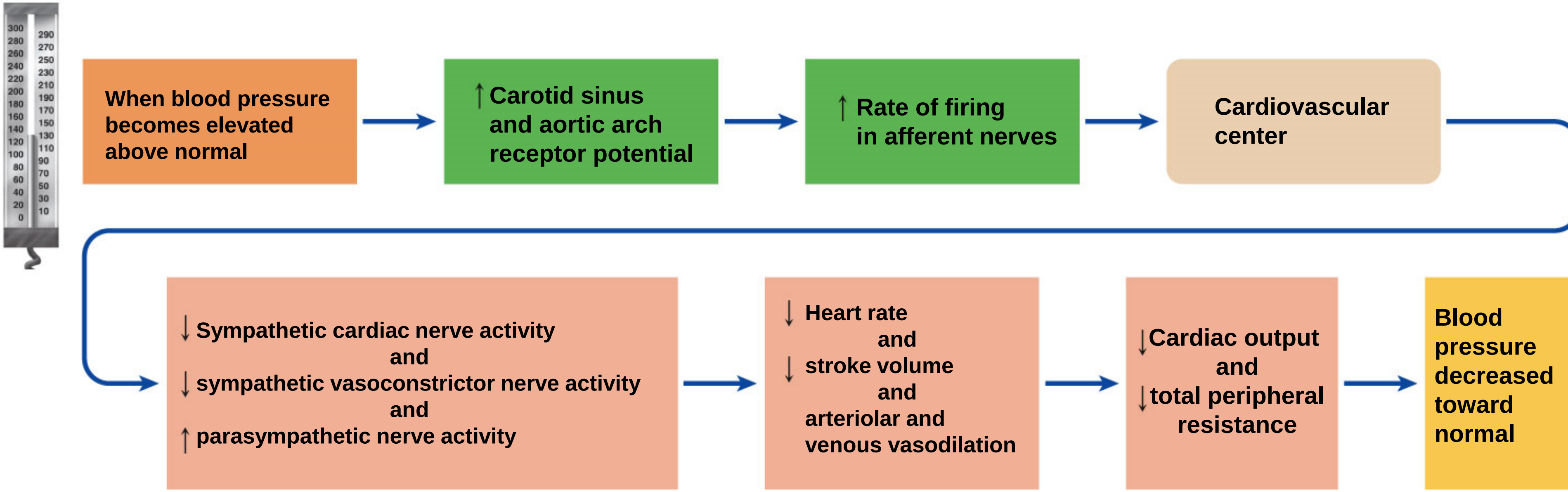


(b) Baroreceptor reflex in response to a fall in blood pressure

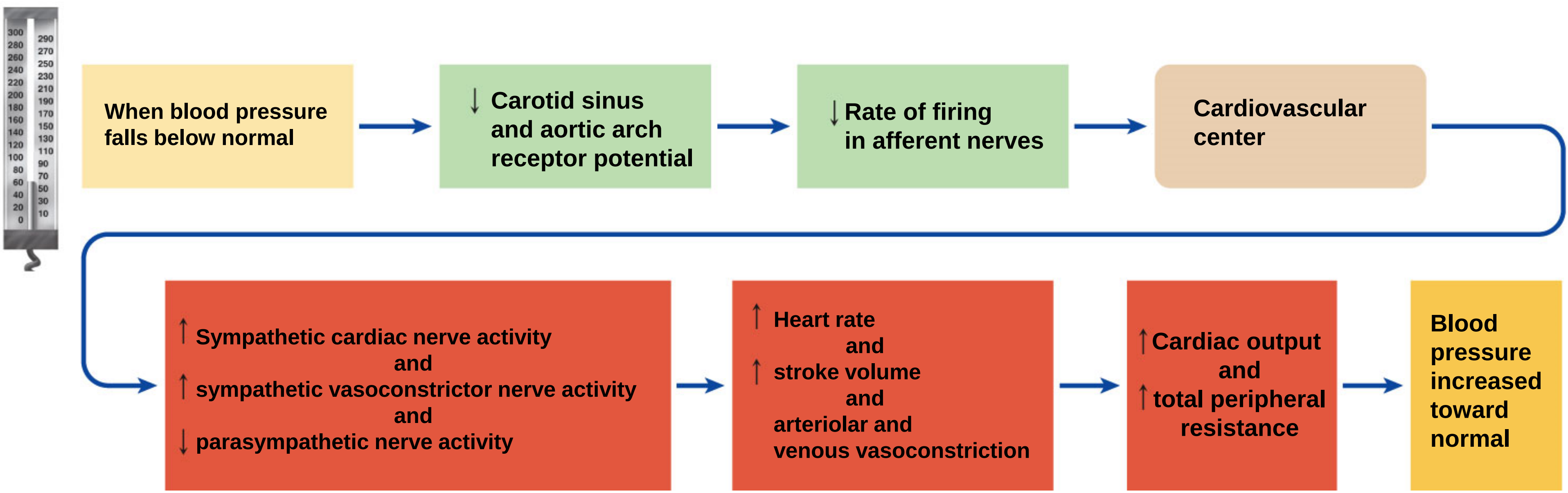


Stepped Art

Fig. 9-65a, p.451

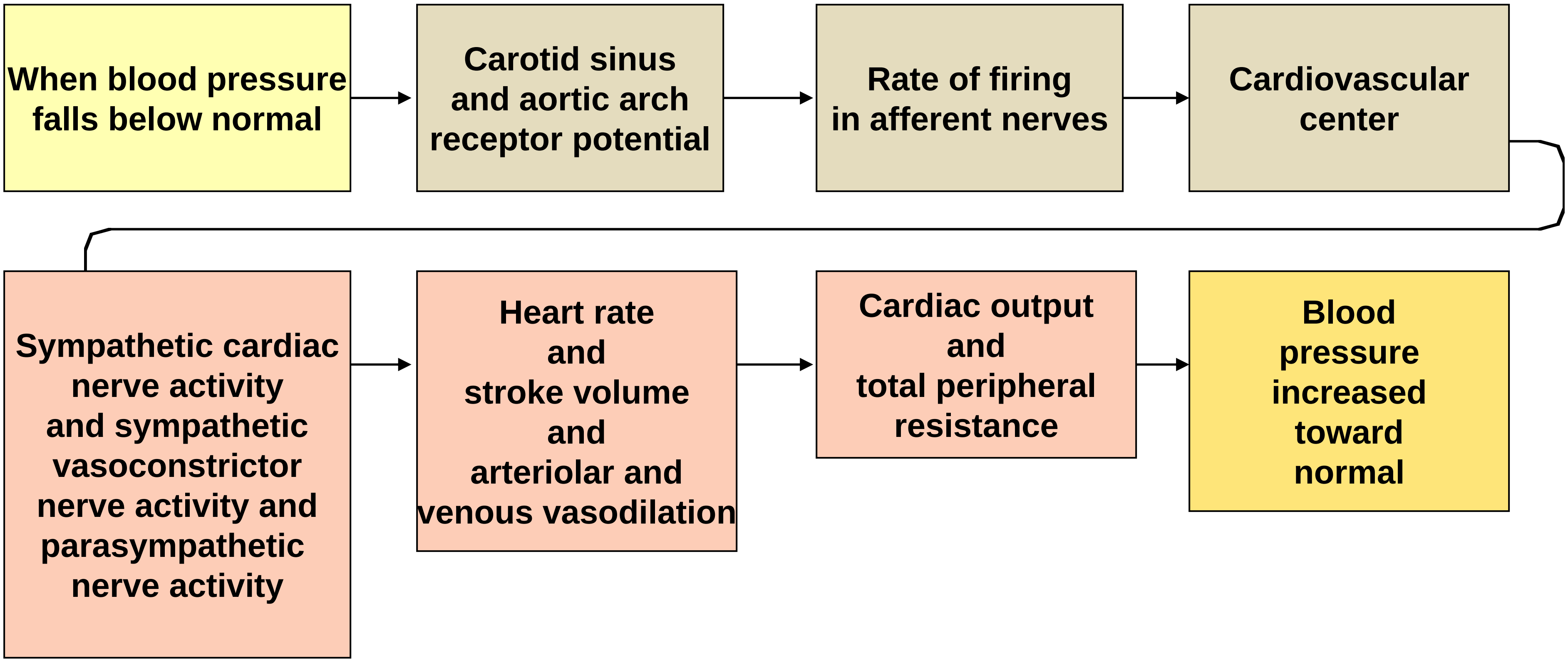


(a) Baroreceptor reflex in response to an elevation in blood pressure



(b) Baroreceptor reflex in response to a fall in blood pressure

Figure 9-65 p451



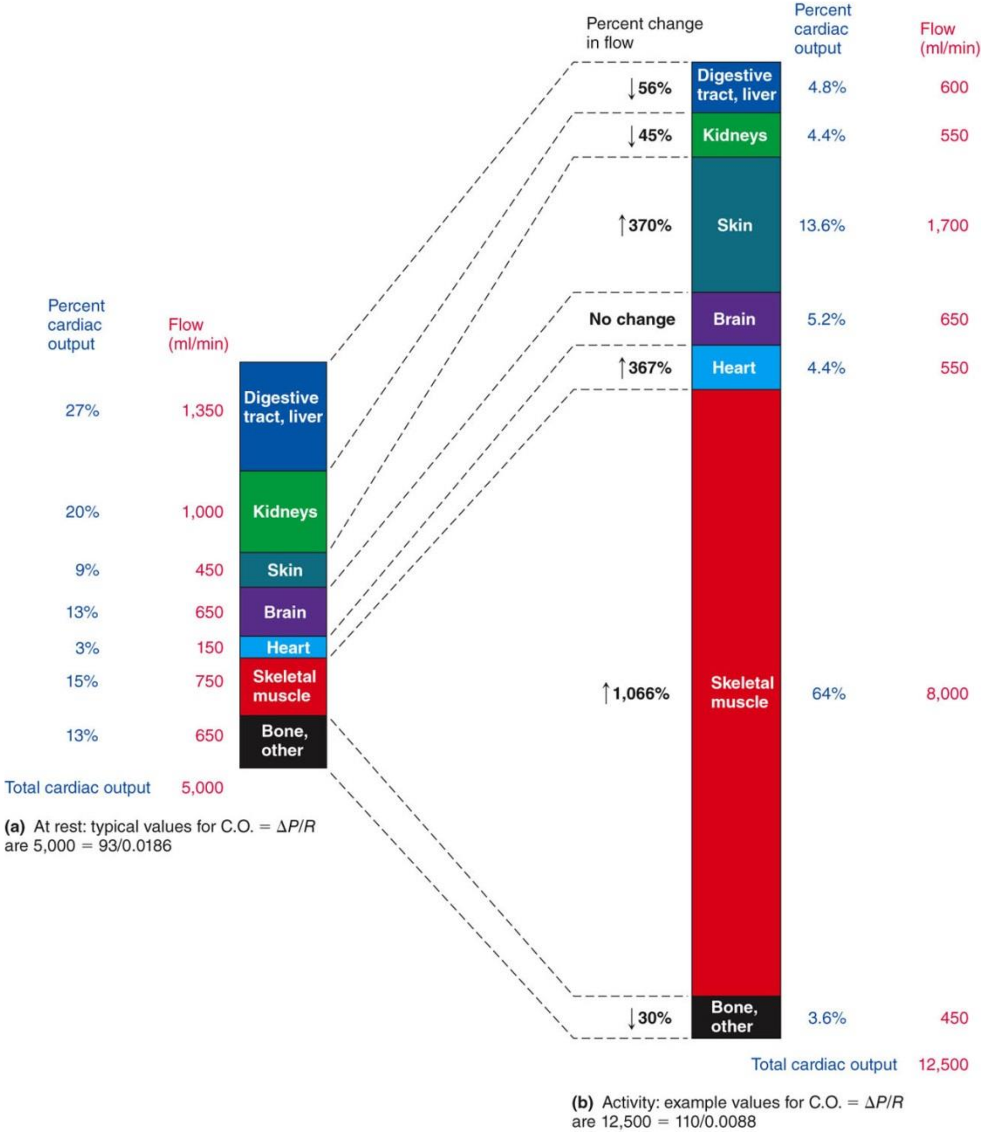
Stepped Art

Fig. 9-65b, p.451

9.16 Integrated Cardiovascular Function

- Response to increased locomotor activity
  - **Increased heart rate** and **stroke volume** result in **increased cardiac output**
  - **Blood flow** to skeletal muscle, heart and skin **increases**
  - **Anticipatory responses**
    - Increased locomotor activity induces cardiac and vascular changes before that activity leads to disturbances in blood gases.
    - Perception of stress can trigger the fight-or-flight response

9.16 Integrated Cardiovascular Function



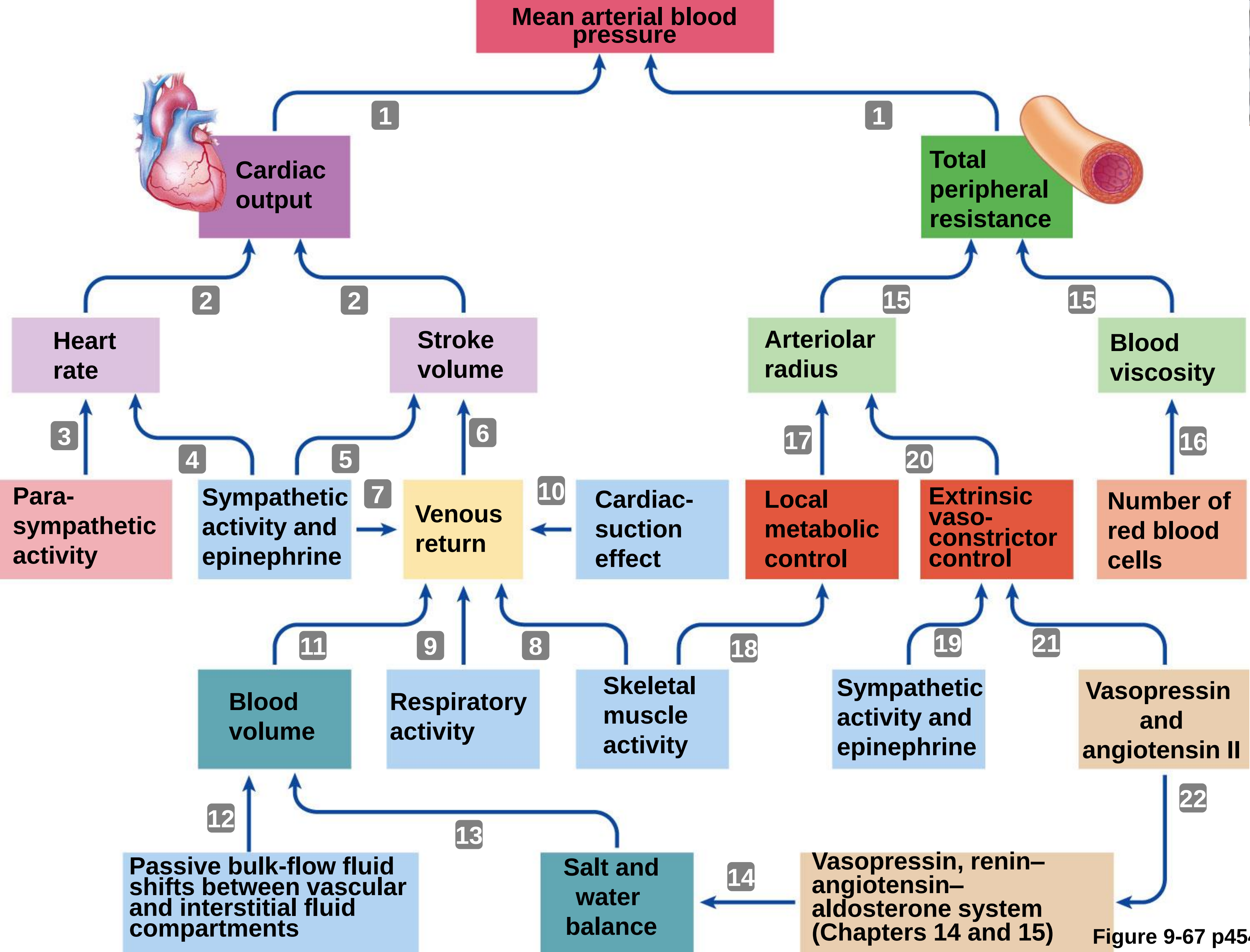


Figure 9-67 p454