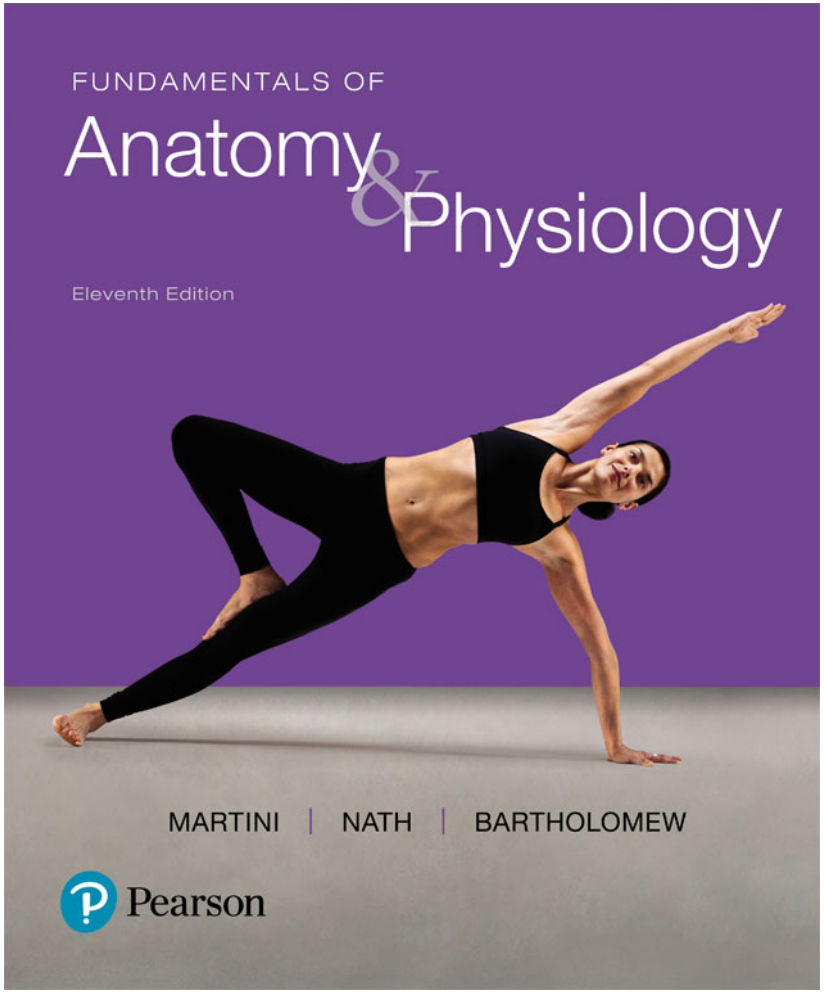
Fundamentals of Anatomy & Physiology

Eleventh Edition



Chapter 20
The Heart

Lecture Presentation by
Lori Garrett,
Parkland College

Learning outcomes

20-1 Describe the anatomy of the heart, including vascular supply and pericardium structure, and trace the flow of blood through the heart, identifying the major blood vessels, chambers, and heart valves.

20-2 Explain the events of an action potential in cardiac muscle, indicate the importance of calcium ions to the contractile process, describe the conducting system of the heart, and identify the electrical events associated with a normal electrocardiogram.

Learning outcomes

20-3 Explain the events of the cardiac cycle, including atrial and ventricular systole and diastole, and relate the heart sounds to specific events in the cycle.

20-4 Define cardiac output, describe the factors that influence heart rate and stroke volume, and explain how adjustments in stroke volume and cardiac output are coordinated at different levels of physical activity.

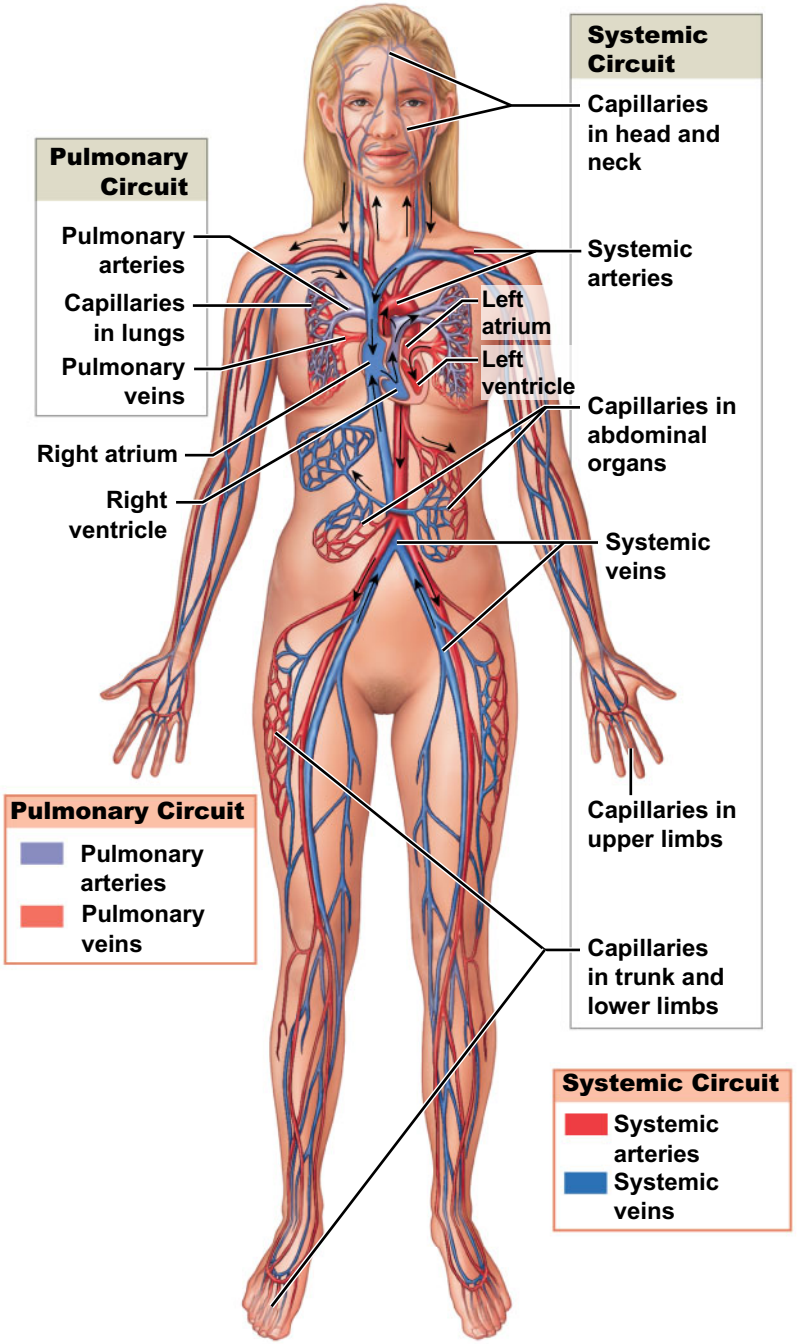
An Introduction to the Heart

- **Cardiovascular system**
  - Heart
  - Blood
  - Blood vessels
- The heart
  - Beats approximately 100,000 times each day
  - Pumping about 8000 liters of blood per day

20-1 Anatomy of the Heart

- **Pulmonary circuit**
  - Carries blood to and from gas exchange surfaces of lungs
- **Systemic circuit**
  - Carries blood to and from the rest of the body
- Each circuit begins and ends at the heart
  - Blood travels through these circuits in sequence

Figure 20–1 An Overview of the Cardiovascular System.



20-1 Anatomy of the Heart

- Types of blood vessels
  - Arteries
    - Carry blood away from heart
  - Veins
    - Return blood to heart
  - Capillaries (exchange vessels)
    - Interconnect smallest arteries and smallest veins
    - Exchange dissolved gases, nutrients, and wastes between blood and surrounding tissues

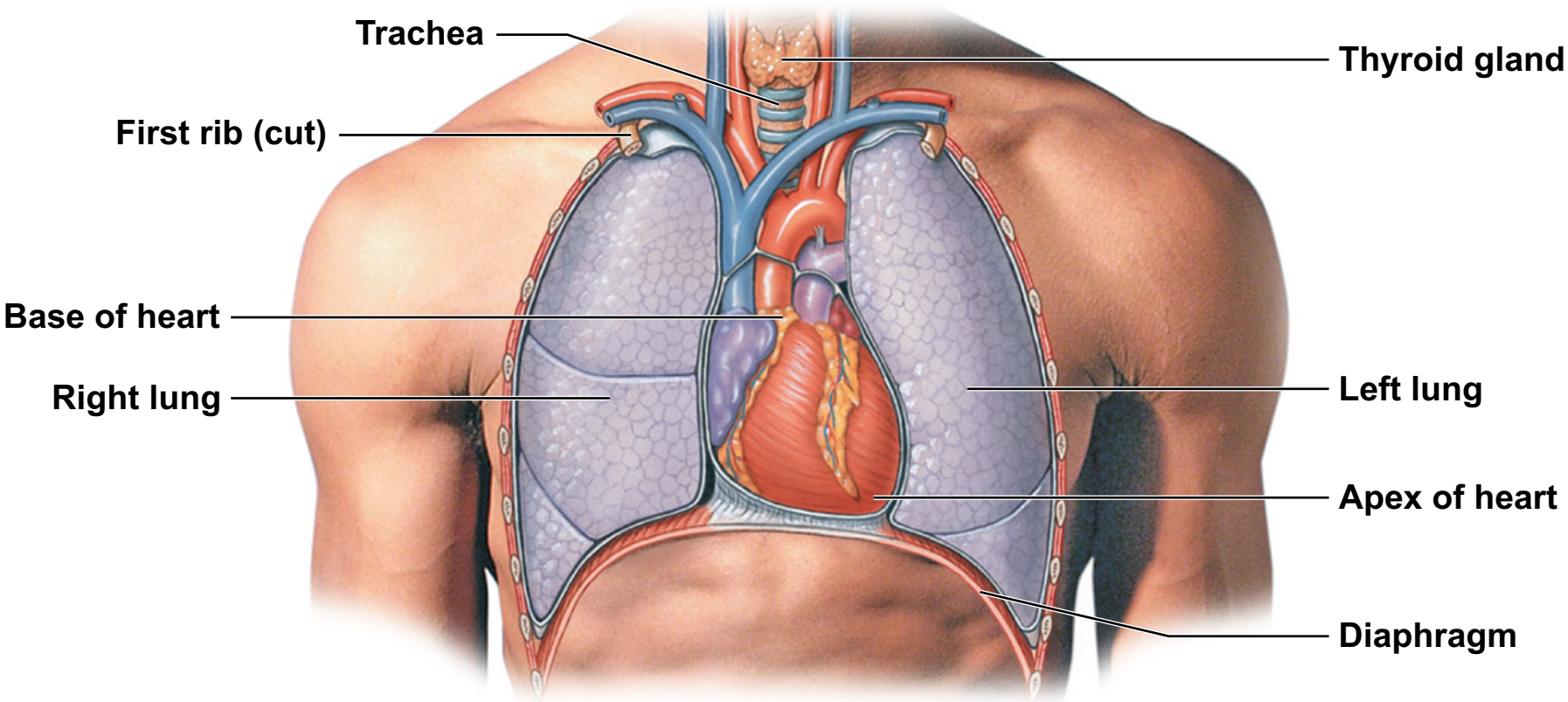
20-1 Anatomy of the Heart

- Four chambers of the **heart**
  - Right atrium
    - Receives blood from systemic circuit
  - Right ventricle
    - Pumps blood into pulmonary circuit
  - Left atrium
    - Receives blood from pulmonary circuit
  - Left ventricle
    - Pumps blood into systemic circuit

20-1 Anatomy of the Heart

- Heart
  - Great vessels connect at **base** (superior)
  - Pointed tip is **apex** (inferior)
  - Sits between two pleural cavities in **mediastinum**

Figure 20–2a The Location of the Heart in the Thoracic Cavity.

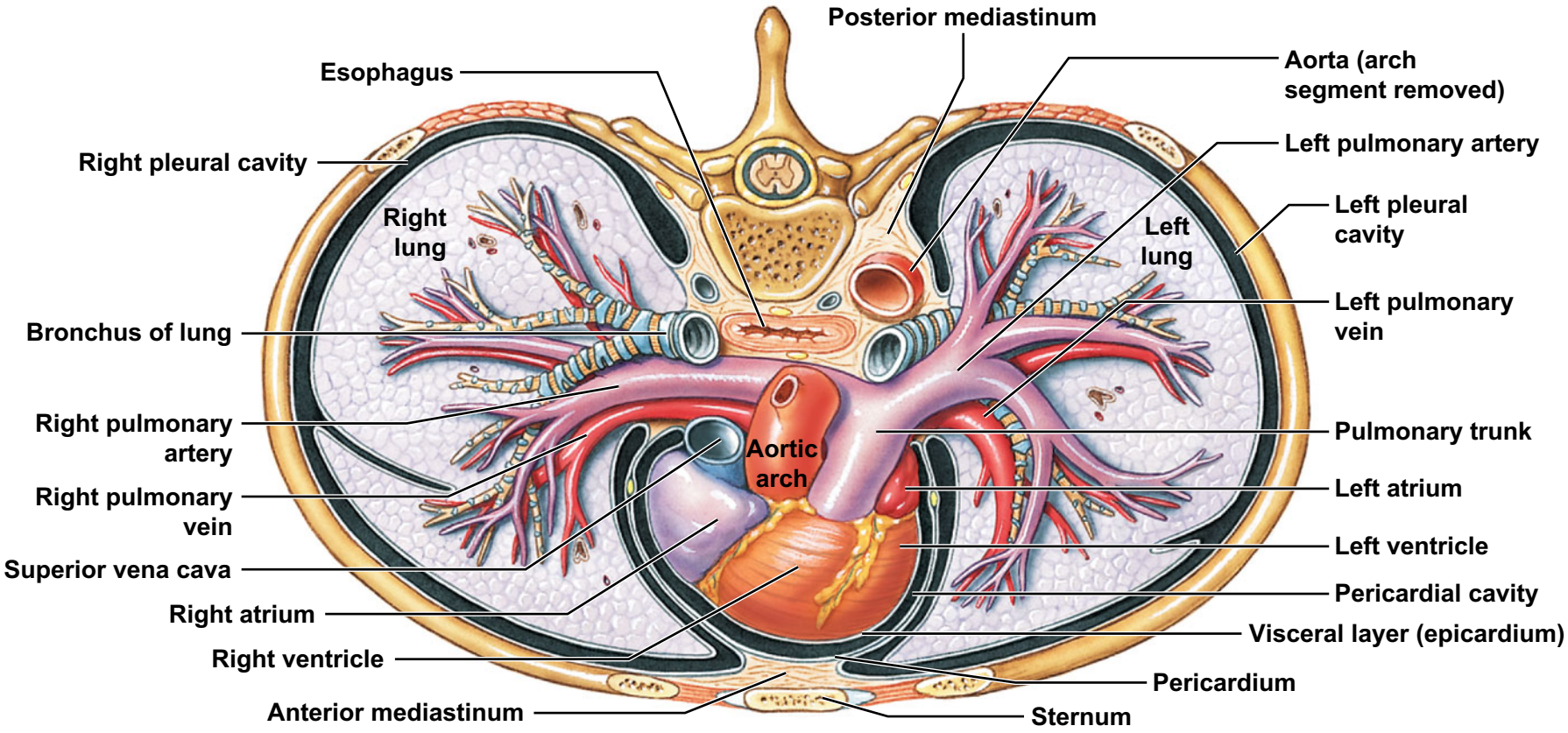


a An anterior view of the chest, showing the position of the heart and major blood vessels relative to the ribs, lungs, and diaphragm.

20-1 Anatomy of the Heart

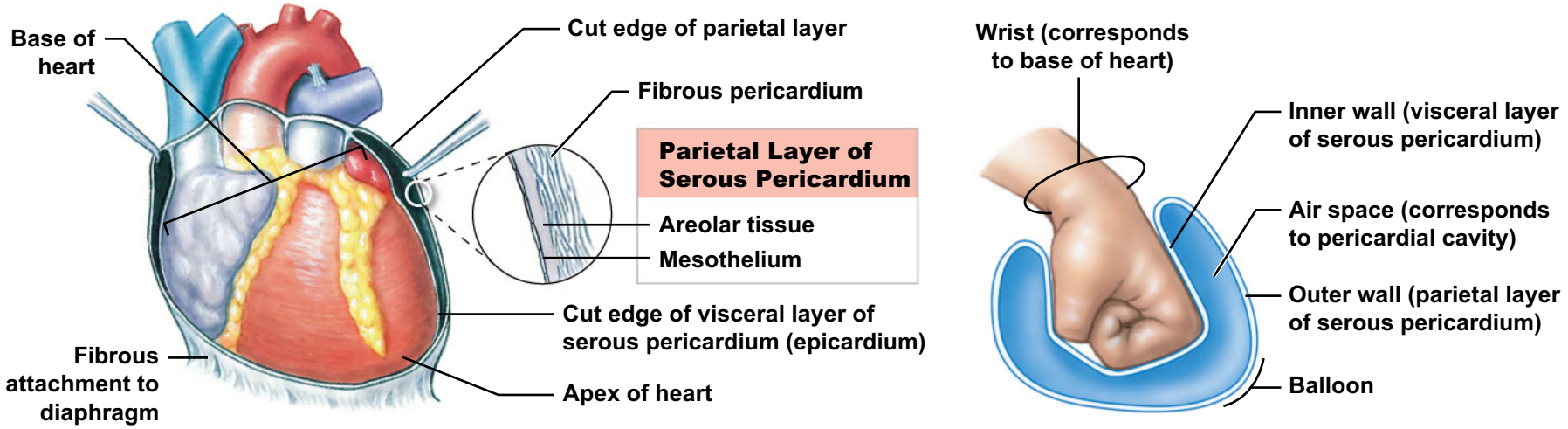
- **Pericardium**
  - Surrounds heart
  - Outer fibrous pericardium
  - Inner **serous pericardium**
    - Outer **parietal layer**
    - Inner **visceral layer** (epicardium)
  - **Pericardial cavity**
    - Between parietal and visceral layers
    - Contains **pericardial fluid**

Figure 20–2b The Location of the Heart in the Thoracic Cavity.



b A superior view of the organs in the mediastinum; portions of the lungs have been removed to reveal blood vessels and airways. The heart is located in the anterior part of the mediastinum, immediately posterior to the sternum.

Figure 20–2c The Location of the Heart in the Thoracic Cavity.



C The relationship between the heart and the pericardial cavity; compare with the fist-and-balloon example.

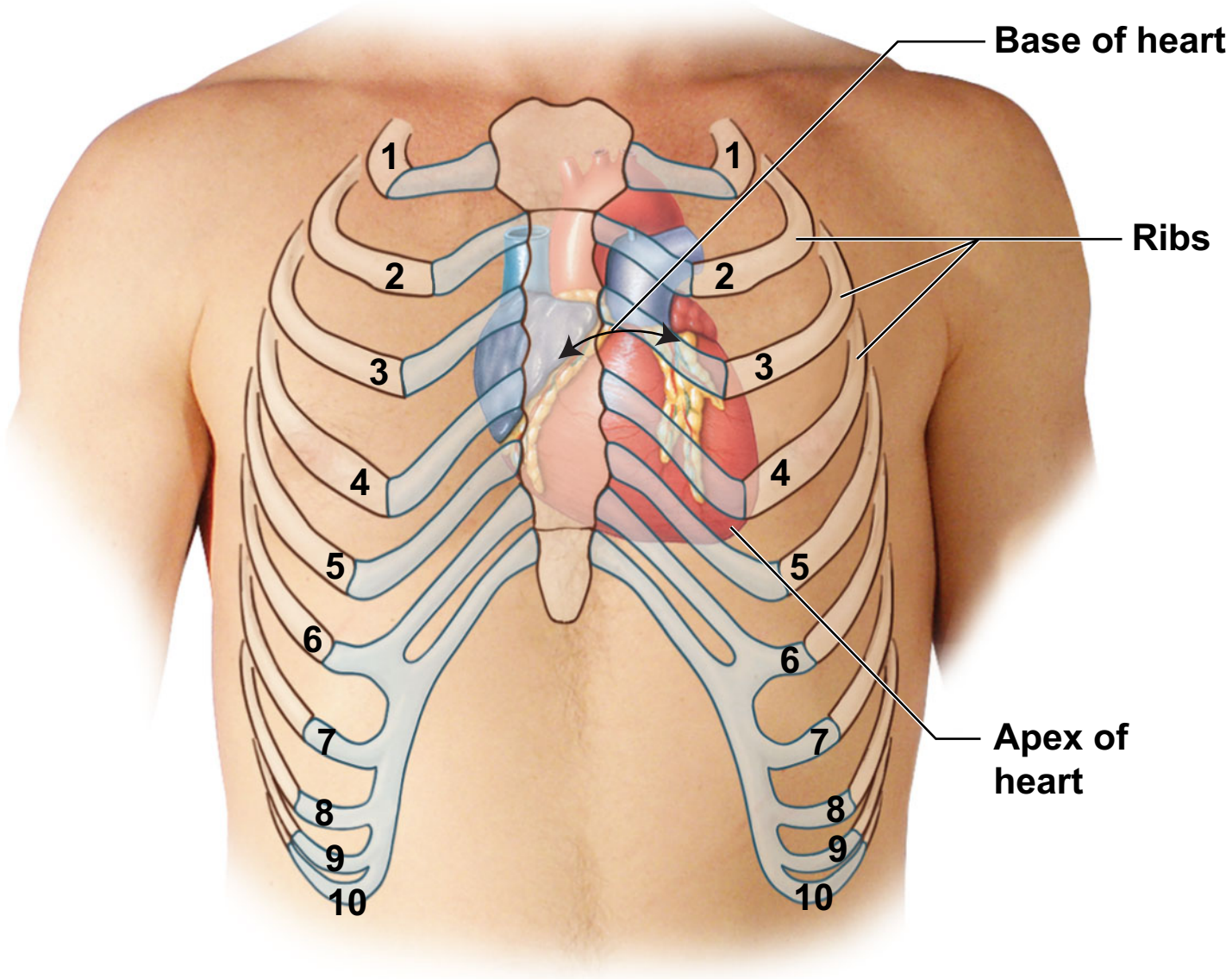
20-1 Anatomy of the Heart

- **Pericarditis**
  - Caused by pathogens in pericardium
  - Inflamed pericardial surfaces rub against each other
    - Producing distinctive scratching sound
  - May cause **cardiac tamponade**
    - Restricted movement of the heart
    - Due to excess fluid in pericardial cavity

20-1 Anatomy of the Heart

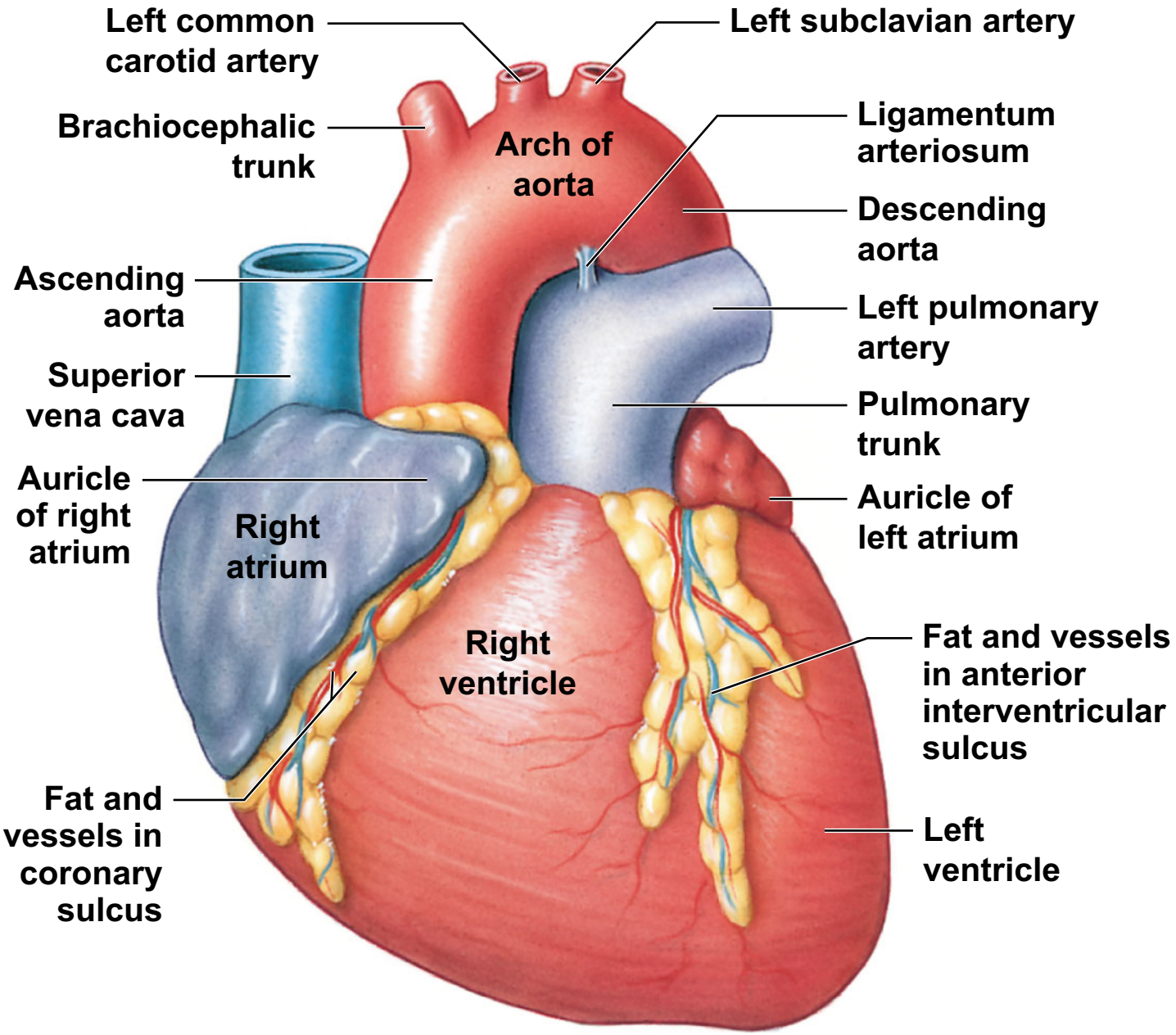
- Superficial anatomy of the heart
  - Two thin-walled atria
    - Each with an expandable outer **auricle**
  - Sulci (grooves)
    - Contain fat and blood vessels
    - **Coronary sulcus**
      - Marks border between atria and ventricles
    - **Anterior interventricular sulcus** and **posterior interventricular sulcus**
      - Mark boundary between left and right ventricles

Figure 20–3a The Position and Superficial Anatomy of the Heart.



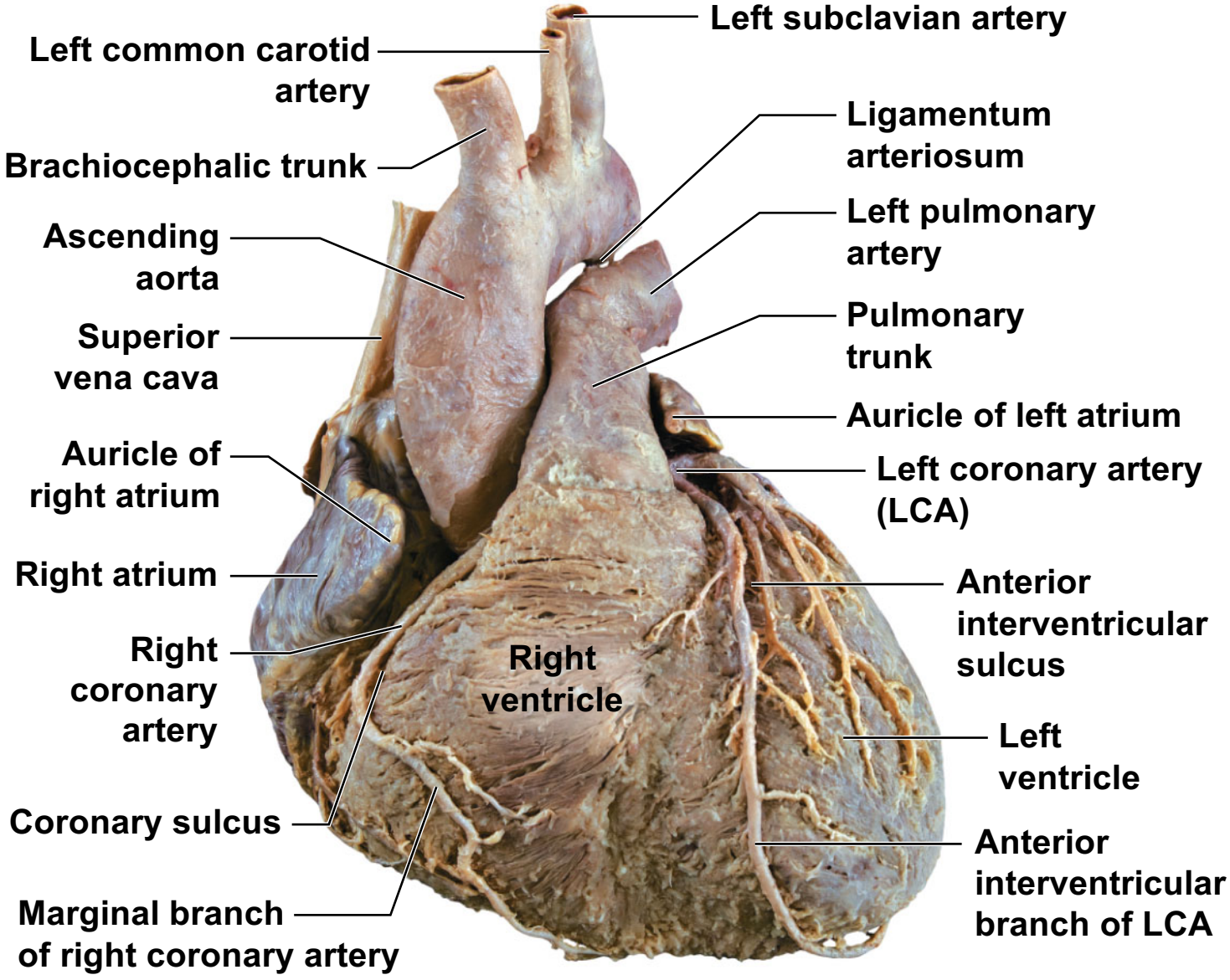
a Heart position relative to the rib cage.

Figure 20–3b The Position and Superficial Anatomy of the Heart.



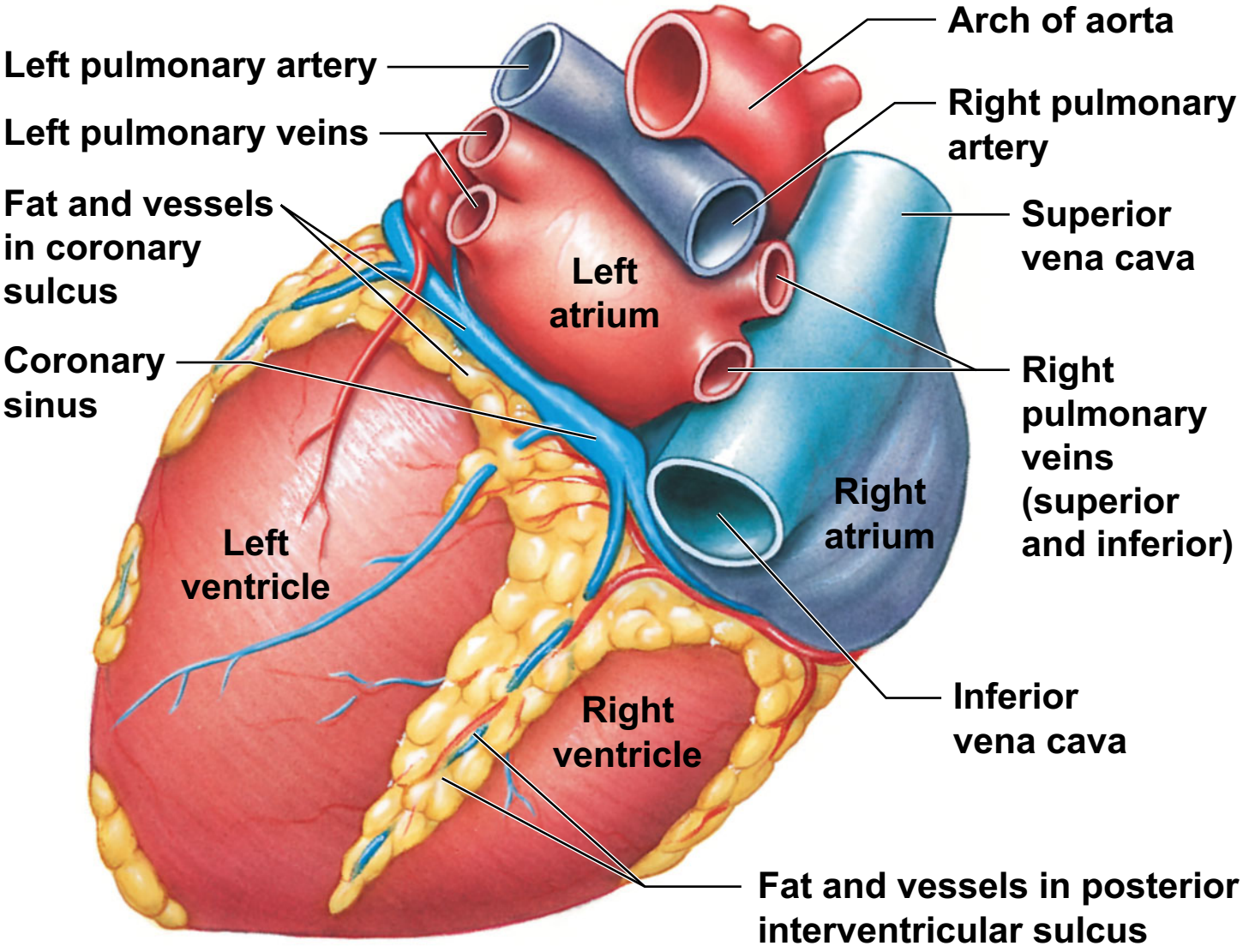
b Major anatomical features on the anterior surface.

Figure 20–3c The Position and Superficial Anatomy of the Heart.



C Anterior surface of the heart, cadaver dissection.

Figure 20–3d The Position and Superficial Anatomy of the Heart.

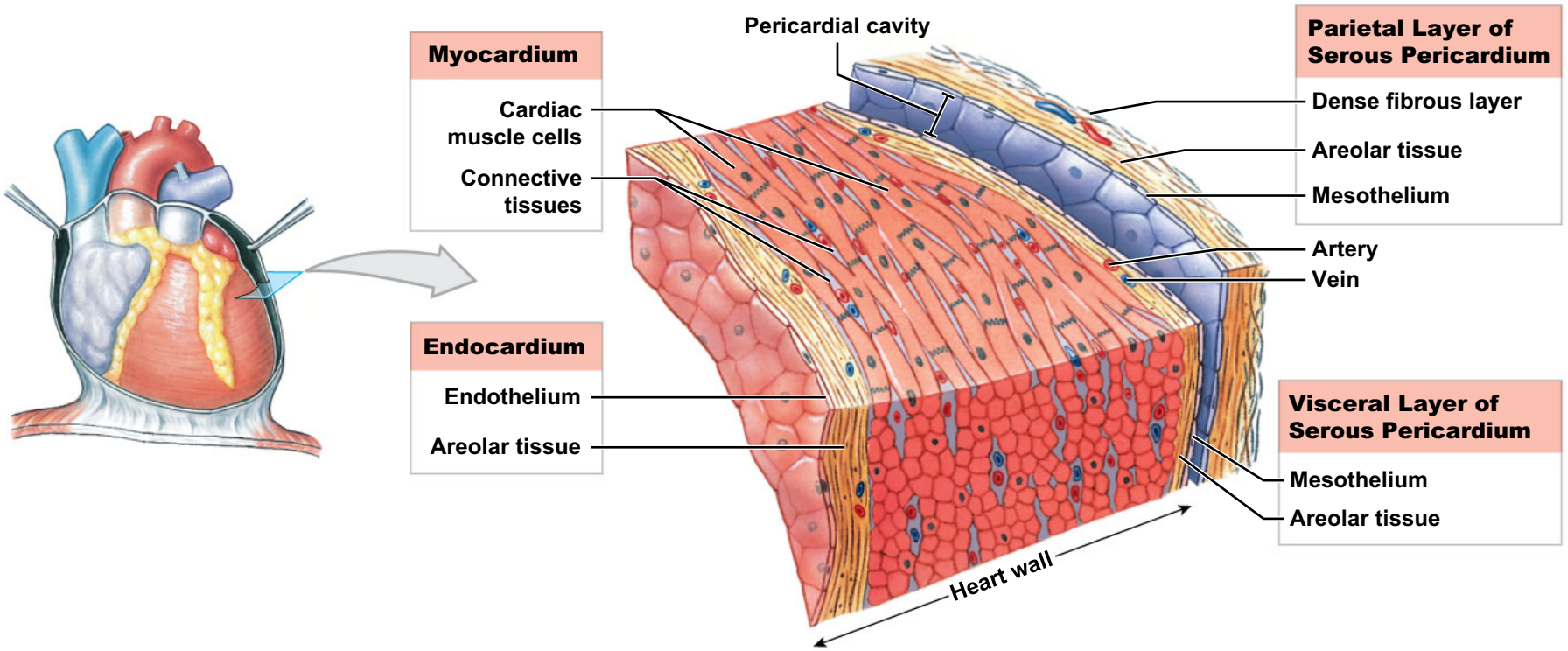


d Major anatomical features on the posterior surface. Coronary arteries (which supply the heart itself) are shown in red; coronary veins are shown in blue.

20-1 Anatomy of the Heart

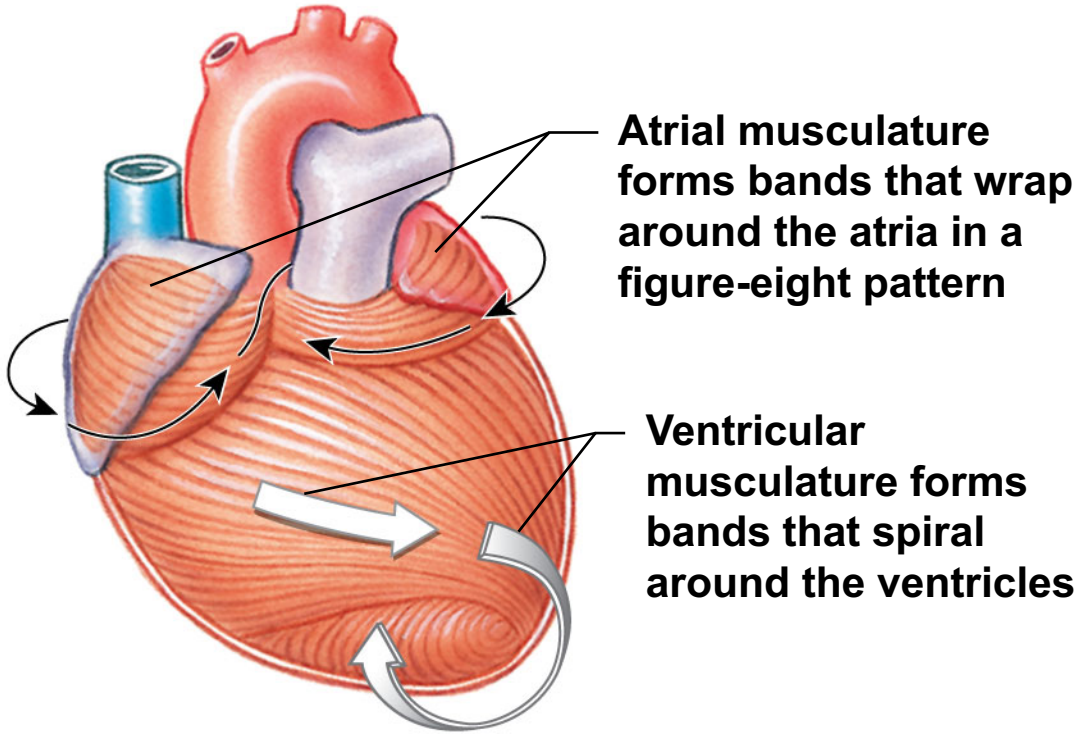
- Heart wall consists of three distinct layers
  - **Visceral layer of serous pericardium** (**epicardium**)
    - Covers surface of heart
    - Covered by **parietal layer of serous pericardium**
  - **Myocardium**
    - Cardiac muscle tissue
  - **Endocardium**
    - Covers inner surfaces of heart
    - Simple squamous epithelium and areolar tissue

Figure 20–4a The Heart Wall.



a A diagrammatic section through the heart wall, showing the relative positions of the myocardium, pericardium, and endocardium. The proportions are not to scale; the thickness of the myocardial wall has been greatly reduced.

Figure 20–4b The Heart Wall.



b Cardiac muscle tissue forms concentric layers that wrap around the atria or spiral within the walls of the ventricles.

20-1 Anatomy of the Heart

- Connective tissues of the heart
  - Physically support cardiac muscle fibers, blood vessels, and nerves of myocardium
  - Distribute forces of contraction
  - Add strength and prevent overexpansion of heart
  - Provide elasticity that helps return heart to original size and shape after contraction

20-1 Anatomy of the Heart

- **Cardiac skeleton**
  - Four dense bands of tough elastic tissue
    - Encircle heart valves and bases of pulmonary trunk and aorta
    - Stabilize positions of heart valves and ventricular muscle cells
    - Electrically insulate ventricular cells from atrial cells

20-1 Anatomy of the Heart

- Internal anatomy and organization
  - Chambers of heart are separated by muscular partitions (septa)
  - **Interatrial septum**
    - Separates atria
  - **Interventricular septum**
    - Separates ventricles
    - Much thicker than interatrial septum

20-1 Anatomy of the Heart

- **Atrioventricular (AV) valves**
  - Tricuspid and mitral valves
  - Folds of fibrous tissue that extend into openings between atria and ventricles
  - Permit blood flow in one direction
    - From right atrium to right ventricle
    - From left atrium to left ventricle
- **Semilunar valves**
  - Pulmonary and aortic valves
  - Prevent backflow of blood into ventricles

20-1 Anatomy of the Heart

- **Right atrium** receives blood from
  - **Superior vena cava**
    - Carries blood from head, neck, upper limbs, and chest
  - **Inferior vena cava**
    - Carries blood from trunk, viscera, and lower limbs

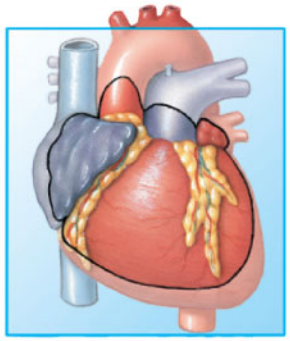
20-1 Anatomy of the Heart

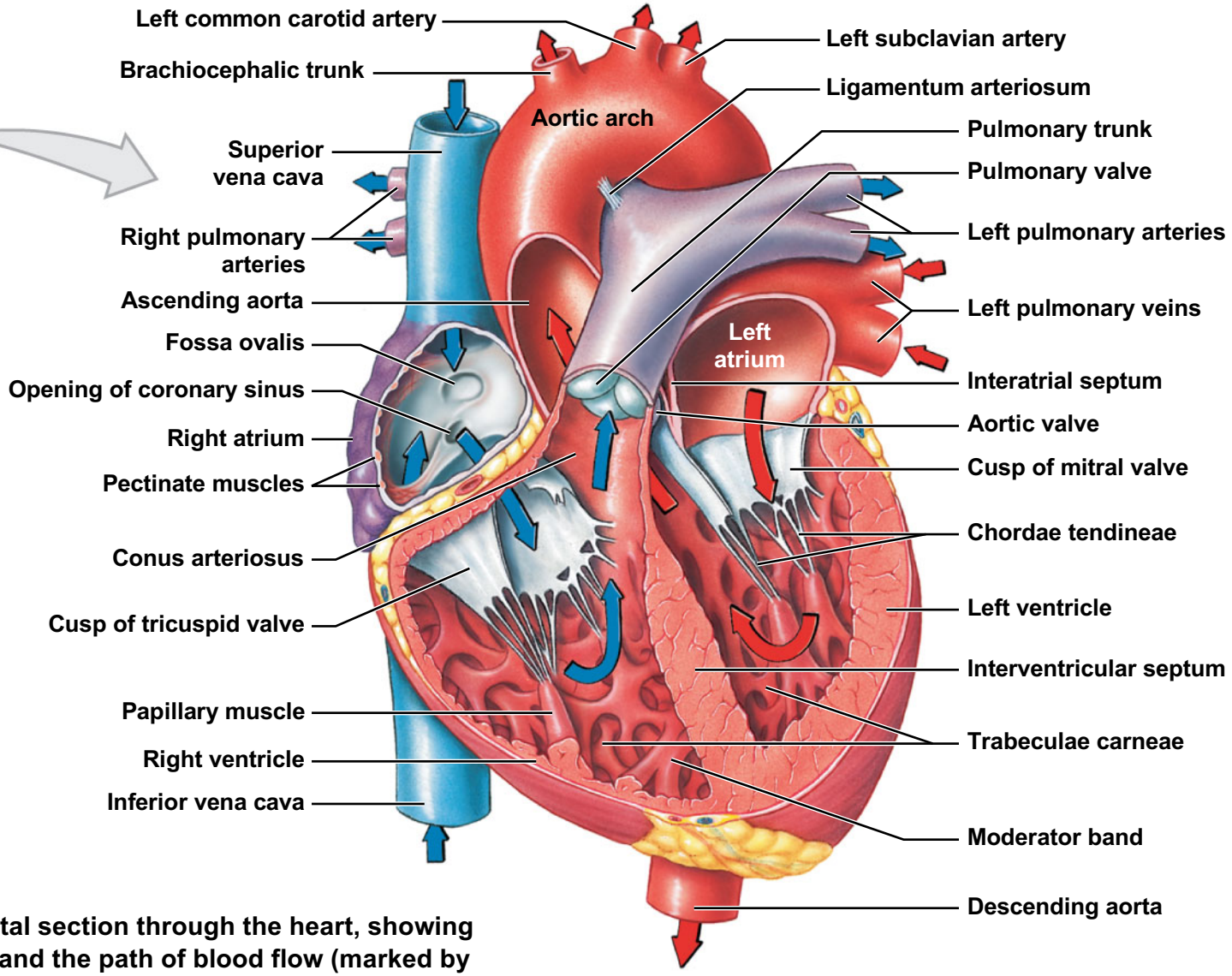
- Right atrium
  - **Foramen ovale**
    - Before birth, is an opening through interatrial septum
    - Connects the two atria of fetal heart
    - Closes at birth, eventually forming **fossa ovalis**
  - **Pectinate muscles**
    - Prominent muscular ridges
    - On anterior atrial wall and inner surface of auricle

20-1 Anatomy of the Heart

- Blood flows from right atrium to right ventricle
  - **Tricuspid valve** (right atrioventricular valve)
    - Has three **cusps**
    - Prevents backflow of blood
  - Free edges of valve attach to **chordae tendineae** from **papillary muscles** of ventricle
    - Prevent valve from opening backward

Figure 20–5a The Sectional Anatomy of the Heart.



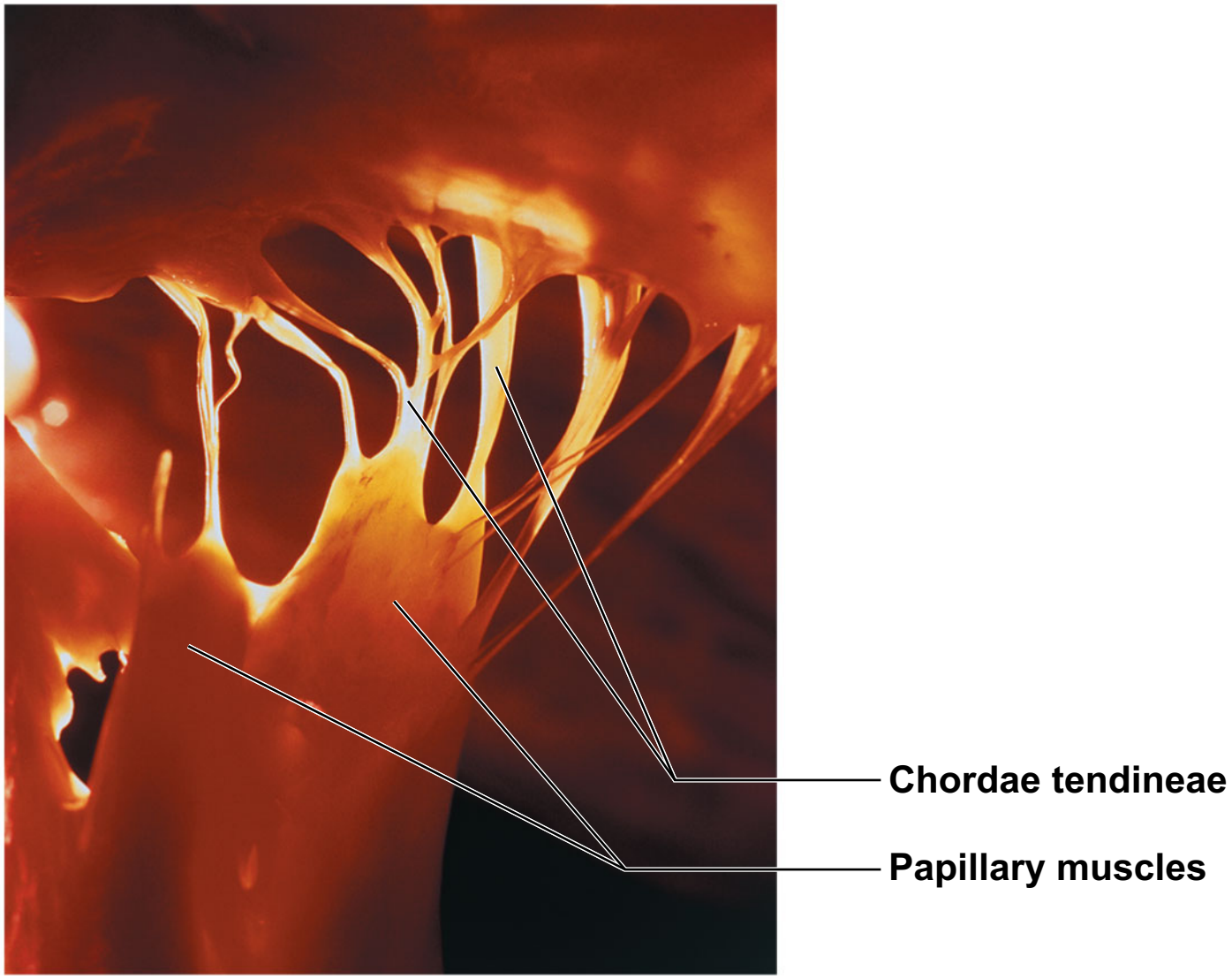


a A diagrammatic frontal section through the heart, showing anatomical features and the path of blood flow (marked by arrows) through the atria, ventricles, and associated vessels.

20-1 Anatomy of the Heart

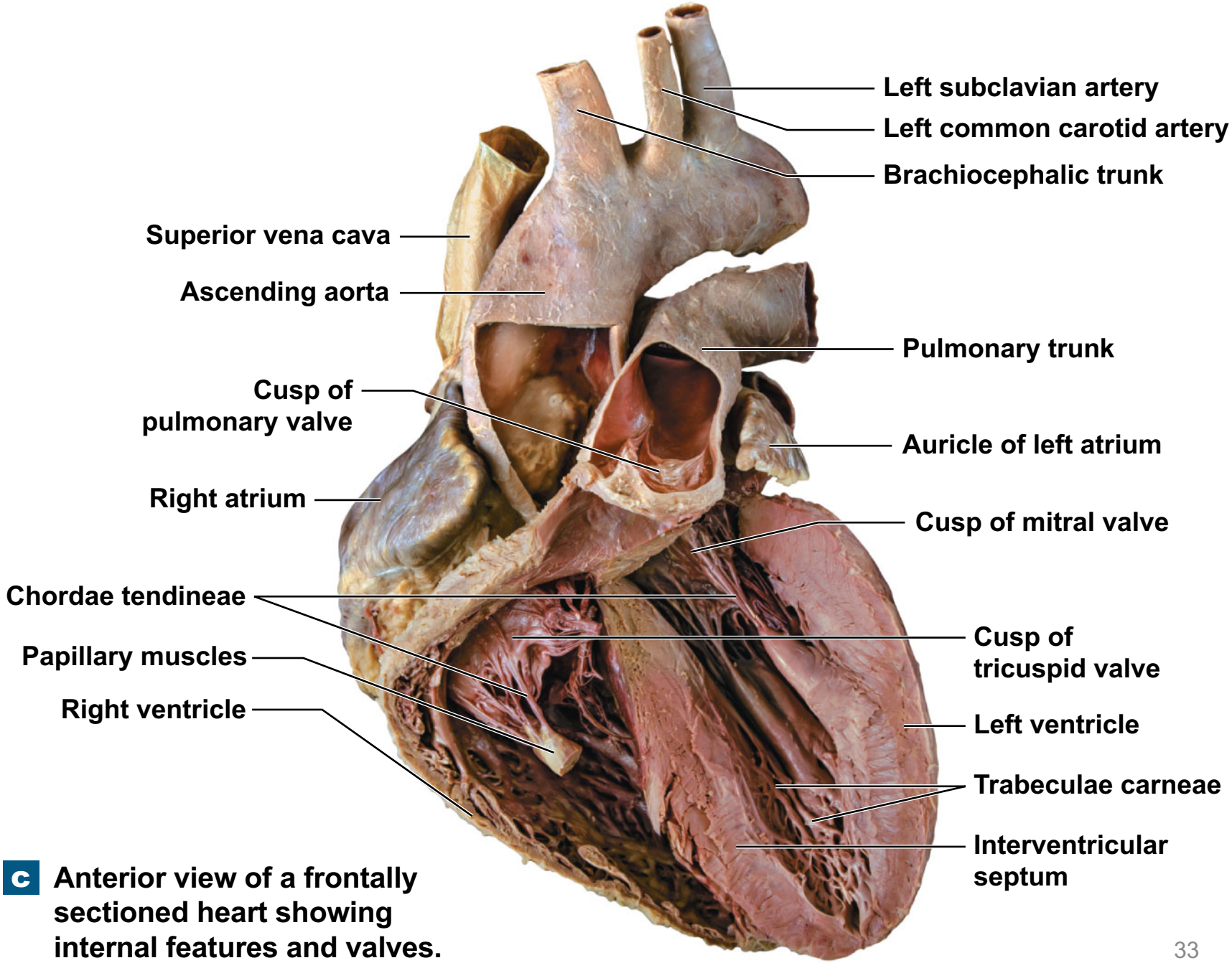
- **Right ventricle**
  - **Trabeculae carneae**
    - Muscular ridges on internal surface (of both ventricles)
  - Moderator band
    - Muscular ridge that delivers stimulus for contraction to papillary muscles

Figure 20–5b The Sectional Anatomy of the Heart.



b Papillary muscles and chordae tendineae support the mitral valve and tricuspid valve.

Figure 20–5c The Sectional Anatomy of the Heart.



c Anterior view of a frontally sectioned heart showing internal features and valves.

20-1 Anatomy of the Heart

- **Conus arteriosus**
  - At superior end of right ventricle
  - Ends at **pulmonary valve**
    - Three semilunar cusps
    - Leads to **pulmonary trunk**
      - Start of pulmonary circuit
      - Divides into **left** and **right pulmonary arteries**

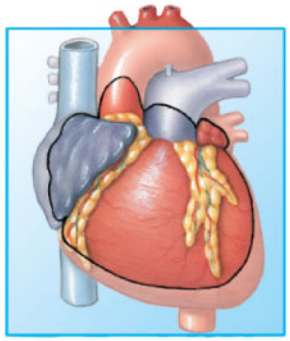
20-1 Anatomy of the Heart

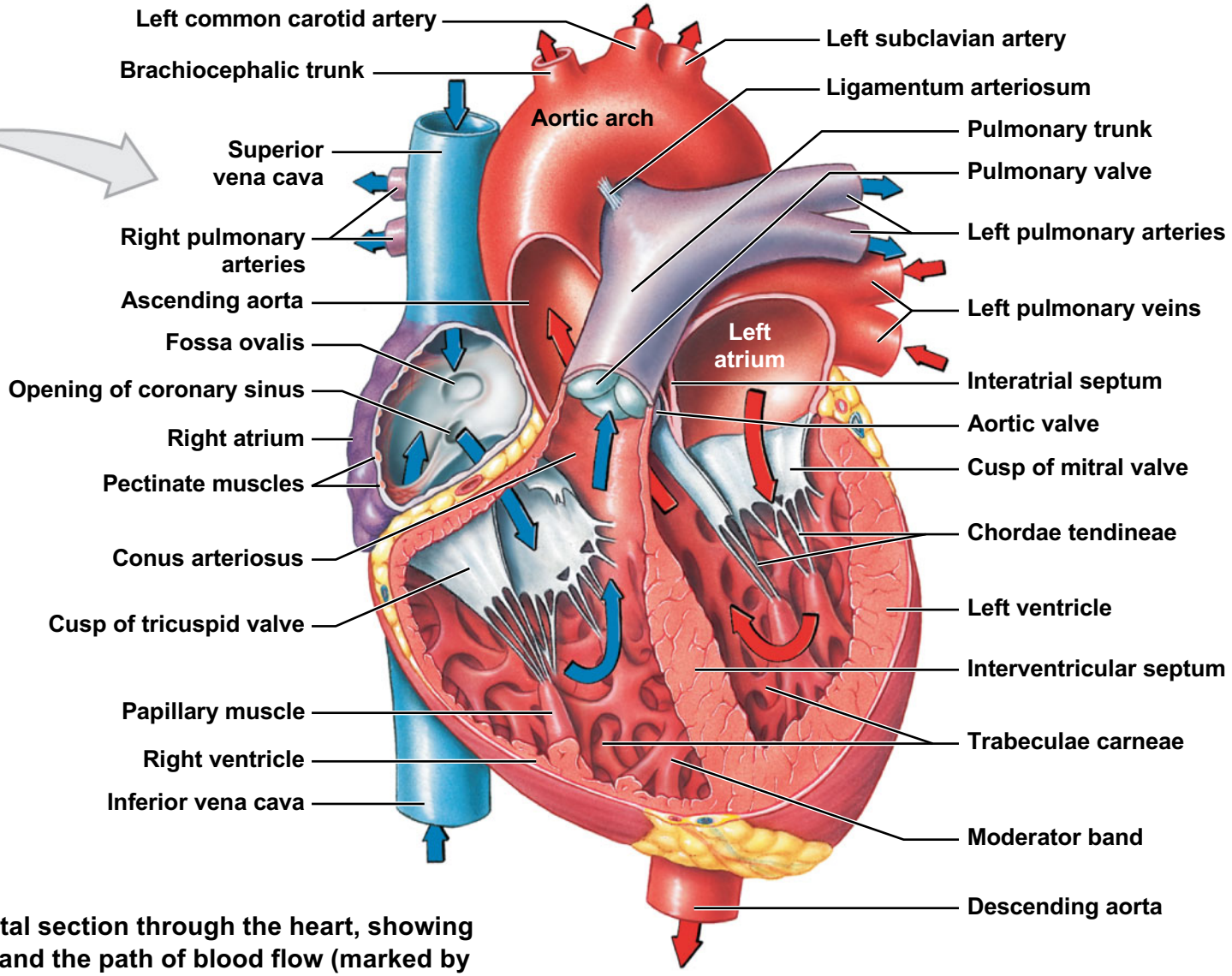
- **Left atrium**
  - Receives blood from **left** and **right pulmonary veins**
  - Blood passes to left ventricle through **mitral valve** (left atrioventricular valve or bicuspid valve)
    - Two cusps

20-1 Anatomy of the Heart

- **Left ventricle**
  - Similar to right ventricle but does not have moderator band
  - Blood leaves left ventricle through **aortic valve** into **ascending aorta**
  - **Aortic sinuses**
    - Saclike expansions at base of ascending aorta
  - Ascending aorta turns to become **aortic arch**
    - Becomes **descending aorta**

Figure 20–5a The Sectional Anatomy of the Heart.



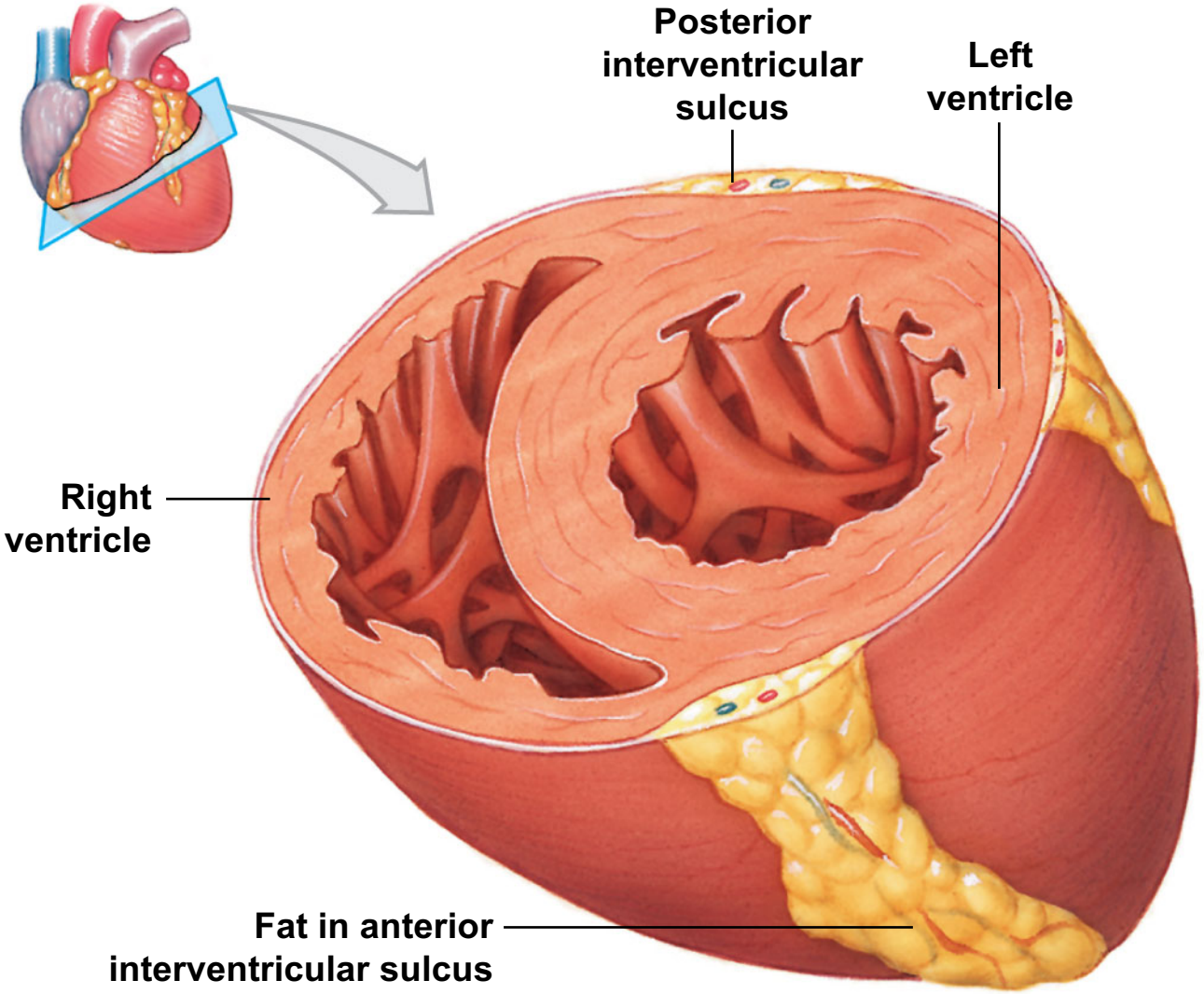


a A diagrammatic frontal section through the heart, showing anatomical features and the path of blood flow (marked by arrows) through the atria, ventricles, and associated vessels.

20-1 Anatomy of the Heart

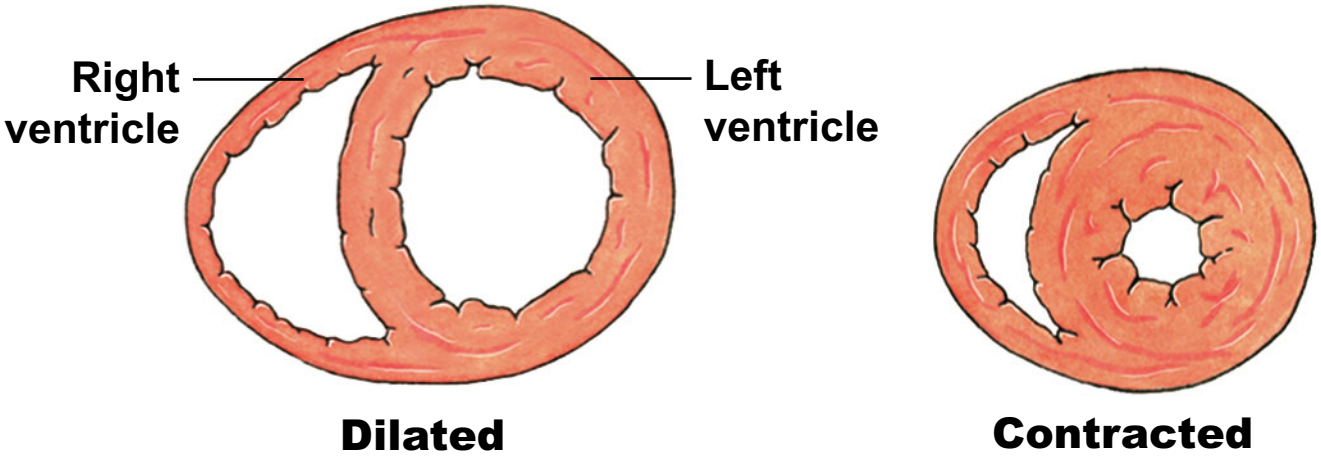
- Compared to left ventricle, the right ventricle
  - Holds and pumps the same amount of blood
  - Has thinner walls
  - Develops less pressure
  - Is more pouch-shaped than round

Figure 20–6a Structural Differences between the Left and Right Ventricles.



a A diagrammatic sectional view through the heart, showing the relative thicknesses of the two ventricular walls. Note the pouchlike shape of the right ventricle and the greater thickness of the left ventricular muscle.

Figure 20–6b Structural Differences between the Left and Right Ventricles.



b Diagrammatic views of the ventricles just before a contraction (dilated) and just after a contraction (contracted).

20-1 Anatomy of the Heart

- Heart valves
  - Prevent backflow of blood
- Atrioventricular (AV) valves
  - Between atria and ventricles
  - When ventricles contract,
    - Blood pressure closes valves
    - Papillary muscles contract and tense chordae tendineae
      - Prevents **regurgitation** (backflow) of blood into atria

20-1 Anatomy of the Heart

- Semilunar valves
  - Pulmonary and aortic valves
  - Prevent backflow of blood into ventricles
  - No muscular braces
  - **Valvular heart disease** (**VHD**)
    - Deterioration of valve function
    - May develop after **carditis** (inflammation of heart)
      - May result from **rheumatic fever** (inflammatory autoimmune response to streptococcal bacteria)

Figure 20–7a Valves of the Heart and Blood Flow (Part 1 of 2).

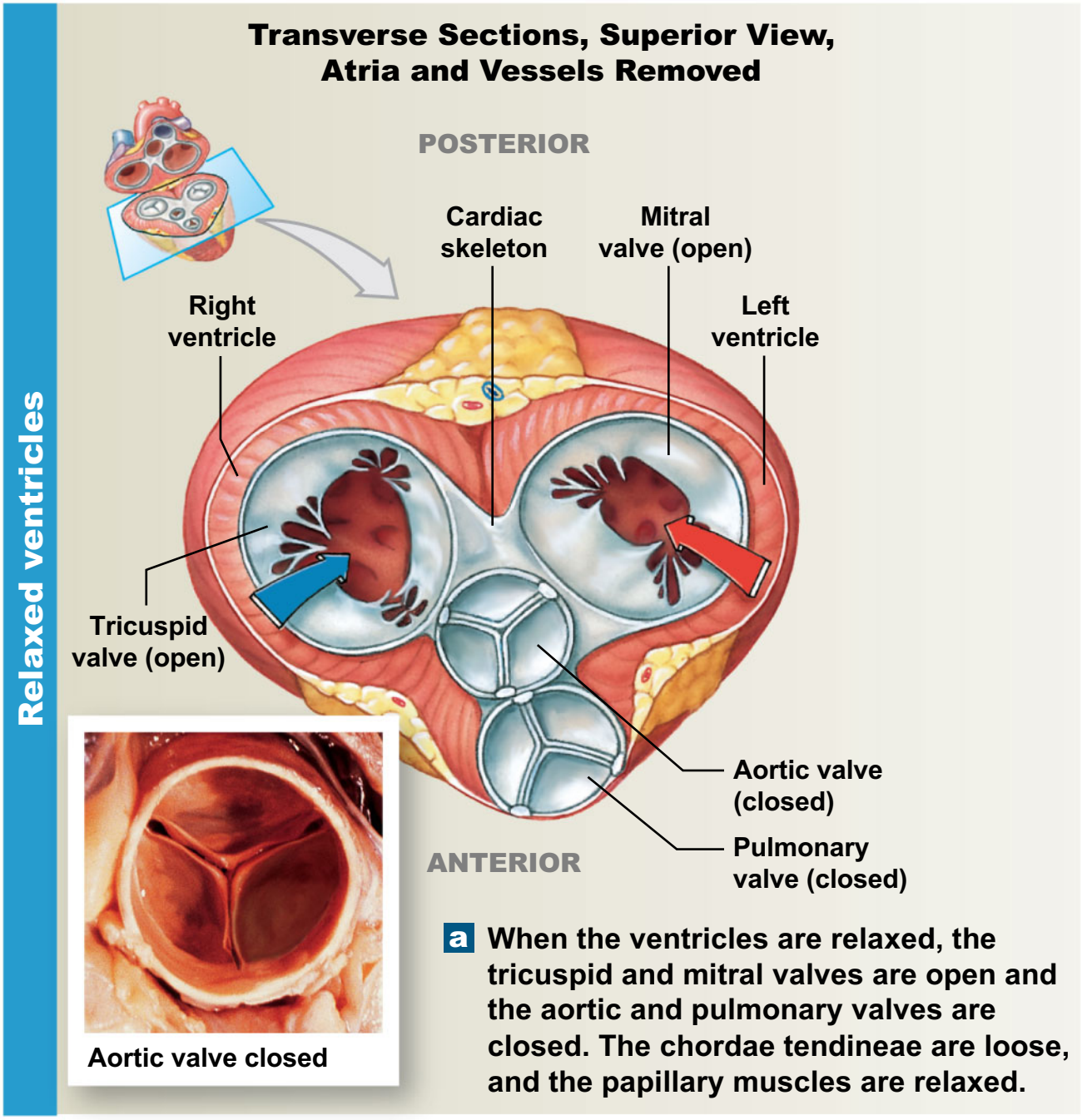


Figure 20–7a Valves of the Heart and Blood Flow (Part 2 of 2).

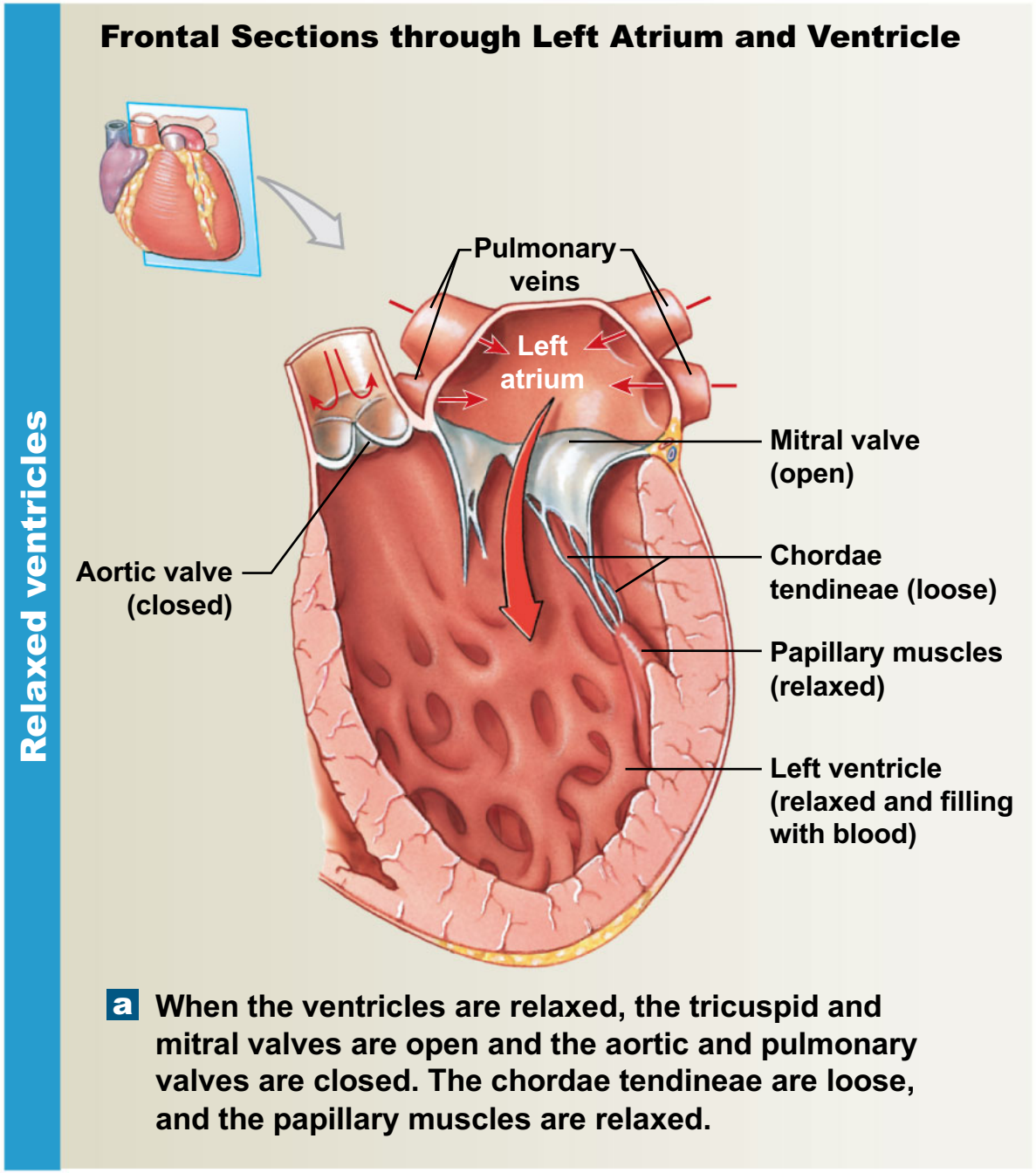
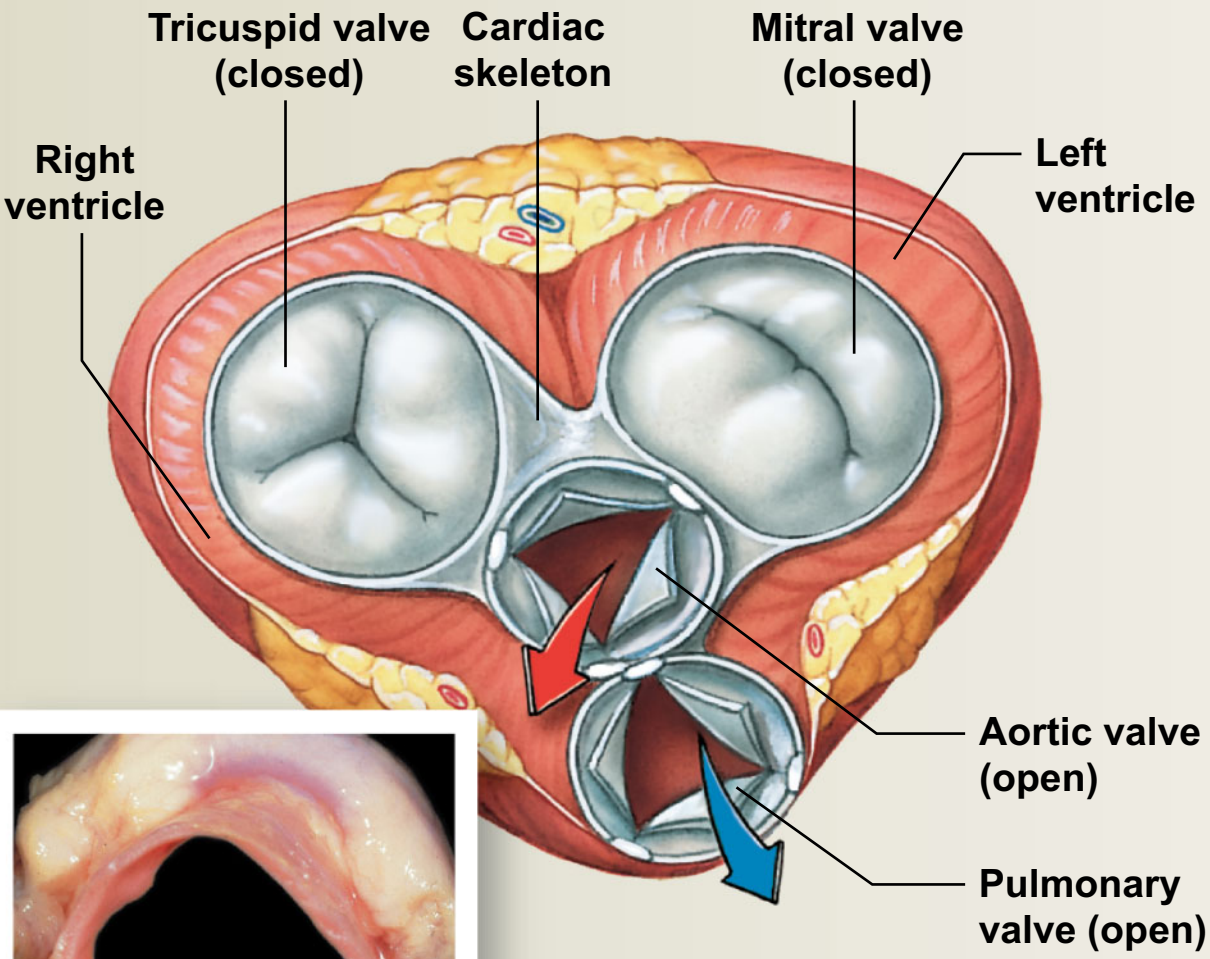


Figure 20–7b Valves of the Heart and Blood Flow (Part 1 of 2).

Contracting ventricles

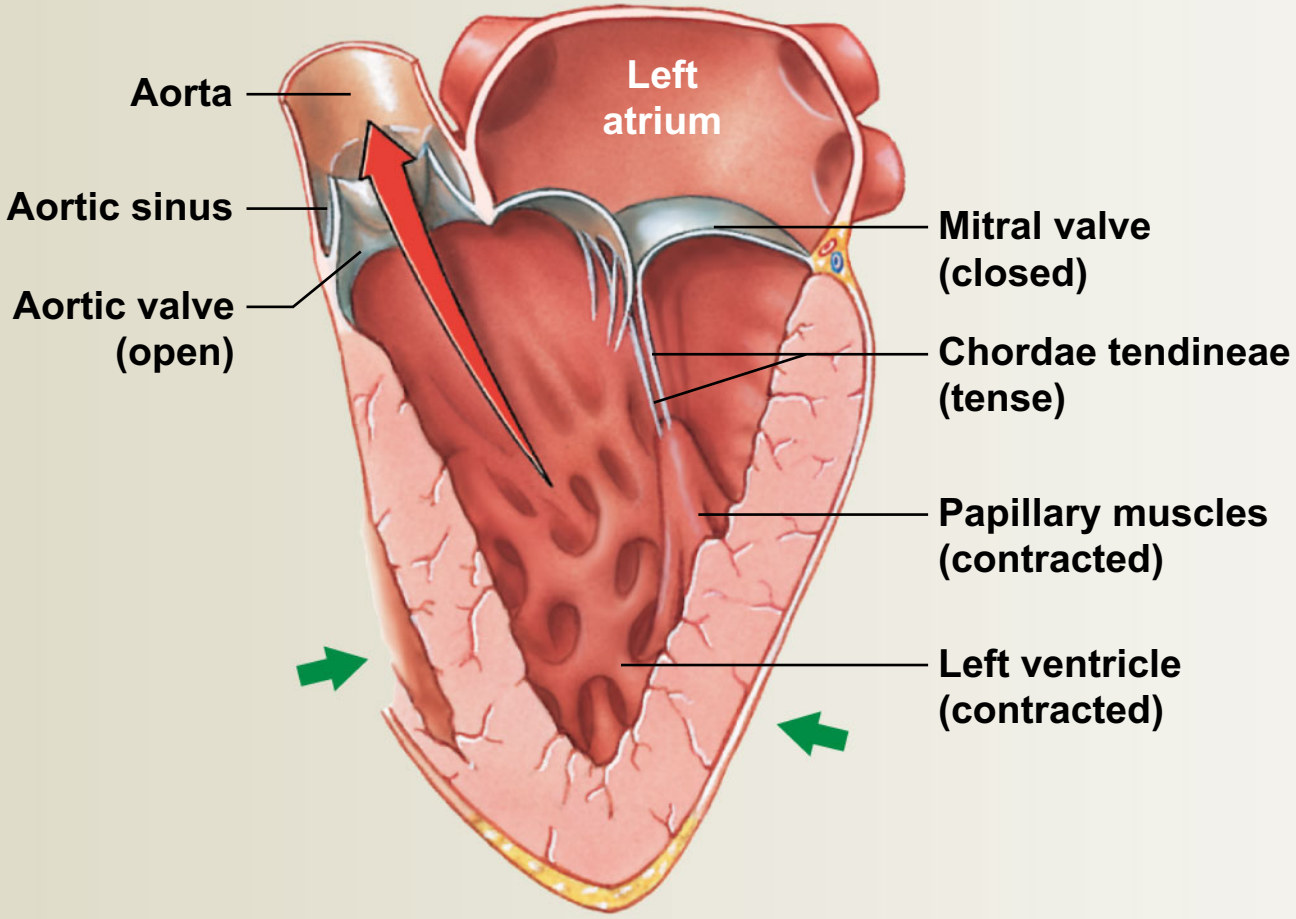


Aortic valve open

b When the ventricles are contracting, the tricuspid and mitral valves are closed and the aortic and pulmonary valves are open. In the frontal section, note the attachment of the mitral valve to the chordae tendineae and papillary muscles.

Figure 20–7b Valves of the Heart and Blood Flow (Part 2 of 2).

Contracting ventricles



b When the ventricles are contracting, the tricuspid and mitral valves are closed and the aortic and pulmonary valves are open. In the frontal section, note the attachment of the mitral valve to the chordae tendineae and papillary muscles.

20-1 Anatomy of the Heart

- **Coronary circulation**
  - Supplies blood to muscle tissue of heart
  - **Coronary arteries**
    - Originate at aortic sinuses
    - Elevated blood pressure and elastic rebound of aorta maintain blood flow through coronary arteries

20-1 Anatomy of the Heart

- **Right coronary artery**
  - Supplies blood to
    - Right atrium
    - Portions of both ventricles
    - Portions of electrical conducting system of heart
  - Gives rise to
    - **Marginal arteries**
    - **Posterior interventricular artery**

20-1 Anatomy of the Heart

- **Left coronary artery**
  - Supplies blood to
    - Left ventricle
    - Left atrium
    - Interventricular septum
  - Gives rise to
    - **Circumflex artery**
    - **Anterior interventricular artery**

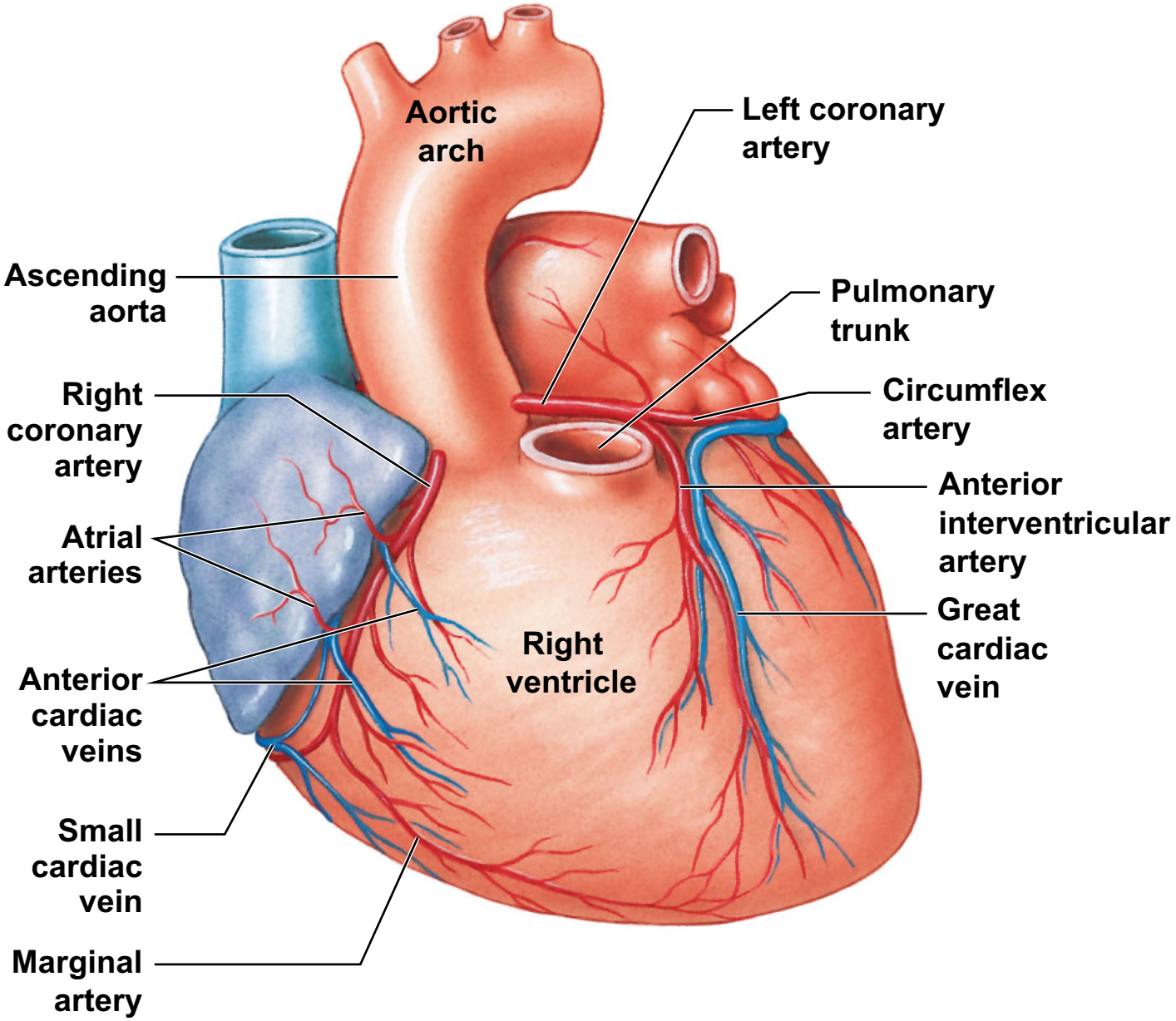
20-1 Anatomy of the Heart

- **Arterial anastomoses**
  - Interconnect anterior and posterior interventricular arteries
  - Maintain constant blood supply to cardiac muscle

20-1 Anatomy of the Heart

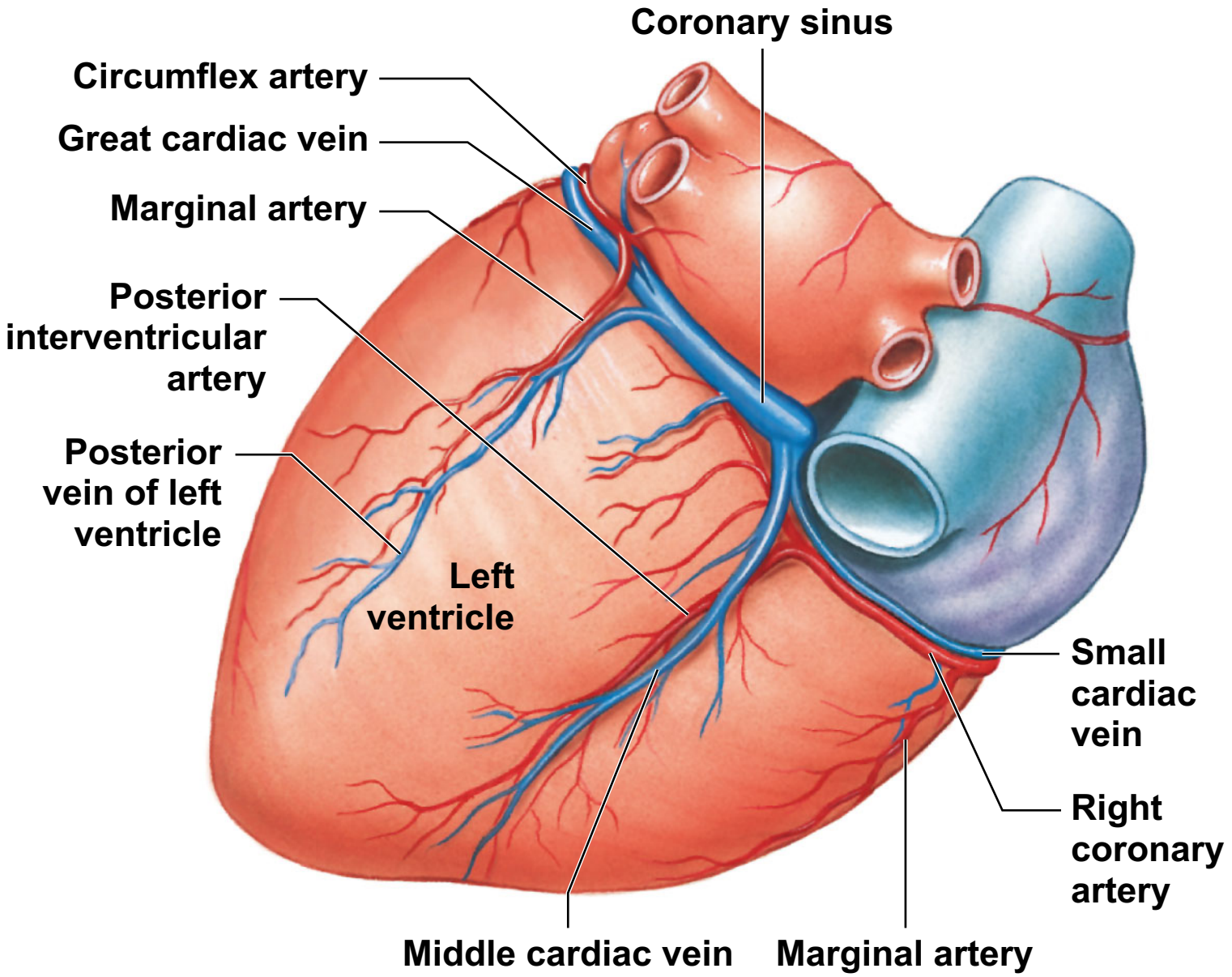
- Cardiac veins
  - **Great cardiac vein**
    - Drains blood from region supplied by anterior interventricular artery
    - Returns blood to **coronary sinus**
      - Opens into right atrium
  - **Posterior vein of left ventricle**, **middle cardiac vein**, and **small cardiac vein**
    - Empty into great cardiac vein or coronary sinus
  - **Anterior cardiac veins** empty into right atrium

Figure 20–8a The Coronary Circulation.



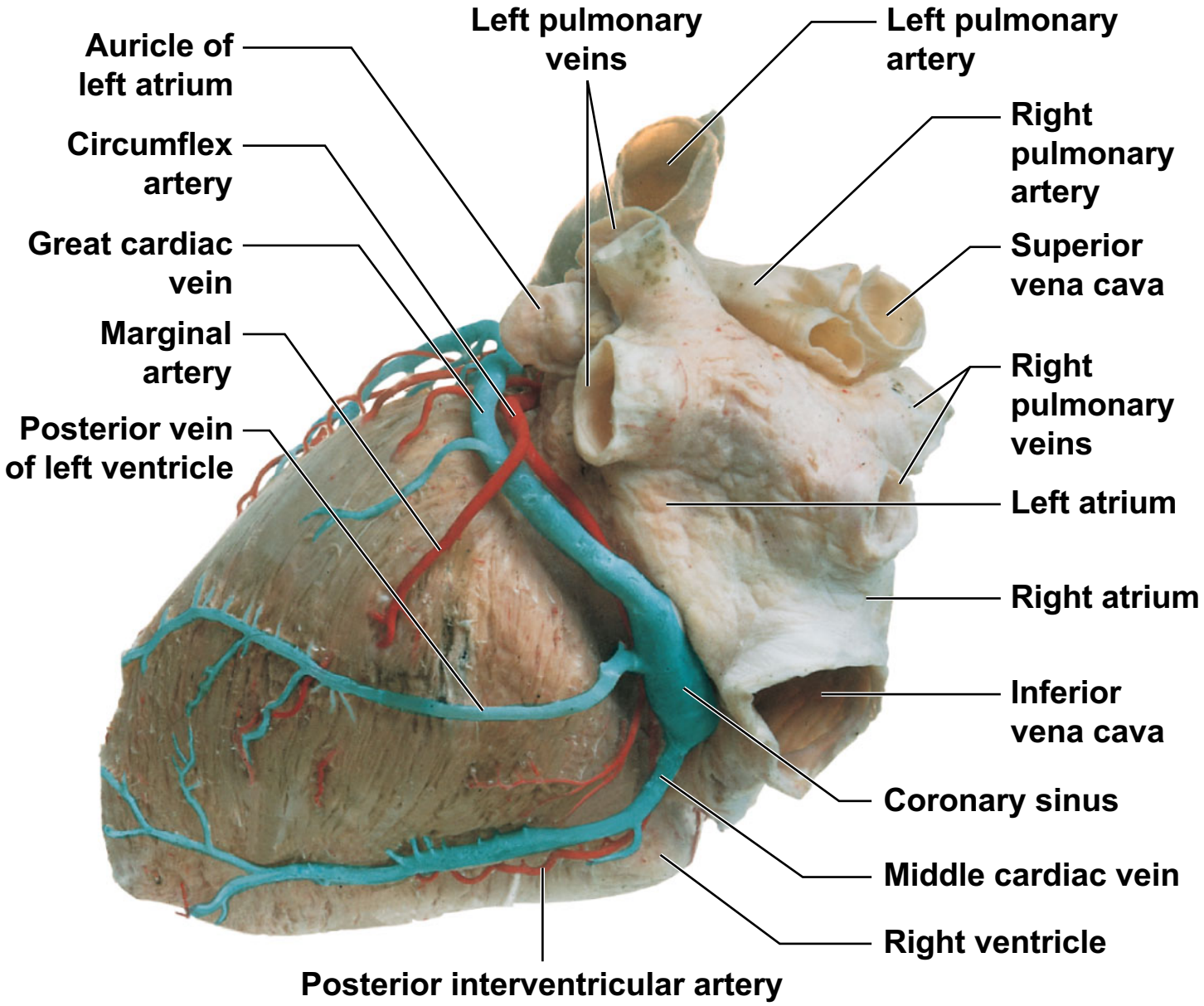
a Coronary vessels supplying and draining the anterior surface of the heart.

Figure 20–8b The Coronary Circulation.



b Coronary vessels supplying and draining the posterior surface of the heart.

Figure 20–8c The Coronary Circulation.

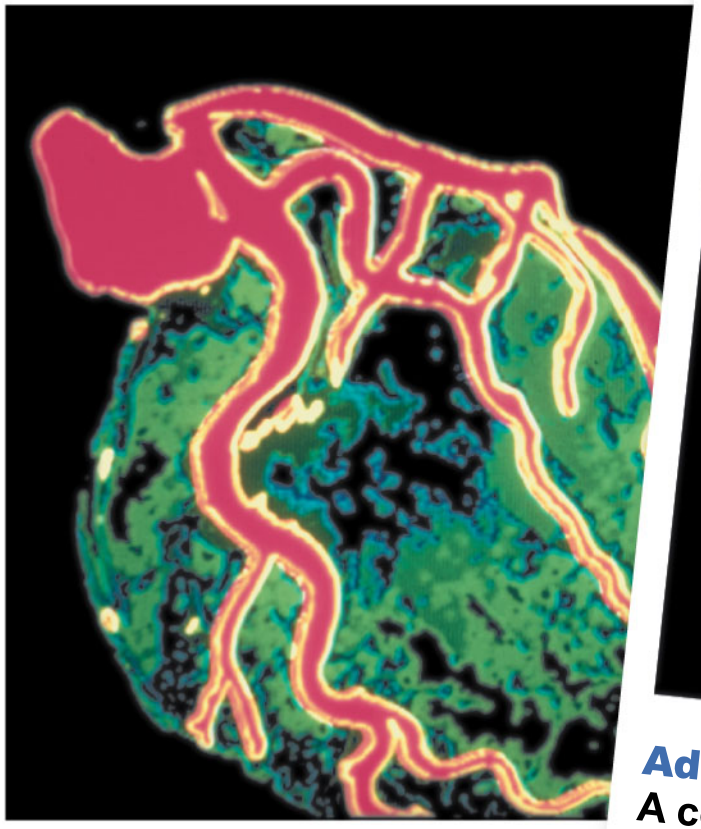


C A posterior view of the heart; the vessels have been injected with colored latex (liquid rubber).

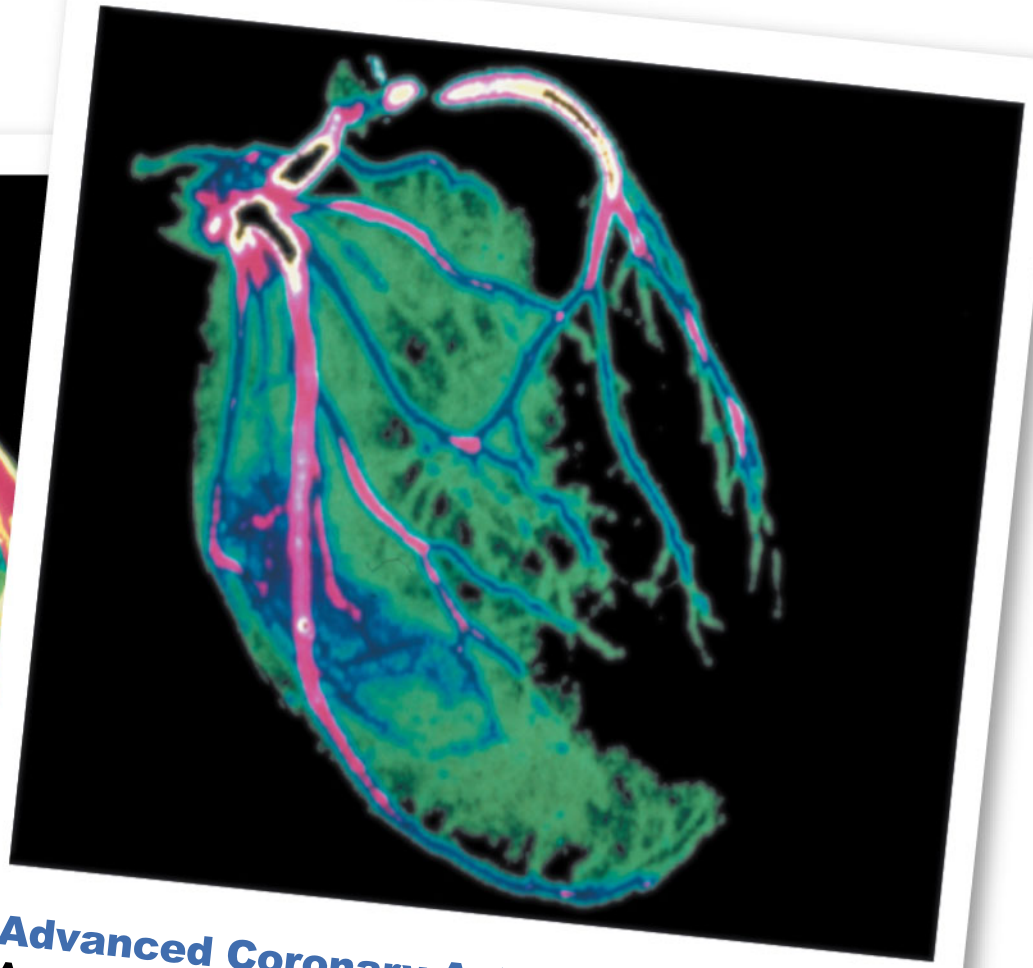
20-1 Anatomy of the Heart

- Coronary artery disease (CAD)
  - Areas of partial or complete blockage of coronary circulation
- Cardiac muscle cells need a constant supply of oxygen and nutrients
  - Reduction in blood flow to heart muscle reduces cardiac performance
- Coronary ischemia
  - Reduced circulatory supply from partial or complete blockage of coronary arteries

Figure 20–9 Heart Disease and Heart Attacks (Part 1 of 4).



Normal Heart
A color-enhanced **digital subtraction angiography (DSA)** scan of a normal heart.

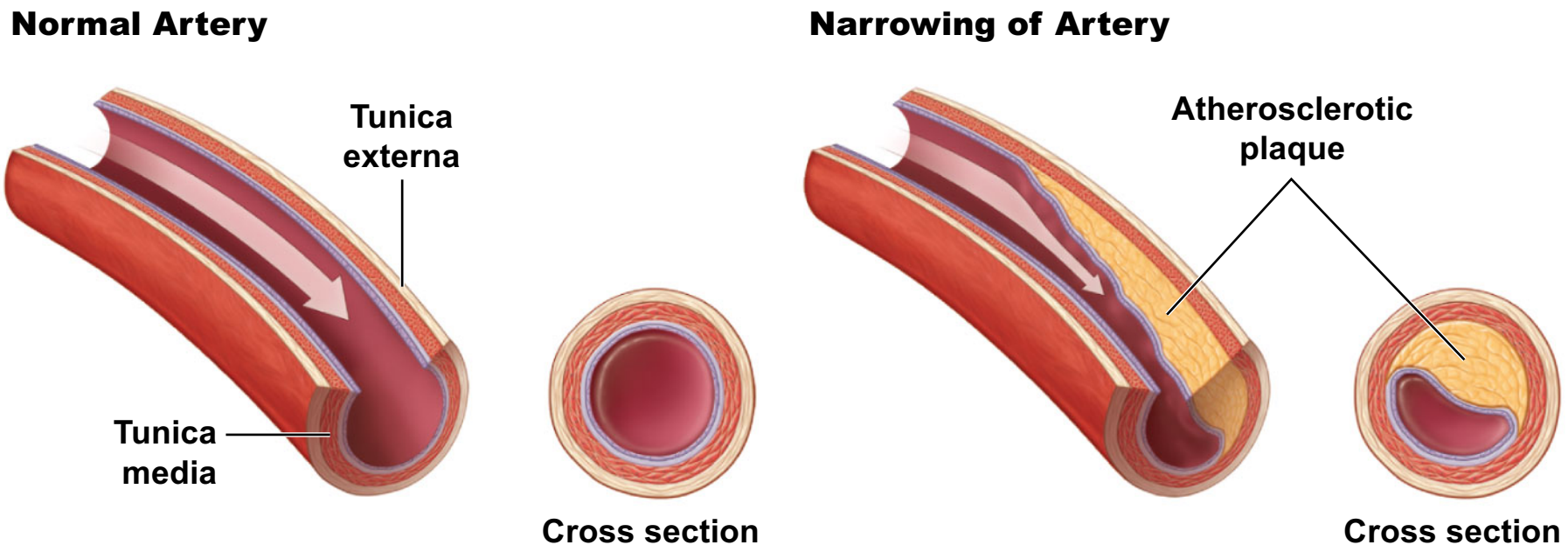


Advanced Coronary Artery Disease
A color-enhanced DSA scan showing advanced coronary artery disease. Blood flow to the ventricular myocardium is severely restricted.

20-1 Anatomy of the Heart

- Coronary artery disease
  - Usual cause is formation of a fatty deposit, or atherosclerotic plaque, in wall of coronary vessel
  - The plaque, or an associated thrombus (clot), narrows passageway and reduces blood flow
  - Spasms in smooth muscles of vessel wall can further decrease or stop blood flow

Figure 20–9 Heart Disease and Heart Attacks (Part 2 of 4).



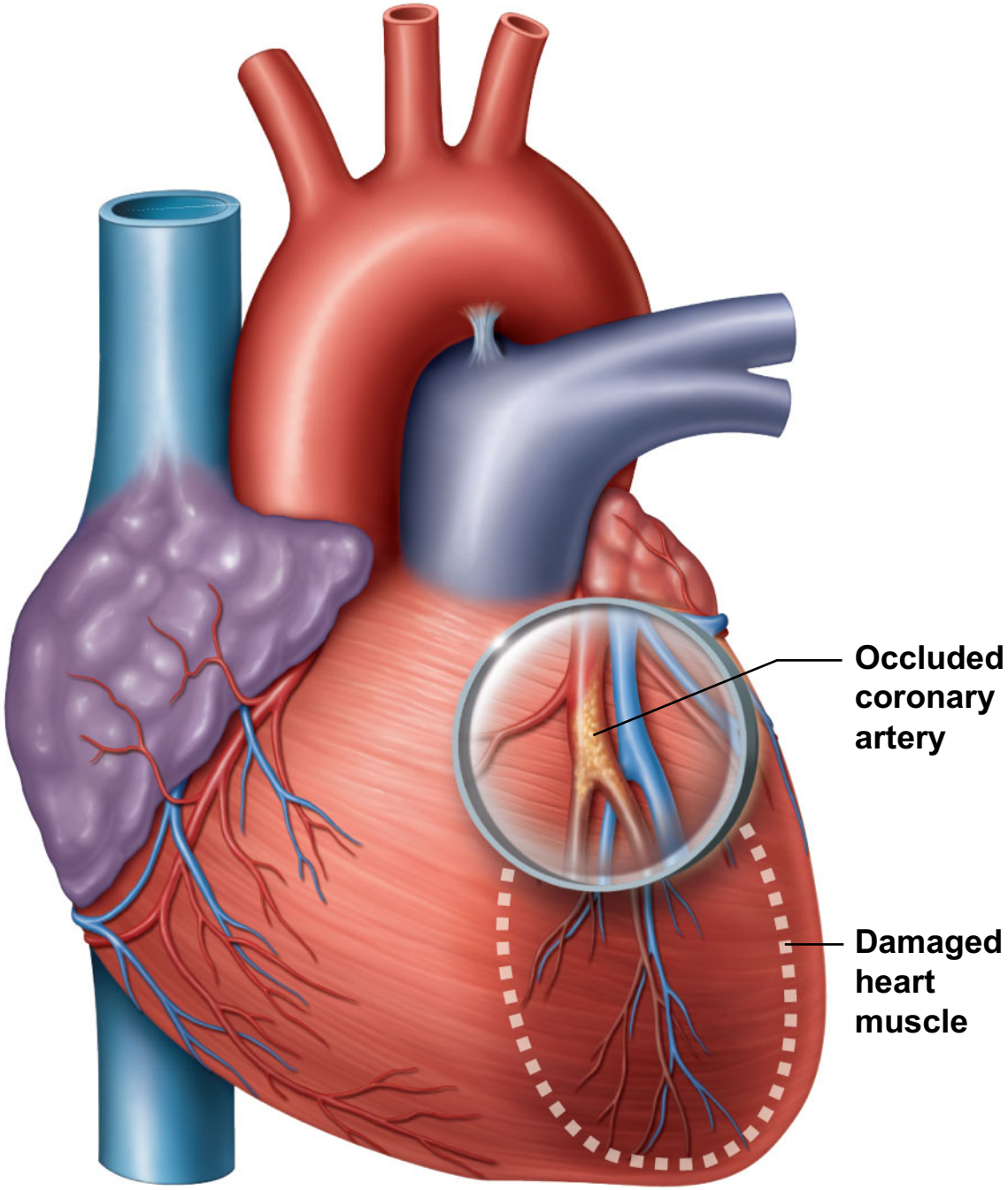
20-1 Anatomy of the Heart

- Angina pectoris
  - Commonly one of the first symptoms of CAD
  - A temporary ischemia develops when workload of heart increases
  - Individual may feel comfortable at rest
  - Exertion or emotional stress can produce sensations of pressure, chest constriction, and pain
  - Pain may radiate from sternal area to arms, back, and neck

20-1 Anatomy of the Heart

- Myocardial infarction (MI), or heart attack
  - Part of coronary circulation becomes blocked
  - Cardiac muscle cells die from lack of oxygen
  - Death of affected tissue creates a nonfunctional area known as an infarct
  - Most commonly results from severe CAD
  - Coronary thrombosis
    - Thrombus formation at a plaque
    - Most common cause of an MI

Figure 20–9 Heart Disease and Heart Attacks (Part 3 of 4).



20-1 Anatomy of the Heart

- Myocardial infarction
  - Consequences depend on site and nature of circulatory blockage
  - If near the start of one of the coronary arteries
    - Damage will be widespread and heart may stop beating
  - If blockage involves small arterial branch
    - Individual may survive the immediate crisis
    - But may have complications such as reduced contractility and cardiac arrhythmias

20-1 Anatomy of the Heart

- Myocardial infarction
  - Causes intense, persistent pain, even at rest
  - Pain is not always felt
    - May go undiagnosed and untreated
  - Often diagnosed with ECG and blood studies
  - Damaged myocardial cells release enzymes into circulation
    - Cardiac troponin T
    - Cardiac troponin I
    - A form of creatinine phosphokinase, CK-MB

20-1 Anatomy of the Heart

- Treatment of CAD and myocardial infarction
  - About 25 percent of MI patients die before obtaining medical assistance
    - 65 percent of MI deaths among people under age 50 occur within an hour

20-1 Anatomy of the Heart

- Risk factor modification
  - Stop smoking
  - Treat high blood pressure
  - Adjust diet to lower cholesterol and promote weight loss
  - Reduce stress
  - Increase physical activity

20-1 Anatomy of the Heart

- Drug treatments are used to
  - Reduce coagulation (e.g., aspirin and coumadin)
  - Block sympathetic stimulation (propranolol or metoprolol)
  - Cause vasodilation (e.g., nitroglycerin)
  - Block calcium ion movement into muscle cells (calcium ion channel blockers)
  - Relieve pain and help dissolve clots (in MI)

20-1 Anatomy of the Heart

- Noninvasive surgery
  - Atherectomy
    - Long, slender catheter is inserted into coronary artery to remove plaque
  - Balloon angioplasty
    - Tip of catheter contains inflatable balloon
    - Inflated balloon presses plaque against vessel walls
    - Plaques commonly redevelop
    - A stent may be inserted to hold vessel open

20-1 Anatomy of the Heart

- Coronary artery bypass graft (CABG)
  - Small section of another vessel is removed
    - Used to create detour around obstructed portion of coronary artery
  - Up to four coronary arteries can be rerouted during a single operation
    - Single, double, triple, or quadruple coronary bypasses

20-2 The Conducting System

- **Heartbeat**
  - A single cardiac contraction
  - All heart chambers contract in series
    - First the atria
    - Then the ventricles
- Two types of cardiac muscle cells
  - Autorhythmic cells
    - Control and coordinate heartbeat
  - Contractile cells
    - Produce contractions that propel blood

20-2 The Conducting System

- **Conducting system**
  - Consists of specialized cardiac muscle cells
    - Initiate and distribute electrical impulses that stimulate contraction
  - **Autorhythmicity**
    - Cardiac muscle tissue contracts without neural or hormonal stimulation

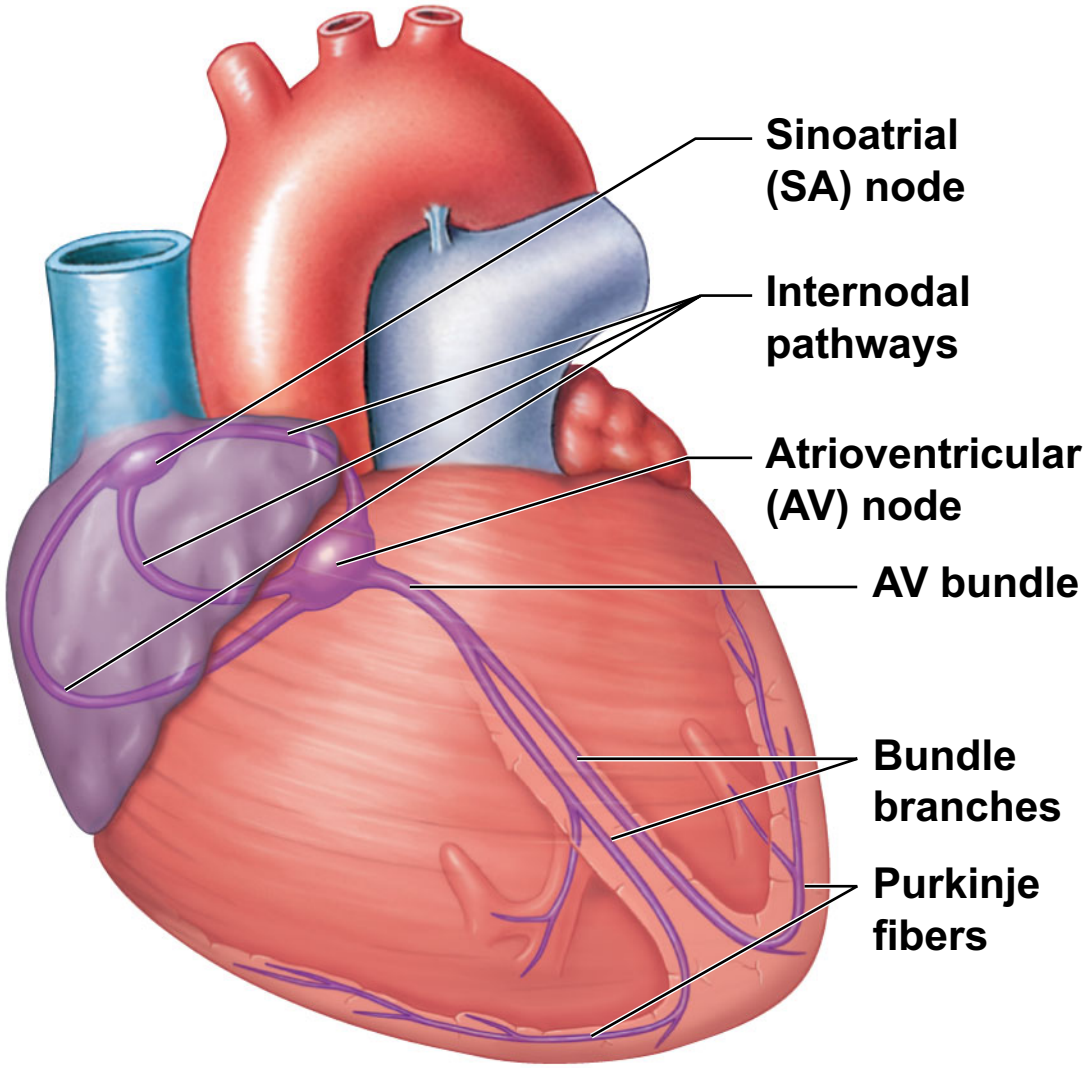
20-2 The Conducting System

- Components of the conducting system
  - **Pacemaker cells** found in
    - **Sinoatrial** (**SA**) **node**—in wall of right atrium
    - **Atrioventricular** (**AV**) **node**—at junction between atria and ventricles
  - **Conducting cells** found in
    - **Internodal pathways** of atria
    - **Atrioventricular** (**AV**) **bundle**, **bundle branches**, and **Purkinje fibers** of ventricles

20-2 The Conducting System

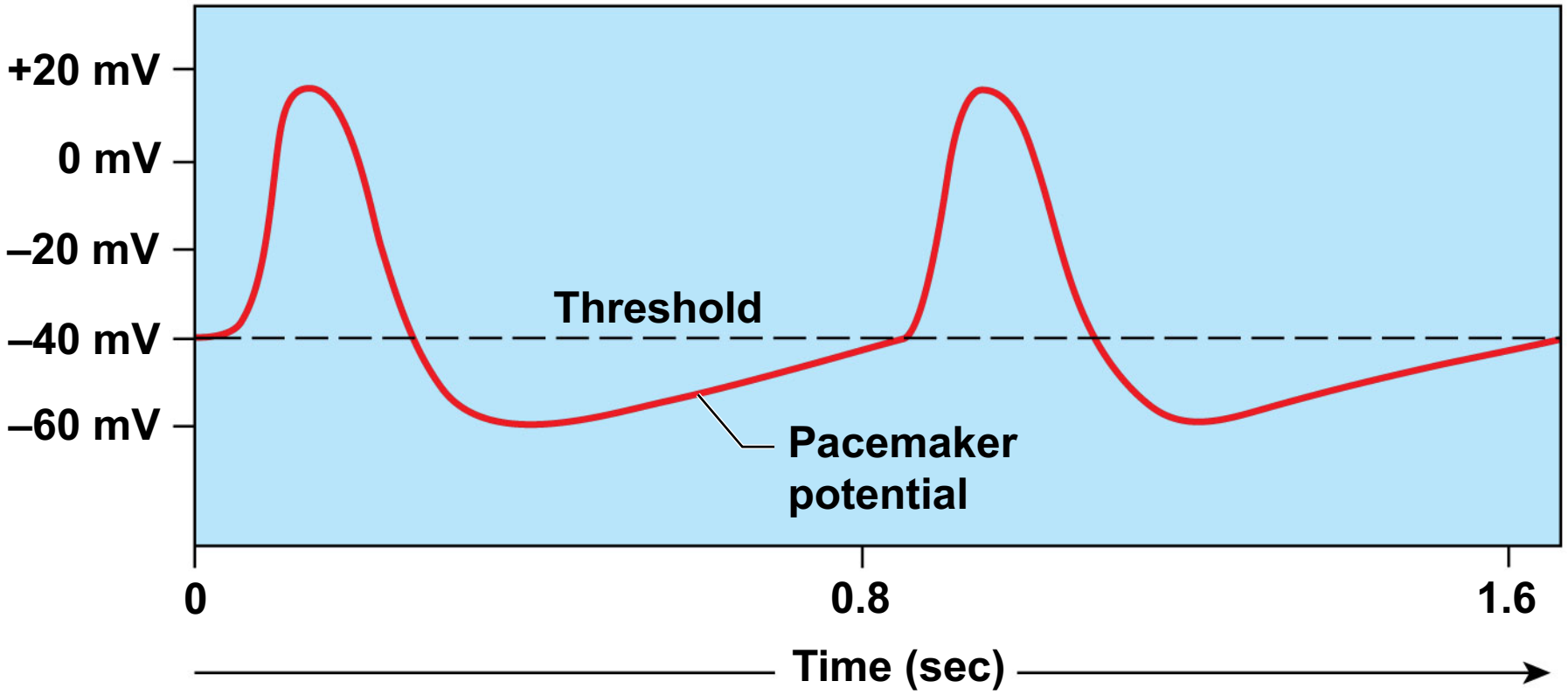
- **Pacemaker potential**
  - Gradual depolarization of pacemaker cells
    - Do not have a stable resting membrane potential
- Rate of spontaneous depolarization
  - SA node: 60–100 action potentials per minute
  - AV node: 40–60 action potentials per minute
- SA node depolarizes first
  - Establishing **sinus rhythm**
- Parasympathetic stimulation slows heart rate

Figure 20–10a The Conducting System of the Heart and the Pacemaker Potential.



a Components of the conducting system.

Figure 20–10b The Conducting System of the Heart and the Pacemaker Potential.



b Changes in the membrane potential of a pacemaker cell in the SA node that is establishing a heart rate of 72 beats per minute. Note the pacemaker potential, a gradual spontaneous depolarization.

20-2 The Conducting System

- Impulse conduction through the heart
  1. SA node activity and atrial activation begin
  2. Stimulus spreads across atria and reaches AV node
  3. Impulse is delayed for 100 msec at AV node
     - Atrial contraction begins

Figure 20–11 Impulse Conduction through the Heart and Accompanying ECG Tracings (Part 1 of 5).

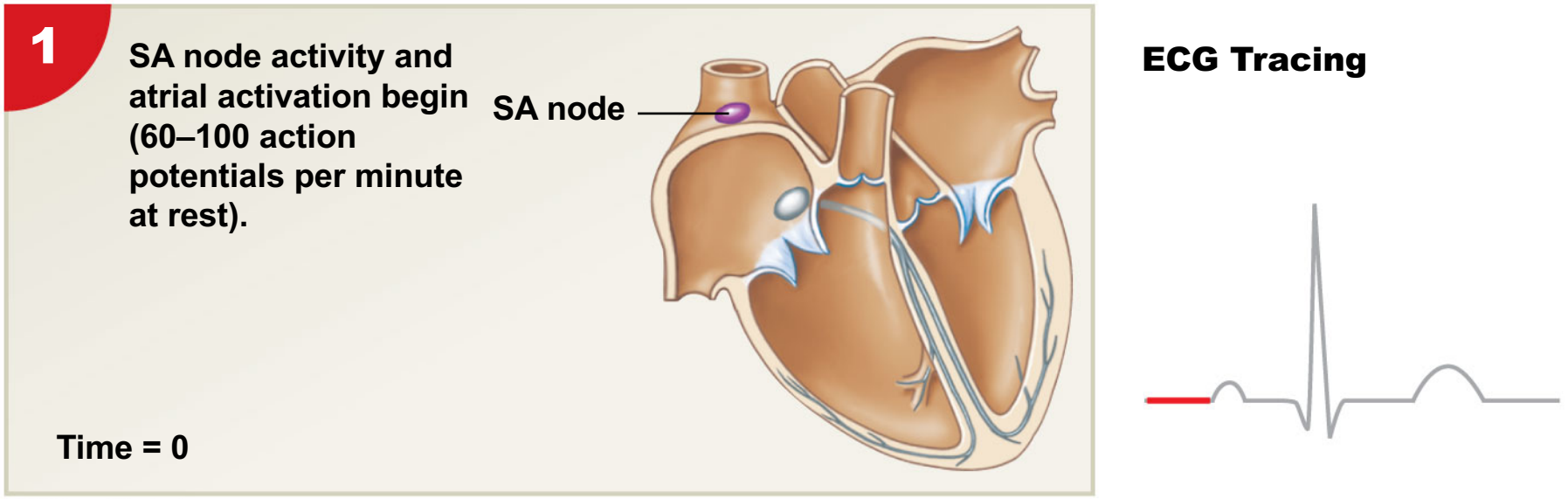


Figure 20–11 Impulse Conduction through the Heart and Accompanying ECG Tracings (Part 2 of 5).

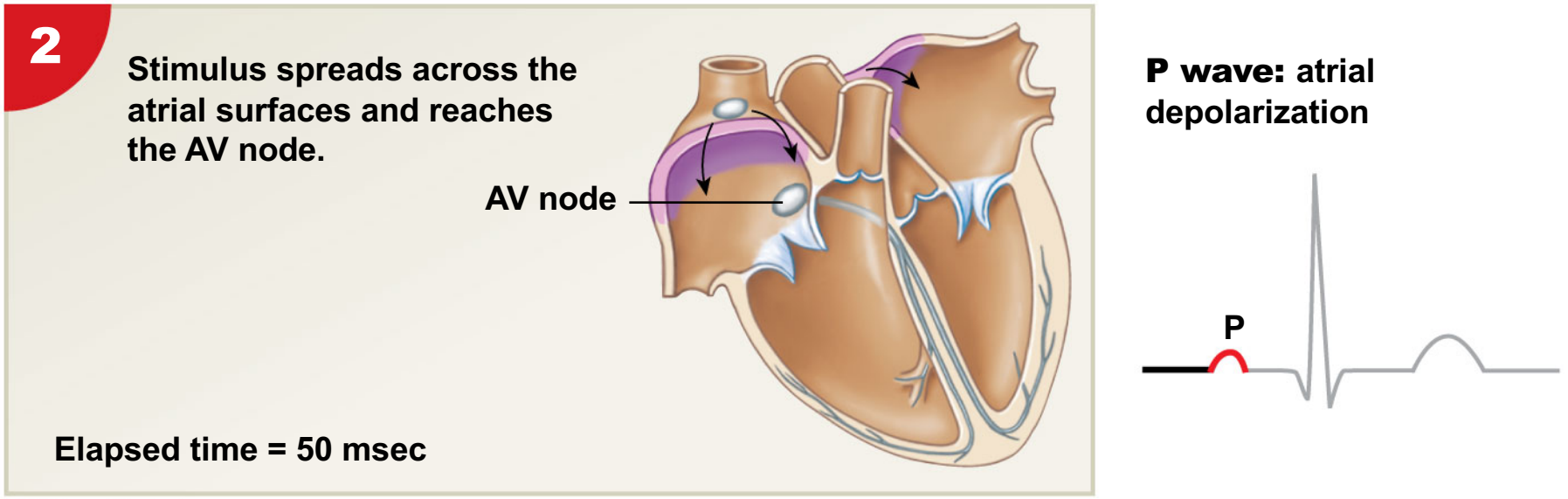
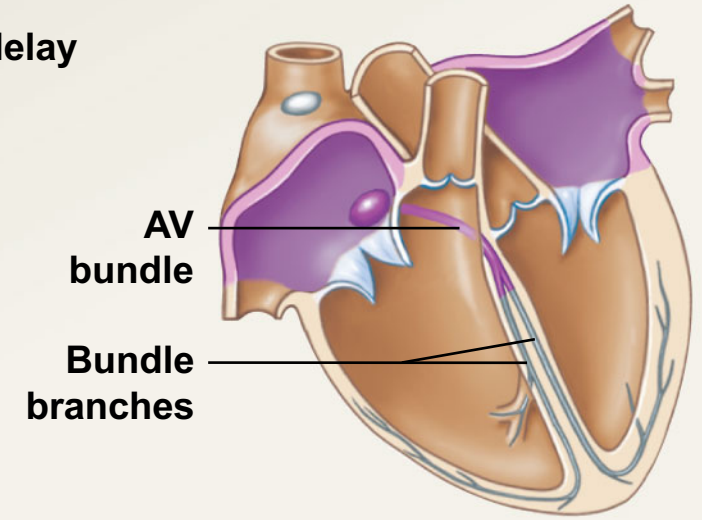


Figure 20–11 Impulse Conduction through the Heart and Accompanying ECG Tracings (Part 3 of 5).

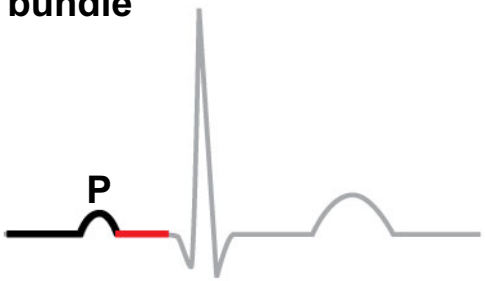
3

There is a 100-msec delay at the AV node. Atrial contraction begins.



Elapsed time = 150 msec

P–R interval: conduction through AV node and AV bundle



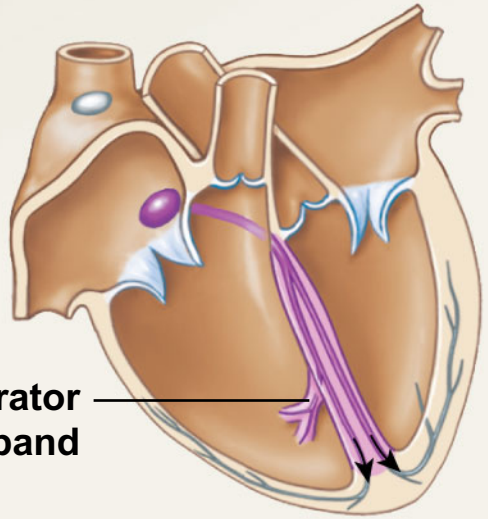
20-2 The Conducting System

- Impulse conduction through the heart
  4. Impulse travels in AV bundle to **left** and **right bundle branches** in interventricular septum
     - To Purkinje fibers
     - And to papillary muscles via moderator band
  5. Purkinje fibers distribute impulse to ventricular myocardium
     - Atrial contraction is completed
     - Ventricular contraction begins

Figure 20–11 Impulse Conduction through the Heart and Accompanying ECG Tracings (Part 4 of 5).

4

The impulse travels along the interventricular septum within the AV bundle and the bundle branches to the Purkinje fibers and, by the moderator band, to the papillary muscles of the right ventricle.



Elapsed time = 175 msec

Q wave:
beginning of ventricular depolarization

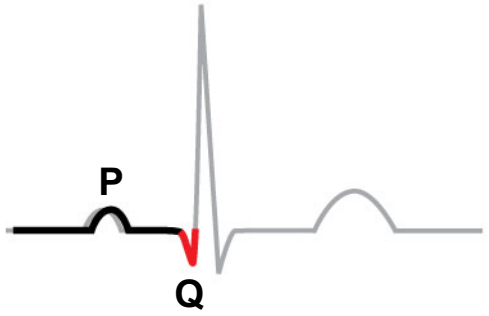
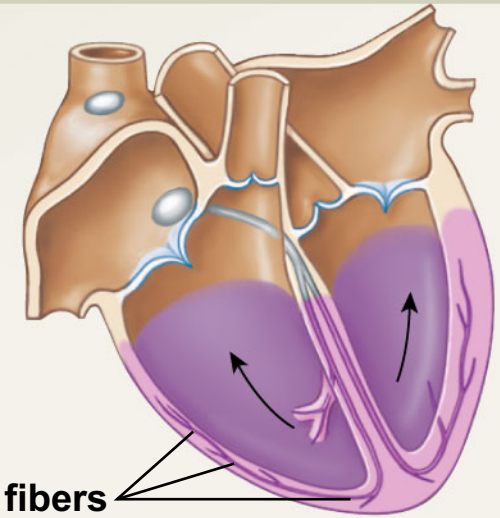


Figure 20–11 Impulse Conduction through the Heart and Accompanying ECG Tracings (Part 5 of 5).

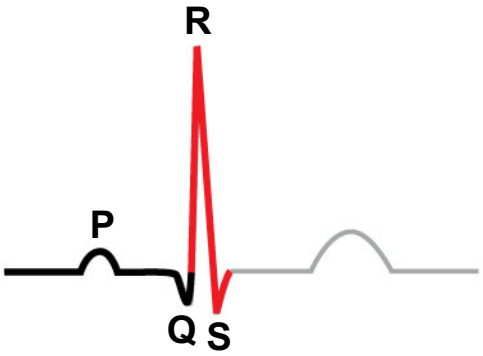
5

The impulse is distributed by Purkinje fibers and relayed throughout the ventricular myocardium. Atrial contraction is completed, and ventricular contraction begins.



Elapsed time = 225 msec

QRS complex:
completion of ventricular depolarization



20-2 The Conducting System

- Disturbances in heart rhythm
  - **Bradycardia**—abnormally slow heart rate
  - **Tachycardia**—abnormally fast heart rate
  - **Ectopic pacemaker**
    - Abnormal cells generate high rate of action potentials
    - Bypasses conducting system
    - Disrupts timing of ventricular contractions

20-2 The Conducting System

- **Electrocardiogram** (**ECG** or **EKG**)
  - A recording of electrical events in the heart
  - Obtained by placing electrodes at specific locations on body surface
  - Abnormal patterns are used to diagnose damage

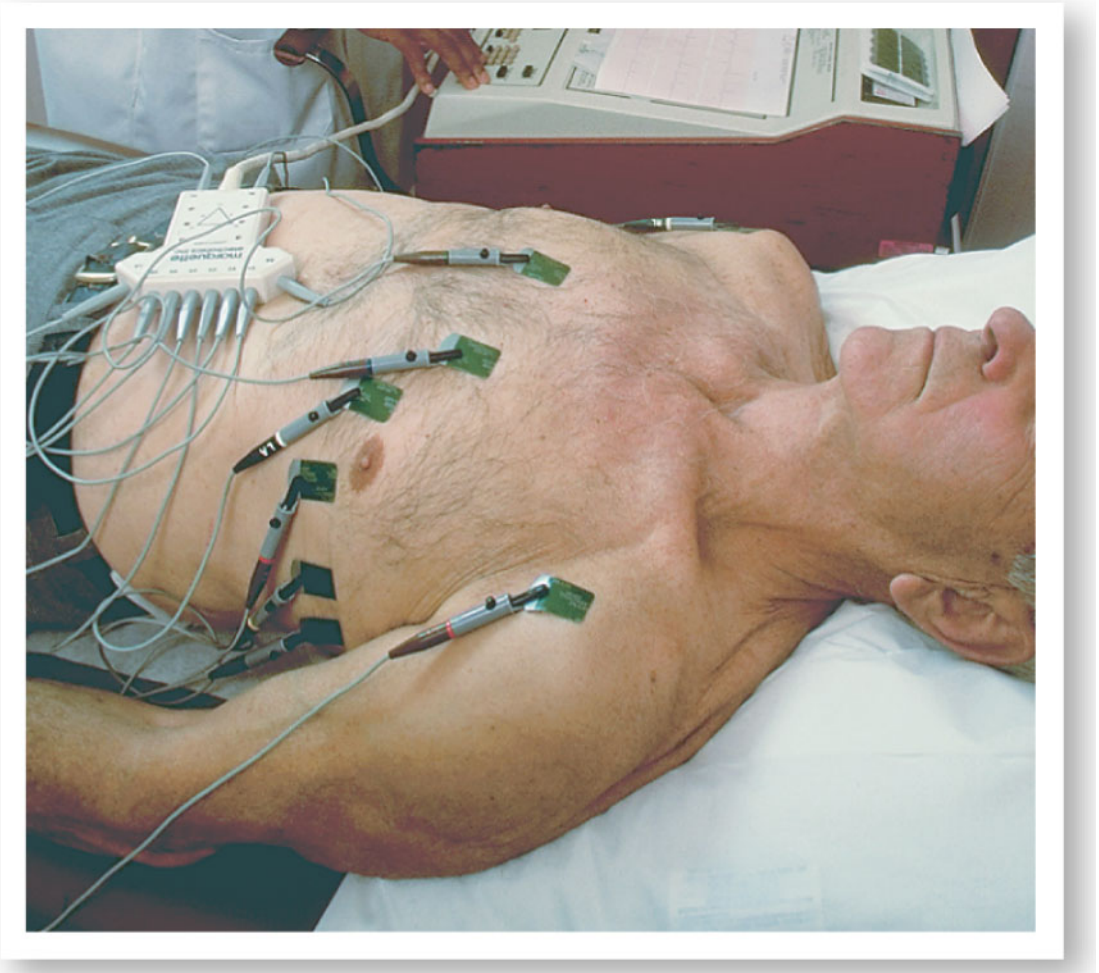
20-2 The Conducting System

- Features of an ECG
  - **P wave**
    - Depolarization of atria
  - **QRS complex**
    - Depolarization of ventricles
    - Ventricles begin contracting shortly after **R wave**
  - **T wave**
    - Repolarization of ventricles

20-2 The Conducting System

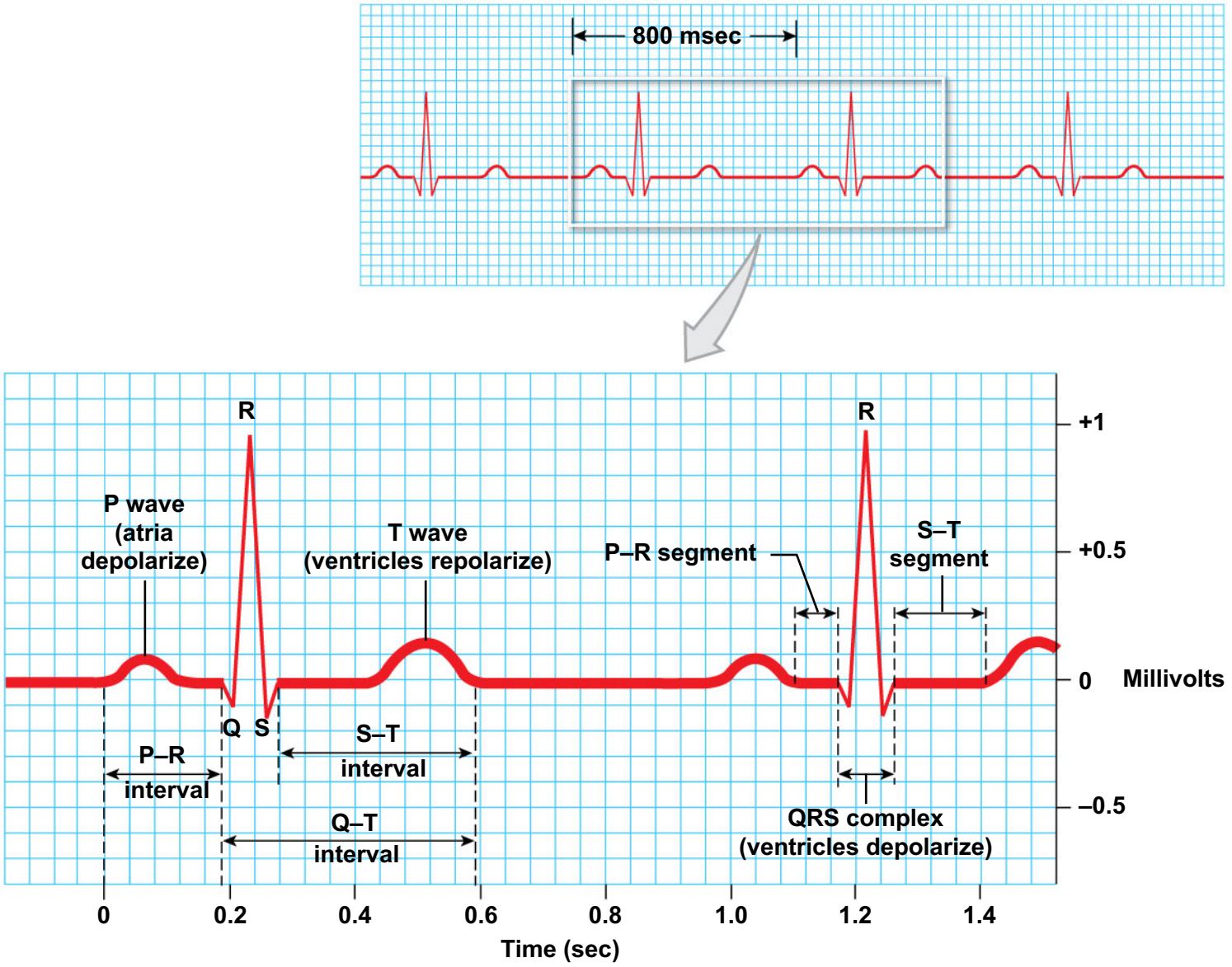
- Time intervals between ECG waves
  - **P–R interval**
    - From start of atrial depolarization
    - To start of QRS complex
  - **Q–T interval**
    - Time required for ventricles to undergo a single cycle of depolarization and repolarization

Figure 20–12a An Electrocardiogram (ECG).



a Electrode placement for recording a standard ECG.

Figure 20–12b An Electrocardiogram (ECG).



b An ECG printout is a strip of graph paper containing a record of the electrical events monitored by the electrodes. The placement of electrodes on the body surface affects the size and shape of the waves recorded. The example is a normal ECG; the enlarged section indicates the major components of the ECG and the measurements most often taken during clinical analysis.

Figure 20–13 Cardiac Arrhythmias (Part 1 of 2).

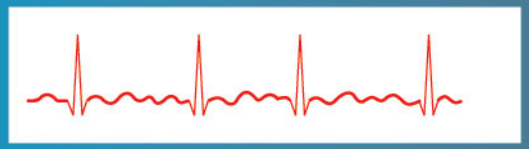
Premature Atrial Contractions (PACs)

Premature atrial contractions (PACs) often occur in healthy individuals. In a PAC, the normal atrial rhythm is momentarily interrupted by a "surprise" atrial contraction. Stress, caffeine, and various drugs may increase the incidence of PACs, presumably by increasing the permeabilities of the SA pacemakers. The impulse spreads along the conduction pathway, and a normal ventricular contraction follows the atrial beat.

Paroxysmal Atrial Tachycardia (PAT)

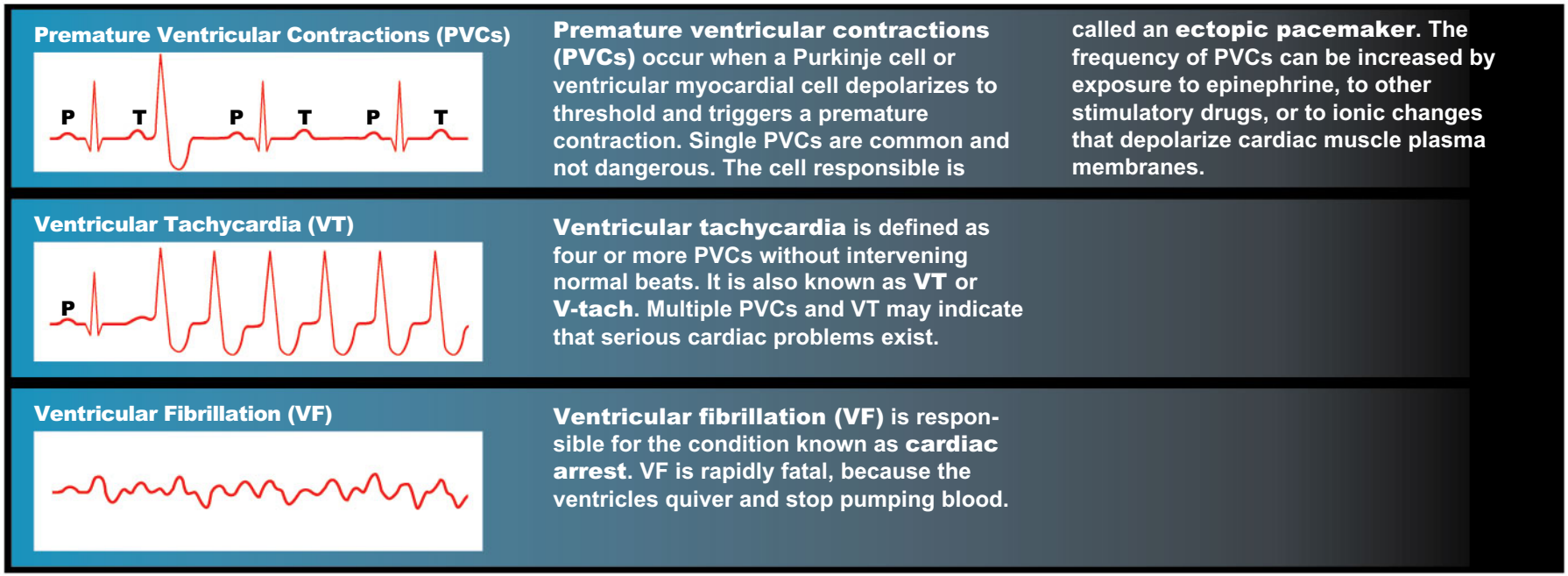
In **paroxysmal** (par-ok-SIZ-mal) **atrial tachycardia**, or **PAT**, a premature atrial contraction triggers a flurry of atrial activity. The ventricles are still able to keep pace, and the heart rate jumps to about 180 beats per minute.

Atrial Fibrillation (AF)



During **atrial fibrillation** (fib-ri-LĀ-shun), the impulses move over the atrial surface at rates of perhaps 500 beats per minute. The atrial wall quivers instead of producing an organized contraction. The ventricular rate cannot follow the atrial rate and may remain within normal limits. Even though the atria are now nonfunctional, their contribution to ventricular end-diastolic volume (the maximum amount of blood the ventricles can hold at the end of atrial contraction) is so small that the condition may go unnoticed in older individuals.

Figure 20–13 Cardiac Arrhythmias (Part 2 of 2).



20-2 The Conducting System

- **Cardiac contractile cells**
  - Form bulk of atrial and ventricular walls
  - Receive stimulus from Purkinje fibers
  - Resting membrane potential
    - Of ventricular cell is about –90 mV
    - Of atrial cell is about –80 mV

20-2 The Conducting System

- **Intercalated discs**
  - Interconnect cardiac contractile cells
  - Membranes of adjacent cells are
    - Held together by desmosomes
    - Linked by gap junctions
  - Transfer force of contraction from cell to cell
  - Propagate action potentials

Figure 20–14a Cardiac Contractile Cells.

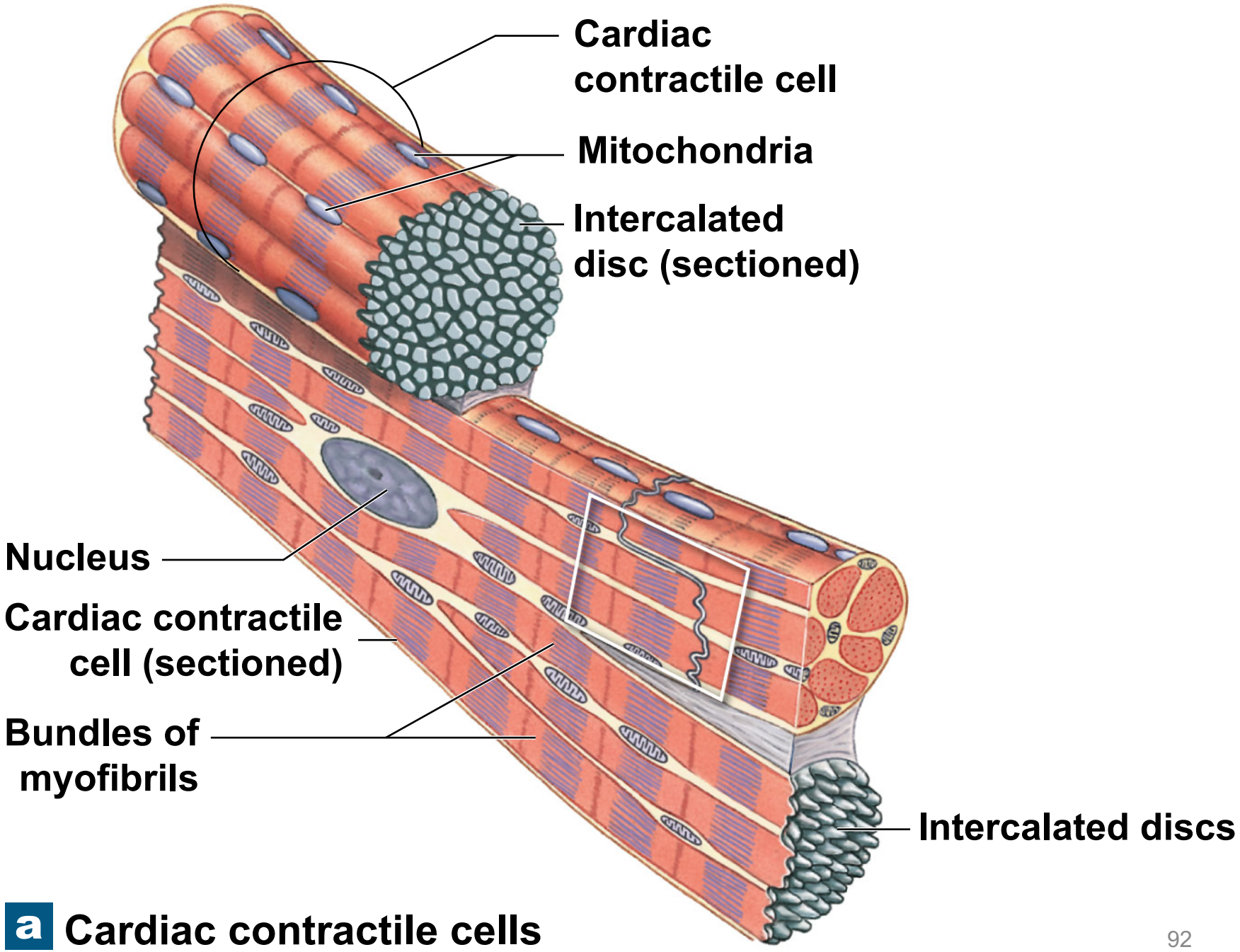


Figure 20–14b Cardiac Contractile Cells.

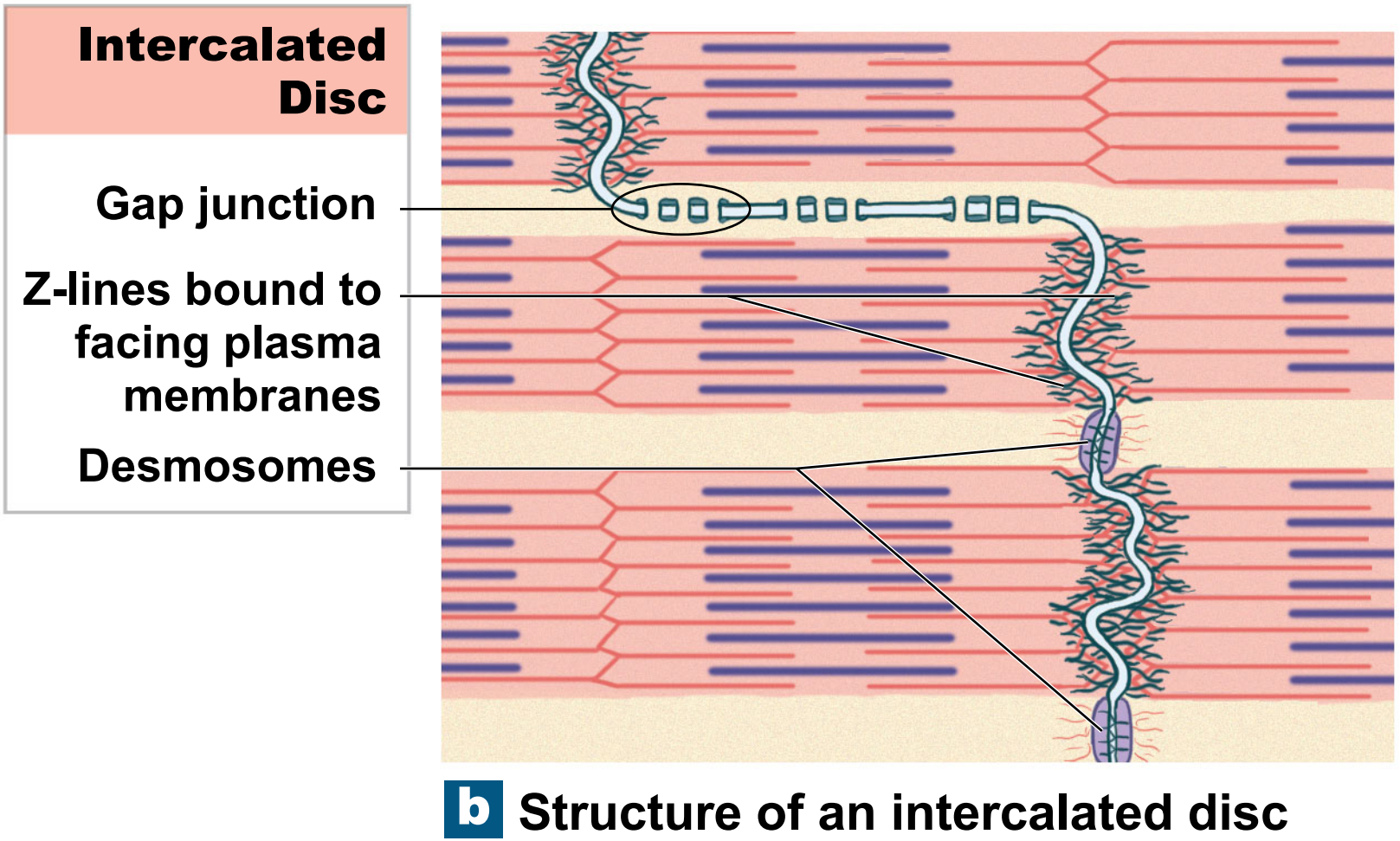
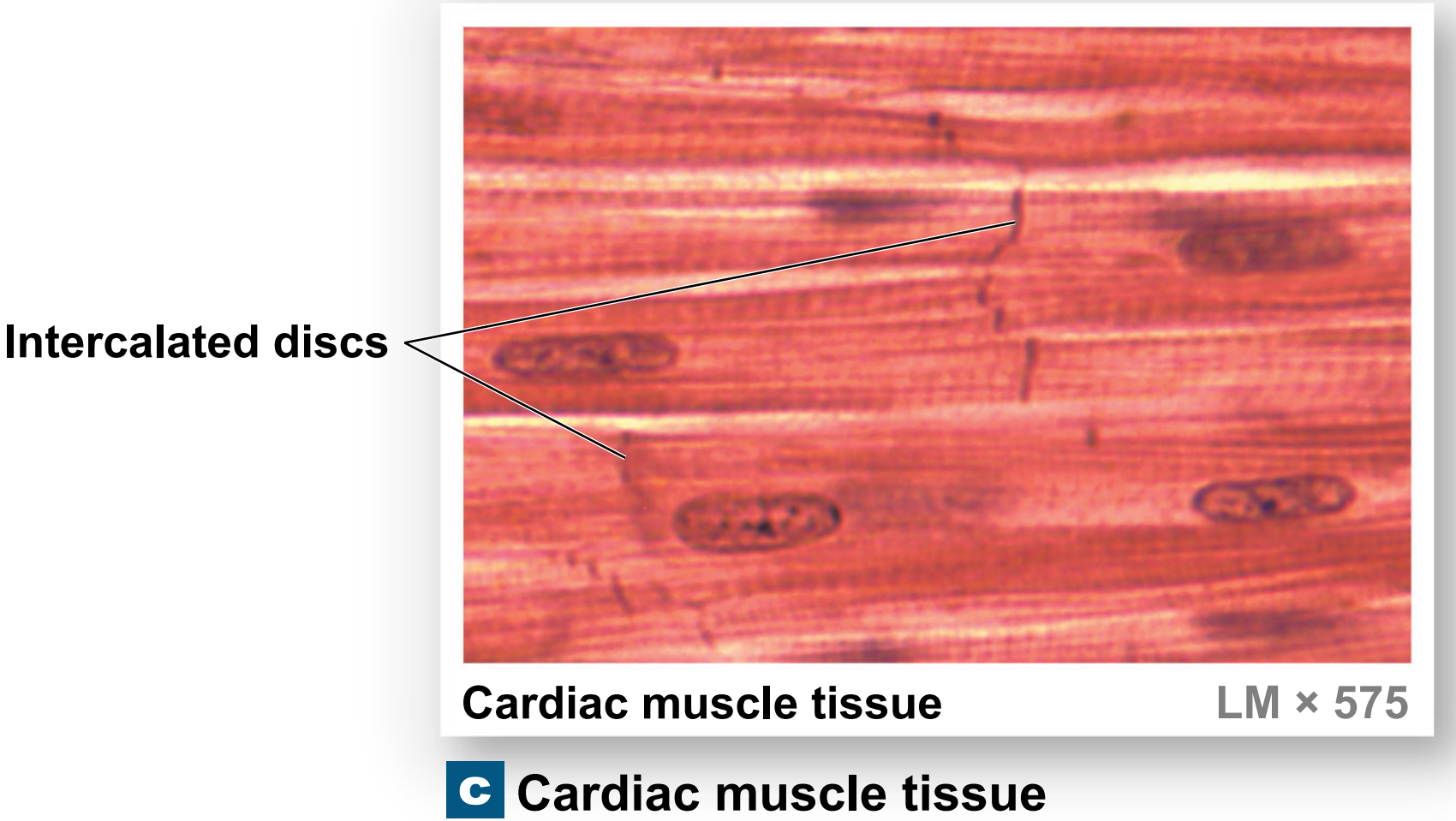


Figure 20–14c Cardiac Contractile Cells.



20-2 The Conducting System

- Characteristics of cardiac contractile cells
  - Small size
  - Single, central nucleus
  - Branching interconnections between cells
  - Intercalated discs

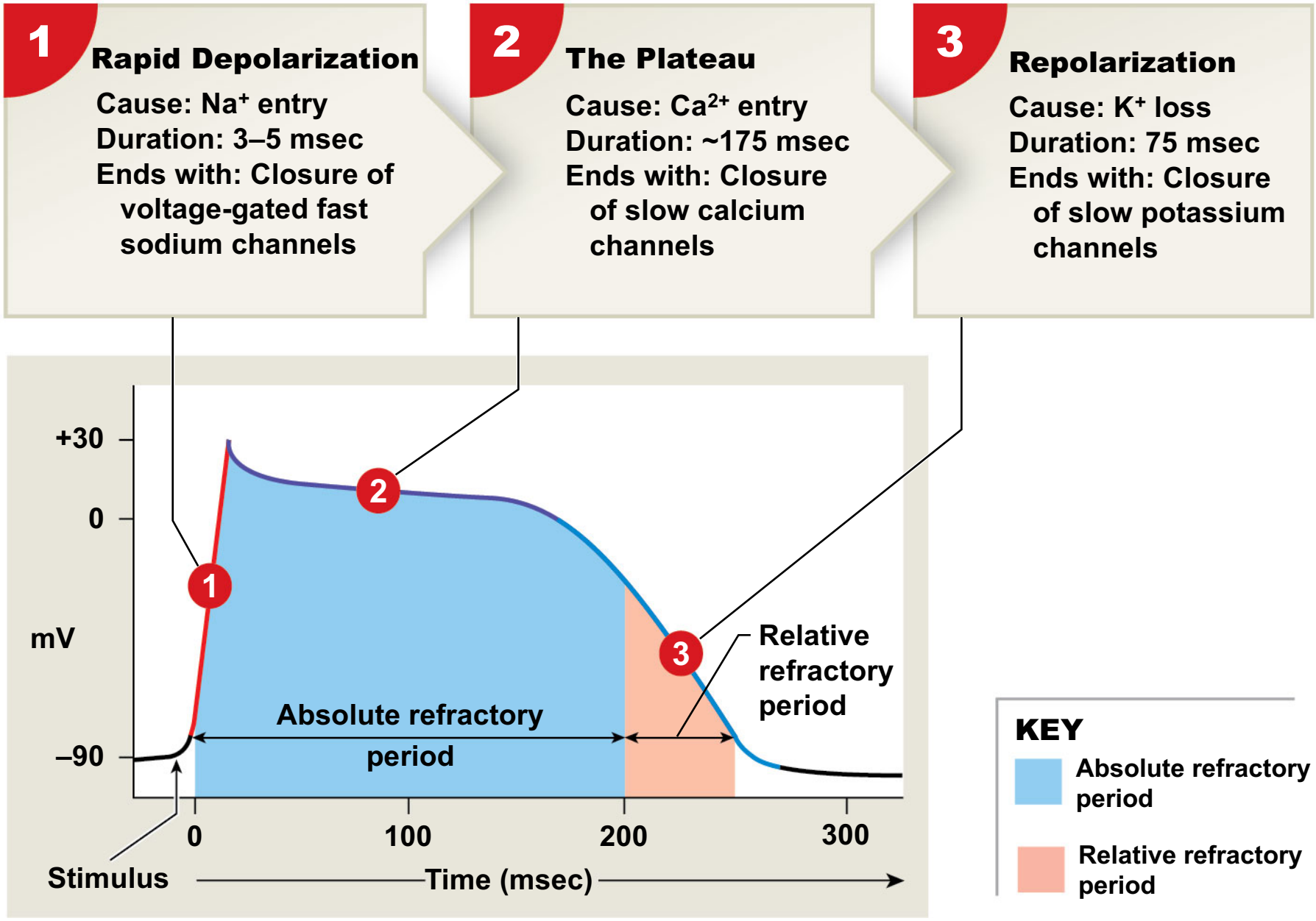
20-2 The Conducting System

- Action potential in cardiac contractile cells
  1. Rapid depolarization
     - Massive influx of Na^+ through **fast sodium channels**
  2. Plateau
     - Extracellular Ca^{2+} enters cytosol through **slow calcium channels**
  3. Repolarization
     - K^+ rushes out of cell through **slow potassium channels**

20-2 The Conducting System

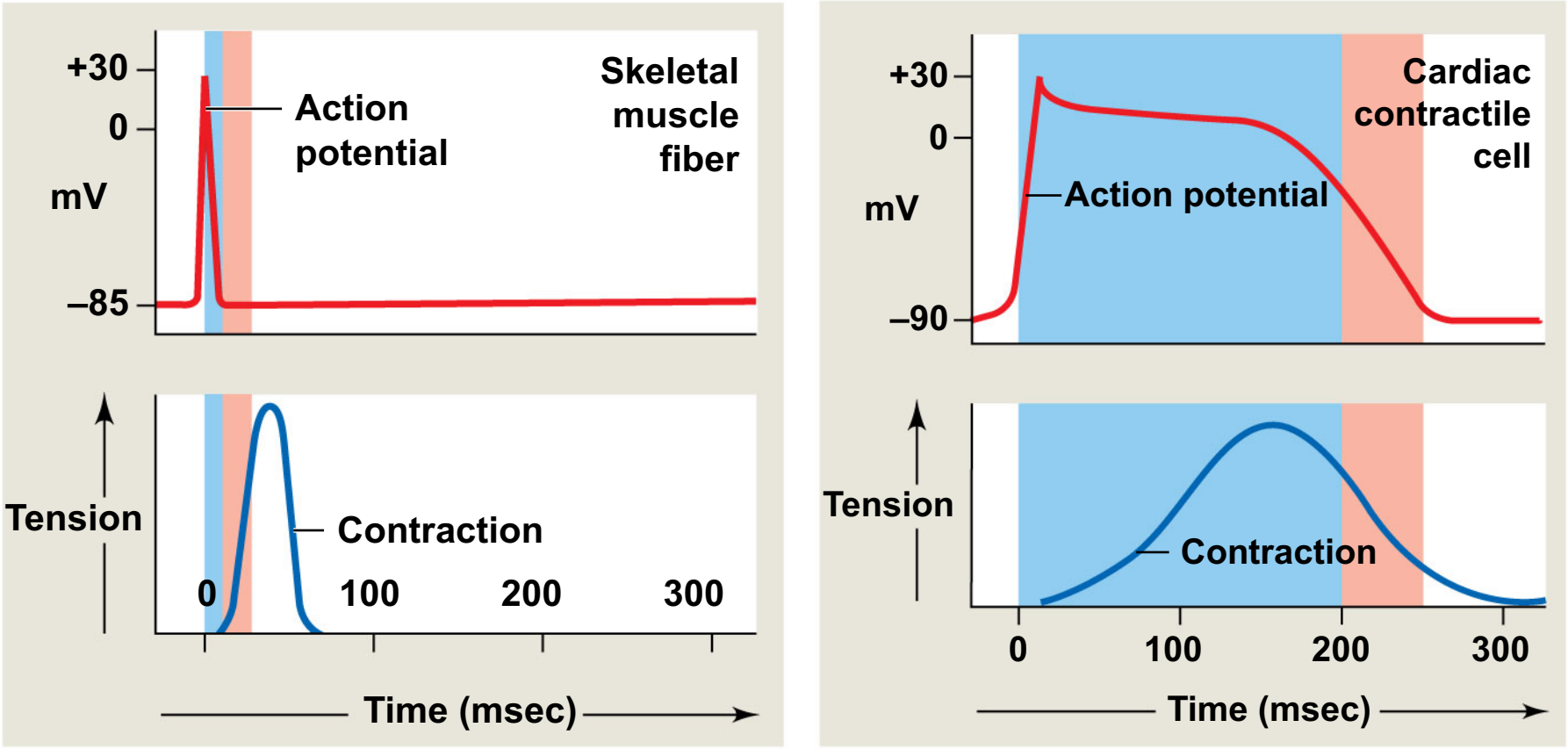
- **Refractory period**
  - Absolute refractory period (200 msec)
    - Cardiac contractile cells cannot respond
  - Relative refractory period (50 msec)
    - Cells respond only to strong stimuli
- Action potential in a ventricular contractile cell
  - 250–300 msec
  - 30 times longer than that in skeletal muscle fiber
  - Prevents summation and tetany

Figure 20–15a Action Potentials in Cardiac Contractile Cells and Skeletal Muscle Fibers.



a Events in an action potential in a ventricular contractile cell.

Figure 20–15b Action Potentials in Cardiac Contractile Cells and Skeletal Muscle Fibers.



b Action potentials and twitch contractions in a skeletal muscle fiber (above) and cardiac contractile cell (below). The shaded areas indicate the durations of the absolute (blue) and relative (beige) refractory periods.

KEY

- Absolute refractory period
- Relative refractory period

20-2 The Conducting System

- Role of calcium ions in cardiac contractions
  1. Extracellular Ca^{2+} crosses plasma membrane during plateau phase
     - Provides roughly 20 percent of Ca^{2+} required for contraction
  2. Entry of extracellular Ca^{2+} triggers release of additional Ca^{2+} from sarcoplasmic reticulum (SR)

20-2 The Conducting System

- Role of calcium ions in cardiac contractions
  - Cardiac muscle tissue
    - Very sensitive to extracellular Ca^{2+} concentrations
  - As slow calcium channels close,
    - Intracellular Ca^{2+} is pumped back into SR or out of cell

20-2 The Conducting System

- Energy for cardiac contractions
  - Aerobic energy
    - From mitochondrial breakdown of fatty acids and glucose
    - Oxygen is delivered by circulation
    - Cardiac contractile cells store oxygen in *myoglobin*

20-3 The Cardiac Cycle

- **Cardiac cycle**
  - From start of one heartbeat to beginning of next
  - Includes alternating periods of contraction and relaxation
- Phases of the cardiac cycle within each chamber
  - **Systole** (contraction)
  - **Diastole** (relaxation)

Figure 20–16 Phases of the Cardiac Cycle.

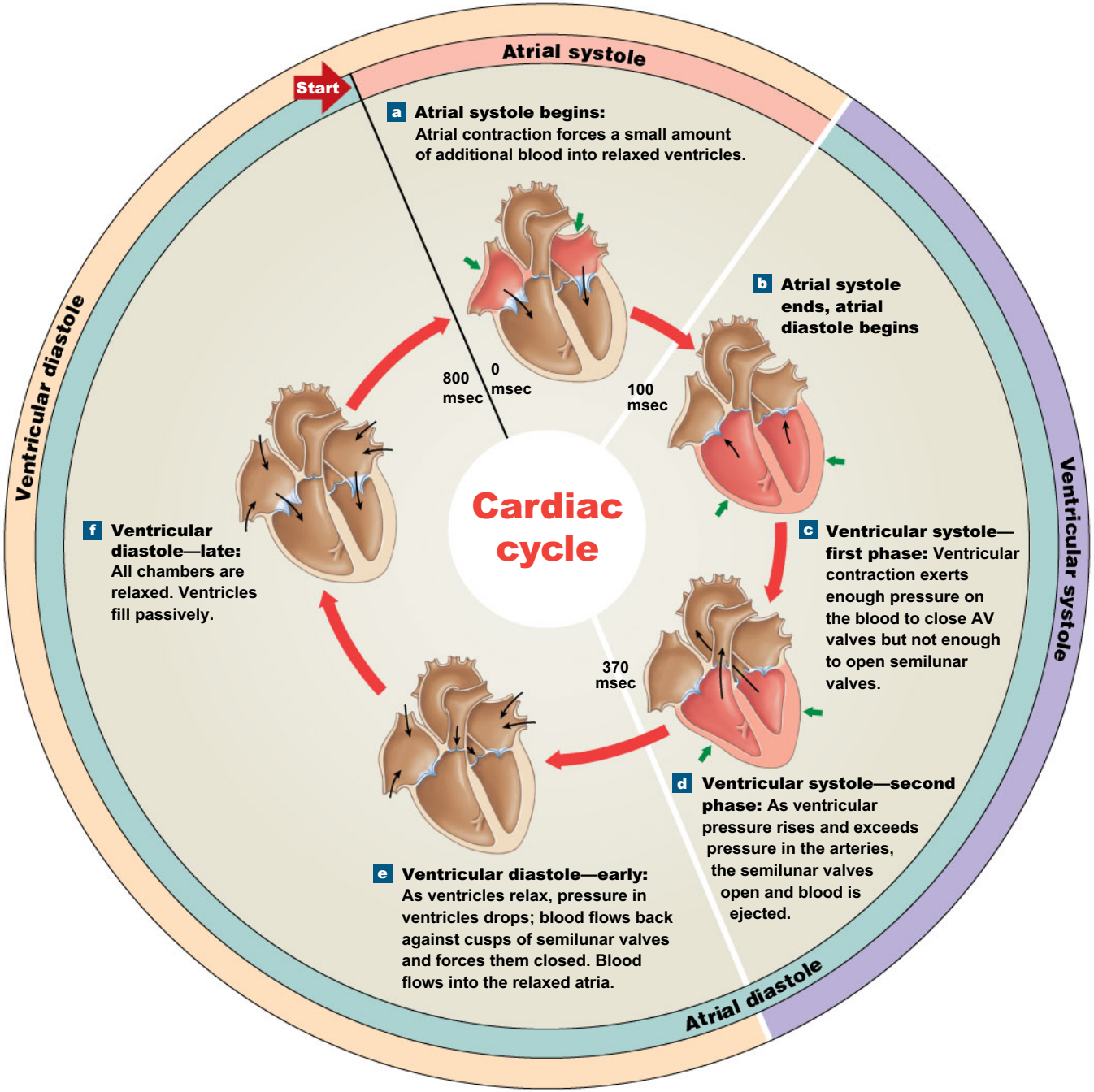


Figure 20–16a Phases of the Cardiac Cycle.

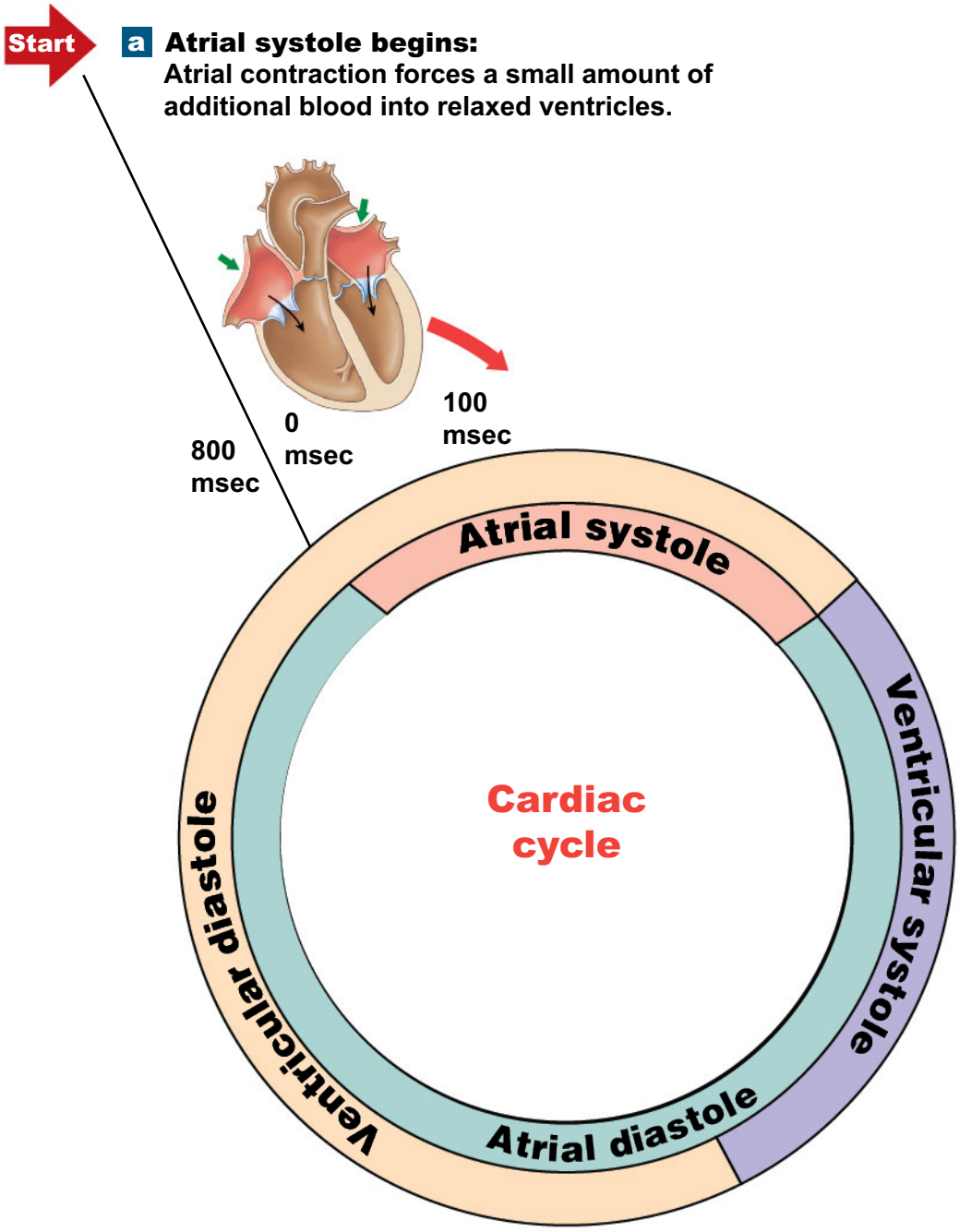


Figure 20–16b Phases of the Cardiac Cycle.

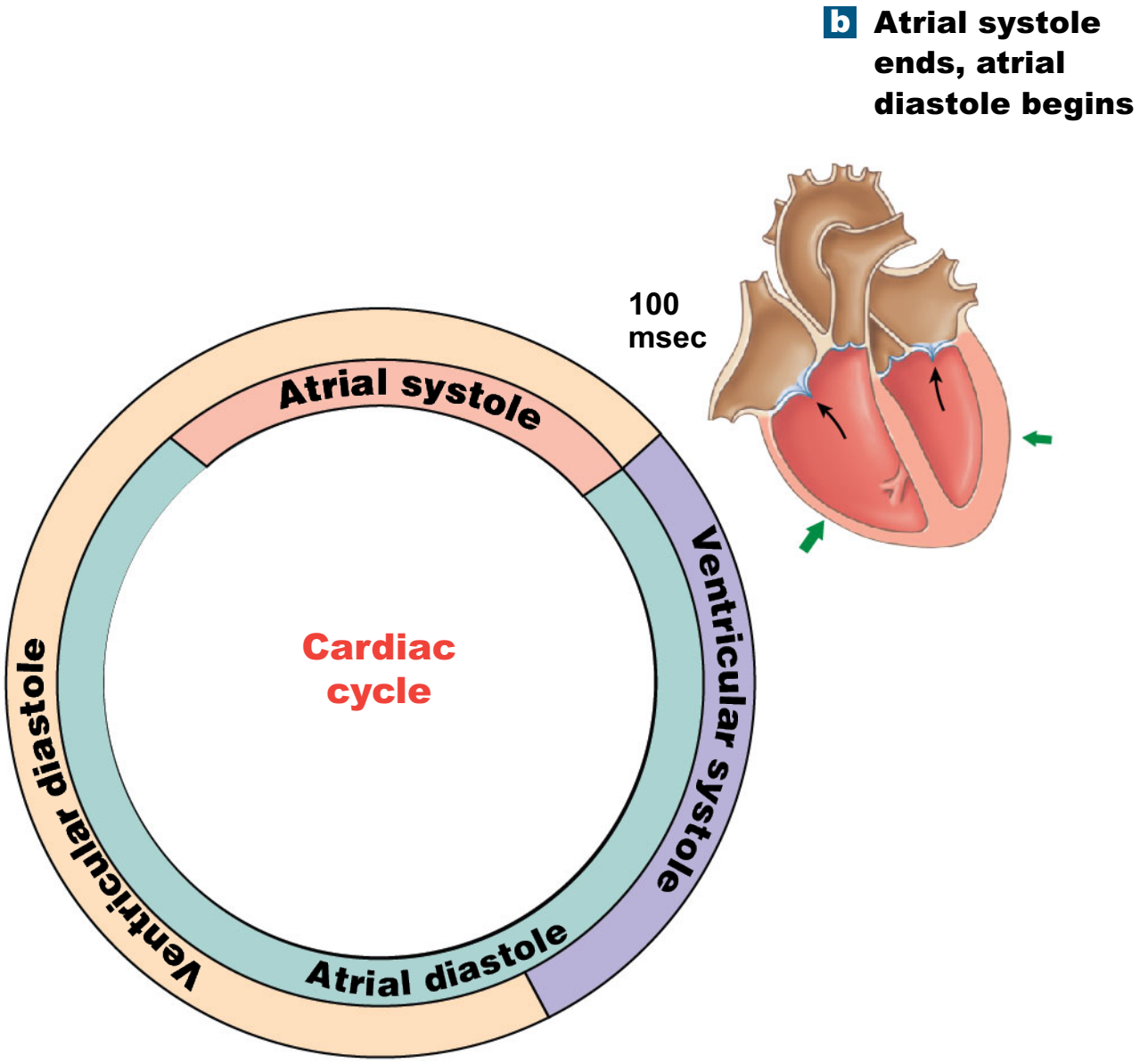
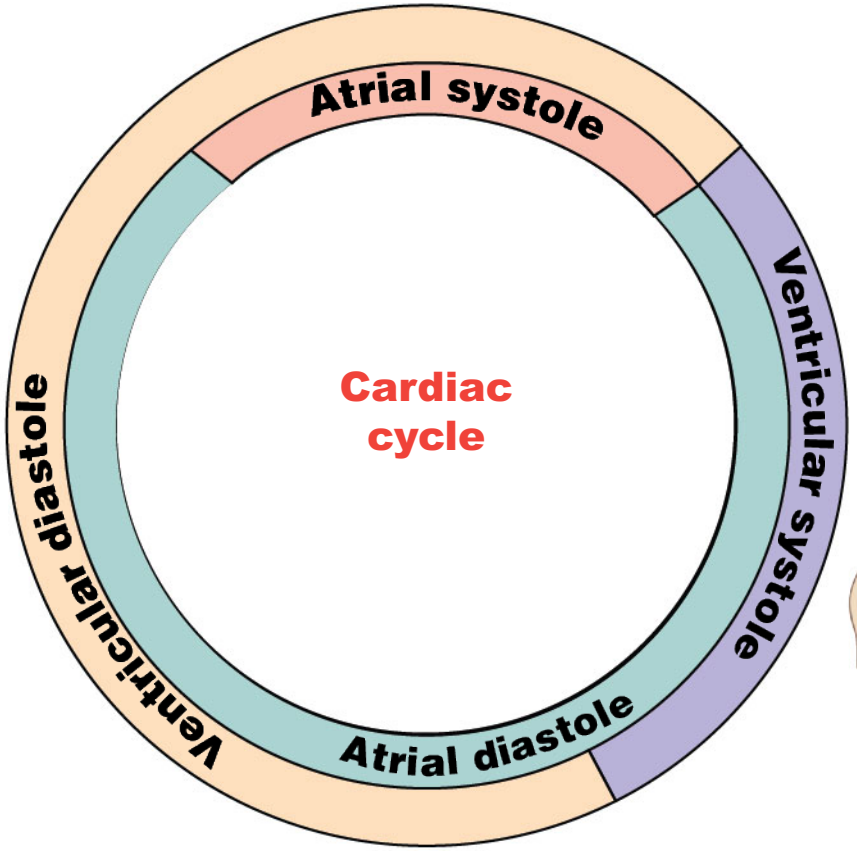
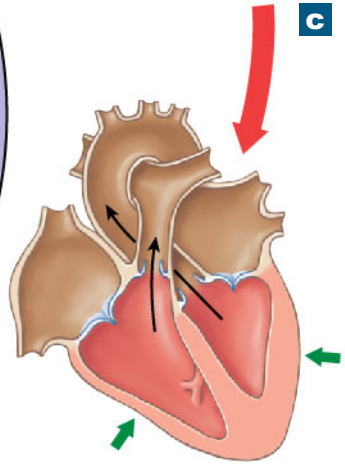


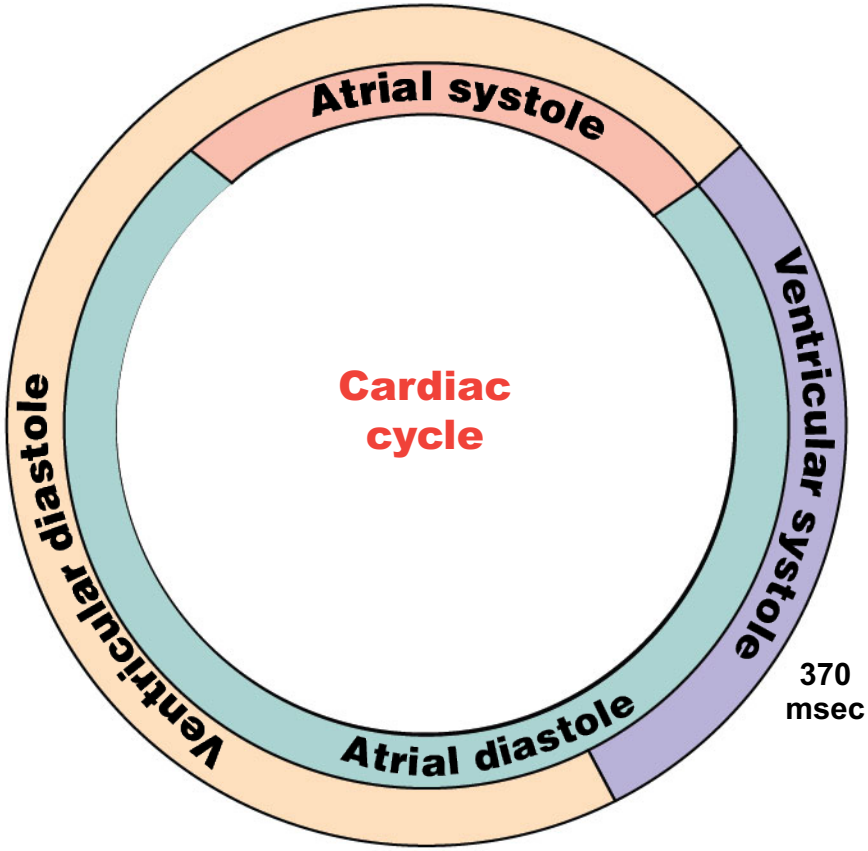
Figure 20–16c Phases of the Cardiac Cycle.



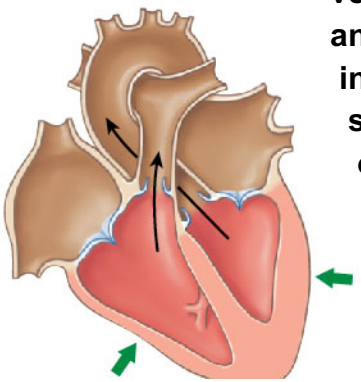


c **Ventricular systole—first phase:** Ventricular contraction exerts enough pressure on the blood to close AV valves but not enough to open semilunar valves.

Figure 20–16d Phases of the Cardiac Cycle.

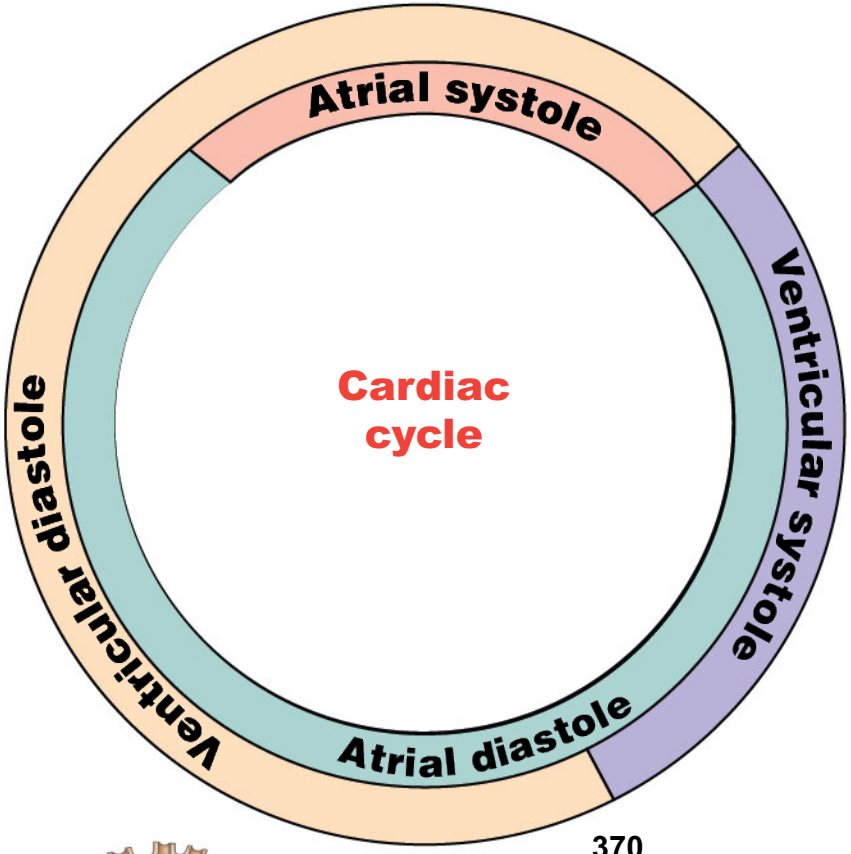


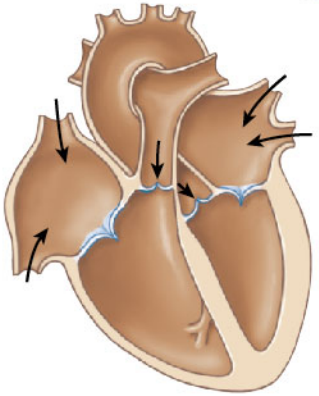
370 msec



d **Ventricular systole—second phase:** As ventricular pressure rises and exceeds pressure in the arteries, the semilunar valves open and blood is ejected.

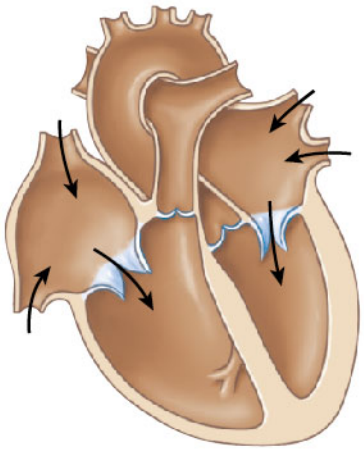
Figure 20–16e Phases of the Cardiac Cycle.

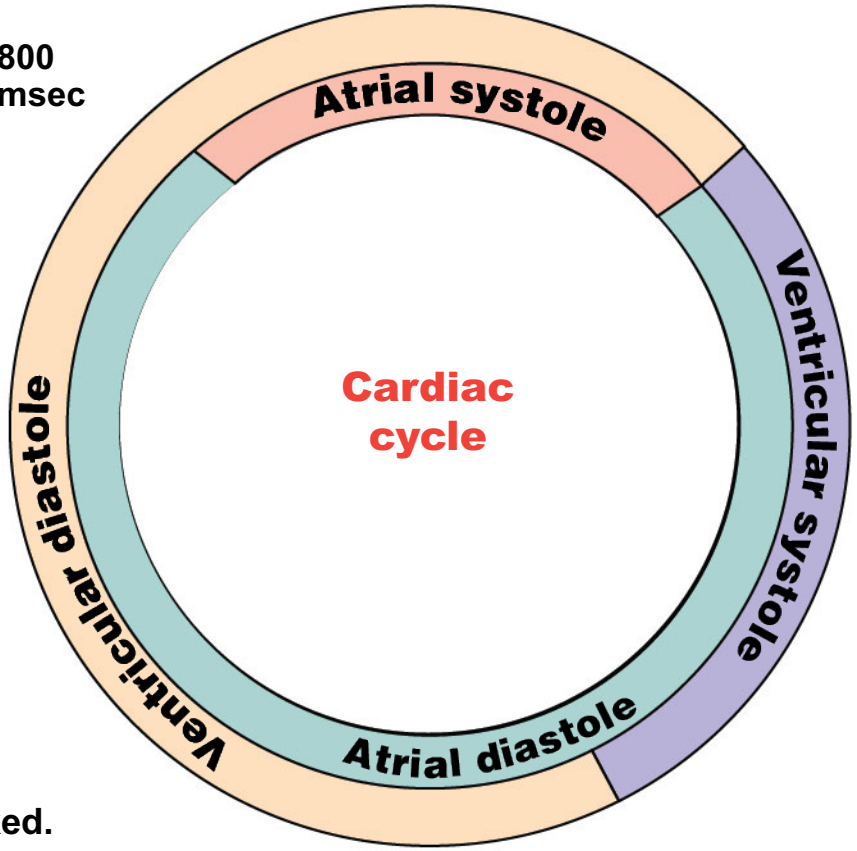




e **Ventricular diastole—early:** As ventricles relax, pressure in ventricles drops; blood flows back against cusps of semilunar valves and forces them closed. Blood flows into the relaxed atria.

Figure 20–16f Phases of the Cardiac Cycle.





f **Ventricular diastole—late:**

All chambers are relaxed.
Ventricles fill passively.

Smart Video: *The Cardiac Cycle*

20-3 The Cardiac Cycle

- Blood pressure in each chamber
  - Rises during systole
  - Falls during diastole
- Blood flows from an area of higher pressure to one of lower pressure
  - Controlled by timing of contractions
  - Directed by one-way valves

20-3 The Cardiac Cycle

- Cardiac cycle and heart rate
  - At 75 beats per minute (bpm),
    - Cardiac cycle lasts about 800 msec
  - When heart rate increases,
    - All phases of cardiac cycle shorten, particularly diastole

20-3 The Cardiac Cycle

- Phases of the cardiac cycle
  - Atrial systole
  - Atrial diastole
  - Ventricular systole
  - Ventricular diastole

20-3 The Cardiac Cycle

- **Atrial systole**
  1. Atrial contraction begins
     - Right and left AV valves are open
  2. Atria eject blood into ventricles

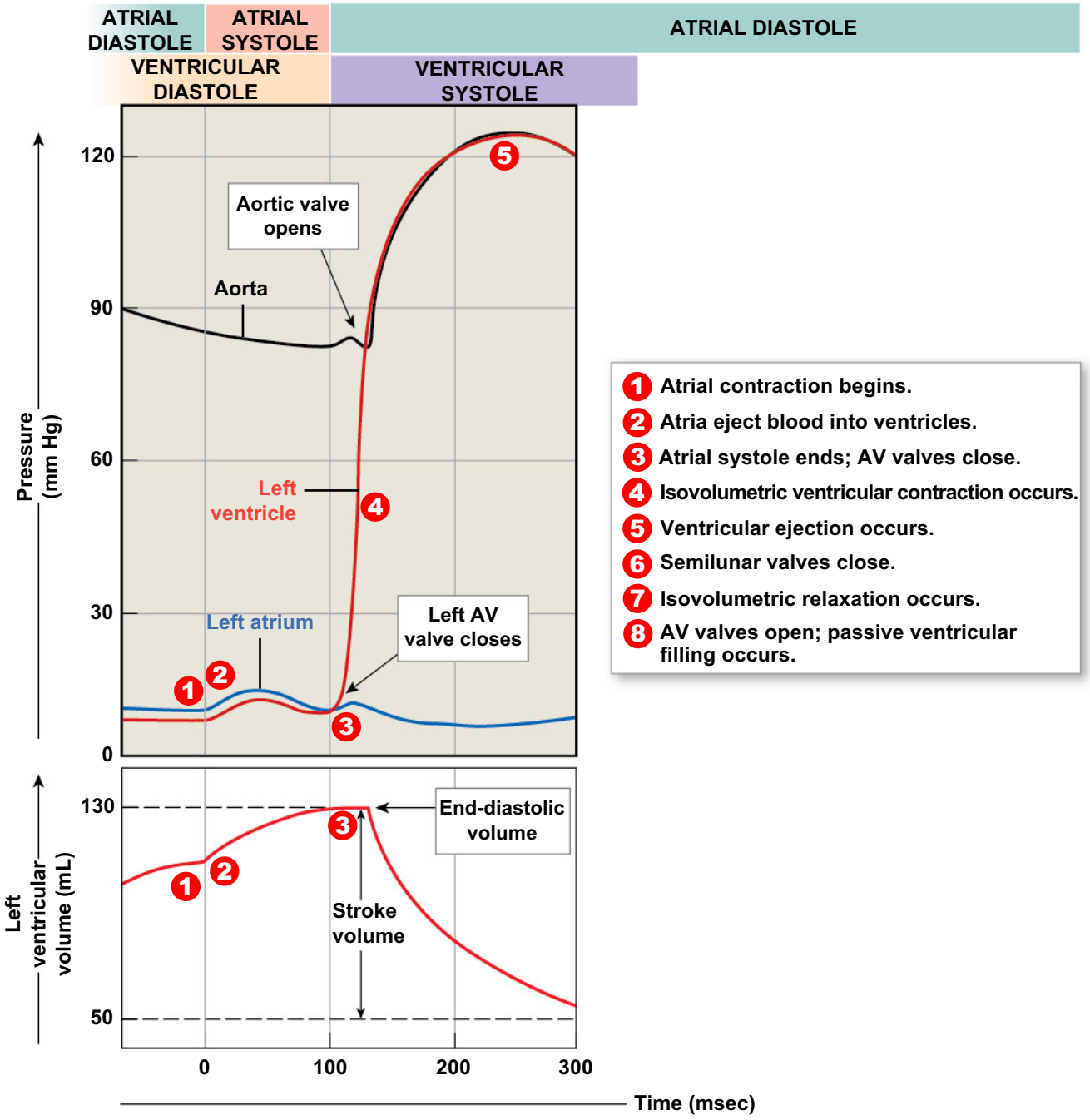
20-3 The Cardiac Cycle

- **Ventricular systole** and **atrial diastole**
  3. Atrial systole ends
     - Atrial diastole begins
     - Ventricles contain maximum blood volume
       - Known as *end-diastolic volume* (*EDV*)
  4. Ventricles contract and build pressure
     - Closing AV valves
     - Producing **isovolumetric contraction**

20-3 The Cardiac Cycle

- Ventricular systole
  5. **Ventricular ejection**
     - Ventricular pressure exceeds arterial pressure
     - Opens semilunar valves, allowing blood to exit
     - Amount of blood ejected = stroke volume (SV)
  6. Semilunar valves close
     - As ventricular pressure falls
     - Ventricles contain end-systolic volume (ESV)
       - About 40 percent of end-diastolic volume

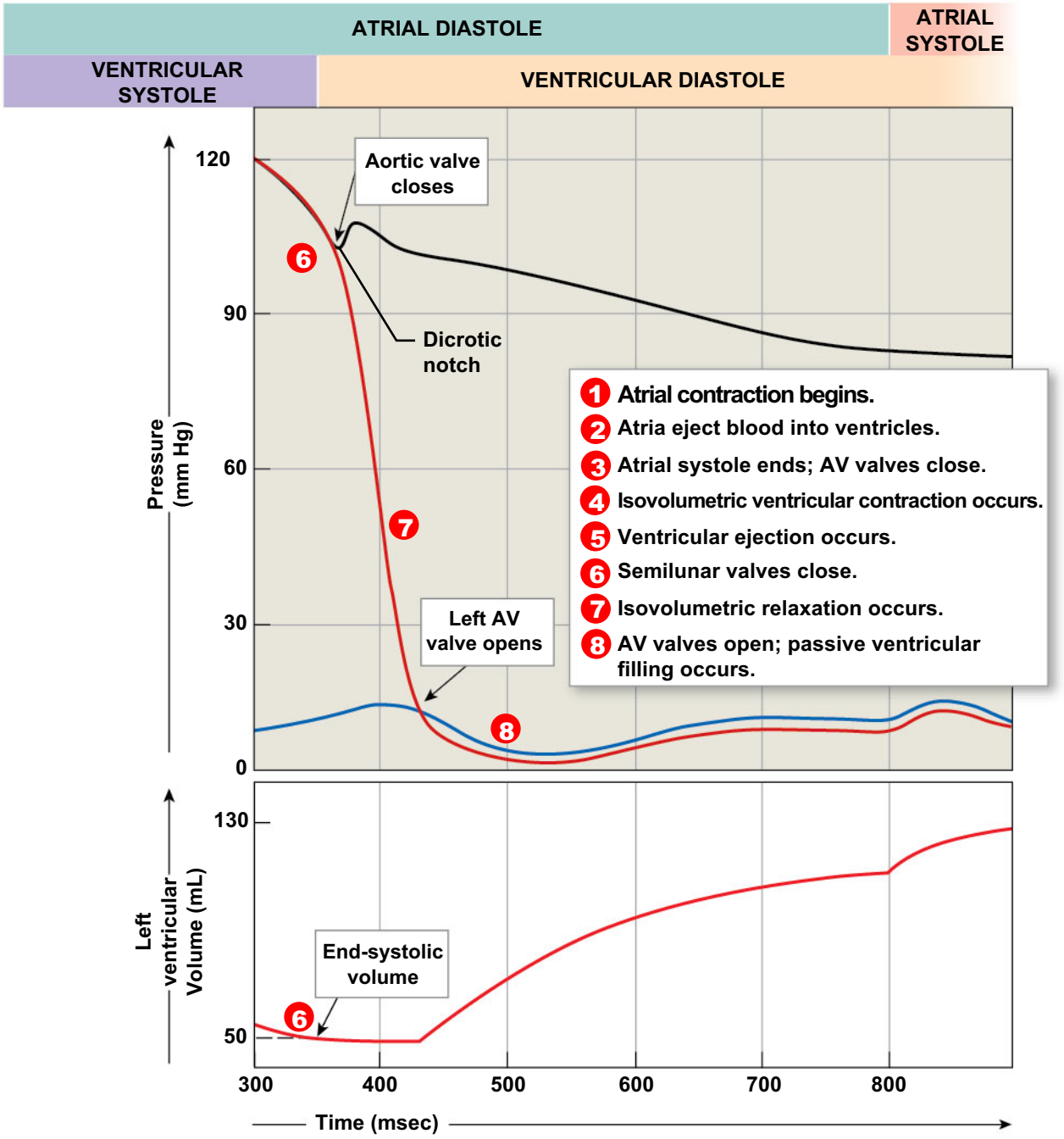
Figure 20–17 Pressure and Volume Relationships in the Cardiac Cycle (Part 3 of 4).



20-3 The Cardiac Cycle

- **Ventricular diastole**
  7. **Isovolumetric relaxation**
     - All heart valves are closed
     - Ventricular pressure is higher than atrial pressure
       - Blood cannot flow into ventricles
  8. AV valves open; ventricles fill passively
     - Atrial pressure is higher than ventricular pressure
- Individuals can survive severe atrial damage
  - Ventricular damage can lead to **heart failure**

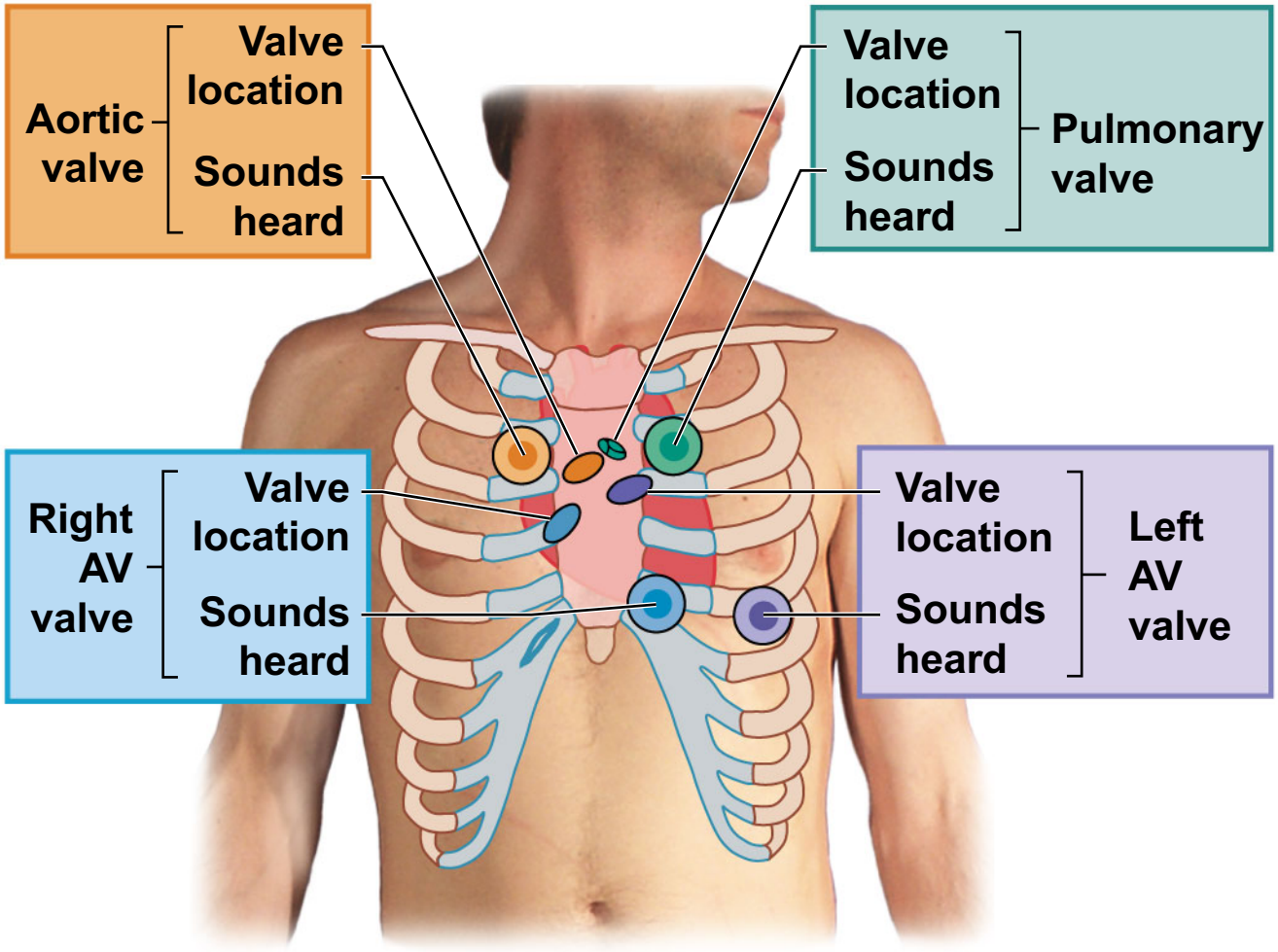
Figure 20–17 Pressure and Volume Relationships in the Cardiac Cycle (Part 4 of 4).



20-3 The Cardiac Cycle

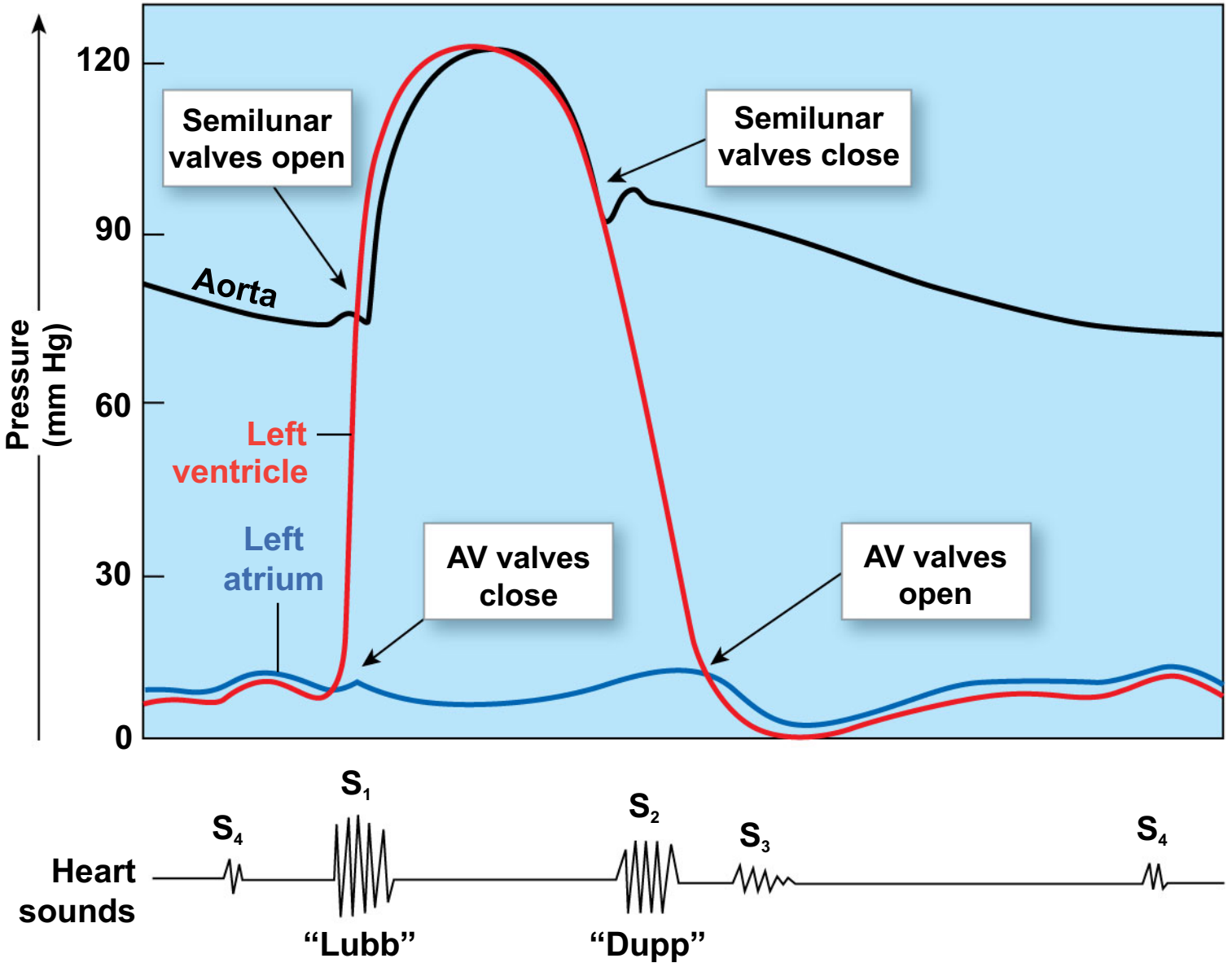
- Heart sounds
  - Detected with a **stethoscope**
  - S_1—Loud sound as AV valves close
  - S_2_Loud sound as semilunar valves close
  - S_3, S_4—Soft sounds
    - Blood flowing into ventricles and atrial contraction
  - **Heart murmur**
    - Sounds produced by regurgitation through valves

Figure 20–18a Heart Sounds.



a Placements of a stethoscope for listening to the different sounds produced by individual valves

Figure 20–18b Heart Sounds.

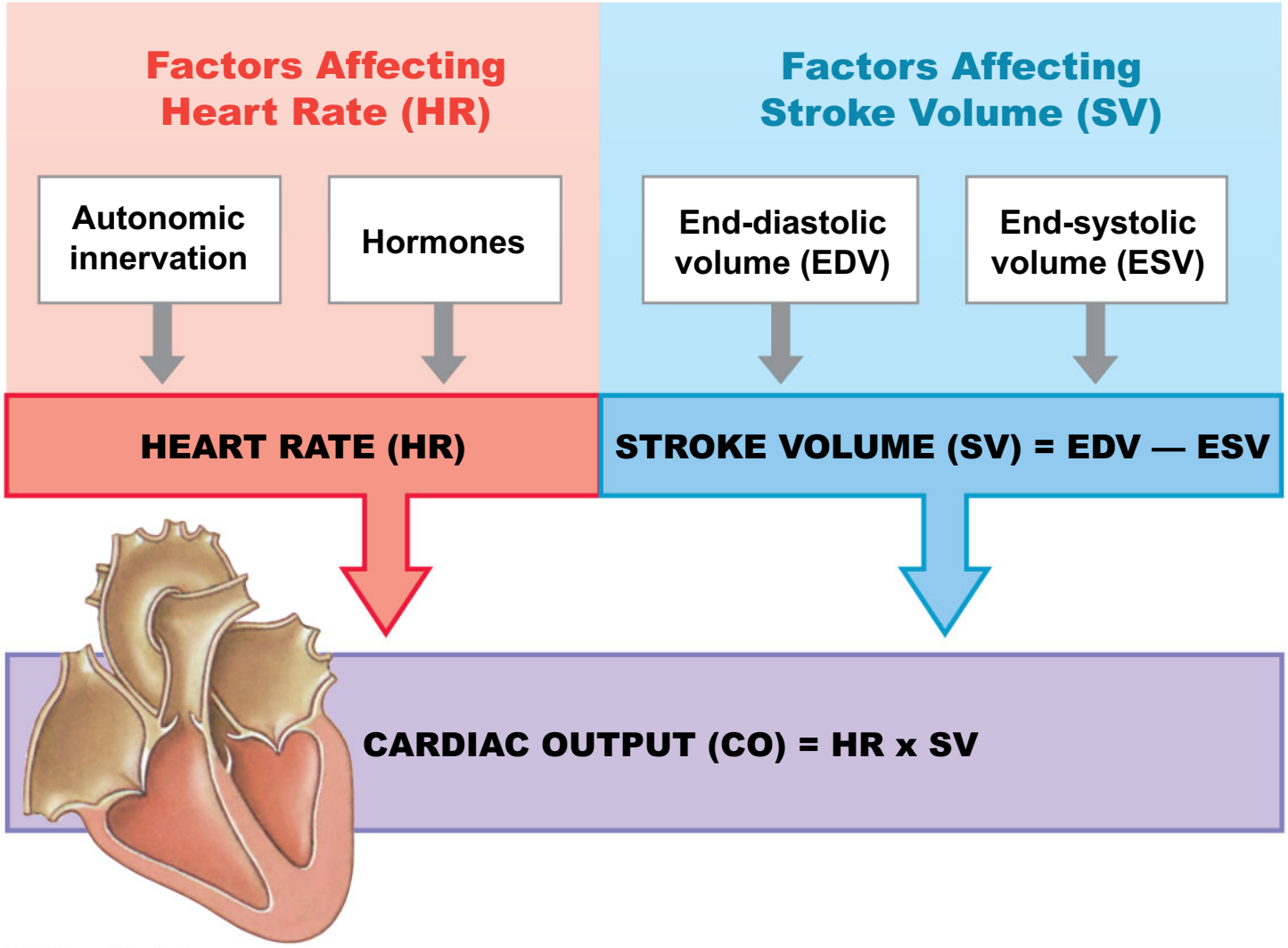


b The relationship between heart sounds and key events in the cardiac cycle

20-4 Cardiac Output

- **Cardiac output** (**CO**)
  - Volume pumped by left ventricle in one minute
  - CO = HR × SV
    - CO = cardiac output (mL/min)
    - **HR** = **heart rate** (beats/min)
    - **SV** = **stroke volume** (mL/beat)

Figure 20–19 Factors Affecting Cardiac Output.



Smart Video: *The Conducting System of the Heart*

20-4 Cardiac Output

- Stroke volume (SV)
  - SV = EDV—ESV
  - **End-diastolic volume** (EDV)
    - Amount of blood in each ventricle at end of ventricular diastole
  - **End-systolic volume** (ESV)
    - Amount of blood remaining in each ventricle at end of ventricular systole
  - Ejection fraction
    - Percentage of EDV ejected during contraction

Figure 20–20 A Simple Model of Stroke Volume (Part 1 of 4).

Start

When the pump handle is raised, pressure within the cylinder decreases, and water enters through a one-way valve. This corresponds to passive filling during ventricular diastole.

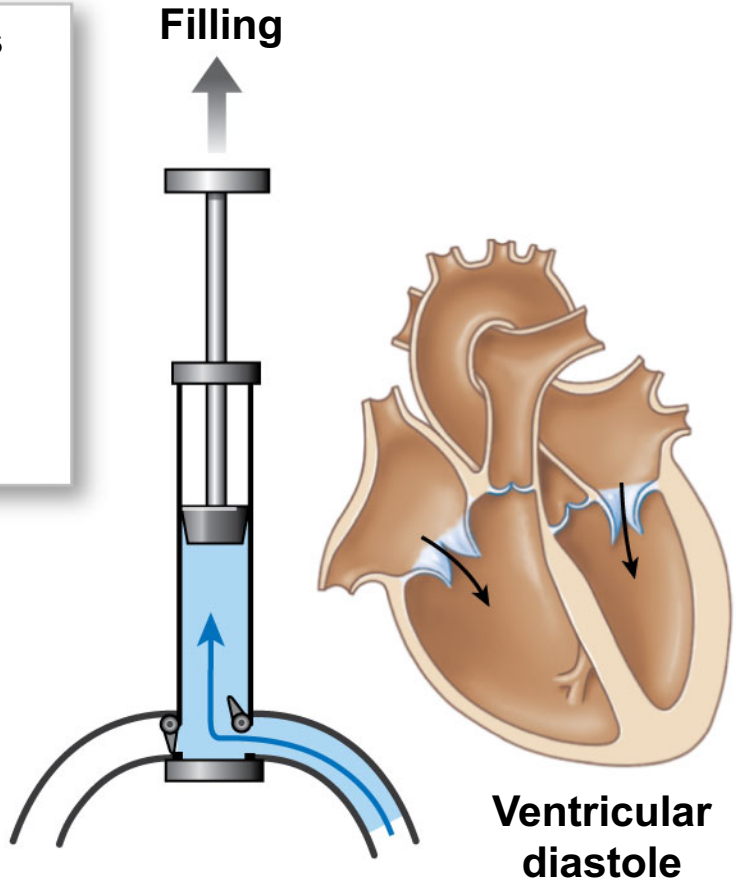
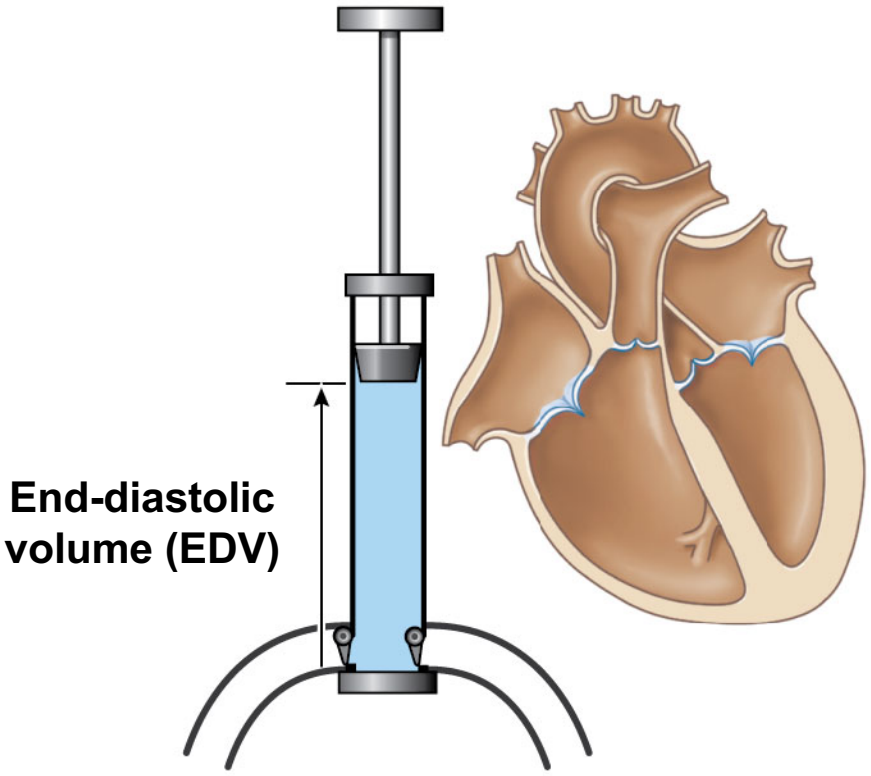
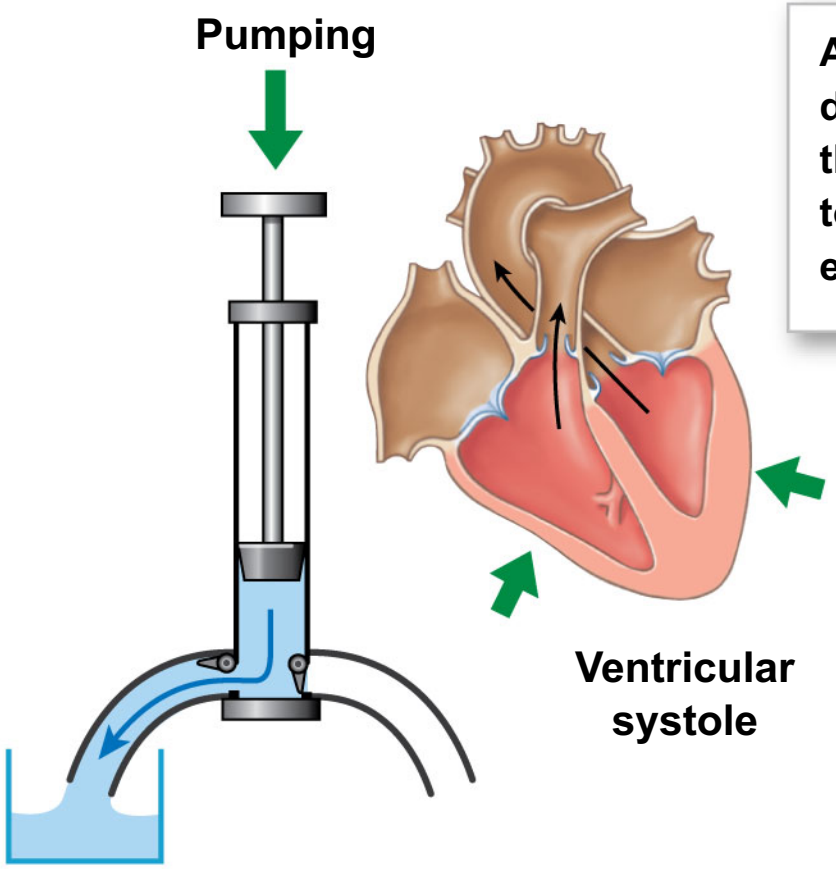


Figure 20–20 A Simple Model of Stroke Volume (Part 2 of 4).



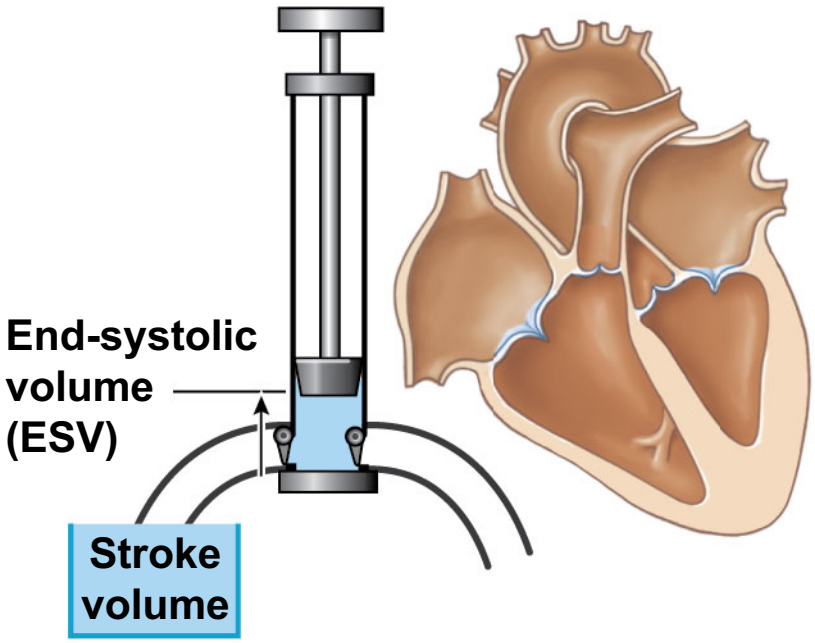
At the start of the pumping cycle, the amount of water in the cylinder corresponds to the amount of blood in a ventricle at the end of ventricular diastole. This amount is known as the **end-diastolic volume (EDV).**

Figure 20–20 A Simple Model of Stroke Volume (Part 3 of 4).



As the pump handle is pushed down, water is forced out of the cylinder. This corresponds to the period of ventricular ejection.

Figure 20–20 A Simple Model of Stroke Volume (Part 4 of 4).



When the handle is depressed as far as it will go, some water will remain in the cylinder. That amount corresponds to the **end-systolic volume (ESV)** remaining in the ventricle at the end of ventricular systole. The amount of water pumped out corresponds to the stroke volume of the heart; the **stroke volume** is the difference between the EDV and the ESV.

20-4 Cardiac Output

- Factors affecting heart rate
  - Autonomic activity
  - Circulating hormones

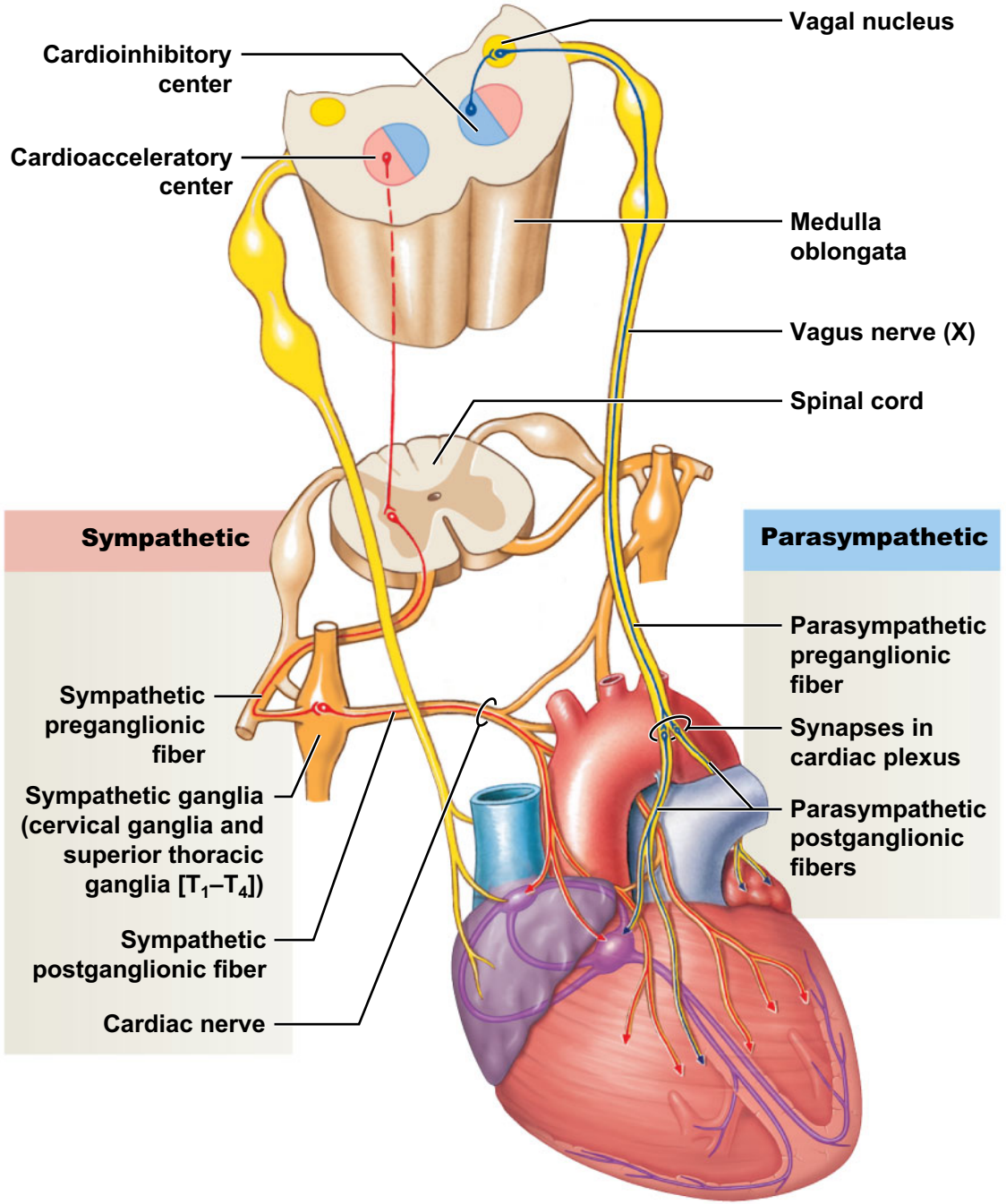
20-4 Cardiac Output

- Autonomic innervation
  - Cardiac plexus innervates heart
  - Vagus nerves (CN X) carry parasympathetic fibers to small ganglia in cardiac plexus
  - Cardiac centers of medulla oblongata
    - **Cardioacceleratory center** controls sympathetic neurons that increase heart rate
    - **Cardioinhibitory center** controls parasympathetic neurons that slow heart rate

20-4 Cardiac Output

- Cardiac reflexes
  - Cardiac centers
    - Monitor blood pressure (baroreceptors)
    - Monitor arterial oxygen and carbon dioxide levels (chemoreceptors)
    - Adjust cardiac activity
- Autonomic tone
  - Maintained by dual innervation and release of ACh and NE
  - Fine adjustments meet needs of other systems

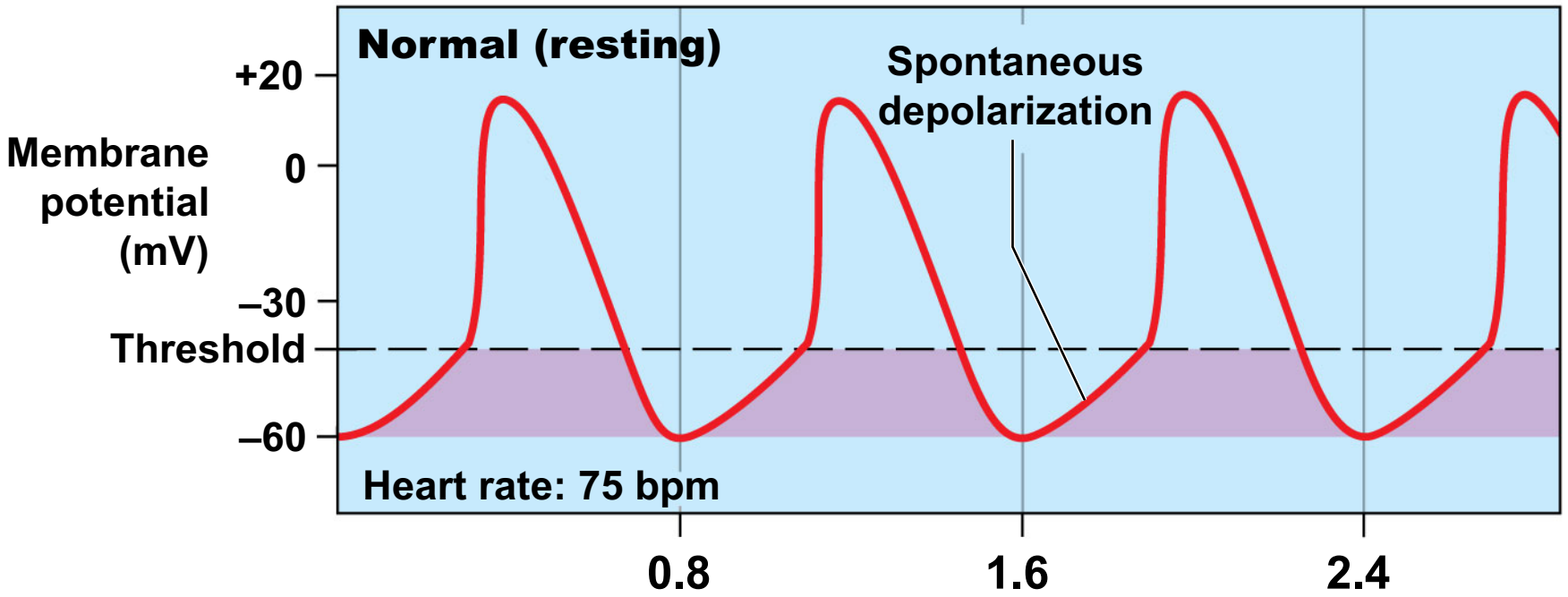
Figure 20–21 Autonomic Innervation of the Heart.



20-4 Cardiac Output

- Effects on pacemaker cells of SA node
  - Membrane potentials of pacemaker cells
    - Are closer to threshold than those of cardiac contractile cells
  - Any factor that changes the rate of spontaneous depolarization or the duration of repolarization
    - Will alter heart rate
    - By changing time required to reach threshold

Figure 20–22a Autonomic Regulation of Pacemaker Cell Function.

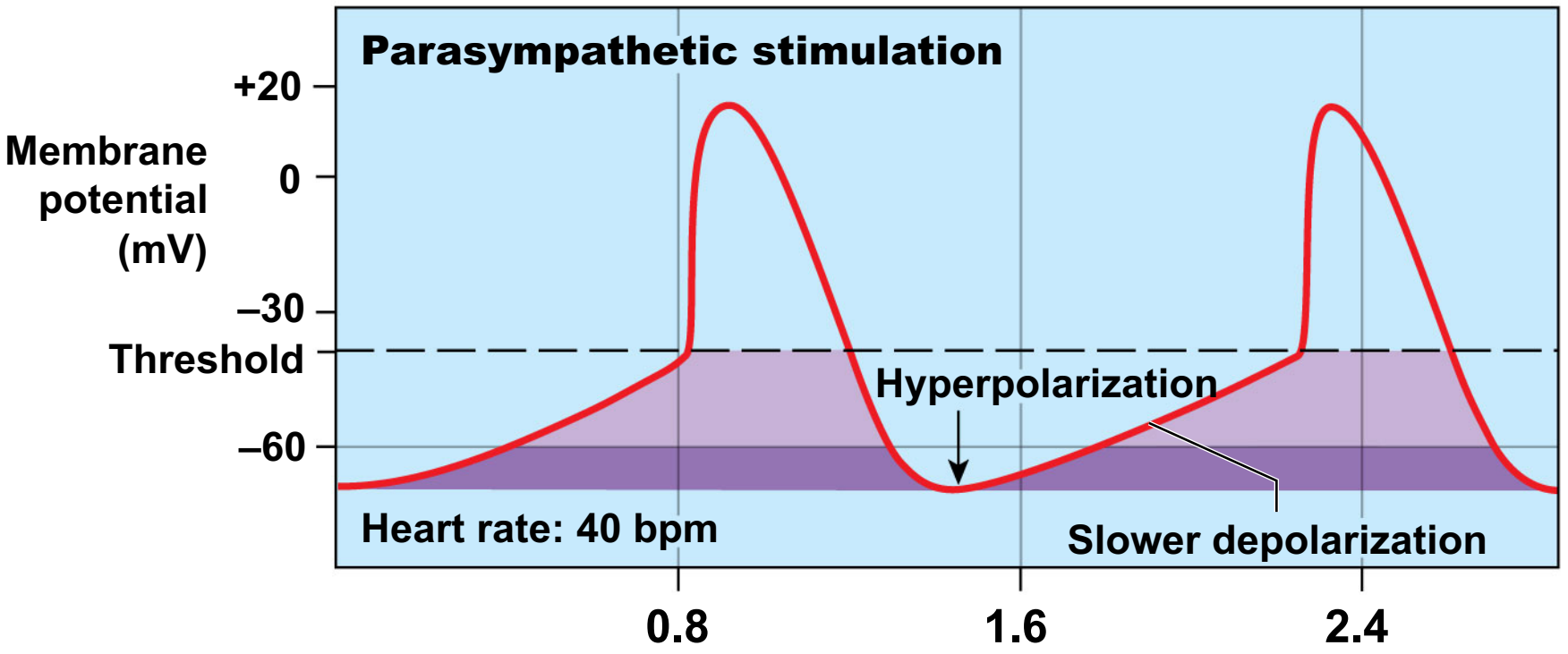


a Pacemaker cells have membrane potentials closer to threshold than those of cardiac contractile cells (–60 mV versus –90 mV). Their plasma membranes spontaneously depolarize to threshold, producing action potentials at a frequency determined by (1) the membrane potential and (2) the rate of depolarization.

20-4 Cardiac Output

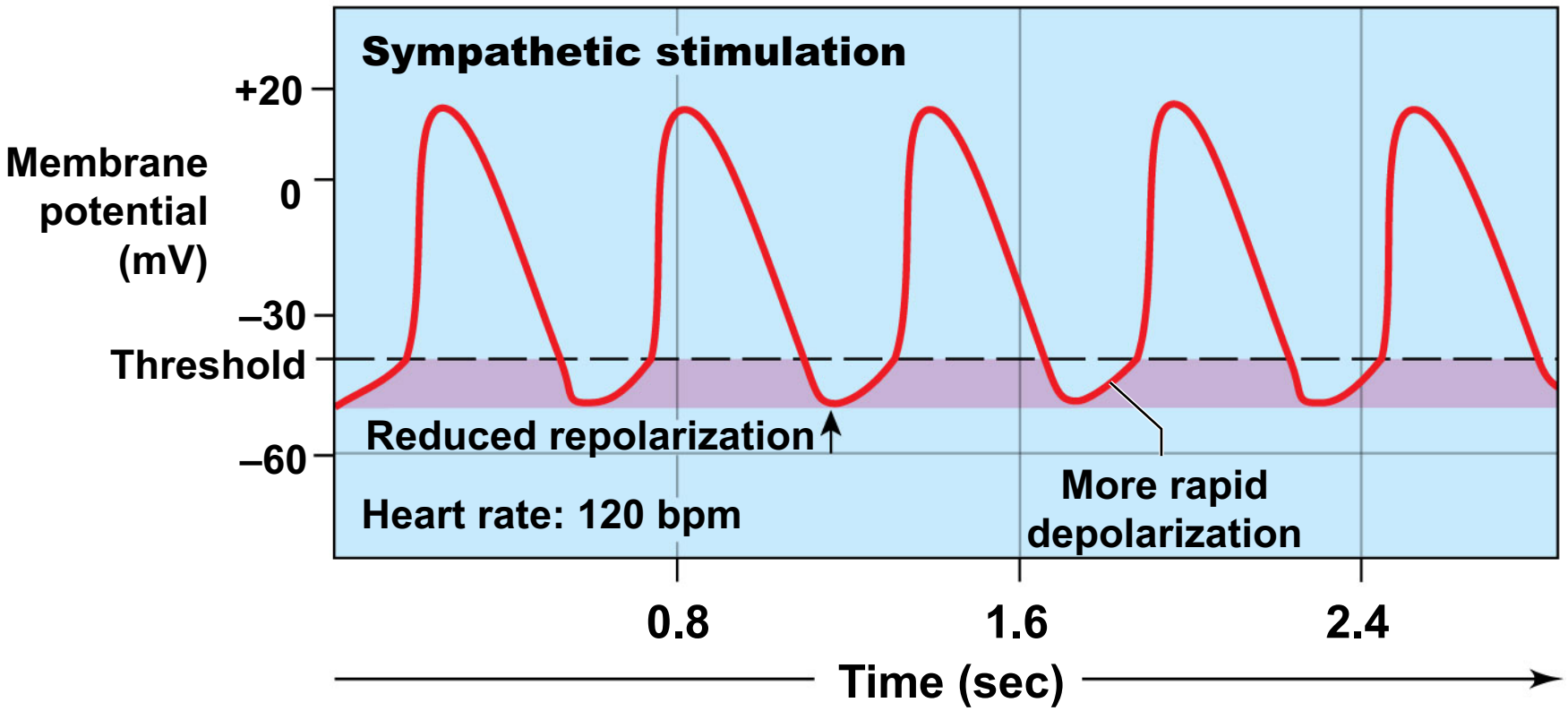
- Effects on pacemaker cells of SA node
  - ACh released by parasympathetic neurons
    - Decreases heart rate
  - NE released by sympathetic neurons
    - Increases heart rate

Figure 20–22b Autonomic Regulation of Pacemaker Cell Function.



b Parasympathetic stimulation releases ACh, which extends repolarization and decreases the rate of spontaneous depolarization. The heart rate slows.

Figure 20–22c Autonomic Regulation of Pacemaker Cell Function.



C Sympathetic stimulation releases NE, which shortens repolarization and accelerates the rate of spontaneous depolarization. As a result, the heart rate increases.

20-4 Cardiac Output

- **Bainbridge reflex** (atrial reflex)
  - Adjustments in heart rate in response to increase in **venous return**
    - Amount of blood returning to heart through veins
  - Stretch receptors in right atrium
    - Trigger increase in heart rate by stimulating sympathetic activity

20-4 Cardiac Output

- Hormonal effects on heart rate
  - Heart rate is increased by
    - Epinephrine (E)
    - Norepinephrine (NE)
    - Thyroid hormone (T_3)

20-4 Cardiac Output

- Factors affecting stroke volume
  - Changes in EDV or ESV affect stroke volume
    - And thus cardiac output
  - Two factors affect EDV
    - **Filling time**
      - Duration of ventricular diastole
    - Venous return

20-4 Cardiac Output

- **Preload**
  - Degree of ventricular stretching during ventricular diastole
  - Directly proportional to EDV
  - Affects ability of muscle cells to produce tension

20-4 Cardiac Output

- EDV and stroke volume
  - At rest,
    - EDV is low
    - Myocardium is stretched very little
    - Stroke volume is relatively low
  - With exercise,
    - Venous return increases
    - EDV increases
    - Myocardium stretches more
    - Stroke volume increases

20-4 Cardiac Output

- **Frank–Starling Principle**
  - As EDV increases, stroke volume increases
- Physical limits
  - Ventricular expansion is limited by
    - Myocardial connective tissues
    - Cardiac skeleton
    - Pericardium

20-4 Cardiac Output

- Three factors affect ESV
  - Preload
    - Ventricular stretching during diastole
  - **Contractility**
    - Force produced during contraction at a given preload
    - Affected by autonomic activity and hormones
  - **Afterload**
    - Tension that must be produced by ventricle to open semilunar valve and eject blood

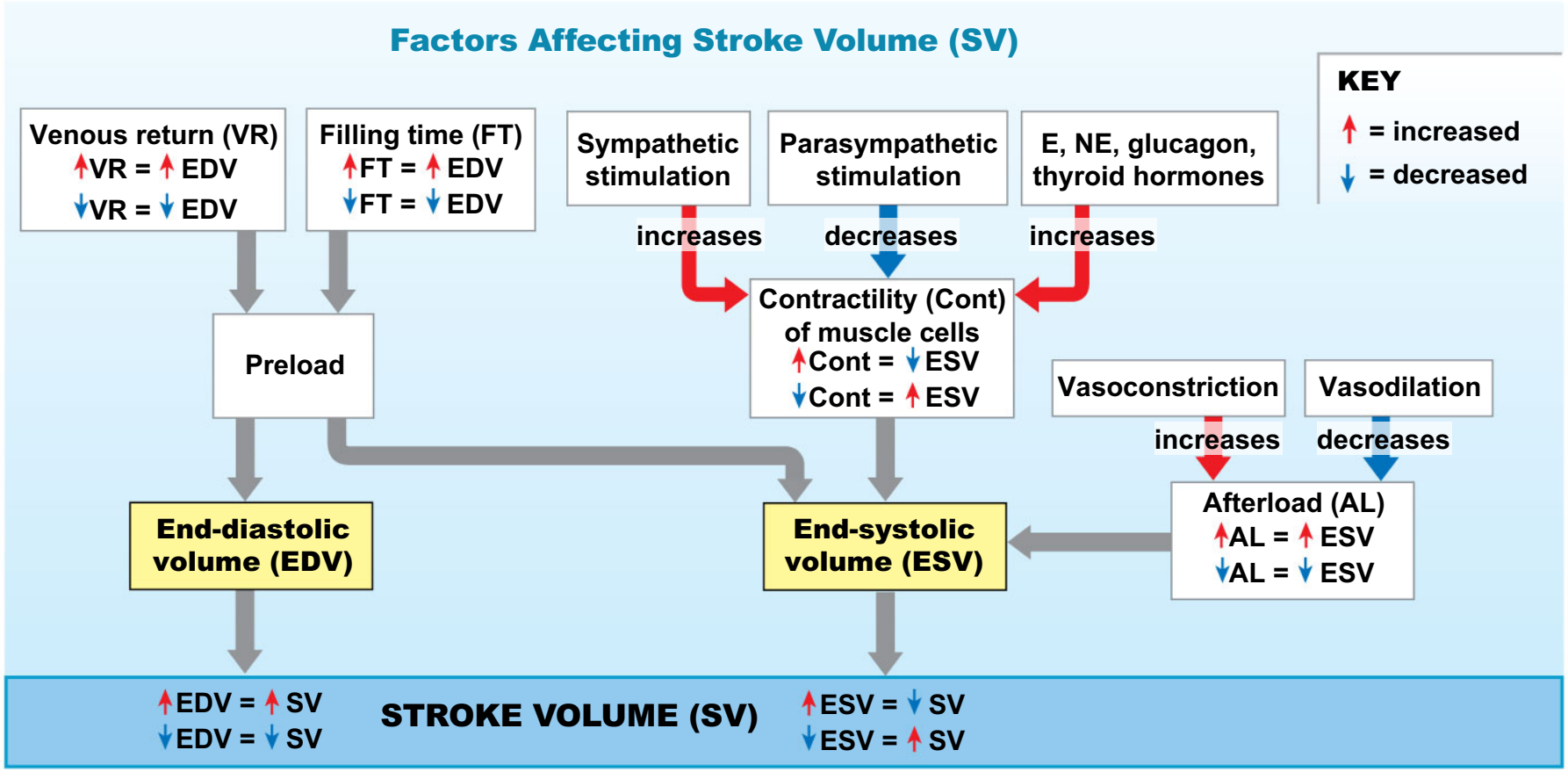
20-4 Cardiac Output

- Effects of autonomic activity on contractility
  - Sympathetic stimulation
    - NE released by cardiac nerves
    - E and NE released by adrenal medullae
    - Causes ventricles to contract with more force
    - Increases ejection fraction, decreases ESV
  - Parasympathetic stimulation
    - ACh released by vagus nerves
    - Reduces force of cardiac contractions

20-4 Cardiac Output

- Hormones
  - Many hormones affect heart contractility
  - Pharmaceutical drugs mimic hormone actions
    - Stimulate or block alpha or beta receptors
    - Block calcium channels
- Afterload
  - Increased by any factor that restricts blood flow
  - As afterload increases, stroke volume decreases

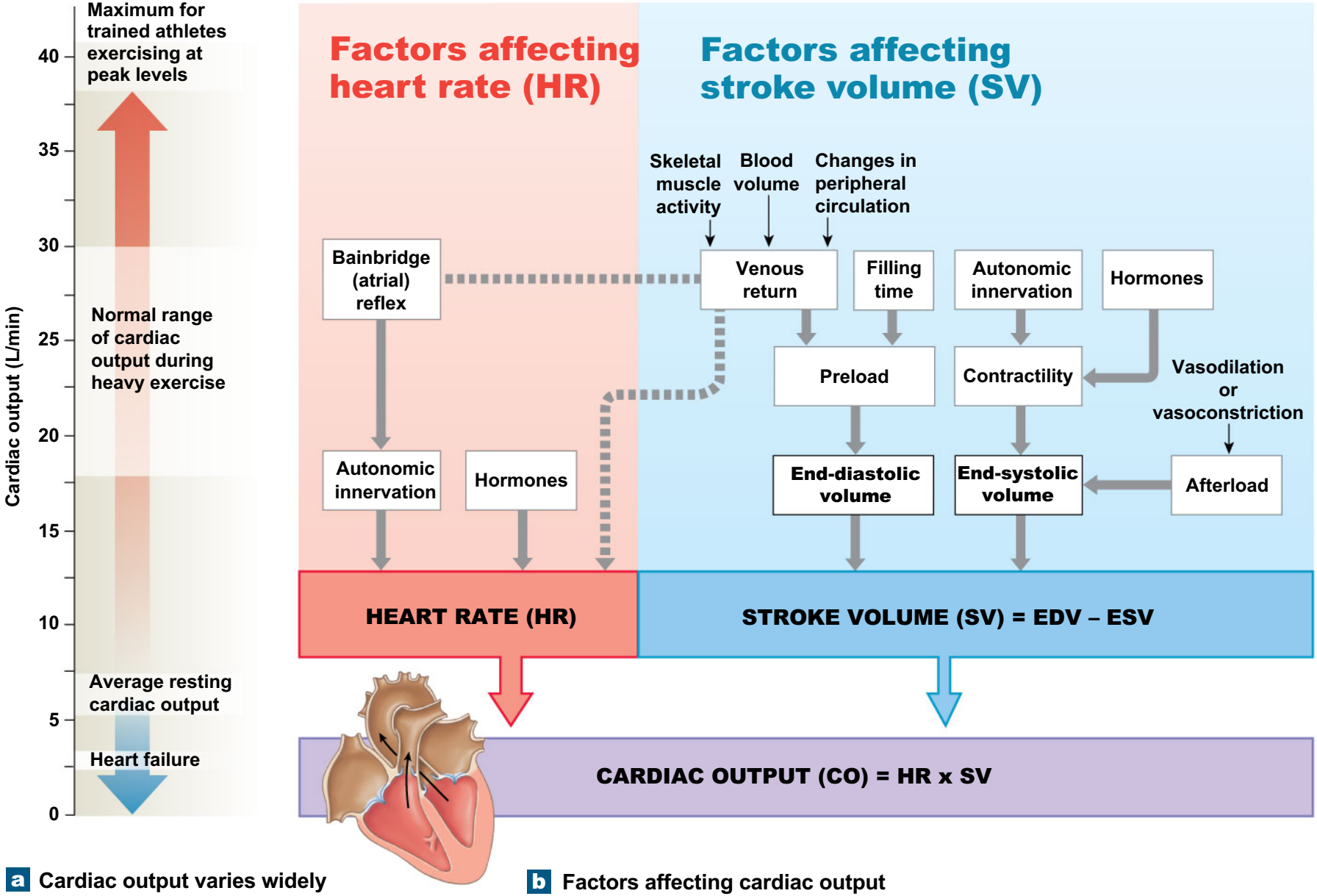
Figure 20–23 Factors Affecting Stroke Volume.



20-4 Cardiac Output

- Summary: the control of cardiac output
  - Heart rate control factors
    - Autonomic nervous system
      - Sympathetic and parasympathetic
    - Circulating hormones
    - Venous return and stretch receptors
  - Stroke volume control factors
    - EDV—filling time and rate of venous return
    - ESV—preload, contractility, and afterload

Figure 20–24 A Summary of the Factors Affecting Cardiac Output.



a Cardiac output varies widely to meet metabolic demands

b Factors affecting cardiac output

20-4 Cardiac Output

- **Cardiac reserve**
  - Difference between resting and maximal cardiac outputs

20-4 Cardiac Output

- Heart and vessels of cardiovascular system
  - Cardiovascular regulation
    - Ensures adequate circulation to body tissues
  - Cardiac centers
    - Control heart rate and peripheral blood vessels
  - Cardiovascular system responds to
    - Changing activity patterns
    - Circulatory emergencies